Lower Duwamish Waterway Group

Port of Seattle / City of Seattle / King County / The Boeing Company

Lower Duwamish Waterway Remedial Investigation

REMEDIAL INVESTIGATION REPORT

APPENDIX B:

BASELINE HUMAN HEALTH RISK ASSESSMENT

FINAL

For submittal to:

The US Environmental Protection Agency
Region 10
Seattle, WA

The Washington State Department of Ecology
Northwest Regional Office
Bellevue, WA

November 12, 2007

Prepared by: Windward environmental LLC

200 West Mercer Street, Suite 401 ♦ Seattle, Washington ♦ 98119

Table of Contents

List of Tables iii

List of Figures xii

List of Maps xiii

List of Acronyms xiv

Executive Summary ES-1
- ES.1 DATA EVALUATION ES-1
- ES.2 CONCEPTUAL SITE MODEL AND EXPOSURE ASSESSMENT ES-2
- ES.3 TOXICITY ASSESSMENT ES-6
- ES.4 RISK CHARACTERIZATION AND UNCERTAINTY ANALYSIS ES-7

B.1 Introduction 1

B.2 Data Evaluation 3
- B.2.1 DATA AVAILABILITY AND SELECTION 5
  - B.2.1.1 Sediment chemistry 5
  - B.2.1.2 Fish and shellfish tissue chemistry 9
- B.2.2 DATA REDUCTION 21
  - B.2.2.1 Averaging duplicate or replicate samples 22
  - B.2.2.2 Selection of best results 22
  - B.2.2.3 Significant figures and rounding 24
  - B.2.2.4 Calculating totals 25
- B.2.3 SUITABILITY OF DATA FOR RISK ASSESSMENT 27
  - B.2.3.1 Representativeness to site-related contamination 27
  - B.2.3.2 Representativeness to expected human exposure 28
  - B.2.3.3 Quality assurance/quality control results 30
  - B.2.3.4 Other factors 30

B.3 Exposure Assessment 33
- B.3.1 SUMMARY OF PREVIOUS LDW EXPOSURE ASSESSMENTS 33
- B.3.2 CONCEPTUAL SITE MODEL 36
  - B.3.2.1 Water recreation 38
  - B.3.2.2 Beach play 39
  - B.3.2.3 Occupational exposure 41
  - B.3.2.4 Clamming 41
  - B.3.2.5 Fishing and shellfishing for consumption 42
  - B.3.2.6 Selection of exposure scenarios for quantification 43
- B.3.3 CHEMICAL SCREENING AND EVALUATION 47
  - B.3.3.1 Sediment 49
  - B.3.3.2 Tissue 52

B.3.4 SELECTION OF EXPOSURE PARAMETERS AND CALCULATION OF CHRONIC DAILY INTAKE 57
B.3.4.1 Seafood ingestion rates 80
B.3.4.2 Sediment exposure parameters 92
B.3.4.3 Exposure point concentrations 95
B.3.4.4 Lead modeling 139
B.3.5 CHRONIC DAILY INTAKE RATES 147

B.4 Toxicity Assessment 147

B.5 Risk Characterization 161
B.5.1 RISK ESTIMATE CALCULATIONS 161
B.5.1.1 Carcinogenic risks 161
B.5.1.2 Non-carcinogenic health effects 163
B.5.1.3 Acceptable risk thresholds 163
B.5.2 RISK CHARACTERIZATION FORMAT 164
B.5.3 RISK CHARACTERIZATION RESULTS 165
B.5.3.1 Seafood consumption 168
B.5.3.2 Netfishing 221
B.5.3.3 Beach play RME 226
B.5.3.4 Clamming 251
B.5.3.5 Total risks for clamming scenarios (clam collection and clam consumption) 259
B.5.4 LEAD 266
B.5.4.1 Children 266
B.5.4.2 Adults 267
B.5.5 BACKGROUND CONCENTRATIONS FOR DETERMINING INCREMENTAL RISK 268
B.5.5.1 Arsenic 270
B.5.5.2 Dioxins/furans 278
B.5.6 RISK CHARACTERIZATION SUMMARY 286

B.6 Uncertainty Analysis 304
B.6.1 EXPOSURE ASSESSMENT 313
B.6.1.1 Exposure point concentrations 313
B.6.1.2 Ingestion rates 330
B.6.1.3 Exposure duration for Asian and Pacific Islander seafood consumption scenario 347
B.6.1.4 Fraction of dose obtained from site 349
B.6.1.5 Dermal exposure 351
B.6.1.6 Representativeness of fish and shellfish COPC data for all potentially exposed populations 358
B.6.1.7 Spatial coverage of sediment chemistry data 359
B.6.1.8 Temporal variability in chemistry data 362
B.6.1.9 Health-protectiveness of sediment exposure scenarios 363

B.6.2 TOXICITY ASSESSMENT 369
B.6.2.1 Toxicity benchmarks 369
B.6.2.2 Total PCBs 370
B.6.2.3 PCB and dioxin/furan TEQs 371
B.6.2.4 Chromium 372
B.6.2.5 Mercury 372
B.6.3 RISK CHARACTERIZATION 373
B.6.3.1 Inclusion of PCBs in estimates of total excess cancer risk 373
B.6.3.2 Risk calculations for undetected chemicals 377
B.6.3.3 Risk calculations for discrete areas within the LDW 388
B.6.3.4 Arsenic risks attributed to clam consumption 401

B.7 Identification of Risk Drivers 404
B.7.1 SEAFOOD CONSUMPTION SCENARIOS 405
B.7.1.1 PCBs, arsenic, cPAHs, and dioxins/furans 406
B.7.1.2 Bis(2-ethylhexyl) phthalate 407
B.7.1.3 Pentachlorophenol 407
B.7.1.4 Other metals 408
B.7.1.5 Organochlorine pesticides 408
B.7.2 DIRECT SEDIMENT CONTACT SCENARIOS 410

B.8 Conclusions 411
8.1 PCBS 411
8.2 ARSENIC 412
8.3 CARCINOGENIC PAHS 412
8.4 DIOXINS/FURANS 413

B.9 References 414

Attachment 1 Occurrence and Selection of Chemicals of Potential Concern

Attachment 2 MTCA-Conforming EPCs

Attachment 3 Chronic Daily Intake Rates for Chemicals of Potential Concern

Attachment 4 Toxicological Profiles for Chemicals of Potential Concern

List of Tables

Table ES-1. Summary of seafood ingestion scenarios ES-10
Table ES-2. Summary of sediment exposure scenarios ES-11
Table B.2-1. Summary of studies included in the baseline surface sediment dataset 7
Table B.2-2. Fish species found in the Lower Duwamish Waterway 10

Table B.2-3. Macroinvertebrate species found in the LDW 17
Table B.2-4. LDW tissue datasets used in the baseline HHRA 18
Table B.2-5. Toxic equivalency and potency equivalency factors for dioxins/furans, PCB congeners, and cPAHs 25
Table B.3-1. Exposure scenarios evaluated in previous risk assessments 34
Table B.3-2. Rationale for the selection or exclusion of exposure pathways 45
Table B.3-3. Identification of COPCs for sediment exposure scenarios 50
Table B.3-4. Identification of COPCs for the seafood consumption scenario 54
Table B.3-5. Summary of seafood ingestion scenarios 58
Table B.3-6. Summary of sediment exposure scenarios 59
Table B.3-7. Daily intake calculations – seafood ingestion, adult tribal RME scenario based on Tulalip data 60
Table B.3-8. Daily intake calculations – seafood ingestion, adult tribal CT scenario based on Tulalip data 61
Table B.3-9. Daily intake calculations – seafood ingestion, child tribal RME scenario based on Tulalip data 62
Table B.3-10. Daily intake calculations – seafood ingestion, child tribal CT scenario based on Tulalip data 63
Table B.3-11. Daily intake calculations – seafood ingestion, adult tribal scenario based on Suquamish data 64
Table B.3-12. Daily intake calculations – seafood ingestion, adult API RME scenario 65
Table B.3-13. Daily intake calculations – seafood ingestion, adult API CT scenario 66
Table B.3-14. Daily intake calculations – seafood ingestion, adult one-meal-per-month scenario 67
Table B.3-15. Daily intake calculations – incidental sediment ingestion during netfishing, adult tribal RME scenario 68
Table B.3-16. Daily intake calculations – dermal contact with sediment during netfishing, adult tribal RME scenario 69
Table B.3-17. Daily intake calculations – incidental sediment ingestion during netfishing, adult tribal CT scenario 70
Table B.3-18. Daily intake calculations – dermal contact with sediment during netfishing, adult tribal CT scenario 71
Table B.3-19. Daily intake calculations – incidental sediment ingestion during child beach play RME 72
Table B.3-20. Daily intake calculations – dermal contact with sediment during child beach play RME 73
Table B.3-21. Daily intake calculations –incidental sediment ingestion during clamming, 7-day-per-year scenario 74

Table B.3-22. Daily intake calculations – dermal contact with sediment during clamming, 7-day-per-year scenario *75*

Table B.3-23. Daily intake calculations – incidental sediment ingestion during tribal clamming RME scenario *76*

Table B.3-24. Daily intake calculations – dermal contact with sediment during tribal clamming RME scenario *77*

Table B.3-25. Daily intake calculations – incidental sediment ingestion during tribal clamming, 183-day-per-year scenario *78*

Table B.3-26. Daily intake calculations – dermal contact with sediment during tribal clamming, 183-day-per-year scenario *79*

Table B.3-27. Seafood species consumed by Tulalip and Suquamish adults and LDW species used to represent consumed species *82*

Table B.3-28. Adult tribal consumption of shellfish (crabs, clams, and mussels) based on Tulalip and Suquamish data *83*

Table B.3-29. Portions of benthic fish and crab consumed – adult tribal RME and CT scenarios based on Tulalip data and adult tribal scenario based on Suquamish data *84*

Table B.3-30. Rates of child tribal (RME and CT) seafood consumption based on Tulalip data associated with different seafood categories *86*

Table B.3-31. Development of percentages and rates of adult API RME and CT seafood consumption associated with different seafood categories *89*

Table B.3-32. API RME and CT consumption of shellfish (crabs, clams, and mussels) *90*

Table B.3-33. Portions of benthic fish and crab consumed – adult API RME and CT scenarios *90*

Table B.3-34. Dermal absorption fractions *93*

Table B.3-35. Seafood consumption categories for developing EPCs *97*

Table B.3-36. Exposure point concentrations and summary statistics for metals and trace elements in tissue *98*

Table B.3-37. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in tissue *102*

Table B.3-38. Exposure point concentrations and summary statistics for SVOCs in tissue *107*

Table B.3-39. Summary of tissue EPCs by seafood category *117*

Table B.3-40. Exposure areas for direct sediment exposure scenarios *120*

Table B.3-41. Exposure point concentrations and summary statistics for metals and trace elements in sediment *121*

Table B.3-42. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in sediment *130*

Table B.3-43. Exposure point concentrations and summary statistics for SVOCs and dioxins/furans in sediment *133*

Table B.3-44. Summary of exposure point concentrations in sediment by exposure scenario 137
Table B.3-45. Input parameters for IEUBK lead model 140
Table B.3-46. Sediment lead values and time-weighted average EPCs 141
Table B.3-47. Median ingestion rate and mean exposure point concentration by seafood category for tribal children based on Tulalip data 142
Table B.3-48. Input parameters for ALM 143
Table B.3-49. Summary of lead data and mean EPC values 144
Table B.3-50. Calculation of median lead concentrations in tissue 145
Table B.4-1. Non-cancer toxicity data (oral) for chemicals of potential concern 151
Table B.4-2. Cancer toxicity data (oral/dermal) for chemicals of potential concern 155
Table B.4-3. Toxicological endpoints for COPCs with non-carcinogenic effects 158
Table B.5-1. Excess cancer risk estimates for the adult tribal RME seafood consumption scenario based on Tulalip data 169
Table B.5-2. Excess cancer risk estimates for the adult tribal CT seafood consumption scenario based on Tulalip data 171
Table B.5-3. Excess cancer risk estimates for the child tribal RME seafood consumption scenario based on Tulalip data 173
Table B.5-4. Excess cancer risk estimates for the child tribal CT seafood consumption scenario based on Tulalip data 175
Table B.5-5. Excess cancer risk estimates for the adult tribal seafood consumption scenario based on Suquamish data 177
Table B.5-6. Excess cancer risks for the API RME seafood consumption scenario 179
Table B.5-7. Excess cancer risks for the API CT seafood consumption scenario 181
Table B.5-8. Excess cancer risk estimates associated with the consumption of one-meal-per-month of seafood by adults 183
Table B.5-9. Non-cancer hazard estimates for the adult tribal RME seafood consumption scenario based on Tulalip data 188
Table B.5-10. Non-cancer hazard estimates for the adult tribal CT seafood consumption scenario based on Tulalip data 190
Table B.5-11. Non-cancer hazard estimates for the child tribal RME seafood consumption scenario based on Tulalip data 192
Table B.5-12. Non-cancer hazard estimates for the child tribal CT seafood consumption scenario based on Tulalip data 194
Table B.5-13. Non-cancer hazard estimates for the adult tribal seafood consumption scenario based on Suquamish data 196
Table B.5-14. Non-cancer hazard estimates for the API RME seafood consumption scenario 198

Table B.5-15. Non-cancer hazard estimates for the API CT seafood consumption scenario 200

Table B.5-16. Non-cancer hazard estimates associated with the consumption of one meal per month of seafood by adults 202

Table B.5-17. Comparison of excess cancer risks and non-cancer hazards by seafood category for selected chemicals in the adult tribal RME and child tribal RME seafood consumption scenarios based on Tulalip data 208

Table B.5-18. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult tribal CT and child tribal CT seafood consumption scenarios based on Tulalip data 210

Table B.5-19. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult tribal seafood consumption scenario based on Suquamish data 212

Table B.5-20. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult API RME seafood consumption scenario 214

Table B.5-21. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult API CT seafood consumption scenario 216

Table B.5-22. Summary for different seafood consumption scenarios of chemicals with greater than 50% of risk derived from seafood categories with no detected values 220

Table B.5-23. Excess cancer risk estimates for the netfishing RME scenario based on exposure by incidental sediment ingestion and dermal absorption 222

Table B.5-24. Excess cancer risk estimates for the netfishing CT scenario based on exposure by incidental sediment ingestion and dermal absorption 223

Table B.5-25. Non-cancer hazard estimates for the netfishing RME scenario based on exposure by incidental sediment ingestion and dermal absorption 224

Table B.5-26. Non-cancer hazard estimates for the netfishing CT scenario based on exposure by incidental sediment ingestion and dermal absorption 225

Table B.5-27. Excess cancer risk estimates for the beach play RME Area 1 scenario based on exposure by incidental sediment ingestion and dermal absorption 227

Table B.5-28. Excess cancer risk estimates for the beach play RME Area 2 scenario based on exposure by incidental sediment ingestion and dermal absorption 228

Table B.5-29. Excess cancer risk estimates for the beach play RME Area 3 scenario based on exposure by incidental sediment ingestion and dermal absorption 229

Table B.5-30. Excess cancer risk estimates for the beach play RME Area 4 scenario based on exposure by incidental sediment ingestion and dermal absorption 230

Table B.5-31. Excess cancer risk estimates for the beach play RME Area 5 scenario based on exposure by incidental sediment ingestion and dermal absorption 231

Table B.5-32. Excess cancer risk estimates for the beach play RME Area 6 scenario based on exposure by incidental sediment ingestion and dermal absorption 232

Table B.5-33. Excess cancer risk estimates for the beach play RME Area 7 scenario based on exposure by incidental sediment ingestion and dermal absorption 233

Table B.5-34. Excess cancer risk estimates for the beach play RME Area 8 scenario based on exposure by incidental sediment ingestion and dermal absorption 234

Table B.5-35. Non-cancer hazard estimates for the beach play RME Area 1 scenario based on exposure by incidental sediment ingestion and dermal absorption 236

Table B.5-36. Non-cancer hazard estimates for the beach play RME Area 2 scenario based on exposure by incidental sediment ingestion and dermal absorption 238

Table B.5-37. Non-cancer hazard estimates for the beach play RME Area 3 scenario based on exposure by incidental sediment ingestion and dermal absorption 240

Table B.5-38. Non-cancer hazard estimates for the beach play RME Area 4 scenario based on exposure by incidental sediment ingestion and dermal absorption 242

Table B.5-39. Non-cancer hazard estimates for the beach play RME Area 5 scenario based on exposure by incidental sediment ingestion and dermal absorption 244

Table B.5-40. Non-cancer hazard estimates for the beach play RME Area 6 scenario based on exposure by incidental sediment ingestion and dermal absorption 246

Table B.5-41. Non-cancer hazard estimates for the beach play RME Area 7 scenario based on exposure by incidental sediment ingestion and dermal absorption 248

Table B.5-42. Non-cancer hazard estimates for the beach play RME Area 8 scenario based on exposure by incidental sediment ingestion and dermal absorption 250

Table B.5-43. Excess cancer risk estimates for the 7-day-per-year clamming scenario based on exposure by incidental sediment ingestion and dermal absorption 252

Table B.5-44. Excess cancer risk estimates for the tribal clamming RME scenario (120 days per year) based on exposure by incidental sediment ingestion and dermal absorption 253

Table B.5-45. Excess cancer risk estimates for the tribal clamming 183-day-per-year scenario based on exposure by incidental sediment ingestion and dermal absorption 254

Table B.5-46. Non-cancer hazard estimates for the 7-day-per-year clamming scenario based on exposure by incidental sediment ingestion and dermal absorption 255

Table B.5-47. Non-cancer hazard estimates for the tribal clamming RME scenario (120 days per year) based on exposure by incidental sediment ingestion and dermal absorption 256

Table B.5-48. Non-cancer hazard estimates for the tribal clamming 183-day-per-year scenario based on exposure by incidental sediment ingestion and dermal absorption 258

Table B.5-49. Risk estimates for example chemicals for the 7-day-per-year clamming scenario and the adult one-meal-per-month clam consumption scenario 260

Table B.5-50. Risk estimates for example chemicals for the tribal clam collection RME scenario and the consumption of clams in the adult tribal RME seafood consumption scenario based on Tulalip data 262

Table B.5-51. Risk estimates for example chemicals for the tribal clamming 183-day-per-year scenario and the consumption of clams in the adult tribal RME seafood consumption scenario based on Tulalip data 264

Table B.5-52. Risk estimates for predicted fetal and adult lead levels using the adult lead model 268

Table B.5-53. Duwamish River surface sediment arsenic data collected upstream of the LDW 271

Table B.5-54. Sediment exposure point concentrations for arsenic in upstream and LDW exposure scenarios 272

Table B.5-55. Incremental risks from exposure to arsenic in sediment 273

Table B.5-56. Inorganic arsenic EPCs for tissue samples collected from the LDW and background areas 275

Table B.5-57. Incremental cancer risk estimates associated with inorganic arsenic for the seafood consumption exposure scenarios 276

Table B.5-58. Incremental cancer risk estimates associated with inorganic arsenic for the seafood consumption categories in the adult tribal RME seafood exposure scenario based on Tulalip data 277

Table B.5-59. Calculated dioxin/furan TEQs in surface sediment samples collected from the greater Seattle area 280

Table B.5-60. Summary of dioxin/furan TEQs used for the characterization of LDW urban background concentrations of dioxins/furans in surface sediment for the greater Seattle area 281

Table B.5-61. Calculated dioxin/furan TEQs in LDW surface sediment samples 282

Table B.5-62. Sediment exposure point concentrations for dioxins/furans (TEQ) in the LDW urban background and LDW exposure scenarios 284

Table B.5-63. Incremental risk estimates from exposure to dioxins/furans (TEQ) in sediment 285
Table B.5-64. Summary of estimated excess cancer risks for the seafood consumption scenarios 288
Table B.5-65. Summary of estimated non-cancer hazards for the seafood consumption scenarios 290
Table B.5-66. Summary of estimated excess cancer risks for direct sediment exposure scenarios 292
Table B.5-67. Risks by chemical for adult and child seafood consumption scenarios 295
Table B.5-68. Risks by chemical for adult and child direct sediment exposure scenarios 298
Table B.5-69. Excess cancer risk estimates across related scenarios 300
Table B.6-1. Summary of uncertainties identified in the baseline HHRA 305
Table B.6-2. Number and tissue types of samples selected for bis(2-ethylhexyl) phthalate and pentachlorophenol reanalysis 317
Table B.6-3. Comparison of original seafood consumption EPCs and risk estimates for bis(2-ethylhexyl) phthalate and pentachlorophenol based on hypothetical tissue reanalyses 319
Table B.6-4. Exposure point concentrations and summary statistics for cPAHs using all available tissue data 321
Table B.6-5. Comparison of excess cancer risks associated with cPAHs based on 2004 and combined datasets 321
Table B.6-6. Comparison of two different methods for calculating PCB totals in LDW tissue samples 324
Table B.6-7. EPCs for total PCBs based on PCB congeners and Aroclor sums 325
Table B.6-8. Excess cancer risks and non-cancer hazards for the adult tribal RME scenario based on Tulalip data: risk estimates for total PCBs as the sum of congeners and as the sum of Aroclors 326
Table B.6-9. Number of participants in child and adult tribal seafood surveys 333
Table B.6-10. Child tribal seafood consumption 334
Table B.6-11. Percentages and rates associated with different seafood categories for the child tribal 95th percentile seafood consumption scenario based on Tulalip data 335
Table B.6-12. Consumption of crabs, clams, and mussels for the child tribal 95th percentile seafood consumption scenario based on Tulalip data 335
Table B.6-13. Portions of crab consumed – child tribal 95th percentile scenario based on Tulalip data 336
Table B.6-14. Excess cancer risk estimates for the child tribal 95th percentile seafood consumption scenario based on Tulalip data 337
Table B.6-15. Non-cancer hazard estimates for the child tribal 95th percentile seafood consumption scenario based on Tulalip data 339

Table B.6-16. Percentages and rates associated with different seafood categories for the child tribal seafood consumption scenario based on Suquamish data 342

Table B.6-17. Consumption of crabs, clams, and mussels for the child tribal seafood consumption scenario based on Suquamish data 342

Table B.6-18. Excess cancer risk estimates for the child tribal seafood consumption scenario based on Suquamish data 343

Table B.6-19. Non-cancer hazard estimates for the child tribal seafood consumption scenario based on Suquamish data 345

Table B.6-20. Comparison of API RME risks with 41-year exposure duration 348

Table B.6-21. Excess cancer risk estimates for seafood consumption exposure scenarios using alternative assumptions for fractional intake of dose obtained from the site 350

Table B.6-22. Excess cancer risk estimates for direct sediment exposure scenarios using alternative assumptions for fractional intake of dose obtained from the site 351

Table B.6-23. Hypothetical non-cancer hazard estimates for vanadium using three dermal absorption factors for the direct sediment exposure scenarios 353

Table B.6-24. Effect of increased dermal adherence factors on risk estimates for adult sediment exposure scenarios 356

Table B.6-25. Effect of increased dermal adherence factor on risk estimates for child sediment exposure scenarios 357

Table B.6-26. Comparison of dioxin/furan TEQ risks with and without cluster of high concentrations at RM 1.5 361

Table B.6-27. PCB risk estimates for sediment exposure scenarios using two EPC calculation methods 362

Table B.6-28. Exposure parameters for dog walking and habitat restoration scenarios 364

Table B.6-29. Cancer risk estimates using hypothetical EPC values for arsenic, cPAHs, and PCBs 365

Table B.6-30. Cancer risk estimates for dog walking at beach play areas 2, 5, and 7 367

Table B.6-31. Non-cancer risk estimates for dog walking at beach play areas 2, 5, and 7 368

Table B.6-32. Chemicals in LDW tissue samples without toxicity benchmarks 370

Table B.6-33. EPCs for sum of Aroclor mass minus the mass of dioxin-like PCB congeners 376

Table B.6-34. PCB cancer risk estimates using various risk calculation methods 377

Table B.6-35. Summary of hypothetical excess cancer risk estimates for seafood consumption scenarios from COPCs that were never detected in LDW tissue samples 379

Table B.6-36. Summary of hypothetical non-cancer hazard estimates for seafood consumption scenarios from COPCs that were never detected in LDW tissue samples 382

Table B.6-37. Summary of hypothetical excess cancer risks for direct sediment exposure scenarios from COPCs that were never detected in LDW sediment samples 385

Table B.6-38. Summary of hypothetical non-cancer hazards for direct sediment exposure scenarios from COPCs that were never detected in LDW sediment samples 386

Table B.6-39. Exposure point concentrations for chemicals on a tissue sampling area-specific basis for the seafood consumption scenarios 389

Table B.6-40. Excess cancer risk estimates for select COPCs for seafood consumption scenarios for all tissue sampling areas compared to the LDW-wide area 394

Table B.6-41. Non-cancer hazard estimates for select COPCs for seafood consumption scenarios for all tissue sampling areas compared to the LDW-wide area 396

Table B.6-42. Comparison of beach play RME excess cancer risks and non-cancer hazards at the Duwamish Waterway Park in the South Park neighborhood and in beach play RME area 5 398

Table B.6-43. Sediment concentrations (mg/kg dw) corresponding to specific risk thresholds for the habitat biologist scenario for arsenic, total PCBs, cPAHs, and dioxins/furans 399

Table B.6-44. Inorganic arsenic concentrations in clam samples and total arsenic concentrations in co-located sediment samples from the LDW 402

Table B.7-1. COCs and identification of risk drivers for seafood consumption scenarios 405

Table B.7-2. Summary of LDW and Puget Sound organochlorine pesticide data 409

Table B.7-3. COCs and identification of risk drivers for direct sediment exposure scenarios 410

List of Figures

Figure ES-1. Seafood consumption scenario risks by chemical ES-15

Figure ES-2. Cancer risks by seafood category for the adult seafood consumption scenarios ES-16

Figure ES-3. Cancer risks by chemical for direct sediment exposure scenarios ES-17

Figure B.2-1. Data flowchart for the HHRA 4

Figure B.3-1. Conceptual site model for baseline human health risk assessment 37

Figure B.3-2. COPC identification flowchart 48

Figure B.3-3. Flowchart showing method for selecting EPC 95

Figure B.5-1. Probability density curves for predicted blood lead concentrations using input data from children's beach play RME and seafood consumption 267

Figure B.5-2. RME seafood consumption scenario risks by chemical 296

Figure B.5-3. Cancer risks by chemical for direct sediment exposure scenarios 299

Figure B.5-4. Excess cancer risks from PCB TEQ for seafood consumption scenarios and rates *302*

Figure B.5-5. Excess cancer risks from total PCBs for seafood consumption scenarios and rates *303*

Figure B.6-1. Inorganic arsenic in LDW clams compared to total arsenic in co-located sediment *403*

List of Maps

Map B.1-1. Lower Duwamish Waterway study area

Map B.2-1. LDW surface sediment sampling locations (subtidal and intertidal)

Map B.2-2a. LDW tissue sampling areas and subareas and individual locations where tissue samples were collected by LDWG in 2004-2005

Map B.2-2b. LDW tissue sampling areas and subareas and individual locations where tissue samples were collected by non-LDWG parties prior to 2004

Map B.3-1. LDW intertidal areas included in the beach play sediment exposure scenarios

Map B.3-2. LDW intertidal areas included in the clam harvesting sediment exposure scenarios

Map B.3-3. PCB TEQ values calculated from individual PCB congeners analyzed in LDW surface sediment using high-resolution methods

Map B.3-4. Dioxin/furan TEQ values calculated from dioxin/furan concentrations in LDW surface sediment samples

Map B.6-1. Total PCBs as Thiessen polygons for the LDW netfishing exposure area

Map B.6-2. Total PCBs as Thiessen polygons for the LDW tribal clamming exposure areas

Map B.6-3. Risk-based concentration comparisons for total PCBs using the habitat biologist exposure scenario

Map B.6-4. Risk-based concentration comparisons for arsenic using the habitat biologist exposure scenario

Map B.6-5. Risk-based concentration comparisons for cPAHs using the habitat biologist exposure scenario

Map B.6-6. Risk-based concentration comparisons for dioxins/furans using the habitat biologist exposure scenario

Map B.6-7. Combination of risks associated with total PCBs, arsenic, dioxins/furans, and cPAHs using the habitat biologist scenario

Map B.6-8. Risk-based concentration comparisons for total PCBs using the netfishing exposure scenario

Map B.6-9. Risk-based concentration comparisons for arsenic using the netfishing exposure scenario

Map B.6-10. Risk-based concentration comparisons for cPAHs using the netfishing exposure scenario

Map B.6-11. Risk-based concentration comparisons for dioxins/furans using the netfishing exposure scenario

Map B.6-12. Combination of risks associated with total PCBs, arsenic, dioxins/furans, and cPAHs using the netfishing scenario

Map B.6-13. Clam habitats, clam sampling locations, areas over which sediments were assumed to potentially serve as sources of particles for the clams, and the distribution of arsenic in surface sediments

List of Acronyms

Acronym	Definition
ABS	dermal absorption factor
AF	adherence factor
ALM	Adult Lead Model
API	Asian and Pacific Islander
ARI	Analytical Resources, Inc.
ATSDR	Agency for Toxic Substance and Disease Registry
BCA	bias-corrected accelerated
BHC	benzene hexachloride
BKSF	biokinetic slope factor
bw	body weight
California EPA	California Environmental Protection Agency
CAS	Columbia Analytical Services, Inc.
CDI	chronic daily intake
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
COC	chemical of concern
COPC	chemical of potential concern
cPAH	carcinogenic polycyclic aromatic hydrocarbon
CSO	combined sewer overflow
CT	central tendency
CTE	central tendency estimate
Cu	copper

Acronym	Definition
dw	dry weight
E/H	environment/habitat
EPA	US Environmental Protection Agency
EPC	exposure point concentration
ERA	ecological risk assessment
ESG	Environmental Solutions Group
FI	fractional intake
GC/ECD	gas chromatography/electron capture detector
GC/MS	gas chromatography/mass spectrometry
GI	gastrointestinal
GSD	geometric standard deviation
H-UCL	UCL based on Land's H-statistic
HEAST	Health Effects Assessment Summary Tables
HHRA	human health risk assessment
HI	hazard index
HPLC/PDA	high-performance liquid chromatography/photodiode array
HQ	hazard quotient
HRGC/HRMS	high-resolution gas chromatography/high-resolution mass spectrometry
HSDB	Hazardous Substance Data Bank
IARC	International Agency for Research on Cancer
IEUBK	Integrated Exposure Uptake Biokinetic Model for Lead in Children
IRIS	Integrated Risk Information System
IR	ingestion rate
J-qualifier	estimated concentration
KC	King County
KM	Kaplan Meier method for calculating a UCL
LDH	lactate dehydrogenase
LDW	Lower Duwamish Waterway
LDWG	Lower Duwamish Waterway Group
MLLW	mean lower low water
MTCA	Model Toxics Control Act
MVUE	minimum-variance unbiased eliminator
N-qualifier	presumptive evidence of a compound

Acronym	Definition
NCEA	National Center for Exposure Assessment
NCP	National Contingency Plan
NMFS	National Marine Fisheries Service
NOAA	National Oceanic and Atmospheric Administration
NPL	National Priorities List
OGWDW	Office of Ground Water and Drinking Water
OSHA	Occupational Safety and Health Administration
PAH	polycyclic aromatic hydrocarbon
Pb	lead
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo-*p*-dioxin
PCDF	polychlorinated dibenzofuran
PCT	polychlorinated terphenyl
PEF	potency equivalency factor
PPRTV	Provisional Peer-Reviewed Toxicity Value
PRG	preliminary remediation goal
PSAMP	Puget Sound Ambient Monitoring Program
PSEP	Puget Sound Estuary Program
QAPP	quality assurance project plan
QC	quality control
RAGS	Risk Assessment Guidance for Superfund
RBC	risk-based concentration
RBTC	risk-based threshold concentration
RCRA	Resource Conservation and Recovery Act
RfD	reference dose
RI	remedial investigation
RI/FS	remedial investigation/feasibility study
RL	reporting limit
RM	river mile
RME	reasonable maximum exposure
SD	storm drain
sd	standard deviation
SF	slope factor

Acronym	Definition
SGOT	serum glutamic oxaloacetic transaminase
SIM	selected ion monitoring
SMS	Washington State Sediment Management Standards
SQS	sediment quality standards of SMS
SVOC	semivolatile organic compound
SWAC	spatially weighted average concentration
t (t-distribution)	statistical method used to calculate the mean for a normally distributed set of samples
TBT	tributyltin
TCDD	tetrachlorodibenzo-*p*-dioxin
TEF	toxic equivalency factor
TEQ	toxic equivalent
TPH	total petroleum hydrocarbons
UCL	upper confidence limit
USACE	US Army Corps of Engineers
USFWS	US Fish and Wildlife Service
VOC	volatile organic compound
WAC	Washington Administrative Code
WCC	Washington Conservation Corps
Windward	Windward Environmental LLC
WDFW	Washington State Department of Fish and Wildlife
WSDOH	Washington State Department of Health
ww	wet weight
Zn	zinc

Executive Summary

This document presents the baseline human health risk assessment (HHRA) for the Lower Duwamish Waterway (LDW) Superfund site, as outlined in the work plan for the remedial investigation (RI) (Windward 2004d). Baseline risk assessments, as defined in US Environmental Protection Agency (EPA) guidance (EPA 1988), "provide an evaluation of the potential threat to human health and the environment in the absence of any remedial action. They provide the basis for determining whether or not remedial action is necessary and the justification for performing remedial actions."

The baseline HHRA presents risk estimates for various scenarios whereby people could be exposed to chemicals of potential concern (COPCs) found in sediment and in fish and shellfish tissues from the LDW (Map B.1-1). Because knowledge of current and future site use is imperfect, the scenarios evaluated in this assessment have been selected in an attempt to not underestimate risks and, as such, may overestimate risks for many site users. The dataset for the baseline HHRA consisted primarily of sediment and tissue chemistry data collected from the LDW during Phase 2 to supplement the historical data that were used in the Phase 1 HHRA (Windward 2003b). The baseline HHRA includes sections on data evaluation, conceptual site model and exposure assessment, toxicity assessment, risk characterization, and uncertainty analysis, each of which is briefly summarized below.

ES.1 DATA EVALUATION

The data evaluation section of the HHRA includes a description of what data were available, a determination of how the data were used in the HHRA, and the suitability of the data for risk assessment purposes.

Sediment chemistry data consisted of chemical concentrations in the uppermost 15 cm of the sediment, which are the appropriate data to evaluate the exposure scenarios identified in this HHRA. Chemical data for evaluating exposures from seafood consumption were available for English sole, starry flounder, crabs, clams, mussels, and perch caught within the LDW. Seafood consumption rates applied to these tissue data were based on consumption studies representative of seafood harvest from other areas of Puget Sound. Many of the species that may be consumed from these other areas (e.g., speckled sanddab, Pacific cod, rockfish, spiny dogfish, walleye pollock) are rarely found in the LDW, and no LDW tissue chemistry data were available for those species. This data gap was addressed by using data from a representative trophic group as a surrogate. In addition, although salmon are a highly preferred and consumed fish from the LDW and tissue data are available for salmon, human health risks were not calculated for consumption of adult salmon. Their exposure to chemicals in LDW sediment is not anticipated to significantly influence the concentrations in their tissues, primarily because of the very small portion of their

lives spent in the LDW. Although risks from salmon consumption were not included in this HHRA, risks to juvenile salmon were addressed in the ecological risk assessment, which is included as Appendix A to the RI report.

Uncertainties in the data may impact risk estimates. For example, pesticides in seafood tissues collected in 2004 were qualified as estimated concentrations with uncertain presence. The tissue concentrations used to estimate the intake of these chemicals are highly uncertain and may not accurately represent exposures to these chemicals from seafood consumption within the LDW. Uncertainties in the data used in this assessment are summarized in Section ES.4, Risk Characterization and Uncertainty Analysis.

ES.2 CONCEPTUAL SITE MODEL AND EXPOSURE ASSESSMENT

The conceptual site model describes scenarios in which people could be exposed to COPCs associated with sediment within the LDW. The primary exposure scenarios were identified through input from site users, including the Muckleshoot and Suquamish Tribes, and through review of prior risk assessments of the LDW. Exposure pathways consisted of direct contact with sediments during commercial netfishing, beach play, and clam harvesting in the LDW and indirect exposure through the consumption of seafood from the LDW. Exposures associated with swimming in the LDW were evaluated through the inclusion of risk estimates developed previously as part of the King County water quality assessment HHRA (King County 1999b).[1]

Several levels of exposure scenarios are used in the risk assessment to describe different intensities (e.g., frequency and duration) of site use or seafood consumption. These scenarios include reasonable maximum exposure (RME) scenarios, upper-bound exposure scenarios, central tendency (CT) exposure scenarios, and a one-meal-per-month seafood consumption scenario. The following describe how each is used in the risk assessment:

- RME is the highest exposure that is reasonably expected to occur at a site. EPA generally uses RME scenarios to evaluate remedial actions at a site (EPA 1989). RME, by definition, likely overestimates exposure for many individuals. With regard to the adult tribal seafood consumption scenarios, application of EPA's tribal seafood consumption framework (EPA 2007b) has resulted in the use of Tulalip seafood consumption survey data to characterize adult tribal RME seafood consumption. An additional tribal scenario is also evaluated based on Suquamish seafood consumption survey data. This scenario represents an upper bound on risk for the LDW site (EPA 2005a).

[1] The highest excess cancer risk estimate from incidental ingestion and direct contact with water from swimming in the LDW was 4×10^{-6}, including estimates for both adults and children. All hazard quotients were less than 1 for both adults and children (King County 1999b).

- In characterizing uncertainty in exposure and risks, it is useful to examine CT exposures (National Research Council 1994). CT risk estimates are intended to reflect average exposures. Average exposure estimates are not favored in decision-making because they will underestimate exposure for a substantial number of individuals (EPA 1989).
- Another method of examining exposure is to identify a unit of exposure that a member of the public can use to assess risks associated with their individual behavior. This last approach was used to characterize seafood consumption exposure on an individual basis. The unit of exposure used in this risk assessment was one meal per month. The one-meal-per-month exposure scenario is not meant to actually describe behavior that is occurring on the LDW because there are no actual data on current seafood consumption rates for the LDW. Instead, it is intended to serve as a basis on which individuals can evaluate their own exposure using a method that is readily scaled to various seafood consumption levels (i.e., a change in the rate of consumption from one meal per month to higher or lower amounts results in proportional change in the amount of chemical exposure and risk). This approach is not intended to represent a measured or established consumption rate for the LDW. Older surveys on seafood consumption in Puget Sound suggest that seafood consumption by recreational anglers is much greater than one meal per month (PSEP 1988).

In the first step of the exposure assessment, a risk-based screening was performed using EPA guidance to identify the COPCs to be evaluated. Sixty-four chemicals were identified as COPCs in sediment or tissue for one or more exposure scenarios; of those, eighteen chemicals[2] were identified for more detailed analysis using both seafood consumption and direct sediment exposure scenarios. Of the 64 COPCs, 26 were never detected in either sediment or tissue (or in neither) but were included because analytical reporting limits (RLs) were above the screening criteria. These undetected COPCs were evaluated in the uncertainty analysis.

The exposure assessment identifies equations and parameters used to quantify exposures to COPCs in each scenario. Quantification of exposure consists of an estimate of the chemical intake people might experience, which is calculated from the concentration data for each COPC and health-protective assumptions regarding intake rates of sediment and seafood and the frequency and duration of the intake. Exposure frequency and duration assumptions for the evaluation of direct sediment exposure under the commercial netfishing scenario were based on site use information collected

[2] COPCs identified for both seafood consumption and direct sediment exposure were antimony, arsenic, benzidine, bis(2-chloroethyl)ether, cadmium, carcinogenic polycyclic aromatic hydrocarbons (cPAHs), chromium, copper, total dichlorodiphenyltrichloroethanes (DDTs), dieldrin, lead, mercury, n-nitrosodimethylamine, n-nitroso-di-n-propylamine, polychlorinated biphenyls, vanadium, zinc, and 4,6-dinitro-o-cresol.

from the Muckleshoot Indian Tribe, which conducts commercial netfishing for adult salmon within the LDW. Exposure parameter values for the beach play and clam harvesting scenarios were based primarily on EPA guidance and best professional judgment because site-specific data on exposure frequency and duration for these scenarios were not available.

There were no seafood consumption surveys specific to the LDW available for individuals (e.g., recreational anglers, tribal members, or other communities) who either currently consume seafood or may consume seafood from this resource in the future. Therefore, the rates of seafood ingestion assumed for the seafood consumption scenarios were developed by EPA based on data collected from several surveys. Specifically, for representing seafood consumption by adult tribal members in general, EPA developed a seafood consumption scenario using survey data for adult Tulalip tribal members. This scenario (the adult tribal RME seafood consumption scenario based on Tulalip data) includes a consumption rate of 97.5 g [3] of seafood per day (three meals per week, assuming 227-g [8 oz] meals), based on a Tulalip tribal study on the consumption of resident species of fish and shellfish from the Puget Sound region. This consumption rate was assumed to be applicable to the ingestion of seafood caught from the LDW and was further divided into seafood categories as follows:

- 8.1 g/day for pelagic fish
- 7.5 g/day for benthic fish
- 43.4 g/day for crabs
- 37.7 g/day for clams
- 0.8 g/day for mussels

In the absence of site-specific seafood consumption surveys of tribal members, it is not known if tribal members currently consume seafood from the LDW at the rates assumed or whether they may do so under future conditions. There is uncertainty about the application of these rates to the LDW, and it is likely that current seafood consumption rates within the LDW are lower than those documented in the Tulalip tribal study because of existing seafood consumption advisories. EPA's Superfund risk assessment guidance requires that exposure estimates be protective of future uses (EPA 1989). Tribes with treaty rights to obtain seafood from the LDW may increase their consumption rate in the future as conditions in the LDW improve with regard to chemical contamination. Habitat improvements may also increase the harvestable population of fish and shellfish to some degree. Consequently, the seafood consumption rates evaluated for the RME scenarios in this HHRA are intended to be protective of both current and future uses.

---

[3] Rate does not include consumption of anadromous fish. Total consumption rate including anadromous fish is 194 g/day (EPA 2006b).

Health risks were also quantified for seafood consumption by Asian and Pacific Islanders (API) (5.3 and 51.5 g/day for resident species of fish and shellfish),[4] a second adult tribal scenario based on Tulalip data that was developed as a CT of the data (18.6 g/day), tribal children based on Tulalip data (8.6 and 55.9 g/day), an adult tribal scenario based on a Suquamish tribal survey (583.5 g/day), and adult one-meal-per-month consumers (7.5 g/day). The tribal seafood consumption scenario based on Suquamish data was included at the request of the Suquamish and Muckleshoot tribes to assist in characterizing the range of potential seafood consumption risks. The seafood consumption rates for the Suquamish Tribe are much higher than for the Tulalip Tribes, primarily because of much higher shellfish consumption rates. The LDW lacks extensive high-quality intertidal shellfish habitat that would be necessary to sustain the higher shellfish consumption rates from the Suquamish study, as previously acknowledged by EPA (2007b).

Exposure scenarios for the tribal children based on Tulalip data, adult tribal members based on Suquamish data, and API adults, included a combination of all the seafood categories listed above for the adult tribal RME scenario based on Tulalip data. For the adult one-meal-per-month scenario, risks were evaluated based on a consumption rate of one meal per month of pelagic fish (such as perch), benthic fish fillets (such as English sole and starry flounder), crab edible meat, or clams. Consistent with EPA risk assessment guidance, all assumptions regarding the amounts of seafood ingested in the RME scenarios were selected to be health-protective to avoid underestimating risks. Consequently, individual risk estimates may be overestimates but are unlikely to be underestimates for most chemicals.

Exposure point concentrations (EPCs) are the concentrations of COPCs in sediment and seafood tissue collected from the LDW that were applied in the exposure equations to calculate COPC intake. The EPC is either the maximum concentration or the upper confidence limit on the mean concentration[5] of a COPC and is intended to represent a long-term exposure concentration. In some cases, the EPC was set equal to one-half the maximum RL if this value was higher than the maximum detected concentration or there were no detected concentrations. EPCs for the direct sediment exposure scenarios (i.e., netfishing, beach play, and clam harvesting) were calculated for the sediment area over which the exposure could potentially occur.

The netfishing scenario assumed that people who engage in commercial netfishing could be exposed to both intertidal and subtidal sediment adhering to their nets. For the beach play RME scenario, EPCs were based only on intertidal sediment data (i.e., data from sediments periodically exposed to air during low tides) from areas that are

---

[4] Two scenarios were created for some populations – one corresponding to a reasonable maximum exposure and one corresponding to a central tendency. Rates do not include consumption of anadromous fish. Total rates including anadromous are 57.1 g/day and 5.8 g/day (Kissinger 2005).

[5] Data management rules for calculating EPCs, as presented in Section B.3.4.3, considered the detection frequency and the number of samples.

accessible to the public. The LDW was divided into eight areas for the beach play RME scenarios, corresponding to contiguous areas where access to intertidal sediments is relatively easy. Separate risk estimates were made for each beach. The exposure frequency selected for the beach play RME scenario (i.e., 65 days per year) was based on a survey conducted by King County of parks adjacent to lakes and represents the 95th percentile of exposure frequency for children up to 6 years old who play in sand near the water (Parametrix 2003). This behavior is consistent with the behavior that is assumed for the beach play RME scenario in the LDW. The clam harvesting scenario assumed that people are exposed to COPCs in sediment as they dig for clams. Two clamming scenarios (the tribal clamming RME scenario, which assumes 120 days of clamming per year, and the tribal clamming 183-day-per-year scenario) included all potential clam habitat areas (as identified during a 2004 survey) that could be accessed either by boat or on foot from the bank. Another clamming scenario used a lower exposure frequency (7 days per year) and included only potential clam habitat areas that could be accessed from the bank. Two additional sediment exposure scenarios, habitat biologist and dog walking, were evaluated in the uncertainty analysis section. Although exposures to intertidal sediment for these scenarios were considered to be lower than exposures from netfishing, beach play, or clam digging, and they are more uncertain to estimate, these scenarios were evaluated to provide information to site users.

EPCs for the seafood consumption scenarios were calculated separately for various types of seafood, called consumption categories. Seven consumption categories were developed based on seafood tissue types available for the LDW: pelagic fish, fillets of benthic fish, whole bodies of benthic fish, edible meat of crabs, and whole bodies of crabs, clams, and mussels. Because both consumption rates and COPC concentrations are different for each tissue, each was characterized by category-specific COPC concentrations. In some cases, chemistry data for more than one species were combined within a single consumption category (e.g., Dungeness crab and slender crab edible meat were combined in the crab edible meat category). A COPC intake rate was then calculated for each consumption category using the COPC tissue dataset and the consumption rate for each category. The chemical intakes for each consumption category were then summed within each seafood consumption scenario (except the adult one-meal-per-month scenario) to yield an overall COPC intake for that scenario.

ES.3 TOXICITY ASSESSMENT

EPA toxicity values (i.e., slope factors [SFs] for evaluation of carcinogenic risks or reference doses [RfDs] for evaluation of effects other than cancer) were identified for all COPCs. Toxicity values for each COPC have been established by EPA and other agencies and are based on either laboratory experiments using animals or epidemiological studies of human populations who were unintentionally exposed in the workplace or in the environment. The SFs provide a health-protective means to evaluate risks because they represent upper bound estimates of carcinogenic potency.

Similarly, non-cancer toxicity values (i.e., RfDs) are health-protective because they are typically based on the most sensitive endpoint and population for which adequate data are available and include uncertainty factors or extrapolations to account for sensitive sub-populations or other limitations of the toxicity study data on which they were based.

ES.4 RISK CHARACTERIZATION AND UNCERTAINTY ANALYSIS

Carcinogenic risks and non-carcinogenic health effects were evaluated separately in the HHRA because of fundamental differences in assumptions about the mechanism of these toxic effects. Carcinogenic risk estimates were calculated by multiplying the estimated chemical intake by the SF. Cancer risk estimates were compared to EPA's acceptable risk range of 10^{-6} to 10^{-4} established in the National Contingency Plan for Superfund sites (40 CFR 300). The lifetime risk of developing cancer in the US population is one in two (i.e., 5×10^{-1}) for men and one in three (i.e., 3×10^{-1}) for women (American Cancer Society 2006). A 1×10^{-6} excess cancer risk represents an additional one-in-one-million probability that an individual may develop cancer over a 70-year lifetime as a result of exposure to chemicals in LDW sediments and surface water (either through direct exposure or indirect exposure through the consumption of seafood).

Chemicals with non-carcinogenic health effects are generally not toxic below a certain threshold; a critical chemical dose must be exceeded before adverse health effects are observed. The potential for non-carcinogenic health effects is represented by the ratio of the estimated chemical intake to the critical chemical dose (called a reference dose), and is expressed as a hazard quotient (HQ). Exposures resulting in an HQ less than or equal to 1 are unlikely to result in non-cancer adverse health effects.

Concentrations of hazardous substances that arise from natural or anthropogenic background conditions, unrelated to specific LDW contaminant sources, may contribute to contaminant concentrations in LDW sediment and tissue and therefore represent a portion of the calculated risks. Background data are discussed in several sections of this HHRA. Although both the federal Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and the Washington State Model Toxics Control Act (MTCA) allow for consideration of background concentrations, they use somewhat different approaches in deriving representative concentrations and in their application in decision making. Under the MTCA regulation (Chapter 173-340 of the Washington Administrative Code [WAC]), cleanup levels may be based on background concentrations in certain cases. This HHRA does not provide evaluations of background data, including the selection of appropriate datasets and their statistical analysis, for the purpose of selecting cleanup levels under CERCLA or MTCA. Where evaluations of background data are presented in this HHRA, they are intended only to provide additional information relevant to exposure

and risk estimates. Additional evaluations of background data to support CERCLA or MTCA determinations of cleanup levels will be provided in the RI and/or FS reports.

Estimated excess cancer risks were highest for the seafood consumption scenarios (Table ES-1). The cumulative risk for all carcinogenic chemicals was 3×10^{-3} for the adult tribal RME seafood consumption scenario based on Tulalip data, with the primary contributors being polychlorinated biphenyls (PCBs) (2×10^{-3}) and inorganic arsenic (1×10^{-3}). The cumulative cancer risk for the seafood consumption scenarios did not include dioxins and furans, which were not analyzed in LDW tissue samples. Dioxins and furans were not measured in tissues because it was assumed that these risks would be unacceptable based on other Puget Sound investigations in areas with sediment concentrations lower than those found in the LDW. These studies have consistently detected dioxins and furans at concentrations that would be associated with unacceptable risk at the consumption rates evaluated in this HHRA. The excess cancer risks from inorganic arsenic are largely attributable to the inorganic arsenic concentrations in clams. Inorganic arsenic concentrations in fish and crabs were much lower and were very similar to background tissue concentrations. In general, the risks from total PCBs calculated as a sum of detected Aroclors were equal to or as much as twice the risk resulting from the PCB toxic equivalent (TEQ). Because of this difference, total risk was calculated two ways, first by including PCB TEQ and excluding total PCBs, and then including total PCBs and excluding PCB TEQ. Cumulative excess cancer risks for the other seafood consumption scenarios (except for the adult tribal scenario based on Suquamish data) were lower (approximately 1.3 to 33% of those for the adult tribal RME scenario based on Tulalip data). The risks for the adult tribal scenario based on Suquamish data were 10 times higher than risks for the adult tribal RME scenario based on Tulalip data, reflecting the much higher seafood consumption rate (almost three meals per day) used in the adult tribal scenario based on Suquamish data.

In the evaluation of non-cancer hazards, arsenic and PCBs had HQs greater than 1 for one or more of the adult RME seafood consumption scenarios, indicating some potential for adverse effects other than cancer. In addition, HQs were greater than 1 for the child tribal RME consumption scenario based on Tulalip data for tributyltin (TBT) and vanadium. For the adult tribal scenario based on Suquamish data, two additional chemicals had HQs greater than 1: chromium and mercury. HQs for these chemicals were 1 or less for all other seafood consumption scenarios.

Excess cancer risks for the direct sediment exposure scenarios were much lower than those for the seafood consumption scenarios (Table ES-2). With the exception of the tribal clamming RME scenario and the tribal clamming 183-day-per-year scenario, all excess cancer risk estimates for direct sediment exposure scenarios were less than or equal to 5×10^{-5} (i.e., only 1.7% of the risks from the adult tribal RME seafood consumption scenario based on Tulalip intake data). Total excess cancer risk from the tribal clamming RME scenario was 1×10^{-4} (excluding PCB TEQ), which is 3.3% of the

risks from the adult tribal RME seafood consumption scenario based on Tulalip data, and 3×10^{-4} (excluding PCB TEQ) for the tribal clamming 183-day-per-year scenario. Total excess cancer risk estimates were greater than 1×10^{-6} for the netfishing scenarios and between 5×10^{-6} and 5×10^{-5} for the beach play RME scenarios. No HQs were greater than 1 for any of the direct sediment exposure scenarios.

Table ES-1. Summary of seafood ingestion scenarios

Scenario Name	Ingestion Rate (g/day)						Meals per Month[c]	Exposure Frequency (days/yr)	Exposure Duration (years)	Body Weight (kg)	Excess Cancer Risk[d]	Non-Cancer HI[e]
	Pelagic Fish	Benthic Fish[a]	Crab[b]	Mussel	Clam	Total						
Adult tribal RME (Tulalip data)	8.1	7.5	43.4	0.82	37.7	97.5	13.1	365	70	81.8	3×10^{-3}	47
Adult tribal CT (Tulalip data)	1.3	1.2	6.6	0.1	5.8	15	2.0	365	30	81.8	1×10^{-4}	5
Child tribal RME (Tulalip data)	3.24	3	17.4	0.33	15.1	39.0	5.2	365	6	15.2	7×10^{-4}	104
Child tribal CT (Tulalip data)	0.52	0.5	2.6	0.04	2.3	6.0	0.8	365	6	15.2	7×10^{-5}	10
Adult tribal (Suquamish data)	56	29.1	54.8	5.0	438.6	583.5	78	365	70	79	3×10^{-2}	348
Adult API RME	4.9	2.4	10.6	4.61	29	51.5	6.9	365	30	63	1×10^{-3}	35
Adult API CT	0.5	0.24	1.1	0.47	3	5.3	0.7	365	9	63	2×10^{-5}	2
One meal per month – benthic	--	7.5	--	--	--	7.5	1.0	365	30	71.8	1×10^{-4}	6
One meal per month – crab	--	--	7.5	--	--	7.5	1.0	365	30	71.8	4×10^{-5}	1
One meal per month – clam	--	--	--	--	7.5	7.5	1.0	365	30	71.8	2×10^{-4}	4
One meal per month – pelagic fish	7.5	--	--	--	--	7.5	1.0	365	30	71.8	2×10^{-4}	10

[a] Includes fillet and whole-body consumption.

[b] Includes edible-meat and whole-body consumption.

[c] It is assumed that one meal is equal to 227g (8 ounces). This assumption was applied to both adult and child scenarios, although a child's meal size may be considerably smaller.

[d] Excess cancer risk excludes PCB TEQ; risk from dioxins and furans is not included..

[e] Total across all chemicals. This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The values indicate that the HI may exceed 1 for individual endpoints.

API – Asian and Pacific Islander

CT – central tendency

HI – hazard index (a sum of the HQs for individual chemicals)

RME – reasonable maximum exposure

TEQ – toxic equivalent

Table ES-2. Summary of sediment exposure scenarios

Scenario Name	Exposure Area	Age Class	Incidental Sediment IR (g/day)	Exposure Frequency (days/yr)	Exposure Duration (years)	Skin Surface Area Exposed (cm^2)	Body Weight (kg)	Excess Cancer Risk[a]
Netfishing RME	all subtidal and intertidal	adult	0.050	119	44	3,600[b]	81.8	3×10^{-5}
Netfishing CT	all subtidal and intertidal	adult	0.050	63	29	3,600[b]	81.8	5×10^{-6}
Beach play RME	area 1	0 - 6 yrs	0.20	65	6	varies by age[c] (1,330 to 2,751)	varies by age (9.1 to 19.7)	2×10^{-5}
	area 2							5×10^{-5}
	area 3							3×10^{-5}
	area 4							3×10^{-5}
	area 5							8×10^{-6}
	area 6							9×10^{-6}
	area 7							5×10^{-6}
	area 8							7×10^{-6}
Clamming 7 days per year	intertidal area accessible from the shore	adult	0.1	7	30	6,040[d]	71.8	1×10^{-6}
Tribal clamming RME scenario	intertidal area accessible from the shore or from a boat	adult	0.1	120	64	6,040[d]	81.8	1×10^{-4}
Tribal clamming 183 days per year	intertidal area accessible from the shore or from a boat	adult	0.1	183	70	6,040[d]	81.8	3×10^{-4}

Note: Non-cancer hazards (HQs) did not exceed 1 for any chemical and are therefore not shown in this table.

[a] Excess cancer risk excludes PCB TEQ.

[b] Recommended surface area for commercial/industrial worker. Assumes that head, hands, and forearms are exposed.

[c] Assumes that 35% of the total child body surface area is exposed, roughly corresponding to a child wearing a short-sleeve shirt and short pants but no shoes.

[d] Assumes that 39% of the total adult body surface area is exposed, roughly corresponding to a barefoot individual wearing a short-sleeve shirt and short pants.

API – Asian and Pacific Islander

CT – central tendency

HQ – hazard quotient

RME – reasonable maximum exposure

TEQ – toxic equivalent

The final step of the risk characterization is to identify risk drivers. Risk drivers are defined in this HHRA as chemicals contributing the majority of the site risks and for which quantitative cleanup levels will be proposed in the feasibility study. The starting point for designating risk drivers is to identify chemicals of concern (COCs), which are defined as chemicals with excess cancer risk estimates greater than 1×10^{-6} or an HQ greater than 1 for any RME exposure scenario. Risk drivers were designated from the COC list based on several considerations, including: 1) risk magnitude, relative to both acceptable risk thresholds and total risk estimates, 2) detection frequency, and 3) data quality considerations.

Nineteen chemicals[6] were identified as COCs for the most health-protective RME seafood consumption scenario (e.g., tribal adult based on Tulalip data). Dioxins/furans were assumed to be COCs for seafood consumption, even though no quantitative risk estimate could be made because no site-specific dioxin/furan tissue data were collected from the LDW. It was assumed that dioxin/furan cancer risk estimates for seafood consumption scenarios would have been greater than 1×10^{-6} if these chemicals had been analyzed in tissue samples, based on their high toxicity, ubiquitous presence in the environment, and elevated concentrations (up to 100 times higher than background concentrations) in limited areas of LDW sediments. In addition, two other COCs (TBT and vanadium) were identified for the child tribal scenario based on Tulalip data. Four risk drivers were identified based on seafood consumption: PCBs, arsenic, carcinogenic polycyclic aromatic hydrocarbons (cPAHs), and dioxins/furans.

Five chemicals, including dioxins/furans, were identified as COCs for the direct sediment exposure scenarios. Four of the five COCs (all except for toxaphene) were identified as risk drivers: PCBs, arsenic, cPAHs, and dioxins/furans. The risk estimate for dioxins/furans based on direct sediment exposure was driven by high concentrations in surface sediments at a small number of sampling locations within the LDW. Outside of these areas, dioxin/furan concentrations were similar to concentrations in background sediment locations in the greater Seattle area. Although risk drivers are identified for setting cleanup levels in the FS, the full list of COCs identified in the risk assessments will be further evaluated in subsequent steps in the cleanup process, in consultation with EPA and Ecology. This evaluation may include:

- Assessment of reductions in sediment concentrations or residual risks from these chemicals following the selection of the preferred alternative in the FS
- Review of any new toxicological effects data, as part of the 5-year review that is conducted once a CERCLA cleanup is completed
- Inclusion of these chemicals as part of the post-cleanup monitoring program

[6] PCBs, arsenic, cPAHs, bis(2-ethylhexyl)phthalate, pentachlorophenol, dioxins/furans, and 11 organochlorine pesticides

There are many uncertainties associated with the risk estimates for each exposure scenario in this HHRA. For example, the RME exposure assumptions were developed to result in high-end estimates of risks associated with the LDW. To be health-protective of all members of the general public, these risk estimates are intended to not underestimate risks even for the reasonable maximally exposed individual, and thus are likely to overestimate risks for most individuals for the chemicals that were evaluated.

Risk estimates were highest for the seafood consumption scenarios, but the uncertainties associated with those risk estimates are also very high. The seafood consumption rates based on tribal and API surveys that were used in this HHRA, although based on well-designed consumption surveys, were not specific to the populations who primarily fish the LDW, and it is uncertain how well they represent the behavior of people who eat fish and shellfish primarily from the LDW, either now or in the future. The Muckleshoot and Suquamish Tribes have made clear their interest in future enhancements of resource quality, quantity, and use in the LDW. These risk estimates are intended to provide information to risk managers in remedial planning at the site but should not be equated with actual risks to people currently consuming LDW seafood. Also note that dioxins and furans were not analyzed in seafood samples, so seafood consumption risk estimates are likely underestimated because these chemicals were not included in the quantitative risk assessment for the seafood consumption scenarios.

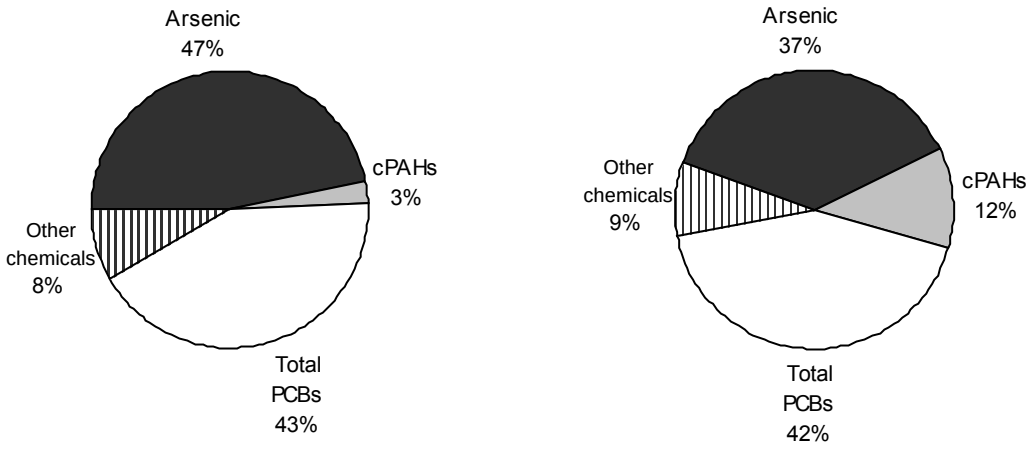
Another important uncertainty is in the methods used to characterize the cancer risks associated with exposures to PCBs. Two methods were used in this HHRA, one based on total PCB data and the cancer SF for total PCBs, and a second based on data for PCB congeners that are thought to have similar toxic effects to dioxins/furans and are evaluated through the cancer SF for dioxins/furans. Because total PCB risk estimation methodology includes, to some degree, the risks posed by dioxin-like PCB congeners, the cancer risk estimates from these two methods were not summed in estimating cumulative risks in order to avoid double-counting cancer risks posed by dioxin-like PCBs. Hence, the risk estimates for the two methods are presented separately in this baseline HHRA. Although this approach avoids the double-counting of dioxin-like PCB cancer risks, it is possible that each method for quantifying PCB cancer risks on its own underestimates the overall PCB health risk. The issues associated with assessing risks posed by environmental PCB mixtures, various approaches for addressing double-counting, and quantitative risk estimates derived using these approaches are discussed in the uncertainty analysis section. It is important that risk managers and interested parties carefully review this information to fully understand the issues involved in characterizing risks from exposure to PCBs.

The excess cancer risk estimates related to inorganic arsenic in the seafood consumption scenarios were almost entirely driven by elevated concentrations of inorganic arsenic in clams. The risk estimates for exposure to inorganic arsenic in

clams are based on eight composite clam tissue samples collected from locations in the LDW where sediment arsenic concentrations were generally similar to or lower than both the average arsenic concentration in LDW sediments and natural background arsenic concentrations. Thus, as described further in the uncertainty analysis section, it is not clear why arsenic concentrations are elevated in LDW clams.

Additional uncertainty analysis provided in this document discusses uncertainties associated with the chemistry data, exposure assumptions, and toxicities of the COPCs. The final risk estimates reflect uncertainties associated with using data and assumptions from multiple sources, but the combined effect of those uncertainties on risk estimates cannot be quantified. However, the assessment tended to overestimate risks more than underestimate them, consistent with the health-protective nature of risk assessment. All or any of the uncertainties are relevant to the risk estimates. In spite of these uncertainties, the baseline characterization of RME risks for the LDW site is considered to be health-protective and sufficient to support risk management decisions.

Risk estimates presented in this HHRA indicate that elevated risks result from exposures to a small number of chemicals, as demonstrated in the distribution of cancer and non-cancer risks by chemical in Figure ES-1 for both adult and child seafood consumption scenarios. The average percent contribution for each chemical or chemical group is shown for the adult scenarios because there was little variability between the adult tribal RME scenario based on Tulalip data, adult API RME scenario, and adult tribal scenario based on Suquamish data. Approximately 85 to 90% of health risks were associated with only or three chemicals (i.e., arsenic, PCBs, or cPAHs), and almost all the risks in the "other chemicals" category were attributed to tentatively identified pesticides. Note that Figure ES-1 does not show risks for dioxins and furans, which were not analyzed in LDW tissue samples. The overwhelming majority of the non-cancer hazards associated with seafood consumption were contributed by total PCBs (> 80% of the total developmental, neurological, and immunological hazard indices). The total PCB HQ for all seafood consumption scenarios, except the adult one-meal-per-month crab scenario, exceeded one.

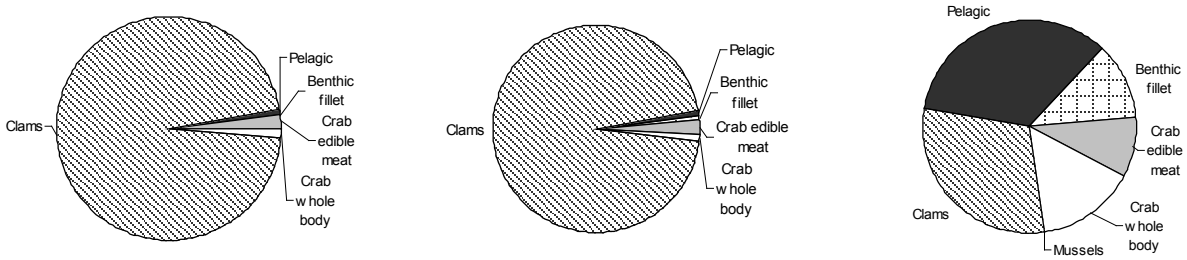


a) Average total cancer risk (excluding PCB TEQ) for adult seafood consumption scenarios

b) Average total cancer risk (excluding PCB TEQ) for child seafood consumption scenarios

Figure ES-1. Seafood consumption scenario risks by chemical

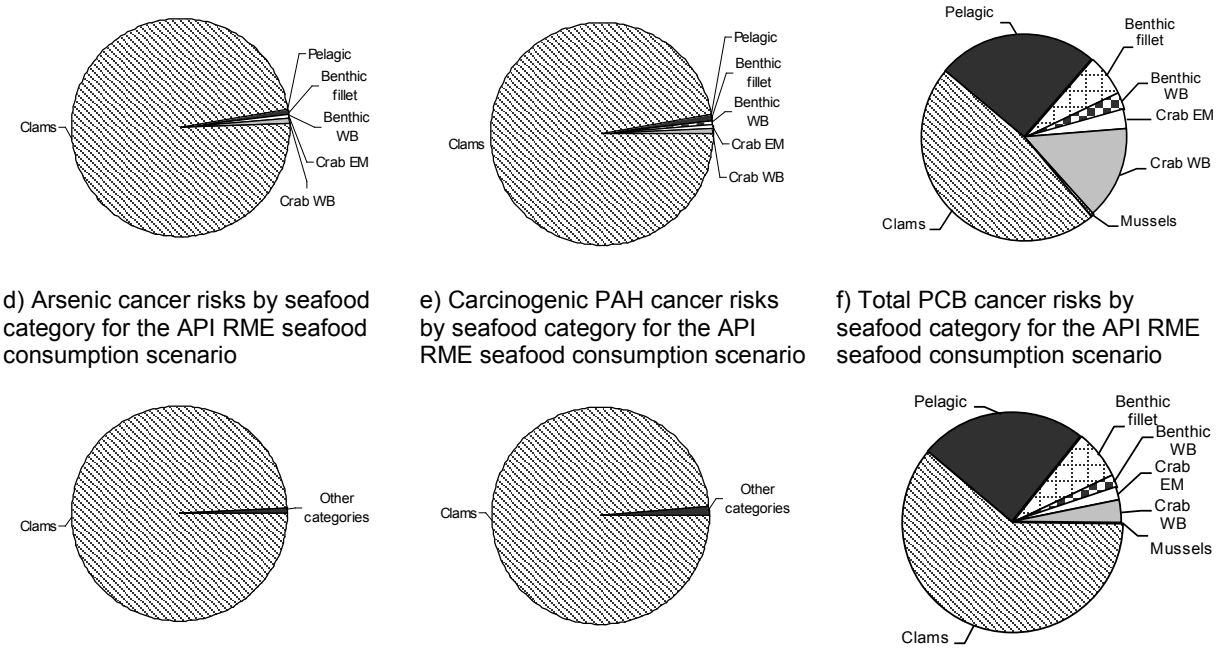
It is also helpful to consider the risk estimates in terms of the proportion of risk estimates related to various seafood types. Elevated risk estimates associated with cPAHs and inorganic arsenic in seafood are largely attributed to clams (Figure ES-2). In contrast to cPAHs and inorganic arsenic, risks from consumption of PCBs in seafood are more evenly divided among the seafood consumption categories (Figure ES-2, panels c, f, and i). There are relatively small differences between the three adult populations (i.e., Tulalip, API, and Suquamish) in the proportion of risks related to seafood types.



a) Arsenic cancer risks by seafood category for the adult tribal RME seafood consumption scenario based on Tulalip data

b) Carcinogenic PAH cancer risks by seafood category for the adult tribal RME seafood consumption scenario based on Tulalip data

c) Total PCB cancer risks by seafood category for the adult tribal RME seafood consumption scenario based on Tulalip data



d) Arsenic cancer risks by seafood category for the API RME seafood consumption scenario

e) Carcinogenic PAH cancer risks by seafood category for the API RME seafood consumption scenario

f) Total PCB cancer risks by seafood category for the API RME seafood consumption scenario

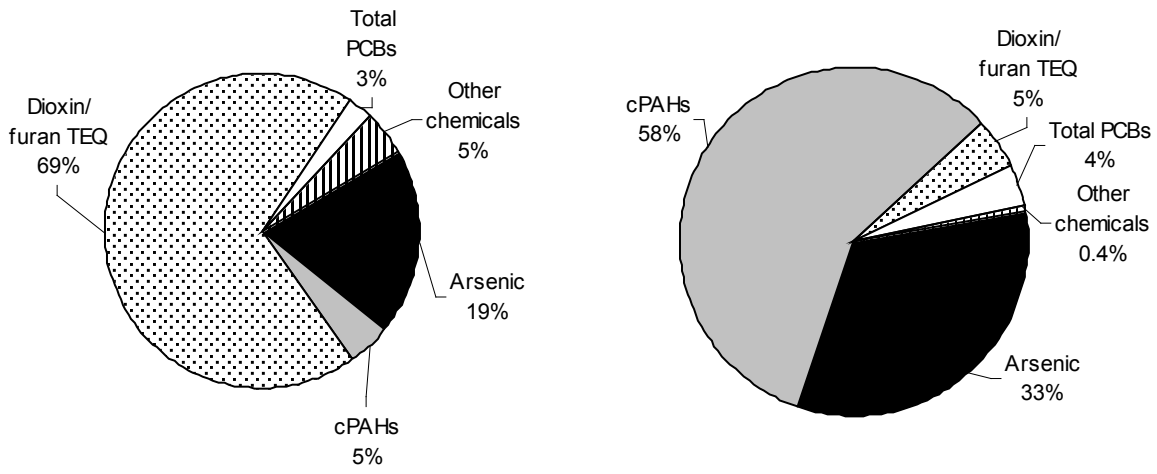
g) Arsenic cancer risks by seafood category for the adult tribal seafood consumption scenario based on Suquamish data

h) Carcinogenic PAH cancer risks by seafood category for the adult tribal seafood consumption scenario based on Suquamish data

i) Total PCB cancer risks by seafood category for the adult tribal seafood consumption scenario based on Suquamish data

Figure ES-2. Cancer risks by seafood category for the adult seafood consumption scenarios

Risk estimates for the direct sediment exposure scenarios were lower than those for seafood consumption. The direct sediment exposure risk estimates were quite different between the adult (i.e., two netfishing and three clamming scenarios) and child (i.e., eight beach play exposure areas) scenarios. The risk contribution by chemical is different for direct sediment exposure scenarios than for seafood exposure scenarios. Dioxin/furan risks contributed the majority (average of 69%) of the risks for the adult direct sediment exposure scenarios, followed by arsenic (average of 19%) (Figure ES-3, panel a). For the beach play RME scenarios, dioxins/furans were much less important to the overall risk estimate (average of 5%), primarily because there were far fewer data available and the highest concentrations from the LDW were not in beach play areas (Figure ES-3, panel b).



a) Average total cancer risk estimate for the two netfishing and three clamming scenarios

b) Average total cancer risk estimate for the eight beach play RME scenarios

Figure ES-3. Cancer risks by chemical for direct sediment exposure scenarios

B.1 Introduction

This document presents the baseline human health risk assessment (HHRA) as part of the remedial investigation and feasibility study (RI/FS) for the Lower Duwamish Waterway (LDW). The LDW was added to the US Environmental Protection Agency's (EPA's) National Priorities List (NPL) under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), also known as Superfund, on September 13, 2001. In February, 2002, the Washington State Department of Ecology (Ecology) also listed the LDW under the authority of the Washington Model Toxics Control Act (MTCA). The key parties involved in the LDW RI/FS are the City of Seattle, King County, the Port of Seattle, and The Boeing Company, working together for this project as the Lower Duwamish Waterway Group (LDWG). Oversight of the LDW RI/FS is being provided by both EPA and Ecology.

The LDW is maintained as a federal navigation channel by the US Army Corps of Engineers (USACE). The shoreline along the majority of the LDW has been developed for industrial and commercial operations (Map B.1-1). Common shoreline features include constructed bulkheads, piers, wharves, sheet piling walls, buildings that extend over the water, and steeply sloped banks armored with riprap or other fill material (Weston 1999). The Seattle neighborhoods of South Park and Georgetown are located to the west and east, respectively, of the LDW (Map B.1-1). These neighborhoods support a mixture of residential, commercial, and industrial uses. The LDW also supports fishing by tribal members and others. Intertidal habitats are dispersed in relatively small patches, with the exception of Kellogg Island, which represents the largest contiguous area of intertidal habitat remaining in the LDW (Tanner 1991). There is great interest in restoring and/or improving the intertidal habitat in the LDW. Several restoration projects have already been completed, some of which included the creation of public parks (Map B.1-1).

Under Superfund regulations, EPA requires that an RI/FS be conducted for all listed sites. An RI evaluates the nature and extent of chemical contamination, estimates baseline human health and ecological risks, and is used by risk managers to identify areas that should be remediated because they pose an unacceptable risk to human health or the environment. An FS proposes alternative approaches to remediating the areas with unacceptable risk and analyzes and compares these alternatives. Cleanup levels are then established in a Record of Decision.

LDWG agreed (in an Administrative Order on Consent, EPA and Ecology 2000) to conduct the RI for the LDW in two phases. The Phase 1 RI is complete (Windward 2003a), including a Phase 1 HHRA (Windward 2003b) based on data that were collected prior to 2003. The Phase 1 HHRA was performed to identify what additional information may be needed to complete the baseline HHRA and was aimed at answering two questions:

- Based on our understanding of current conditions, are there areas within the LDW that might be candidates for early remediation based on risks to human health?
- What are the data gaps? What additional data are needed to better understand risks to human health resulting from exposures to chemicals in LDW sediment?

A work plan (Windward 2004d) was subsequently developed for the second phase of the RI. Although the work plan identified this phase as the Phase 2 RI, the simpler term "RI" is used throughout this document to emphasize the fact that the RI is a stand-alone document that does not rely on any previous documents, such as the Phase 1 RI report. The work plan identified a series of supplemental data collection efforts to fill the identified data gaps. All additional data collection efforts have since been completed.

This baseline HHRA was conducted according to the RI work plan (Windward 2004d). Baseline risk assessments, as defined by EPA (1988) guidance for conducting an RI/FS, "provide an evaluation of the potential threat to human health and the environment in the absence of any remedial action. They provide the basis for determining whether or not remedial action is necessary and the justification for performing remedial actions." The baseline HHRA presents risk estimates for various scenarios whereby people may be exposed to chemicals of potential concern (COPCs) found in sediment and in fish and shellfish tissues from the LDW and summarizes prior risk evaluations related to exposure to COPCs in water. Because knowledge of current and future site use is imperfect, the scenarios evaluated in this assessment have been selected in an attempt to not underestimate risks, and, therefore, may overestimate risks for many site users.

Early cleanup is of great interest because the Superfund cleanup process can take many years. After completion of the Phase 1 RI, LDWG recommended seven areas within the LDW for early cleanup, based on both human health and ecological risks. Partial cleanups at two of those early action areas, the south storm drain near the Norfolk combined sewer outfall/storm drain (CSO/SD) and the Duwamish/Diagonal CSO/SD, were completed in 2003 and 2004, respectively. The designs for cleanup at three other areas (Boeing Plant 2, Terminal 117, and Slip 4) are underway. Although some early actions have been completed, this risk assessment evaluates pre-cleanup, baseline conditions.

The baseline HHRA is based on data previously summarized in the Phase 1 HHRA (Windward 2003b) and data collected since the Phase 1 RI was completed. It has been developed in accordance with both national and regional EPA guidance (EPA 1989, 1991a, 1996a, 1998, 1999d, 2001b). Pursuant to the RI work plan (Windward 2004d), this HHRA was not developed according to the specific MTCA protocols necessary for the development of cleanup levels under Washington Administrative Code (WAC) 173-240-708.

Along with the results of the baseline ecological risk assessment (ERA) (Windward 2003b), the results of this HHRA will be considered in the process for identifying cleanup levels in the FS. The results of selected exposure scenarios presented in the risk assessments will be used in the RI to calculate risk-based threshold concentrations (RBTCs) for chemical contaminants identified as risk drivers. RBTCs are chemical concentrations in sediment or tissue associated with acceptable risk thresholds for specific human exposure scenarios.

This HHRA reports risks associated with a wide variety of human exposure scenarios (e.g., several different seafood consumption scenarios, netfishing, clam digging, beach play). The breadth of the human exposure scenarios evaluated informs risk managers and other interested parties of the potential range of health risks to humans who might be exposed to contaminated media from the LDW on a regular basis. However, not all of these scenarios are appropriate for calculating sediment RBTCs or for establishing cleanup levels. The FS will consider the conservatism and uncertainty associated with RBTCs, along with factors such as background concentrations, when setting the preliminary remedial goals.

This baseline HHRA includes the following sections:

- Section B.2 – Data evaluation
- Section B.3 – Exposure assessment
- Section B.4 – Toxicity assessment
- Section B.5 – Risk characterization
- Section B.6 – Uncertainty analysis
- Section B.7 – Identification of risk drivers
- Section B.8 – Conclusions

Details on site background, previous investigations, and environmental setting are provided in the RI report and are referenced accordingly.

B.2 Data Evaluation

A large amount of chemical and other data was used in the HHRA to derive risk estimates. Figure B.2-1 is a flowchart that shows the various steps in data compilation and calculation that are described in this document. This figure references the section where each step in the process is discussed.

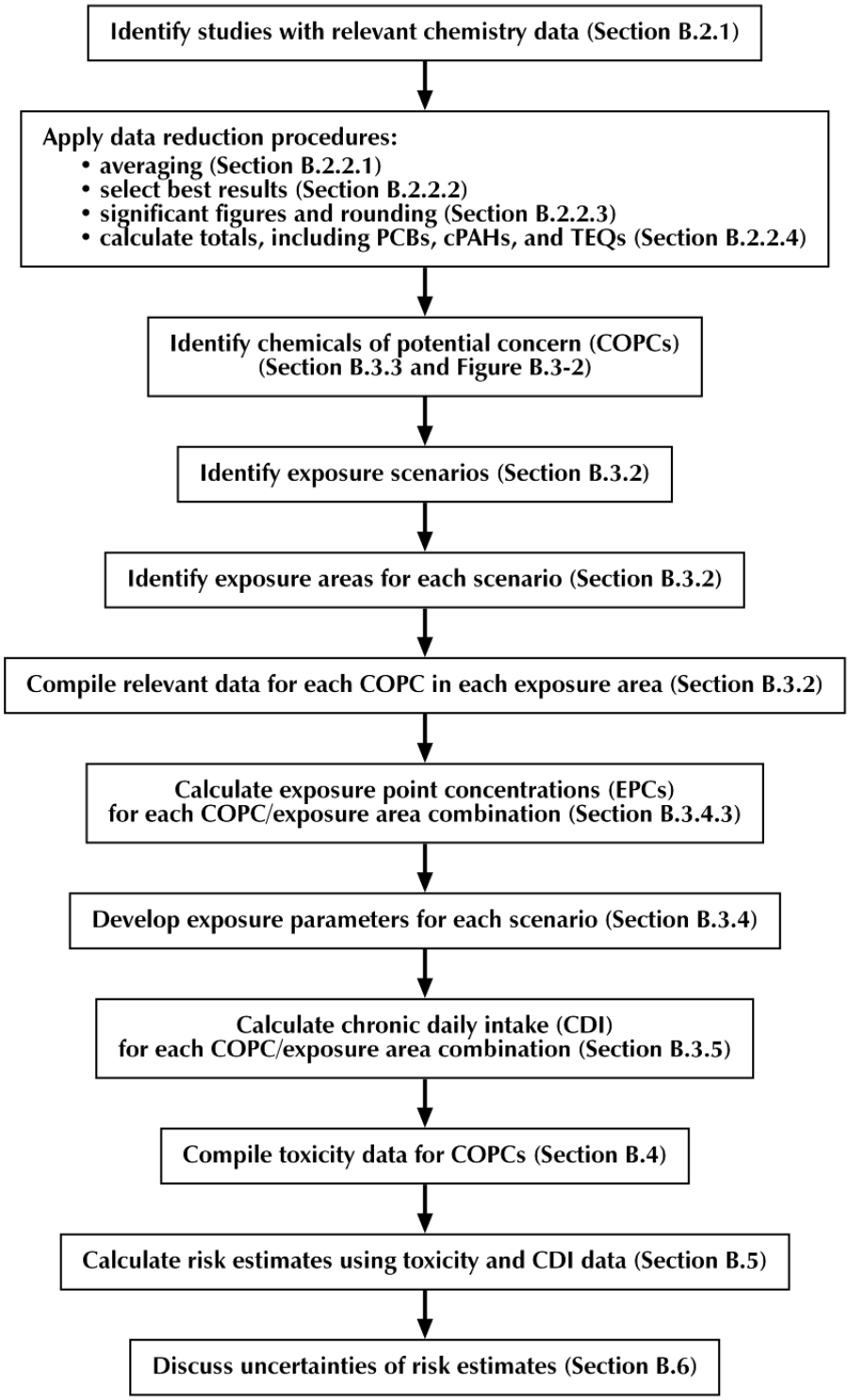


Figure B.2-1. Data flowchart for the HHRA

People may be exposed to chemicals found in LDW sediments either through direct exposure to sediment or indirectly through the consumption of fish and shellfish or contact with water (Section B.3.2). Accordingly, tissue, sediment, and water chemistry data are relevant. The following subsections describe data availability (Section B.2.1), data reduction (Section B.2.2), and the suitability of data for risk assessment purposes (Section B.2.3). Details on data aggregation and calculations are provided in Section B.2.3 and in applicable sections of the exposure assessment (Section B.3) where such calculations are used.

B.2.1 DATA AVAILABILITY AND SELECTION

Many environmental investigations conducted within the LDW have included the collection of chemistry data from samples of fish and shellfish tissue, sediment, or water. The data sources for sediment and tissue are summarized below. Although water data are also available from the LDW, a previous risk assessment conducted by King County has shown that exposure pathways related to water result in much lower exposure compared to other pathways (King County 1999d). Excess cancer risk estimates for hypothetically highly exposed adult and child swimmers in the LDW were less than 1×10^{-6}, which is lower than risk estimates for sediments and much lower than estimates related to seafood consumption. For that reason, water exposure pathways and water data are not quantitatively evaluated in this HHRA. However, risks associated with water exposure, as quantified in the King County (1999d) report, are used in this HHRA to fully characterize health risks from all potential exposures in the LDW. Since the completion of the King County HHRA (1999d), additional polychlorinated biphenyl (PCB) water data have been collected by King County (Mickelson and Williston 2006). The modeled PCB water concentrations used in the 1999 assessment were higher than the empirical PCB water concentrations from 2005, suggesting that the 1999 risk estimates are overestimated and therefore health-protective. Additional details on the assumptions of the King County assessment are presented in the risk characterization section. The representativeness of the risk estimates in the King County study for the LDW site risk assessment was previously discussed in the Phase 1 HHRA (Windward 2003b) and it was concluded that they provided a health-protective means to evaluate risk.

B.2.1.1 Sediment chemistry

The surface sediment chemistry dataset used in the baseline HHRA (hereafter called the baseline surface sediment chemistry dataset) is described in a separate memorandum (Windward 2006d) and summarized briefly below. The baseline surface sediment chemistry dataset was compiled from numerous sampling events that occurred from 1990 to 2005. As agreed by EPA and Ecology, some of the data from these sampling events are not adequate for representing site conditions in the baseline risk assessments for one or more of the following reasons:

- The sediment sampling horizon was too deep to be considered surface sediment.[7]
- The sediment characterized by the sample was removed during maintenance dredging activities after the sample was collected.
- The sample was collected after removal actions at two early action areas (Duwamish/Diagonal and Boeing Developmental Center south storm drain outfall near the Norfolk CSO), and these data were determined not to represent "baseline conditions"
- The sediment sampling location was resampled at a later date, either as part of a monitoring program or to characterize temporal trends. In either case, the latter data replaced the earlier data.
- The quality of the data does not meet Superfund standards, or the data quality could not be confirmed. Details of the data quality are described in the Technical Memorandum: Criteria for Defining the Baseline Surface Sediment Dataset for Use in the Lower Duwamish Waterway RI/FS (Windward 2006d).

Since the RI/FS began, there have been two sediment removal actions at early action areas within the LDW (Duwamish/Diagonal and Boeing Developmental Center south storm drain outfall). There is no EPA policy or guidance about whether baseline risk assessments should include or exclude risk reduction achieved by removal actions that occur during the RI/FS. LDWG plans to evaluate the risk reduction achieved at these two areas in the FS as part of the residual risk assessment. Therefore, data that characterize areas prior to remediation activities at early action areas are included in this baseline HHRA to represent baseline conditions. Data collected after remediation activities at those areas will be included in the FS.

The baseline surface sediment chemistry memorandum (Windward 2006d) summarizes, in a table, all the surface sediment samples that have been collected in the LDW from 1990 to 2005 and identifies those that were included in the baseline surface sediment dataset and those that were excluded, along with the rationale for their exclusion. The summary table from that memorandum will also be included as an appendix to the RI report. The sediment sampling events that are included in the baseline dataset are listed in Table B.2-1. The baseline surface sediment sampling locations are shown on Map B.2-1.

[7] For the purposes of this risk assessment, surface sediment samples are those collected from the top 15 cm of the sediment horizon. Sediment samples that include less than 15 cm of sediment are included; samples that include the top 15 cm but also include deeper sediment in the same sample are not included here because analyses were not performed separately on the two horizons (< 15 cm and > 15 cm). The uncertainty associated with defining the baseline surface sediment chemistry dataset in this manner is discussed in Section B.6.

Table B.2-1. Summary of studies included in the baseline surface sediment dataset

Sampling Event	Event Code	Year	Chemicals	Number of Sampling Locations[a]	Source
LDW RI. Chemical analyses of benthic invertebrate and clam tissue samples and co-located sediment samples.	LDWRI-Benthic	2005	metals, SVOCs, PCB Aroclors, selected PCB congeners on subset of samples, butyltins	35	Windward (2005b)
LDW RI. Data report: surface sediment sampling for chemical analyses and toxicity testing.	LDWRI-Surface Sediment	2005	metals, SVOCs, PCB Aroclors, selected PCB congeners and dioxins and furans on subset of samples, butyltins	160	Windward (2005d; 2005e)
Boyer Towing dock replacement	Boyer Towing	2004	metals, SVOCs, PCB Aroclors, TBT	3	WR Consulting (2004)
Slip 4 early action area site characterization	Slip4-EarlyAction	2004	PCB Aroclors, mercury	30	Integral (2004)
Rhône-Poulenc surface/subsurface sampling	RhônePoulenc 2004	2004	metals, organochlorine pesticides, SVOCs	21	EPA (2005d)
Norfolk CSO sediment remediation project 5-year monitoring program: annual monitoring report – year 5, April 2004	Norfolk-monit7	2004	metals, PCB Aroclors, SVOCs	4	King County (2005)
Triad approach to characterize PCB in a Washington riverine sediment site (USACE)	Jorgensen August 2004	2004	metals, PCB Aroclors, SVOCs	43	unpublished data
Duwamish/Diagonal perimeter monitoring – pre-dredge	DuwDiag-October2003	2003	metals, PCB Aroclors, organochlorine pesticides, SVOCs	12	King County (2005)
Terminal 117 early action area site characterization	T117 Boundary Definition	2003-2004	PCB Aroclors; metals, TBT and SVOCs on selected samples	54	Windward (2004a; 2004b)
Boeing Plant 2 transformer investigation – Phase 1	Plant 2-Trans-former Phase1	2003	PCB Aroclors	6[b]	Floyd Snider McCarthy (2004)
Norfolk combined sewer overflow (Duwamish River) sediment cap recontamination. Phase I investigation.	Ecology-Norfolk	2002	PCB Aroclors	17	Ecology (2003)
Norfolk CSO sediment remediation project 5-year monitoring program: Annual monitoring report – year 3, April 2002	Norfolk-monit5	2002	metals, PCB Aroclors, SVOCs	1	King County (2002)
Norfolk CSO 5-year monitoring program, year 2, April 2001	Norfolk-monit4	2001	metals, PCB Aroclors, SVOCs	1	King County (2001b)
Norfolk CSO 5-year monitoring program – 12-month post-construction	Norfolk-monit3	2000	metals, PCB Aroclors, SVOCs	1	King County (2000c)
Norfolk CSO 5-year monitoring program – supplemental nearshore sampling	Norfolk-monit2b	2000	metals, PCB Aroclors, SVOC	3	King County (2000b)
Outfall and nearshore sediment sampling report, Duwamish facility	James Hardie Outfall	2000	metals, PCB Aroclors, SVOCs	9	Weston (2000)
Norfolk CSO 5-year monitoring program – 6-month post-construction	Norfolk-monit2a	1999	metals, PCB Aroclors, SVOCs	2	King County (2000d)

Sampling Event	Event Code	Year	Chemicals	Number of Sampling Locations[a]	Source
Norfolk CSO 5-year monitoring program – post-backfill	Norfolk-monit1	1999	metals, PCB Aroclors, SVOCs	2	King County (1999e)
EPA site inspection: Lower Duwamish River	EPA SI	1998	metals, organochlorine pesticides, PCB Aroclors and selected congeners, dioxins and furans, TBT, SVOCs, VOCs	251	Weston (1999)
King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay	KC WQA	1997	metals, PCB Aroclors, SVOCs, tetrabutyltin	14	King County (1999d)
Duwamish Waterway Phase 1 site characterization	Boeing SiteChar	1997	metals, PCB Aroclors, SVOCs	79[b]	Exponent (1998)
Duwamish Waterway sediment characterization study	NOAA SiteChar	1997	total PCBs, selected PCB congeners, total PCTs	299	NOAA (1997; 1998)
Seaboard Lumber site, Phase 2 site investigation	Seaboard-Ph2	1996	metals, PCB Aroclors, SVOCs	20	Herrera (1997)
Rhône-Poulenc seep sampling	Rhône-Poulenc RFI-3	1996	metals, phenols	14	Rhône-Poulenc (1996)
RCRA Facility Investigation Duwamish Waterway sediment investigation, Plant 2 – Phase 2b	Plant 2 RFI-2b	1996	metals, PCB Aroclors, phthalates	36	Weston (1998a)
Duwamish/Diagonal cleanup Study – Phase 2	Duw/Diag-2	1996	metals, PCB Aroclors, SVOCs	10	King County (2000a)
Duwamish/Diagonal cleanup Study – Phase 1.5	Duw/Diag-1.5	1995	metals, PCB Aroclors, SVOCs	9	King County (2000a)
Norfolk CSO sediment cleanup study – Phase 3	Norfolk-cleanup3	1995	PCB Aroclors, SVOCs	12	King County (1996)
Norfolk CSO sediment cleanup study – Phase 2	Norfolk-cleanup2	1995	metals, organochlorine pesticides, PCB Aroclors and selected congeners, SVOCs	2	King County (1996)
RCRA Facility Investigation Duwamish Waterway sediment investigation, Plant 2 – Phase 2a	Plant 2 RFI-2a	1995	metals, PCB Aroclors SVOCs	54	Weston (1998a)
RCRA Facility Investigation Duwamish Waterway sediment investigation, Plant 2 – Phase 1	Plant 2 RFI-1	1995	metals, PCB Aroclors, TPH, SVOCs, VOCs	66	Weston (1998a)
Duwamish/Diagonal cleanup study – Phase 1	Duw/Diag-1	1994	metals, organochlorine pesticides, PCB Aroclors, SVOCs	31	King County (2001a)
Norfolk CSO sediment cleanup study – Phase 1	Norfolk-cleanup1	1994	metals, organochlorine pesticides, SVOCs, PCB Aroclors	13	King County (1996)
Rhône-Poulenc RCRA Facility Investigation for the Marginal Way facility – Round 2	Rhône-Poulenc RFI-2	1994	SVOCs	6	Rhône- Poulenc (1995)
Results of sampling and analysis, sediment monitoring plan, Duwamish Shipyard, Inc.	Duwamish Shipyard	1993	metals, SVOCs, TBT	1	Hart Crowser (1993)
Harbor Island remedial investigation	Harbor Island RI	1991	metals, organochlorine pesticides, PCB Aroclors, SVOCs, VOCs, TPH, TBT	9	Weston (1993)

[a] Samples are surface sediment grab samples from 0-to-15-cm depth unless otherwise noted.

[b] Samples collected were from 0-to-5-cm depth. Sample total does not include three reference samples that were collected upstream of the study area.

CSO – combined sewer overflow
EPA – US Environmental Protection Agency
KC – King County
LDW – Lower Duwamish Waterway
NOAA – National Oceanic and Atmospheric Administration
PCB – polychlorinated biphenyl
PCT – polychlorinated terphenyl
RCRA – Resource Conservation and Recovery Act
RI – remedial investigation
SVOC – semivolatile organic compound
TBT – tributyltin
TPH – total petroleum hydrocarbons
USACE – US Army Corps of Engineers
VOC – volatile organic compound

Both intertidal and subtidal sediment chemistry data are used in the baseline HHRA. An elevation of -2 ft mean lower low water (MLLW) was used to divide intertidal and subtidal locations, which corresponds to the shoreline (i.e., land/water interface) elevation defined by the aerial photos taken by the US Fish and Wildlife Service (USFWS) in 1999 (USFWS 2000). Approximately 600 surface sediment samples (i.e., 15 cm depth or less) were collected from intertidal locations, and approximately 750 surface sediment samples were collected from subtidal locations (Map B.2-1). A summary of baseline surface sediment chemistry data is provided in Attachment 1 (Table 1) of this HHRA.

B.2.1.2 Fish and shellfish tissue chemistry

Tissue chemistry data for the study area are available for several different tissue types from several sampling events conducted since 1995. Site-specific tissue chemistry data are available for the following species: chinook salmon, coho salmon, English sole, starry flounder, Dungeness crab, red rock crab, slender crab, mussels, soft-shell clam, shiner surfperch, striped perch, pile perch, and Pacific staghorn sculpin. People may consume many of these species. There may be other species found in the LDW that are also consumed by people to some degree, but there are no available tissue chemistry data for these other species. Table B.2-2 lists the fish species that have been found in the LDW. Note that the HHRA seafood consumption rates are based on seafood consumption data for Puget Sound. There are many species in Puget Sound that may be consumed by people (e.g., speckled sanddab, Pacific cod, rockfish, spiny dogfish, walleye pollock) but are rarely found in the LDW, and no LDW tissue chemistry data are available for these species.

Table B.2-2. Fish species found in the Lower Duwamish Waterway

Common Name	Scientific Name	Family	Abundance[a]	Abundance Citation	Environment	Habitat	E/H Citation	Diet	Diet Citation
American shad	*Alosa sapidissima*	Engraulidae	rare	9, 10, 11, 12	anadromous	bays, estuaries, freshwater	32	plankton, copepods, mysids, small fish	33
Bay goby	*Lepidogobius lepidus*	Gobiidae	rare	2, 3, 6	marine (estuary)	benthic (mud bottom)	13	benthic organisms	28
Bay pipefish	*Syngnathus grisiolineatum*	Syngnathidae	common	11	marine	demersal (associated with eel grass in the intertidal areas)	15	isopods, amphipods	14
			rare	6, 10					
Big skate	*Raja binoculata*	Rajidae	rare	7, 11	marine	benthic (sandy and gravelly bottoms)	16	crustaceans, fish	14
Blackbelly eelpout	*Lycodopsis pacifica*	Zoarcidae	rare	11	marine	over soft bottoms	32	worms, crustaceans, small bivalves, brittle stars	34
Brown rockfish	*Sebastes auriculatus*	Scorpaenidae	rare	11, 12	marine	shallow, low-profile, rocky reefs	32	finfish, benthic crustaceans, fish eggs, larvae	35
Buffalo sculpin	*Enophrys bison*	Cottidae	rare	1, 2, 3, 4, 7, 11, 12	marine (estuary)	benthic (inshore rocky and sandy areas)	13	mainly algae, also amphipods, small fishes, crabs, polychaetes, nudibranchs, isopods	13, 29
Bull trout	*Salvelinus confluentes*	Salmonidae	rare	6, 9	anadromous	benthopelagic (near shore)	21	mainly fish, plus zooplankton	31
Butter sole	*Isopsetta isolepis*	Pleuronectidae	common	6	marine (estuary)	benthic (sandy bottom)	13	worms, fish, shrimps	14
			rare	7					
Chinook salmon[b]	*Oncorhynchus tshawytscha*	Salmonidae	abundant	1, 4, 5, 6, 9, 10	anadromous	benthopelagic	27	juveniles: insects, epibenthic crustaceans, pelagic organisms	30
			rare	2					
Chum salmon	*Oncorhynchus keta*	Salmonidae	abundant	5, 6, 9	anadromous	benthopelagic	27	juveniles: copepods, amphipods, cumaceans, euphausiids	29
			common	10					
			rare	1, 4					
C-O sole	*Pleuronichthys coenosus*	Pleuronectidae	rare	7, 11	marine	benthic (flat bottoms, rocky areas)	13	isopods, fish, polychaetes, amphipods, turbellarians, bivalves	29

Table B.2-2, cont. Fish species found in the Lower Duwamish Waterway

Common Name	Scientific Name	Family	Abundance[a]	Abundance Citation	Environment	Habitat	E/H Citation	Diet	Diet Citation
Coho salmon[b]	*Oncorhynchus kisutch*	Salmonidae	abundant	6, 9, 10	anadromous	benthopelagic	27	juveniles: insects, epibenthic crustaceans, pelagic organisms, small fish	29
			common	4, 10					
			rare	1, 2					
Crescent gunnel	*Pholis laeta*	Pholidae	rare	6, 9, 11	marine (estuary)	demersal (intertidal areas, under rocks)	13	gammarid amphipods, copepods, tanaids, isopods	29
Cutthroat trout	*Oncorhynchus clarki*	Salmonidae	rare	1, 4, 5, 6, 9, 10	anadromous	benthopelagic	22	fish, epibenthic crustaceans, pelagic organisms, insects	18
Dolly Varden	*Salvelinus malma*	Salmonidae	rare	1, 4	freshwater	benthopelagic	21	fish, epibenthic crustaceans, pelagic organisms, insects	14
Dover sole	*Microstomus pacificus*	Pleuronectidae	common	2, 11	marine	benthic (mud bottom)	13	benthic invertebrates, echinoderms, mollusks, polychaetes	24
			rare	3					
English sole	*Parophrys vetulus*	Pleuronectidae	abundant	2, 3, 4, 7, 11, 12	marine (estuary)	benthic (sand and mud bottoms)	18	cumaceans, gammarid amphipods, polychaetes, tanaids, crabs, bivalves	29
			rare	1, 6					
Eulachon	*Thaleichthys pacificus*	Osmeridae	rare	3	anadromous	pelagic	13	plankton (feeds only while at sea)	20
Flathead sole	*Hippoglossoides elassodon*	Pleuronectidae	rare	2, 11, 12	marine	benthic (soft mud bottom, adults below 180 m)	13	polychaetes, cumaceans, gammarid amphipods, isopods, bivalves	29
Gunnel sp.	*Apodichthys sp.*	Pholidae	rare	10	marine	intertidal zone among rocks and shallow eelgrass beds	32	small crustaceans, mollusks	13
Great sculpin	*Myoxocephalus polyacanthocephalus*	Cottidae	rare	11	marine	intertidal areas, sand and mud bottoms	13	small fish	13
Hybrid sole	*Inopsetta Isopsetta ischyra*	Pleuronectidae	rare	1, 12	marine (estuary)	benthic	13	benthic organisms	14
Kelp perch	*Brachyistius frenatus*	Embiotocidae	rare	9	marine	among fronds in kelp beds from near surface to depths of about 30 m	32	small crustaceans, parasites	13

Table B.2-2, cont. Fish species found in the Lower Duwamish Waterway

Common Name	Scientific Name	Family	Abundance[a]	Abundance Citation	Environment	Habitat	E/H Citation	Diet	Diet Citation
Largescale sucker	*Catostomus macrocheilus*	Catostomidae	rare	1, 2, 4, 6	freshwater	demersal	21	algae, diatoms, insects, amphipods, and mollusks	20
Longfin sculpin	*Jordania zonope*	Cottidae	rare	11	marine	demersal, intertidal areas, rocky areas and kelp	13	amphipods, benthic copepods, crabs, shrimp, gastropods, polychaetes	38
Longfin smelt	*Spirinchus thaleichthys*	Osmeridae	abundant	1, 2, 11	anadromous	benthopelagic (close to shore, in bays and estuaries)	21	crab larvae, copepods, mysid shrimp	29
			common	12					
			rare	7, 9					
Longnose dace	*Rhinichthys cataractae*	Cyprinidae	rare	6	freshwater	demersal	21	mayflies, blackflies, and midges	20
Longnose skate	*Raja rhina*	Rajidae	rare	11	marine	partially or entirely buried in sand or silt bottoms	36	small fish, crustaceans, worms, mollusks	36
Mountain whitefish	*Prosopium williamsoni*	Salmonidae	rare	1, 6, 9	freshwater	benthopelagic	14	insects, invertebrates, eggs, small fish	14
Northern pikeminnow	*Ptychocheilus oregonensus*	Cyprinidae	rare	1, 6	freshwater	benthopelagic	20	insects, fish	20
Northern ronquil	*Ronquilus jordani*	Bathymasteridae	rare	11	marine	demersal	13	polychaetes, plankton, invertebrates, cladocerans, copepods	14
Northern sculpin	*Icelinus borealis*	Cottidae	rare	6	marine	demersal	13	benthic crustaceans, shrimps/prawns	14, 29
Pacific cod	*Gadus macrocephalus*	Gadidae	rare	2, 3, 4	marine	(demersal, continental shelf and upper slopes)	23	fish, octopi, large crustaceans, worms, amphipods	26, 29
Pacific herring	*Clupea pallasi*	Clupeidae	abundant	4, 9, 11	marine	benthopelagic (coastal, first year in bays)	14	planktonic crustaceans, fish larvae	14, 29
			common	1, 2, 7, 12					
			rare	6, 10					
Pacific sand dab	*Citharichthys sordidus*	Paralichthyidae	common	11	marine	over soft sand bottoms	13	benthic crustaceans, worms	24
			rare	12					

Table B.2-2, cont. Fish species found in the Lower Duwamish Waterway

Common Name	Scientific Name	Family	Abundance[a]	Abundance Citation	Environment	Habitat	E/H Citation	Diet	Diet Citation
Pacific sandlance	*Ammodytes hexapterus*	Ammodytidae	abundant	6, 9	marine (brackish)	benthopelagic (surface or burrowed in sand)	13	zooplankton	17, 29
			common	4					
			rare	1, 10, 11					
Pacific staghorn sculpin	*Leptocottus armatus*	Cottidae	abundant	1, 2, 3, 4, 6, 9, 10, 11, 12	marine (lower estuary, offshore)	benthic (sandy bottom)	13	isopods, bivalve siphons, polychaetes, crabs, fish, tanaids, shrimp	19
			common	7					
Pacific tomcod	*Microgadus proximus*	Gadidae	abundant (juveniles)	7, 11	marine (brackish)	benthic (over sand)	23	shrimp, amphipods, isopods, gastropods, mussels, fishes	24
			common	2, 3, 12					
			rare	1, 4					
Padded sculpin	*Artedius fennestralis*	Cottidae	common	2, 3	marine	benthic	13	gammarid amphipods, isopods, tanaids, shrimp, copepods, small fish	18, 29
			rare	7, 12					
Peamouth chub	*Mylocheilus caurinus*	Cyprinidae	rare	9	freshwater	demersal (brackish)	21	aquatic insects, larvae, terrestrial insects, crustaceans, mollusks, small fish	21
Penpoint gunnel	*Apodichthys flavidus*	Pholidae	rare	5, 6, 9	marine (estuary)	demersal (intertidal tide pools)	13	isopods, amphipods, shrimp, gastropods, other epibenthic crustaceans	29
Pile perch	*Rhacochilus vacca*	Embiotocidae	abundant	12	marine	demersal (rocky shores; near kelp, pilings, underwater structures)	13	isopods, bivalves, crabs, amphipods	29
			common	4, 7, 11					
			rare	1, 2, 3, 6, 9					
Pink salmon[b]	*Oncorhynchus gorbuscha*	Salmonidae	rare	6	anadromous	benthopelagic	27	juveniles: copepods, amphipods, barnacle larvae, cumaceans	27, 28
Plainfin midshipman	*Porichthys notatus*	Batrachoididae	common	11	marine	benthic (nearshore shelf, sand/mud bottom)	18	crustaceans, fish	14
			rare	2					
Prickly sculpin	*Cottus asper*	Cottidae	common	12	marine	benthic	13	benthic organisms	20
			rare	1, 2, 3, 4, 6, 9, 11					

Table B.2-2, cont. Fish species found in the Lower Duwamish Waterway

Common Name	Scientific Name	Family	Abundance[a]	Abundance Citation	Environment	Habitat	E/H Citation	Diet	Diet Citation
Pygmy poacher	*Odontopyxis trispinosa*	Agonidae	rare	2, 3, 7, 11	marine	demersal (soft bottoms)	13	epibenthic invertebrates	14
Ratfish	*Hydrolagus colliei*	Chimeridae	rare	2, 7, 11	marine	demersal (sandy bottom)	13	worms, bivalves, crustaceans, fishes	17, 29
Redsided shiner	*Richardsonius balteatus*	Cyprinidae	common	6	freshwater	demersal	20	zooplankton, algae, insects	20
Rex sole	*Errex zachirus*	Pleuronectidae	rare	11	marine	demersal	37	worms, benthic crustaceans, mollusks	24
River lamprey	*Lampetra ayresi*	Petromyzontidae	rare	1, 4, 6, 9	anadromous	demersal	14	adult: fish juveniles: detritus, algae	20
Rock sole	*Lepidopsetta bilineata*	Pleuronectidae	abundant	7,11	marine (estuary)	benthic (more pebbly bottom than most other flatfish)	13	isopods, gammarid amphipods, polychaetes, cumaceans, bivalves, crabs, fish	29
			common	2, 3, 12					
Rockfish	*Sebastes* spp.	Scorpaenidae	rare	1, 8	marine	demersal (near structure)	25	crabs, gammarid amphipods, mysids, shrimp, fish	26
Roughback sculpin	*Chitonotus pugeteneis*	Cottidae	common	11,12	marine	benthic (sand/mud bottom)	13	shrimps and other crustaceans	18
			rare	2, 3, 7					
Saddleback gunnel	*Pholis ornata*	Pholidae	rare	3, 5, 6, 9, 11, 12	marine (estuary)	demersal (sandy bottom)	13	amphipods, isopods, polychaetes, copepods, cumaceans	29
Sand sole	*Psettichthys melanostictus*	Pleuronectidae	common	1, 2, 3, 7, 11, 12	marine, estuary	benthic (sandy bottom)	14	fishes, worms, crustaceans, and mollusks	14, 29
			rare	1					
Sailfin sculpin	*Nautichthys oculofasciatus*	Hemitripteridae	rare	11	marine	over rocks from inshore to depths of 110 m, often with algae	32	finfish, benthic crustaceans	19
Sharpnose sculpin	*Clinocottus acuticeps*	Cottidae	rare	6	marine	benthic (sand/vegetation)	13	benthic organisms	22
Shiner surfperch	*Cymatogaster aggregata*	Embiotocidae	abundant	1, 4, 5, 6, 7, 9, 10, 11, 12	marine (estuary)	demersal (in shallow water, around eelgrass beds, piers and pilings commonly in bays and quiet back waters)	13	amphipods, cumaceans, polychaetes, copepods, isopods, algae	22, 29
			common	2, 3					

Table B.2-2, cont. Fish species found in the Lower Duwamish Waterway

Common Name	Scientific Name	Family	Abundance[a]	Abundance Citation	Environment	Habitat	E/H Citation	Diet	Diet Citation
Slender sole	*Lyopsetta exilis*	Pleuronectidae	rare	3, 11	marine	benthic (> 200 m depth)	13	carnivore	24
Snake prickleback	*Lumpenus saggita*	Stichaeidae	abundant	1, 2, 3, 4, 6	marine	benthopelagic (shallow bays and offshore waters)	13	bivalves, marine worms, amphipods	29
			common	9, 10, 11, 12					
			rare	7					
Sockeye salmon[b]	*Oncorhynchus nerka*	Salmonidae	rare	40	anadromous	benthopelagic	27	juveniles: insects, epibenthic crustaceans, pelagic organisms	28
Soft sculpin	*Gilbertidia sigalutes*	Cottidae	rare	4	marine	demersal	13	epibenthic crustaceans, phytoplankton, fish eggs/larvae	14
Speckled sanddab	*Citharichthys stigmaeus*	Bothidae	rare	7, 9, 11	marine	benthic (sandy bottom)	13	crustaceans, fish	19
Spiny dogfish	*Squalus acanthias*	Squalidae	rare	2, 11	marine	benthopelagic	26	primarily fish	27
Starry flounder	*Platichthys stellatus*	Pleuronectidae	abundant	1, 2, 3, 4, 6, 7, 9, 10, 11, 12	marine (estuary, brackish)	benthic	22	isopods, fish, gammarid amphipods, polychaetes, gastropods, worms	14
			common	5					
Steelhead[b]	*Oncorhynchus mykiss*	Salmonidae	common	9, 10	anadromous	benthopelagic	39	juveniles: insects, epibenthic crustaceans, pelagic organisms	29
			rare	1, 4, 5, 6, 11					
Striped seaperch	*Embiotoca lateralis*	Embiotocidae	common	1, 4, 12	marine	demersal	13	amphipods, isopods, crabs, shrimp	29
			rare	2, 3, 5, 6, 7, 9, 10					
Sturgeon poacher	*Podothecus acipenserinus*	Agonidae	rare	3, 11	marine	demersal (soft bottom)	13	cumaceans, gammarid amphipods, shrimp, copepods, polychaetes, tanaids	29
Surf smelt	*Hypomesus pretiosus*	Osmeridae	abundant	9	marine (brackish)	benthopelagic	22	isopods, cumaceans, larvaceans, copepods, amphipods	29
			common	1, 4, 6, 7					
			rare	11					
Three-spine stickleback	*Gasterosteus aculeatus*	Gasterosteidae	common	1, 5, 6,10, 11	marine, anadromous	benthopelagic (in/near vegetation)	21	worms, crustaceans, insects/larvae, small fish	20, 29
			rare	4, 12					

Table B.2-2, cont. Fish species found in the Lower Duwamish Waterway

Common Name	Scientific Name	Family	Abundance[a]	Abundance Citation	Environment	Habitat	E/H Citation	Diet	Diet Citation
Torrent sculpin	*Cottus rhotheus*	Cottidae	rare	11	freshwater	demersal	21	crustaceans, midges and mayflies larvae, minnows	21
Tubesnout poacher	*Pallasina barbata*	Agonidae	rare	3, 11	marine	demersal (eelgrass & seaweeds)	13	amphipods, polychaetes, copepods, mysids	29
Walleye pollock	*Theragra chalcogramma*	Gadidae	rare	1, 2, 4	freshwater	benthopelagic	23	insects, midge larvae, fish	14
Whitespotted greenling	*Hexagrammos stelleri*	Hexagrammidae	common	7	marine (intertidal)	demersal (nearshore, near rocks, pilings and eelgrass beds)	23	gammarid amphipods, shrimp, crabs, fish, polychaetes	29
			rare	2, 11					

[a] Abundance: abundant (numerically dominant); common (occurs in most samples); rare (occurs in few samples). Abundance characterizations reflect LDW data collected by authors in the cited study. These data may reflect sampling gear bias for the species identified.

[b] Adults are found in the LDW only as they migrate to spawning ground upstream of the LDW and include wild and hatchery species.

E/H – environment/habitat

Citations

1. Matsuda et al. (1968)
2. Miller et al. (1975)
3. Miller et al. (1977a)
4. Weitkamp and Campbell (1980)
5. Taylor et al. (1999)
6. Warner and Fritz (1995)
7. West et al. (2001)
8. Malins et al. (1980)
9. Shannon (2006)
10. Windward (2004b)
11. Windward (2005c)
12. Windward (2006b)
13. Eschmeyer et al. (1983)
14. Hart (1973)
15. Dawson (1985)
16. McEachran and Dunn (1998)
17. Armstrong (1996)
18. Clemens and Wilbey (1961)
19. Fitch and Lavenberg (1975)
20. Scott and Crossman (1973)
21. Page and Burr (1991)
22. Morrow (1980)
23. Cohen et al. (1985)
24. Pearcy and Hancock (1978)
25. Lamb and Edgel (1986)
26. Cox and Francis (1997)
27. Groot and Margolis (1998)
28. Grossman (1979)
29. Miller et al. (1977b)
30. Cordell et al. (2001)
31. Rieman and McIntyre (1993)
32. Gilbert and Williams (2002)
33. Whitehead (1985)
34. Anderson (1994)
35. Hobson (2000)
36. Florida Museum of Natural History (2005)
37. Cooper and Chapleau (1998)
38. Demetropoulos et al. (1990)
39. Gall and Crandell (1992)
40. Kerwin and Nelson (2000)

Marine invertebrates are also found in the LDW. Table B.2-3 lists the invertebrate species found during LDWG sampling efforts for clams (Windward 2004a) and fish and crabs (Windward 2005c, 2006b). Some of these invertebrates, notably some of the clams, crabs, shrimp, and mussels, may be consumed by people.

Table B.2-3. Macroinvertebrate species found in the LDW

Common Name	Scientific Name
Anemone, plumose	*Metridium senile*
Anemone	unknown
Ascidian	unknown
Clam, Baltic macoma	*Macoma baltica*
Clam, bent-nosed	*Macoma nasuta*
Clam, eastern soft-shell	*Mya arenaria*
Clam, stained macoma	*Macoma inquinata*
Clam, white sand macoma	*Macoma secta*
Crab, black-clawed	*Lophopanopeus bellus*
Crab, decorator	*Loxorhynchus crispatus*
Crab, Dungeness	*Cancer magister*
Crab, hermit	*Pagurus* sp.
Crab, kelp	*Pugettia producta*
Crab, red rock	*Cancer productus*
Crab, slender	*Cancer gracilis*
Frilled dogwinkle	*Nucella lamellosa*
Mussel, blue	*Mytilus edulis*
Moon snail	*Polinices lewisii*
Nudibranch, striped	*Armina californica*
Sea star, mottled	*Evasterias troschelii*
Sea star, sunflower	*Pycnopodia helianthoides*
Sea star, sand	*Luidia*
Sea star	*Pisaster* sp.
Sea star, sun	*Solaster stimpsoni*
Sea pen	unknown
Shrimp, coonstripe	*Pandalus danae*
Shrimp, crangon	*Crangon* sp.
Tunicate	unknown
Urchin	unknown

Source: Windward (2004a; 2005c; 2006b)

Over 200 composite samples for crab, English sole, starry flounder, perch, clams, and mussels were used in this HHRA (Table B.2-4). Species for which tissue chemistry data are available were used as surrogates for all other species of the same general type, as

described further in Section B.3.4.1.1. For example, tissue chemistry for both English sole and starry flounder were used to represent other benthic fish species.

Table B.2-4. LDW tissue datasets used in the baseline HHRA

Title	Year	Species	Number of Samples	Individuals per Sample	Sample Type	Chemicals
Chemical analyses of fish and crab tissue samples collected in 2005 (Windward 2006b)[a]	2005	English sole	10	5	skin-on fillet	PCB Aroclors
			21	5	whole body[b]	
		Dungeness crab	3	5	edible meat	
			3	5	hepatopancreas[c]	
		slender crab	1	5	edible meat	
			1	10	hepatopancreas[c]	
		shiner surfperch	22	10	whole body	
Chemical analyses of fish and crab tissue samples collected in 2004 (Windward 2005c)[a]	2004	English sole	7	5	skin-on fillet	metals, SVOCs, organochlorine pesticides, PCB Aroclors (PCB congeners in subset of samples), TBT
			21	5	whole body	
		starry flounder	1	5	skin-on fillet	
			3	5	whole body	
		Dungeness crab	7	5	edible meat	
			3	6 – 15	hepatopancreas[c]	
		slender crab	12	5	edible meat	
			4	15 – 18	hepatopancreas[c]	
		shiner surfperch	24	9 – 10	whole body	
		Striped perch	1	12	skin-on fillet	
		pile perch	1	12	skin-on fillet	
Chemical analyses of benthic invertebrate and clam tissue samples and co-located sediment samples (Windward 2005b)	2004	soft-shell clam	14	19 – 52	whole body	metals, SVOCs, organochlorine pesticides, PCB Aroclors (PCB congeners in subset of samples), TBT
Waterway Sediment Operable Unit Harbor Island Superfund Site – Assessing human health risks from the consumption of seafood (ESG 1999)	1998	English sole	3	5	skinless fillet	mercury, TBT, PCB Aroclors
		red rock crab	2	5	edible meat	
		Dungeness crab	1	5	edible meat	
		red rock crab/ Dungeness crab	1	5	edible meat	
		Striped perch	2	5 – 8	skin-on fillet	
King County Combined Sewer Overflow Water Quality Assessment for the Duwamish River and Elliott Bay (King County 1999d) [d]	1996-1997	Dungeness crab	2	3	edible meat	metals, TBT, SVOCs, PCB Aroclors
			1	3	hepatopancreas[c]	
		English sole	3	20	skinless fillet	
		mussels	22	50 – 100	whole body	
Puget Sound Ambient Monitoring Program – annual sampling (West et al. 2001)[e]	1992	English sole	3	5 – 20	skinless fillet	SVOCs, organochlorine pesticides, PCB Aroclors, arsenic, copper, lead, mercury

Title	Year	Species	Number of Samples	Individuals per Sample	Sample Type	Chemicals
	1995	English sole	3	5 – 20	skinless fillet	organochlorine pesticides, PCB Aroclors, arsenic, copper, lead, mercury
Elliott Bay/Duwamish River Fish Tissue Investigation (Battelle 1996; Frontier Geosciences 1996)	1995	English sole	3	6	skinless fillet	PCB Aroclors, mercury, methylmercury, TBT

[a] Pacific staghorn sculpin samples were also collected during this sampling event, but these data were not used in the baseline HHRA because available data suggest that this species is rarely consumed by humans

[b] Whole-body samples include 11 composite samples analyzed as whole bodies and 10 composite samples, the concentrations of which were estimated using results from the analyses of fillet and remainder (i.e., all remaining tissue and fluids after fillets were removed from the specimens used to create English sole fillet composite samples) composite samples. The estimated English sole whole-body concentrations were based on the relative weights and total PCB concentrations in skin-on fillet and remainder tissues collected in 2005.

[c] Data from hepatopancreas composite samples were mathematically combined with data from composite samples of edible meat to form composite samples of edible meat plus hepatopancreas. Whole-body (i.e., edible meat plus hepatopancreas) crab concentrations were calculated assuming 69% (by weight) edible meat and 31% hepatopancreas, based on the relative weight of these tissues in a 16.6-cm Dungeness crab dissected by Windward in 2004 (unpublished data).

[d] Additional samples of cooked crab and English sole were collected during the King County water quality assessment (King County 1999d) but were not used to characterize risks in that assessment and are not used in this HHRA. Approximately 30 additional mussel samples, in addition to the 22 samples listed here, were analyzed as part of the caged mussel deployment designed to assess impacts from the combined sewer overflows. These data are not included in this HHRA because they are not representative of concentrations in mussels that people could collect.

[e] Approximately 140 samples of chinook and coho salmon filets (both composites and individuals) were collected from the LDW from 1992 to 1998. Data from these samples were not included in the HHRA because the chemical concentrations in these adult fish are unrelated to site-specific contamination (see text later in this section for additional explanation).

HHRA – human health risk assessment

PCB – polychlorinated biphenyl

SVOC – semivolatile organic compound

TBT – tributyltin

Some site-specific tissue chemistry data were excluded from the HHRA for one or more of the following reasons (as described below): they are unrelated to sediment contamination in the LDW (e.g., salmon), the species is rarely consumed by people (e.g., Pacific staghorn sculpin), or the lack of representativeness or comparability with other human seafood consumption data. For example, data from Varanasi et al. (1993) were not used in the HHRA because only juvenile chinook salmon were sampled in that study, and juvenile salmonids are not consumed by people. Juvenile chinook salmon data collected by LDWG (Windward 2004b) were also not used for the same reason. Juvenile salmon data from the National Marine Fisheries Service (NMFS) (2002) were also excluded from the HHRA, as were two individual shiner surfperch specimens that were analyzed for PCBs. The latter were excluded because only data from composite samples were included in the HHRA. Although composite samples do not allow for the evaluation of individual fish or shellfish variability in chemical concentrations, they are considered to be more representative of average concentrations. The average concentration is the basis for assessing human contaminant exposure.

The adult salmon that migrate through the Duwamish estuary on their way to upstream spawning areas were exposed to chemicals within the LDW very briefly as juveniles. In addition, adult salmon could be exposed to chemicals transported from the LDW to Puget Sound. The magnitude of such exposure is highly uncertain but is likely to be small relative to other Puget Sound sources. The contribution of these exposures to adult body burdens is likely to be insignificant because the large majority of a salmon's growth occurs in marine waters outside the LDW (O'Neill et al. 1998). For example, a 10-g juvenile chinook salmon with a total PCB concentration of 140 µg/kg wet weight (ww), the mean concentration reported by Varanasi et al. (1993), contains 1.4 µg of PCBs. A 15-kg returning adult chinook salmon captured in the LDW with a total PCB concentration of 56 µg/kg ww, the mean concentration reported by West et al. (2001), contains 840 µg of PCBs, almost all of which are derived from the ingestion of food in Puget Sound and the Pacific Ocean. Based on these data and the analysis presented by O'Neill et al. (1998), less than 1% of the PCB body burden contained in adult salmon migrating through the LDW could have been obtained from prey items consumed in the LDW. Therefore, because this assessment is focused on the evaluation of risks from exposures to chemicals related to the LDW system, adult salmon were not included in the HHRA. Because of the small amount of PCBs in adult salmon that may be related to LDW sediment, the exclusion of salmon from seafood consumption scenarios resulted in some underestimation of human exposures and risk associated with the LDW, as discussed in Section B.6.1.2.2.

Data from several samples of cooked edible portions of seafood were available from the King County water quality assessment (King County 1999d) but were excluded because the highly variable nature of cooking methods and equipment would make comparison to other datasets difficult. Available data suggest that cooking alters the tissue concentrations of PCB congeners (Skea et al. 1979; Zabik et al. 1979; 1982) and mercury (Morgan et al. 1997) on a wet-weight basis. Data from cooked samples are relevant for the evaluation of human health because most people cook seafood before eating it. However, using a combination of data for cooked and uncooked fish does not provide a consistent means for evaluating risks. The site-specific data on cooked seafood and consumption of crab hepatopancreas are discussed in the uncertainty analysis (Section B.6).

For the 1996 Elliott Bay/Duwamish River fish tissue study, both total mercury and methylmercury were analyzed in three English sole composite samples. Methylmercury and total mercury concentrations were within 15% of each other. Because the majority of mercury in fish tissue samples is in the form of methylmercury (EPA 2000d), total mercury concentrations are used in this HHRA as a surrogate for methylmercury concentrations, and the toxicity criterion for methylmercury is used to evaluate all mercury exposures. This represents a health-protective approach because methylmercury is the more toxic form via the oral route. Although the three fish tissue methylmercury data are identified in Attachment 1 to this HHRA, only total mercury

data are used in the exposure and risk calculations for purposes of consistency with the other larger datasets that included only total mercury and not methylmercury.

Data on PCBs, as Aroclors, are available from almost all tissue samples (99%). Pesticides and semivolatile organic compounds (SVOCs) were also analyzed frequently (60 to 70% of the samples). Mercury, arsenic, lead, copper, and tributyltin (TBT) were analyzed in a similar percentage of samples. These chemicals were analyzed in all the tissue samples collected by LDWG in 2004, which comprise the primary dataset for the risk assessment. Chemicals that have not been analyzed in LDW tissue samples but were analyzed in surface sediment samples are discussed in the uncertainty analysis (Section B.6).

Collection locations for LDW tissue samples listed in Table B.2-4 are presented on Map B.2-2. A summary of LDW tissue chemistry data is provided in Attachment 1 (Table 3) of this HHRA.

B.2.2 DATA REDUCTION

Data reduction refers to computational methods used to aggregate data. Data that were selected according to Tables B.2-1 and B.2-4, and the description in Section B.2.1, were used in the determination of exposures on a dry-weight basis for sediment chemistry and on a wet-weight basis for tissue chemistry. All concentrations qualified as estimates (i.e., J-flagged data) were assumed to indicate positive identification of the chemical and were used without modification in subsequent calculations. Some J-flagged data, most notably the 2004 pesticide results, were also N-flagged, indicating a tentative identification of the chemical. In the case of pesticides, the N flag was necessary because of the high potential for interferences from PCBs that were also present in the samples (Windward 2005c). JN-flagged data were still used in the risk assessment, but the uncertainty associated with these results is higher than the uncertainty associated with J-flagged results. Accordingly, the JN-flagged pesticide data are presented separately in the risk characterization (Section B.5) from data for chemicals that were not JN-flagged. Analytical results for pesticides used in this risk assessment from events prior to 2004 were likely to have been complicated by interference from PCBs however, the detected results were not all JN-qualified. Less than 1% of the data were rejected by data validators for quality issues and flagged with an R qualifier. R-flagged data were not used at all in the risk assessment because the data validator determined these results to be unusable.

The most significant use of aggregated data was for the calculation of exposure point concentrations (EPCs), which are intended to represent long-term estimates of exposure in the HHRA. The EPC computation methods are described in detail in the exposure assessment (Section B.3.4.3).

Additional procedures related to averaging, selection of the best data points when multiple data are available, selection of significant figures and rounding procedures,

and calculating totals for chemical groupings (i.e., PCBs, cPAHs, DDTs and dioxins/furans) are described below.

B.2.2.1 Averaging duplicate or replicate samples

Chemical concentrations obtained from the analysis of laboratory duplicates or replicates (two or more analyses on the same sample) were averaged for a closer representation of the "true" concentration compared to the results of a single analysis. Averaging rules were dependent on whether the individual results were detected or undetected chemicals. If all concentrations were detected for a given parameter, the values were averaged arithmetically. If all concentrations were undetected for a given parameter, the minimum reporting limit (RL) was reported. If the concentrations were a mixture of detected and undetected, any two or more detected concentrations were averaged arithmetically and undetected concentrations were excluded. If there was a single detected concentration and one or more undetected concentrations, the detected concentration was reported. The latter two rules were applied regardless of whether the RL was higher or lower than the detected concentration. Note that computation of total PCBs, TEQs for dioxins/furans, and potency equivalents for cPAHs used a different treatment of RLs (see Section B.2.2.4).

Identical averaging rules were applied in situations where multiple sediment samples were collected from the same location at the same time, such as field duplicate samples, or when multiple sediment samples were collected at a single location (i.e., a location with specific x and y coordinates) within a 6-month period. In these instances, a single "average" result for each chemical was generated for that sediment sampling location.

B.2.2.2 Selection of best results

In some instances, the laboratory generates more than one result for a chemical for a given sample. Multiple results can occur for several reasons, including: 1) the original result did not meet the laboratory's internal quality control (QC) guidelines, and a reanalysis was performed; 2) the original result did not meet other project data quality objectives, such as a sufficiently low RL, and a reanalysis was performed; or 3) two different analytical methods were used for that chemical. In each case, a single best result was selected for use. The procedures for selecting the best result differed depending on whether a single or multiple analytical methods were used for that chemical.

For the same analytical method, if the results were:

- Detected and not qualified, then the result from the lowest dilution was selected, unless multiple results from the same dilution were available, in which case, the result with the highest concentration was selected.
- A combination of estimated and unqualified detected results, then the unqualified result was selected. This situation most commonly occurred when

the original result was outside of calibration range, thus requiring a dilution. No results outside the calibration range were used in the HHRA.

- All estimated, then the "best result" was selected using best professional judgment in consideration of the rationale for qualification. For example, a result qualified based on laboratory replicate results outside of QC objectives for precision would be preferred to a qualified result that was outside the calibration range.
- A combination of detected and undetected results, then the detected result was selected. If there was more than one detected result, the applicable rules for multiple results (as discussed above) were followed.
- All undetected results, then the lowest RL was selected.

If the multiple results were from different analytical methods, then the result from the preferred method specified in the quality assurance project plan (QAPP) or based on the consensus of the professional opinions of project chemists was selected.

The following rules were applied to multiple results from different analytical methods:

- For detected concentrations analyzed by the SVOC full-scan and selected ion monitoring (SIM) methods, the highest detected concentration was selected. If the result by one method was detected and the result by the other method was not detected, then the detected result was selected for reporting, regardless of the method. If results were reported as non-detected by both methods, the undetected result with the lowest RL was selected. The SIM method is more analytically sensitive than the full-scan SVOC method, and the undetected results were generally reported at a lower RL by the SIM method than by the full-scan method. Therefore, the SIM method was selected for non-detected results unless an analytical dilution or analytical interferences elevated the SIM RL above the SVOC full-scan RL.
- Hexachlorobenzene, hexachlorobutadiene, and hexachlorocyclopentadiene were analyzed using multiple analytical methods for some samples (EPA Methods 8081A, 8270, and/or 8270-SIM). The result from the method with the greatest sensitivity (i.e., lowest RL) was selected if all results were undetected. EPA Method 8081A results were generally selected, when available, because the standard laboratory RLs from this analysis are significantly lower than those from EPA Methods 8270 and 8270-SIM. When chemicals were detected, the detected result with the highest concentration was selected unless the detected concentration was qualified as estimated or tentatively identified, in which case the rule designating treatment of qualified and unqualified data would apply.
- A subset of the fish and crab tissue samples were analyzed for bis(2-ethylhexyl) phthalate using EPA Method 8270D and for pentachlorophenol using EPA Method 8041, to achieve RLs lower than those achieved in the original analyses

using EPA Method 8270-SIM. For the re-analyses, all sample extracts underwent a silica gel cleanup to separate matrix interferences (i.e., lipids) from the chemicals of interest. After this cleanup step, analytical dilutions were either not needed or were prepared using lower dilution factors than those in the original analyses, resulting in significantly lower RLs. In addition, the lowest points of the initial calibrations used for the re-analyses were lower than the lowest points for the initial calibrations used for the original Method 8270-SIM analyses, which further reduced the RLs achieved for the re-analyses. The re-analysis results for these two analytes were selected for reporting because of the greater sensitivity of the re-analysis methods for these analytes.

B.2.2.3 Significant figures and rounding

Analytical laboratories reported results with various numbers of significant figures depending on QAPP instructions, the instrument, parameter, and the concentration relative to the RL. The reported (or assessed) precision of each observation was explicitly stored in the project database by recording the number of significant figures assigned by the laboratory. Tracking of significant figures becomes important when calculating averages and performing other data summaries.

When a calculation involves addition, such as totaling PCBs or polycyclic aromatic hydrocarbons (PAHs), the calculation can be only as precise as the least precise number that went into the calculation. For example (assuming two significant figures):

> 210 + 19 = 229 would be reported as 230 because 19 is reported only to 2 significant digits, and the enhanced precision of the trailing zero in the number 210 is not significant.

When a calculation involves multiplication or division, such as carbon normalization, the original figures for each value are carried through the calculation (i.e., individual values are not adjusted to a standard number of significant figures, instead the appropriate adjustment is made to the resultant value at the end of the calculation). The result is rounded at the end of the calculation to reflect the value used in the calculation with the fewest significant figures. For example:

> 59.9 × 1.2 = 71.88 would be reported as 72 because there are two significant figures in the number 1.2.

When rounding, if the number following the last significant figure is less than 5, the digit is left unchanged. If the number following the last significant figure is equal to or greater than 5, the digit is increased by 1.

B.2.2.4 Calculating totals

Concentrations for several analyte sums were calculated as follows:

- Total PCBs were calculated using only detected concentrations for seven Aroclor mixtures[8] (1016, 1221, 1232, 1242, 1248, 1254, and 1260) in accordance with Ecology's Sediment Management Standards (WAC 173-204). For individual samples in which none of the seven Aroclor mixtures was detected, total PCBs were given a value equal to the highest RL of the seven Aroclors. An alternate approach for computing total PCBs has been used for other HHRAs in EPA Region 10 and was evaluated in Section B.6.1.1.8.
- Toxic equivalents (TEQs) were used for totaling certain groups of chemicals, specifically dioxin/furan TEQ, PCB TEQs, and carcinogenic PAHs (cPAHs). The 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) toxic equivalency factors (TEFs) for coplanar PCBs and certain polychlorinated dibenzo-*p*-dioxin or furan (dioxin and furan) congeners are presented in Table B.2-5. The TEFs relate the toxicity of the co-planar PCB congeners and certain dioxin and furan congeners to the toxicity of 2,3,7,8-TCDD. Similarly, potency equivalency factors (PEFs) relate the toxicity of certain PAH compounds to that of benzo(a)pyrene. PEFs for cPAHs are also shown in Table B.2-5. PCB TEQ, dioxin/furan TEQ, and cPAH totals were calculated for each sample by summing the products of the concentrations of each individual congener or compound and its specific TEF or PEF for each group (PCB TEQ, dioxin/furan TEQ, and cPAHs, respectively). Congeners or compounds that were undetected for a given sample were assigned a value equal to one-half the sample-specific RL for use in the TEQ calculation.
- Total DDTs were calculated from detected concentrations of three to six isomers: 2,4'-DDD, 2,4'-DDE, 2,4'-DDT, 4,4'-DDD, 4,4'-DDE, and 4,4'-DDT. For samples in which all individual isomers were undetected, the single highest RL for that sample was assigned to represent the sum of the three to six isomers.

Table B.2-5. Toxic equivalency and potency equivalency factors for dioxins/furans, PCB congeners, and cPAHs

Compound	Toxic Equivalency or Potency Equivalency Factor
Dioxins and furans[a]	
2,3,7,8-tetrachlorodibenzo-*p*-dioxin	1
1,2,3,7,8-pentachlorodibenzo-*p*-dioxin	1
1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin	0.1
1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin	0.1
1,2,3,7,8,9-hexachlorodibenzo-*p*-dioxin	0.1

[8] For several sediment samples, Aroclors 1262 and 1268 were also included in the total PCB calculation, but these Aroclors are rarely quantified.

Compound	Toxic Equivalency or Potency Equivalency Factor
1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin	0.01
Octachlorodibenzo-*p*-dioxin	0.0003
2,3,7,8-tetrachlorodibenzofuran	0.1
1,2,3,7,8-pentachlorodibenzofuran	0.03
2,3,4,7,8-pentachlorodibenzofuran	0.3
1,2,3,6,7,8-hexachlorodibenzofuran	0.1
1,2,3,7,8,9-hexachlorodibenzofuran	0.1
1,2,3,4,7,8-hexachlorodibenzofuran	0.1
2,3,4,6,7,8-hexachlorodibenzofuran	0.1
1,2,3,4,6,7,8-heptachlorodibenzofuran	0.01
1,2,3,4,7,8,9-heptachlorodibenzofuran	0.01
Octachlorodibenzofuran	0.0003
PCB congeners[a]	
PCB-77	0.0001
PCB-81	0.0003
PCB-105	0.00003
PCB-114	0.00003
PCB-118	0.00003
PCB-123	0.00003
PCB-126	0.1
PCB-156	0.00003
PCB-157	0.00003
PCB-167	0.00003
PCB-169	0.03
PCB-189	0.00003
cPAHs[b]	
Benzo[a]pyrene	1
Benz[a]anthracene	0.1
Benzo[b]fluoranthene	0.1
Benzo[k]fluoranthene	0.1
Chrysene	0.01
Dibenz[a,h]anthracene[c]	0.4
Indeno[1,2,3-cd]pyrene	0.1

[a] TEFs for dioxin and furans and PCB congeners from the World Health Organization (Van den Berg et al. 2006).

[b] PEFs for cPAHs were defined by the California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (California EPA 1994). PEFs are available for PAHs that were not analyzed in LDW sediments. The PEFs for these compounds are not shown here and are not used in this risk assessment.

[c] The PEF was determined by California EPA by dividing the inhalation unit risk factor for this compound by the inhalation unit risk factor for benzo[a]pyrene.

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

PEF – potency equivalency factor

TEF – toxic equivalency factor

B.2.3 SUITABILITY OF DATA FOR RISK ASSESSMENT

There are several factors to consider in assessing the suitability of environmental data for risk assessments (EPA 1989, 1992b). Of primary importance is the degree to which the data adequately represent site-related contamination and the expected human exposures at the site. Also important to consider are the data quality criteria goals and the source, documentation, analytical methods, RLs, and level of review associated with the data. Because data from many different investigations were available for the LDW, the factors described above were evaluated for each dataset to determine whether it was reasonable to combine all data for use in this HHRA. These suitability determinations were performed in consultation with EPA and Ecology.

B.2.3.1 Representativeness to site-related contamination

B.2.3.1.1 Sediment

Sediment studies within the LDW have been designed for both the reconnaissance (e.g., EPA site inspection and National Oceanic and Atmospheric Administration [NOAA] site characterization) and focused investigations (e.g., early action investigations at Terminal 117 and Slip 4) of areas of concern. In addition, there has been significant and sediment chemistry data collection as part of this RI. The reconnaissance events focused primarily on subtidal sediments, and most of the focused investigations included the collection of both intertidal and subtidal sediments. The extensive coverage of the reconnaissance surveys and the focused intensity of the individual facility investigations indicate that the available sediment chemistry data are representative of the general range of environmental conditions within the LDW. Far more samples have been collected in areas where chemical concentrations were high (i.e., near known sources). Therefore, standard statistical measures (e.g., mean, median) may not be representative of the overall distribution of all chemicals in the LDW because of the sampling bias toward more contaminated areas. However, because a good spatial coverage of samples is available for most chemicals, spatially weighted average concentrations (SWACs) are likely to be fairly representative of overall conditions.[9] A discussion of the distribution of sediment chemistry data and the manner in which they have been used in the HHRA to calculate EPCs is provided in Section B.3.4.3.

B.2.3.1.2 Tissue

Representativeness of tissue data to contamination in LDW sediment was evaluated by reviewing the migratory behavior of the target species and tissue collection locations. Many field events focused on fish and crab collection have occurred in the LDW over the past 30 years. The experience from these events, coupled with information obtained from regional fish biologists and the technical literature on the

[9] Although the interpolation algorithm can still generate a SWAC where large spatial gaps exist, the resulting SWAC is not likely to be very accurate, particularly if steep concentration gradients exist.

target fish and crab species, suggest that all of the target species reside in the LDW most of the time. One exception is for adult English sole and starry flounder, which migrate out of the LDW into deeper waters of Puget Sound, including Elliott Bay, for spawning.

Within the LDW, samples of perch, crab, clam, and English sole were collected from multiple locations. Most of the samples used in this HHRA were collected during the 2004 and 2005 tissue sample collection efforts (Windward 2005c, 2006b). The study design for fish and crab included four tissue sampling areas located from river miles (RMs) 0.2 to 1.0, 1.6 to 2.4, 2.9 to 3.7, and 4.2 to 5.2 (see Map B.2-2). These sampling areas were chosen to represent areas associated with different sediment PCB concentrations found in the LDW, as explained in the QAPP (Windward 2004c). Clams were also collected throughout the LDW from areas where they were expected to be present based on clam habitat quality, as identified by Windward during a reconnaissance survey (Windward 2004a). Eight of the ten clam tissue collection locations were identified as high-abundance areas during that reconnaissance survey. The other two clam tissue collection locations were designated as low-abundance areas but were included in the study design to adequately represent the range of sediment concentrations for chemicals expected to be of concern based on Phase 1 analysis (i.e., PCBs, arsenic, and cPAHs). Given the variety of collection locations and the objective to evaluate the consumption of seafood collected throughout the LDW, the available tissue chemistry data adequately represent site-related exposures with respect to the seafood consumption exposure route.

B.2.3.2 Representativeness to expected human exposure

B.2.3.2.1 Sediment

People may come in contact with LDW sediment through various activities, such as netfishing,[10] clamming, beach play, swimming, wading, beachcombing, dog walking, and habitat restoration. A more detailed description of these activities is given in Section B.3.2. Most of these activities focus on intertidal regions, although netfishing occurs throughout the LDW in both subtidal and intertidal regions. Several large sediment sampling events have collected sediment samples from throughout the entire LDW. Therefore, the overall distribution of sediment samples appears to reflect the expected human exposure from activities such as netfishing, that encompass the entire LDW.

Intertidal sampling density is high in some areas where focused investigations have occurred. Areas with high sampling density do not necessarily correspond with intertidal areas where human exposure is expected to occur (e.g., from clamming or children playing in sediment). For example, many intertidal sediment samples were

[10] Netfishing in this context refers to gill nets deployed from boats by the Muckleshoot Indian Tribe for salmon fishing. Incidental sediment ingestion and dermal exposure to sediment may occur while retrieving nets, as discussed in Section B.3.2.3.

collected as part of the Boeing Plant 2 Resource Conservation and Recovery Act (RCRA) Facility Investigation (RM 2.9 to RM 3.6 on east side of the LDW), but public access to this site is strictly controlled. The Phase 2 study design for the 2005 surface sediment sampling event (Windward 2005h) included the identification and characterization of areas that people might use more often (Windward 2005i). Consequently, sediment chemistry data exist for intertidal areas most likely to be visited by people.

B.2.3.2.2 Tissue

Representativeness of the tissue data for estimating potential human exposure was evaluated by reviewing 1) which species are consumed by humans, 2) the time of sample collection (i.e., does it coincide with a time during which harvest normally occurs?), and 3) the size range of the samples collected and analyzed (i.e., is the size range normally consumed?). An extensive review conducted by Environmental Solutions Group (ESG) (1999) of existing seafood consumption surveys for Puget Sound indicated that all of the species listed in Table B.2-4 are potentially consumed by anglers in the LDW and Elliott Bay. Flatfish and perch may be consumed year-round, although these species are not favored during seasons when adult salmon can be legally harvested (Landolt et al. 1985). In addition, most of the tissue samples analyzed since 1992 (Table B.2-4) were collected during the late summer or early fall. A few of the sampling events conducted before 1997 also occurred in the spring. Therefore, data are available on fish and crab samples collected during most seasons. Clams were collected only in 2004 during the late summer, but these animals can be harvested in other seasons as well.

The Washington State Department of Health (WSDOH) recently updated their fish consumption advisory for the LDW (WSDOH 2005). WSDOH now recommends no consumption of resident fish and shellfish from the LDW. The Washington State Department of Fish and Wildlife (WDFW) is responsible for enforcing fishing regulations, but with respect to the consumption advisory, their role is to remind anglers of the advisory and to point out warning signs that have been installed at multiple locations in the LDW. They have no other enforcement responsibility pertaining to citations or other penalties.

In spite of an existing consumption advisory for the LDW (WSDOH 2005), it is likely that some people consume resident fish and shellfish from the LDW. Regardless of current LDW seafood consumption, the objective of this risk assessment is to examine what the risks to consumers might be, given patterns of seafood consumption that could exist in the absence of chemical contamination. The size of fish and shellfish potentially consumed is more likely related to availability rather than any legal size restriction. The fish and crab specimens included in the composite samples were all adults and are considered to be representative of sizes typically consumed by people fishing and crabbing the LDW. The smallest specimens included in the composite

samples were 20 cm for English sole, 8 cm for shiner surfperch, and 9 cm (carapace width) for Dungeness and slender crabs (Windward 2005c, 2006b).

B.2.3.3 Quality assurance/quality control results

All datasets used in the HHRA have been validated by the original authors of the individual studies or by outside third parties. Summaries of data validations conducted by others for historical sampling events are presented in the technical memoranda: *Summary of Sediment and Tissue Chemistry Datasets to be Used in the Phase 2 RI/FS* (Windward 2005j), *Summary of Sediment and Tissue Chemistry Datasets to be Used in the Phase 2 RI/FS: Addendum 1* (Windward 2005k), and the *Summary of Sediment and Tissue Chemistry Datasets to be Used in the Phase 2 RI/FS: Addendum 2* (Windward 2007b). Data validation reports for samples collected by LDWG for the RI are included in the data reports (Windward 2005a, b, c, d, e, f, 2006a, b, c, 2007a). No additional data validation is planned for this HHRA. Some results were qualified as unusable[11] by the data validators. Data qualified as unusable were not used in this HHRA.

B.2.3.4 Other factors

B.2.3.4.1 Documentation of field and laboratory practices

Documenting field and laboratory procedures makes it possible to assess the impact of any deviation from these procedures on data usability. As described in the technical memorandum describing data quality objectives (Windward 2001), such procedures were documented during the verification process that was conducted during database construction. A thorough review of the documentation provided (e.g., method descriptions, QC results) for the various studies did not reveal any issues that would adversely affect the usability of the data for risk assessment purposes. Data collected by LDWG followed field and laboratory procedures that were approved by EPA and Ecology and that were similar to historical sampling events.

B.2.3.4.2 Analytical data review

The level of analytical data review can also affect data usability. All data used in this risk assessment were subjected to a thorough data reduction and validation process. Other factors that could potentially impact data usability for specific data types are described below.

---

[11] Approximately 1,000 results were qualified as unusable out of more than 150,000 analytical results. Reasons for exclusion reflect quality concerns identified by the analytical laboratory or data validator, such as extremely low matrix or surrogate spike recoveries. Almost all of the rejected results were SVOCs that are often difficult to quantify analytically (1,2,4-trichlorobenzene, 1,2-dichlorobenzene, 2,4-dimethylphenol, 3,3'-dichlorobenzidine, 3-nitroaniline, 4-chloroaniline, 4-nitroaniline, aniline, benzidine, benzoic acid, hexachlorocyclopentadiene, pentachlorophenol, and pyridine). In addition, some sediment results for metals (antimony, cadmium, silver, and selenium) were rejected, as were several results for organochlorine pesticides (delta-BHC, endosulfan sulfate, and endrin aldehyde).

B.2.3.4.3 Analytical methods

Sediment

The sediment surveys from which the baseline surface sediment chemistry dataset was compiled used similar or identical analytical methods for most analytes, with one notable exception. PCB analyses for the NOAA site characterization were conducted using a high-performance liquid chromatography/photodiode array (HPLC/PDA) detector, in contrast to PCB analyses for all the other events, which were conducted using a gas chromatography/electron capture detector (GC/ECD). NOAA laboratory data for total PCBs are based on a nonstandard analytical method and may not be quantitatively comparable to data generated using standard analytical techniques. Specifically, the NOAA laboratory data for total PCBs reflect the difference between the results of one analysis for the sum of PCBs and polychlorinated terphenyls (PCTs) and the results of a separate analysis for PCTs alone.

Krahn et al. (1998) reported the results for 30 samples that were analyzed using both HPLC/PDA and GC/ECD methods by two different laboratories.[12] The two laboratories calculated total PCBs for each sample, which were then compared to each other. Total PCB concentrations between the two laboratories varied by as much as a factor of 6. Regression analyses conducted for the two sets of results indicate that the GC/ECD results were lower than the HPLC/PDA results at high (> 10 mg/kg dry weight [dw]) PCB concentrations and higher than the HPLC/PDA results at low (< 0.1 mg/kg dw) PCB concentrations. The regression coefficient (R^2) between the two sets of analyses was 0.92. The differences between the total PCB concentrations calculated by the two laboratories are not surprising given the differences between the two methods, including: 1) different ranges of linear response for the two detectors, 2) differences in methods for calculating total PCBs, 3) differences in methods of quantifying and/or removing analytical interferences, and 4) differences in RLs.

Despite the differences between the two analytical methods for PCBs, data from both methods were used in this risk assessment, although the uncertainty associated with total PCB concentrations may be significant in some areas. Alternate risk calculations are presented in the uncertainty analysis (Section B.6) using total PCB data derived solely from summing Aroclor concentrations analyzed using GC/ECD.

Tissue

Although different laboratories and in some cases different methods were used for the various tissue analyses, all tissue data summarized in Table B.2-4 met the data quality objectives established for the project. Therefore, combining data from various sources is acceptable for this risk assessment.

---

[12] HPLC/PDA analyses were conducted by the NOAA laboratory in Seattle; GC/ECD analyses were conducted by Analytical Resources, Inc., in Seattle.

RLs for undetected chemicals can affect data usability if they are higher than risk-based concentrations (RBCs).[13] RLs higher than the corresponding RBCs were noted for several undetected chemicals (Section B.3.3.2), many of which were subsequently identified as COPCs for tissue based solely on this observation. The uncertainty associated with the risk characterization for these chemicals is high. Consequently, a quantitative analysis of the risks associated with chemicals that were never detected is presented in the uncertainty analysis (Section B.6).

Carcinogenic PAHs (Table B.2-5) were analyzed in 70% of the historical tissue samples and all the samples collected by LDWG in 2004. Because this chemical group was identified as a COPC in the Phase 1 HHRA based on elevated RLs, LDWG focused on achieving lower RLs in 2004 to reduce uncertainties in cPAH risk analysis. This objective was achieved. The 2004 data are thought to be more representative of actual concentrations as a result of better detection limits. Because these datasets provide adequate data for risk assessment and these data also represent the bulk of the tissue chemistry data, the older cPAH data with elevated RLs were not used in the risk characterization to reduce uncertainty in the analysis. An alternative exposure scenario that included the older data with the 2004 data was quantitatively evaluated in the uncertainty analysis (Section B.6).

Analytical methods for tissues were generally consistent among studies, but some variations were noted. PCBs were quantified using an electron capture detector (i.e., EPA Method 8081) in all studies except ESG (1999). In that study, PCBs were quantified with a low-resolution mass spectrometer. The two types of detectors should give similar results, and there should be little if any impact on data comparability and usability. Consequently, data from both methods are considered usable for risk assessment purposes. All analyses quantified individual Aroclors, which were then summed in an identical manner. Total PCB concentrations derived from Aroclor data are presented as the sum of only detected values. In cases where all Aroclors were undetected, the total PCB concentration was assumed to be equal to the highest RL from among all the individual Aroclors.

PCBs were also analyzed in 49 fish, crab, and clam tissue samples collected by LDWG in 2004 by high-resolution gas chromatography/high-resolution mass spectrometry (HRGC/HRMS). Aroclors and all 209 PCB congeners were analyzed in each sample, although some congeners coelute with each other and must be reported as a group. Total PCBs were calculated as Aroclors and as the sum of congeners. For these samples, concentrations of total PCBs (as Aroclors) and total PCBs (as congeners) were always within a factor of 2 of each other (Windward 2005c, f). Concentrations of total PCBs (as congeners) were lower than concentrations of total PCBs (as Aroclors) in fish and crab samples but higher in clam samples. Although all PCB data were considered suitable for use in risk assessment, data for total PCBs (as Aroclors) were used in the

[13] RBCs are concentrations associated with specific risk levels that are used for screening purposes.

calculation of health risks in this HHRA, because the tissue concentrations were higher in fish and crab than for total PCBs (as congeners). This data use is considered a health-protective approach and is evaluated in Table B.6-8 of the uncertainty section.

B.3 Exposure Assessment

This exposure assessment describes scenarios in which people may come in contact with sediment-associated COPCs and provides equations and parameters so that potential exposures can be quantified. Section B.3.1 summarizes previous exposure assessments conducted for the LDW. Section B.3.2 presents the conceptual site model that introduces the exposure scenarios that were evaluated in this HHRA. Section B.3.3 describes a risk-based screening procedure to identify which chemicals were evaluated in detail in the HHRA. Section B.3.4 describes how the exposure scenarios were quantified, including equations used for calculations, and Section B.3.5 presents chronic daily intake (CDI) estimates for all chemicals evaluated.

B.3.1 Summary of Previous LDW Exposure Assessments

Several prior risk assessments have addressed the LDW or portions of the LDW. Exposure scenarios evaluated previously for the LDW, including Harbor Island, are summarized in Table B.3-1. The most recent HHRA was conducted by LDWG as part of the Phase 1 RI for this site (Windward 2003b). Three exposure scenarios, consumption of fish/shellfish, water recreation (which includes swimming), and exposure to sediment by commercial fishers, were evaluated in more than one risk assessment (Table B.3-1). The risk assessment conducted by King County (1999b) evaluated two pathways, scuba diving and windsurfing, that were based on exposures to water. King County (1999b) also evaluated a swimming scenario, which was based on exposure to water and sediment. The Phase 1 HHRA (Windward 2003b) also included a beach play scenario, which evaluated direct contact with intertidal sediment by children.

Table B.3-1. Exposure scenarios evaluated in previous risk assessments

Site/Project	Activity	Route/Exposure Medium	Group	Key Exposure Variables	Source
LDWG Phase 1 human health risk assessment	non-commercial fishing	consumption of fish/shellfish	adults, children	seafood consumption rate = 84 g/day (adult tribal)	Windward (2003b)
	commercial netfishing	incidental ingestion of sediment dermal contact with sediment	adults	exposure frequency = 119 days/yr	
	water recreation[a]	incidental ingestion of water dermal contact with water	adults, children	exposure frequency = 24 days/yr	
	beach play	incidental ingestion of sediment dermal contact with sediment	children	exposure frequency = 41 days/yr	
	industrial work on the LDW	incidental ingestion of sediment dermal contact with sediment	adults	not quantitatively evaluated	
Waterway Sediment Operable Unit Harbor Island Superfund Site	non-commercial fishing	consumption of fish/shellfish	adults	seafood consumption rate = 77 g/day (adult tribal)	Environmental Solutions Group (1999)
LDW and Elliott Bay water quality assessment	swimming	incidental ingestion of water dermal contact with water incidental ingestion of sediment dermal contact with sediment	adults, children	exposure frequency = 2, 12, 24 days/yr (low, medium, high)	King County (1999b)
	scuba diving	incidental ingestion of water dermal contact with water	adults	exposure frequency = 2, 12, 24 days/yr (low, medium, high)	
	windsurfing	incidental ingestion of water dermal contact with water	adults	exposure frequency = 2, 12, 24 days/yr (low, medium, high)	
	commercial fishing	incidental ingestion of water dermal contact with water incidental ingestion of sediment dermal contact with sediment	adults	exposure frequency = 2, 24, 91 days/yr (low, medium, high)	
	non-commercial fishing	consumption of fish and shellfish	adults, children	seafood consumption rate = 93, 152, 305 g/day (low, medium, high)	

Site/Project	Activity	Route/Exposure Medium	Group	Key Exposure Variables	Source
Boeing Plant 2 RCRA facility investigation (LDW)	commercial fishing	incidental ingestion of sediment dermal contact with sediment	adults	exposure frequency = 215 days/yr	Weston (1998b)
	recreational fishing	consumption of fish and shellfish	adults	not quantitatively evaluated	
Harbor Island RI	commercial fishing	incidental ingestion of sediment dermal contact with sediment	adults	exposure frequency = 215 days/yr	Weston (1993)

[a] Based on results presented by King County (1999b).

LDWG – Lower Duwamish Waterway Group

RCRA – Resource Conservation and Recovery Act

RI – remedial investigation

B.3.2 CONCEPTUAL SITE MODEL

A conceptual site model is a graphical representation of chemical sources, transport mechanisms, exposure pathways, exposure routes, and potentially exposed populations. It provides the basis for developing exposure scenarios to be evaluated in the exposure assessment component of the HHRA.

The human health conceptual site model is presented in Figure B.3-1. For the purposes of this HHRA, sediments are the assumed source of chemicals for all exposures at the site, regardless of actual exposure medium (e.g., tissue, sediment). The risks from direct exposure to surface water were previously evaluated quantitatively by King County (1999b) and found to be lower than risks associated with the sediment or fish consumption pathways. The surface water risk estimates from that HHRA have been incorporated in this HHRA. Although chemical sources other than sediment exist in the LDW, the exposure assessment focuses only on scenarios that include a direct (i.e., ingestion or dermal contact) or indirect (i.e., consumption of fish or shellfish) pathway to chemicals in sediments. Sources of chemical contamination of the sediments are discussed further in the main body of the RI report.

Five exposure scenarios are represented in Figure B.3-1, corresponding to potentially exposed populations described below. Each exposure scenario (e.g., beach play reasonable maximum exposure [RME]) involves at least one potential exposure pathway to contaminated sediments (e.g., dermal contact with sediments, incidental ingestion of sediments) and a potential exposure route through which contaminants can enter the body of an exposed individual (e.g., dermal absorption of contaminants through exposed skin surfaces, gastrointestinal absorption of ingested contaminants), although the importance of some pathway/route combinations is minor or incomplete for some scenarios. For example, ingestion of drinking water was considered to be an incomplete pathway for all scenarios considered. The scenarios presented are not mutually exclusive. Several of the scenarios are evaluated cumulatively in the risk characterization, as described in Section B.3.2.6 (e.g., exposure pathways associated with swimming, beach play, and seafood consumption). For simplicity, the volatilization pathway through air is not shown in Figure B.3-1. Existing sediment chemistry data for volatile organic compounds (VOCs) indicate that these chemicals are rarely detected in LDW sediments. Other organic chemicals, such as PCBs, are not expected to volatilize significantly from sediment. HHRAs conducted on the Hudson River in New York, where PCB concentrations are much higher than they are in the LDW, concluded that the calculated cancer risk from the inhalation of volatilized PCBs was insignificant (TAMS and Gradient 2000).

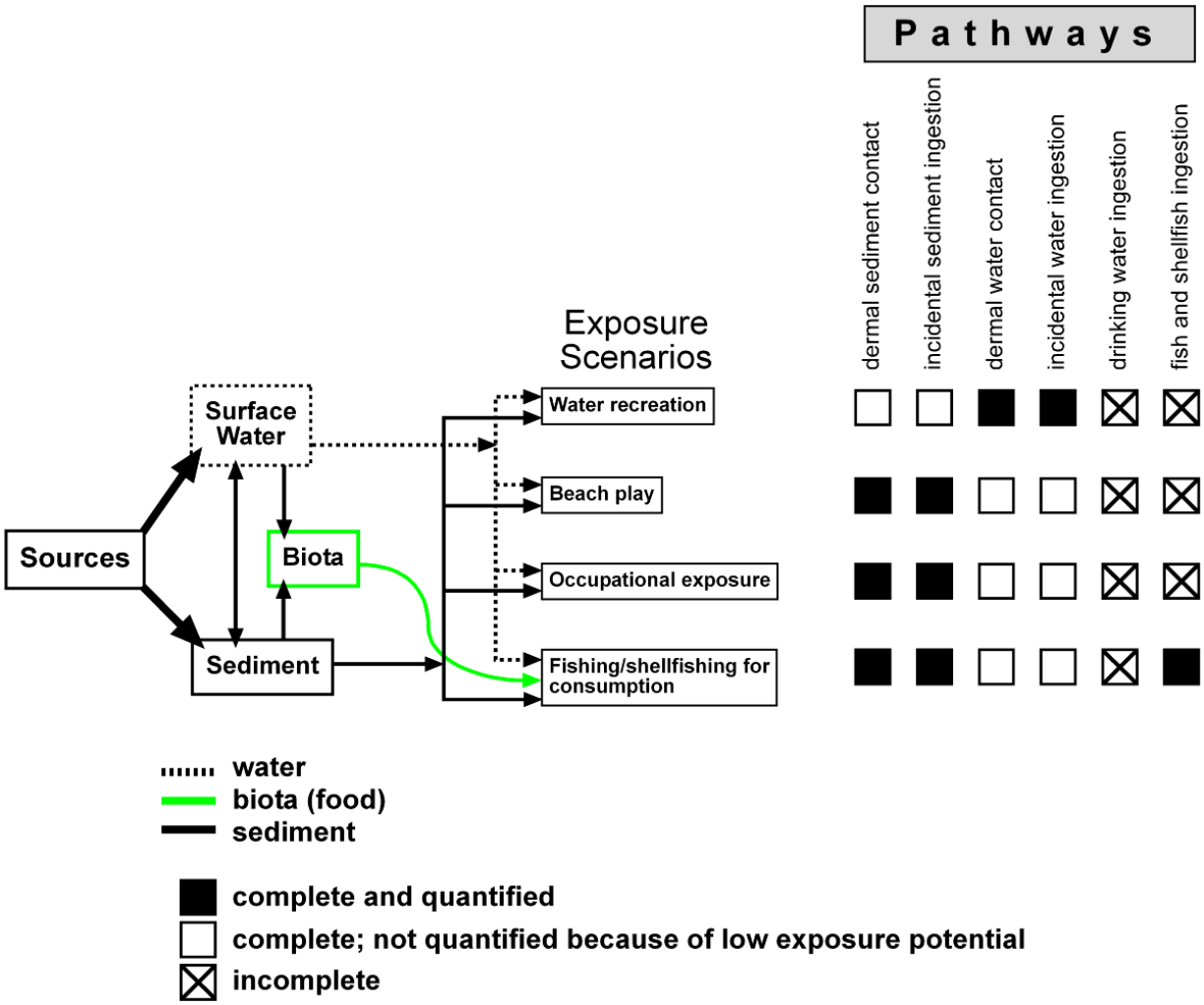


Figure B.3-1. Conceptual site model for baseline human health risk assessment

Several levels of exposure scenarios are used in the risk assessment to describe different intensities (e.g., frequency and duration) of site use or seafood consumption. These scenarios include RME scenarios, upper-bound exposure scenarios, central tendency (CT) exposure scenarios, and a one-meal-per-month seafood consumption scenario. RME is the highest exposure that is reasonably expected to occur at a site. EPA generally uses RME scenarios to evaluate remedial actions at a site (EPA 1989). RME by definition likely overestimates exposure for many individuals. With regard to the adult tribal seafood consumption scenarios, application of EPA's tribal seafood consumption framework has resulted in the use of Tulalip seafood consumption survey data to characterize adult tribal RME seafood consumption. An additional tribal scenario is also provided here based on Suquamish seafood consumption survey data. This scenario represents an upper bound on risk for the LDW site (EPA 2005a). In characterizing uncertainty in exposure and risks, it is useful to examine CT exposures (National Research Council 1994). CT risk estimates are intended to reflect

average exposures. Average exposure estimates are not favored in decision-making because they will underestimate exposure for a substantial number of individuals (EPA 1989). Another method for examining exposure is to identify a unit of exposure that a member of the public can use to assess risks associated with their individual behavior. This last approach was used to characterize exposure for seafood consumers on a one-meal-per-month basis. The one-meal-per-month scenario was included, not to represent actual exposure rates but to provide the public with an estimate that could be readily scaled to individual behaviors. The one-meal-per-month scenarios are not intended to represent a measured or established consumption rate for the LDW.

Each scenario shown in Figure B.3-1 is discussed qualitatively below. For some pathways, both RME scenarios and CT scenarios were developed to describe some of the range of possible exposures and risks. The exposure parameters for each scenario are discussed in detail in Section B.3.4, and summarized in Tables B.3-5 and B.3-6 in that section.

B.3.2.1 Water recreation

Water recreation could include kayaking/canoeing, swimming, scuba diving, and windsurfing. The primary exposure medium for these activities is water, although individuals may also come in contact with contaminated sediments that have been resuspended in the water column or as they enter the water from the shore. King County, in their issue paper on human site use in the Duwamish River and Elliott Bay (King County 1999c), concluded that the frequency of these recreational activities would be low in the LDW as compared to Elliott Bay, because of the industrial nature of the waterway and limited access to the water from the shoreline. Future remedial and restoration actions could increase the frequency of these recreational activities, particularly kayaking and canoeing.

Although there is the potential for mixed use of the river in the future for recreational activities (as well as industrial and tribal activities), a specific water recreation scenario was not developed for two reasons. First, risks associated with water contact were previously deemed to be very low in the King County risk assessment (King County 1999b). Second, the magnitude of sediment contact likely to occur during the launching and retrieval of small boats would be less than exposures that are covered under other risk scenarios included in this risk assessment; therefore, any risks related to specific water recreation scenarios can be considered through review of the other direct contact scenarios (i.e., beach play, netfishing, and clamming). The King County risk assessment (King County 1999b) estimated health risks associated with swimming in the LDW and concluded that the risks were generally within the range of risks considered to be acceptable by EPA. Swimming risks were associated with chemicals in the water. Excess cancer risks were highest for arsenic and PCBs, ranging from a low of 1×10^{-10} for older children exposed to PCBs (Table 9, Appendix B.1 of Phase 1 HHRA) to 4×10^{-6} for young children exposed to arsenic. All hazard indices for non-cancer risks were less than 1. The risks associated with the water component of the

swimming scenario were small (25% or less) compared to the risks associated with the sediment component. Additional PCB water data recently collected by King County (Mickelson and Williston 2006) suggest that the modeled PCB water concentrations used in the 1999 assessment likely overestimate the true concentrations. Exposures of children to LDW sediment in this HHRA are quantified under the beach play RME scenario described below. The King County risk estimates for the swimming scenario are incorporated in the cumulative risk characterization, as described in Section B.3.2.6.

B.3.2.2 Beach play

Beach play RME scenarios were developed to assess the risk to young children (i.e., up to 6 years of age) playing in intertidal sediments of LDW beaches with public access from shore, both now and in the future. Although these exposure scenarios focus on young children, they also serve to represent health-protective estimates of the risk to older children and adults who live in homes bordering the LDW, volunteers and public-sector employees responsible for habitat restoration within intertidal areas, and individuals who may access intertidal areas by passing through private property boundaries. Because young children have a higher incidental soil ingestion rate (IR) and a lower body weight than adults, exposure estimates for children are higher than those for adults who might visit at the same frequency. Thus, because of the higher exposures for children, the beach play RME scenarios are health-protective for the evaluation of other potential visitors to the LDW.

The exposure areas for beach play were chosen based on the information gathered during the human use survey of the LDW as presented in *Technical Memorandum: Results from Survey of Potential Human Access Locations on the Lower Duwamish Waterway* (Windward 2005i). The survey identified the portions of the LDW shoreline that are accessible to the public.

An exposure frequency of 65 days per year was selected from a King County survey of lake beaches (Lake Union, Lake Washington, and Lake Sammamish) (Parametrix 2003). This exposure frequency represents the 95th percentile for children from birth to 6 years of age who engage in playing and digging in sand adjacent to the water. This behavior is consistent with the behavior that is assumed for the beach play RME scenario in the LDW.

Many of the beaches in other areas that were surveyed by King County are located adjacent to, or within a few blocks of, residential areas, similar to the proximities in some LDW areas, particularly the South Park neighborhood. This proximity of residences to the LDW makes it possible for certain individuals to visit the shoreline area of the LDW on a daily basis. The Duwamish River Cleanup Coalition has reported daily park visits in South Park occur (EPA and Ecology 2007). However, the beach play RME scenario is designed to address a specific type of behavior, which includes not just walking along the beach but also contact with moist sediment in

intertidal areas over a significant portion of the body. The study conducted by King County (Parametrix 2003) identified this type of exposure and quantified the high end (95th percentile) of this exposure frequency as being 65 days per year for children from 0 to 6 years old. The study also showed daily visits to beaches for other activities associated with exposure to areas within parks above the water line (i.e., walking, running, hiking, sunbathing, sitting, reading, biking, nature observation) for some adults and children. The lakeside beaches included in the King County survey are not associated with chemical contamination; therefore, the frequency of use should not be suppressed because of contamination concerns by the public. No similar survey has been conducted for the LDW. Any results from such a survey of the LDW could reflect use suppression from LDW chemical contamination. Therefore, the King County survey provides the most unbiased data source from the region for quantifying the exposure frequency for this scenario.

The beach play RME scenario is aimed at assessing exposure to young children playing in the mud (sediment) along the intertidal areas of the LDW and receiving significant exposure to intertidal sediments. Another scenario (dog walking) was created to assess risks for individuals who visit the LDW more frequently but receive a lower exposure to intertidal sediments because of the nature of the activity (Section B.6).

To better assess risk from exposure to particular sections of the LDW, the LDW was divided into eight exposure areas. The exposure areas for the beach play RME scenarios, along with the rationale for the exclusion of certain beaches, are shown on Map B.3-1. The exposure areas were delineated based on potential break points between access points to the river, as can be seen on Map B.3-1.

The primary exposure pathways for the beach play RME scenario are dermal contact and incidental ingestion of intertidal sediment. While direct contact with surface water may occur, the frequency and magnitude of this contact during beach play is likely to be very low when compared to the magnitude and frequency of contact with the intertidal sediment that may occur during beach play activities. In addition, direct exposure to surface water was quantitatively evaluated under the swimming scenario in the King County (1999c) risk assessment, as indicated above for the water recreation scenario. Therefore, exposure to water was not included in the beach play RME risk scenarios. However, exposure to water and sediment through swimming [based on King County (1999c) estimates] in combination with beach play RME is presented in the risk characterization section (see Section B.5.6).

The exposure frequency selected for the beach play RME scenario is based on King County beaches with more amenities than LDW intertidal areas. Consequently, this exposure frequency may overestimate the current exposure frequency for the LDW. However, although the future use of the LDW is expected to remain industrial in nature, increased public access and habitat restoration activities may not only expand the areas where people currently access the river, but also increase the frequency with

which they do so. Consequently, the potential overestimation of current risks in the beach play RME scenario is intended to be protective of future uses as well.

B.3.2.3 Occupational exposure

The LDW supports a large number of water-dependent commercial uses. Many of the facilities adjacent to the LDW rely on vessel traffic on the waterway. Workers on these vessels could potentially come in contact with sediment and surface water, but most workers are typically aboard vessels and well above the water surface. Consequently, the contact frequency is expected to be low relative to other direct contact scenarios quantified in this HHRA.

Workers involved in commercial netfishing in the LDW may come in contact with sediment and surface water. Individuals from the Muckleshoot Indian Tribe participate annually in a commercial gillnetting operation in the LDW. The gillnet lead lines typically come in contact with sediments during normal operations. The netfishers may contact this sediment incidentally upon net retrieval and may then also have incidental contact with surface water and sediment suspended in surface water. The exposure area for netfishing was assumed to cover the entire study area of the LDW. Data from sediment samples taken throughout the waterway, including intertidal and subtidal areas, were included (see Map B.2-1). Two commercial netfishing scenarios are evaluated for adult exposures: a CT scenario that assumes typical frequency and duration of netfishing activity, as recommended by EPA, and an RME scenario that assumes more frequent and longer term netfishing. Other occupational scenarios involving sediment exposure including a biologist conducting restoration work and a King County special operations crew are described and evaluated in the uncertainty analysis (Section B.6.1.9).

B.3.2.4 Clamming

Two surveys conducted in 2004, one for clam abundance (based on quality of habitat and clam abundance evaluations) and the other for human access locations (Windward 2004a, 2005i), indicated that there is a potential for clamming to occur throughout the LDW. Because of different human use patterns observed along the LDW, three separate clamming scenarios were developed.

A 7-day-per-year clamming scenario was generated to estimate the risk to individuals who are assumed to use only public access points to reach the shoreline. The exposure area for this scenario included all intertidal areas of the LDW that have direct upland access or that can be reached by walking along the shore from an upland access point at low tide. Upland access points were identified based on the human access survey (Windward 2005i), which characterized the portions of the LDW shoreline that are accessible to the public. The exposure areas for this scenario are shaded green on Map B.3-2, which also provides additional information regarding public access along the river.

A 120-day-per-year tribal clamming RME scenario was developed to address greater access available to individuals collecting clams by shore and/or by boat, particularly tribal members. Tribal members have access via treaty rights to harvest clams and other shellfish on both public and private property along the entire shoreline of the LDW. The assumed exposure area for this tribal clamming RME scenario included nearly all of the intertidal areas within the LDW and is illustrated by both the blue and green shaded areas on Map B.3-2. In addition, a tribal clamming 183-day-per-year scenario was developed at the request of the Suquamish and Muckleshoot Tribes to represent a high-end clamming frequency. This scenario uses the same exposure area as the tribal clamming RME scenario.

The exposure area for both tribal clamming scenarios is equivalent to the potential clam habitat areas identified during the intertidal clam survey conducted in 2004 (Windward 2004a). The clam populations were not quantified at all the potential clam habitat areas. Clam populations may be limited in the upstream-most extent of the LDW because of habitat constraints such as salinity. During certain times of the year, the uppermost reaches of the LDW are entirely freshwater (King County 1999a). Consequently, the area over which tribal clamming is estimated to occur may be overestimated.

The clamming scenarios include exposure to contaminants via dermal contact with sediment and incidental ingestion of sediment. While direct contact with surface water may occur, the frequency and magnitude of this contact during clamming is likely to be very low compared to the magnitude and frequency of contact with the intertidal sediment that occurs during clamming activities. Therefore, exposure to water is not included in the clamming scenarios. Exposure from the ingestion of clams harvested from the LDW is included in the seafood consumption pathway (Section B.3.2.5). In addition, risk estimates associated with a combination of activities, including clamming, swimming (as estimated by King County [1999c]), and clam consumption, are presented in the risk characterization section (Section B.5.6).

B.3.2.5 Fishing and shellfishing for consumption

Harvesting of fish, crabs, and clams can occur throughout the LDW. Seafood consumed by people fishing in the LDW may be contaminated following exposures to chemicals in sediments and surface water in the LDW. Fishers may also come in direct contact with surface water and sediment. Contact with these media is likely only incidental for fishers, but sediment contact would be common for individuals harvesting clams. As described in Section B.3.2.4, incidental ingestion and dermal contact with sediment is included in the clamming scenario to address this exposure. Several seafood consumption scenarios are evaluated, including an adult tribal scenario based on Suquamish data, adult and child tribal scenarios based on Tulalip data, adult Asian and Pacific Islander (API) scenarios, and one-meal-per-month scenarios (see Section B.3.4.1 for a discussion of consumption rates applied to evaluate these scenarios).

EPA's application of the tribal framework for conducting seafood consumption risk assessments indicates that the tribal scenarios based on Tulalip data are the most appropriate for the LDW (EPA 2005a) and were therefore designated as the tribal RME scenario. The tribal scenario based on Suquamish data will be used to characterize the upper end of the risk range and to characterize the range of uncertainties in risk estimates. Additional information on the specific seafood consumption surveys is provided in Section B.3.4.1. The scenarios for seafood consumption were developed to be protective of future use inasmuch as ingestion rates were derived from studies of tribal seafood consumption in populations that have access to resources that are less polluted than the LDW and can be harvested from larger areas than the LDW.

Two seafood consumption scenarios each are evaluated for the adult tribal (Tulalip data), child tribal (Tulalip data), and API populations: a CT scenario that is intended to represent average exposures from seafood consumption and an RME scenario that assumes more frequent and longer-term seafood consumption. The cumulative risks associated with consuming clams, coming into contact with the sediment during clam harvesting, and swimming (as estimated by King County [1999c]) are addressed in Section B.5.6.

B.3.2.6 Selection of exposure scenarios for quantification

Specific exposure assumptions were developed to quantify exposure pathways for the scenarios shown in Figure B.3-1. A complete exposure pathway includes an exposure medium and exposure point; a potentially exposed population, including receptor age (i.e., adult vs. child); and an exposure route. The exposure parameters and the likelihood of exposure under both current and future land use at the site are discussed in Section B.3.4 for all exposure pathways quantified. Section B.3.4 also presents details on the multiple versions of each scenario that are evaluated.

EPA guidance (1989) states that "actions at Superfund sites should be based on an estimate of the RME expected to occur under both current and future land-use conditions." As discussed previously, EPA defines the RME as "the highest exposure that is reasonably expected to occur at a site." The scenarios developed in this HHRA are consistent with RME guidelines.

The exposure scenarios evaluated in this HHRA represent both current and future conditions, depending on the scenario. Separate scenarios for current and future land use were not evaluated for the following reasons:

- Future land use within the LDW is not expected to differ greatly from current land use. The use of the waterway for commercial and industrial purposes is expected to continue into the foreseeable future, although certain recreational and tribal activities that are consistent with these land uses may be more common in the future.

- Because site-specific parameters based on current land-use practices are unavailable for many exposure parameters (see Section B.3.4), reasonable maximum values were selected. These values likely overestimate current exposure but are derived here to provide additional information to risk managers in evaluating potential future increases in site use represented by the selected exposure scenarios (Table B.3-2).

Table B.3-2. Rationale for the selection or exclusion of exposure pathways

Exposure Scenario	Exposure Point	Exposure Medium	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Water recreation	Water recreation areas in LDW	sediment	resident	adult	dermal, ingestion[a]	qualitative	Exposure via swimming less than exposure via beach play.
				child	dermal, ingestion[a]	qualitative	Exposure via swimming less than exposure via beach play.
		surface water	resident	adult	dermal, ingestion[a]	numeric	Most likely direct contact pathway for surface water.
				child	dermal, ingestion[a]	numeric	Most likely direct contact pathway for surface water.
Beach play in intertidal area[b]	LDW beaches	sediment	resident	adult	dermal, ingestion[a]	qualitative	Adult's exposure during beach play likely to be less than child's exposure on a per kilogram body weight basis.
				child	dermal, ingestion[a]	numeric	Residents may play at the shoreline near or adjacent to their houses.
		surface water	resident	adult	dermal, ingestion[a]	qualitative	Exposure attributable to resuspended sediment in water column is insignificant compared to that from direct contact with bedded sediment.
				child	dermal, ingestion[a]	qualitative	Exposure attributable to resuspended sediment in water column is insignificant compared to that from bedded sediment.
Human consumption of resident seafood	Fishing/shellfishing locations in the LDW	resident fish and shellfish tissue	resident , visitor, worker	adult, child	ingestion	numeric	Although available data suggest current seafood consumption from LDW is low, tribal members have treaty harvest rights and the public also has recreational expectations for a fishable and swimmable estuary.
				adult, child	dermal	qualitative	Exposure via dermal pathway is insignificant.

Exposure Scenario	Exposure Point	Exposure Medium	Receptor Population	Receptor Age	Exposure Route	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Fishing/ shellfishing in intertidal areas	fishing locations in the LDW	sediment	resident, visitor, worker	adult	dermal, ingestion[a]	numeric	Recreational clamming may occur, given the abundance of clams in some areas. Incidental exposure during fishing is insignificant.
				child	dermal, ingestion[a]	qualitative	Incidental exposure during fishing likely to be less than that assumed in beach play scenario; potential exposure during clamming likely to be much lower compared to adult exposures.
		surface water	resident, visitor, worker	adult	dermal, ingestion[a]	qualitative	Incidental exposure is insignificant.
				child	dermal, ingestion[a]	qualitative	Incidental exposure is insignificant.
Occupational exposure (netfishing)	commercial netfishing locations in LDW, which potentially include all LDW sediments	sediment	worker	adult	dermal, ingestion[a]	numeric	Commercial fishers are active at the site throughout the fishing season; nets contact the sediment.
		surface water		adult	dermal, ingestion[a]	qualitative	Exposure attributable to resuspended sediment in water column is insignificant compared to that from bedded sediment.
Other occupational exposure[c]	industrial facilities adjacent to LDW	sediment	worker	adult	dermal, ingestion[a]	qualitative	Exposure expected to be much less than that evaluated in other sediment exposure scenarios.
		surface water	worker	adult	dermal, ingestion[a]	qualitative	Exposure expected to be much less than that evaluated in other scenarios.

[a] Incidental sediment ingestion associated with dermal contact.

[b] Although the beach play scenario is expected to be protective of adults who may participate in beach play activities, they may receive exposure through other activitiess, such as dog walking. Thus, a dog-walking scenario is evaluated in the uncertainty analysis (Section B.6.1.9).

[c] Alternate occupational exposure scenarios are evaluated in the uncertainty section (B.6.1.9), including exposure scenarios for a habitat biologist, Washington Conservation Corps and citizen volunteers, and King County special operations staff.

LDW – Lower Duwamish Waterway

Risk assessment guidance (EPA 2004d; National Research Council 1994) also describes a CT scenario, which is intended to represent average exposures, as compared to the RME. CT exposure estimates may provide the public with important information, enabling people to evaluate their own risks from exposure to the site. Several CT scenarios were included in this HHRA: one for seafood consumption for an adult tribal population based on adult Tulalip data, one for seafood consumption for a child tribal population based on Tulalip data, one for seafood consumption for a population of API, and one for netfishing. The CT scenarios for adult and child tribal populations based on Tulalip data and the adult API were based on the 50th percentile of seafood consumption rates from applicable surveys, as discussed in Section B.3.4.1. For other scenarios (e.g., clamming), the data necessary to determine the range of likely exposures are not available, and thus no CT exposure estimates were made. A CT scenario could be developed for the beach play scenario; but because eight different exposure areas were evaluated, the resulting risk estimates were considered to provide a reasonable range for evaluating this scenario.

Summing risks from multiple exposure pathways is reasonable if multiple pathways are relevant to the same person or group of people. EPA (1989) suggests that summing risks from multiple RME scenarios that do not occur simultaneously could be overly conservative. Three sets of summed scenarios are created in the risk characterization (see Section B.5.6): adult Tulalip (netfishing RME, swimming, and adult Tulalip RME seafood consumption), child (beach play RME, swimming, and child Tulalip RME seafood consumption), and adult low-end clamming (clamming – 7 days per year, swimming, clam consumption – one meal per month). Although CT scenarios for netfishing and seafood consumption are available, the netfishing RME scenario was summed with a seafood consumption RME scenario when evaluating risks across different exposure pathways because these activities are not mutually exclusive and both could be practiced by some individuals. No CT scenarios were included when risks were summed for any other multiple-pathway combinations in Section B.5.6 because other summed scenarios did not include more that one RME scenario.

Table B.3-2 documents the decision process for selecting exposure pathways for quantification. Risk estimates were not quantified for occupational exposure scenarios other than netfishing because exposures are likely to be lower than for the other scenarios. Additional discussion and analysis of the health protectiveness of the sediment exposure scenarios is provided in Section B.6.1.9.

B.3.3 CHEMICAL SCREENING AND EVALUATION

A comprehensive set of chemicals has been analyzed in both sediment and tissue collected from the LDW. In accordance with EPA (1996a) guidelines, risk-based screening was conducted to determine which chemicals should be quantitatively evaluated in the baseline HHRA. Screening helps to focus the HHRA on the parameters that may pose a risk.

The decision process for identifying COPCs is shown in Figure B.3-2. The screening process and results are presented in Attachment 1. For detected chemicals with RBCs, the maximum detected concentration was compared to the applicable RBC (Step 3a). RLs were also evaluated relative to the RBCs for chemicals that had maximum detected concentrations that did not exceed the RBCs, as shown in Figure B.3-2 (Steps 4a and 4b). If a chemical was detected in greater than 10% of the samples, and those detected values never exceeded the RBC, the chemical was excluded from further analysis. For those chemicals with a detection frequency less than 10%, the number of times the RL exceeded the RBC was determined (the right side of Figure B.3-2; Step 4b). If RLs exceeded the RBC with a frequency greater than 10% (Step 4b), that was considered sufficient uncertainty that the RBC could have been exceeded, and the chemical was retained as a COPC. Risks related to COPCs identified based on RLs greater than RBCs alone are considered in the uncertainty analysis (Section B.6). Chemicals without RBCs could not be screened or quantitatively evaluated, but were considered in the uncertainty analysis.

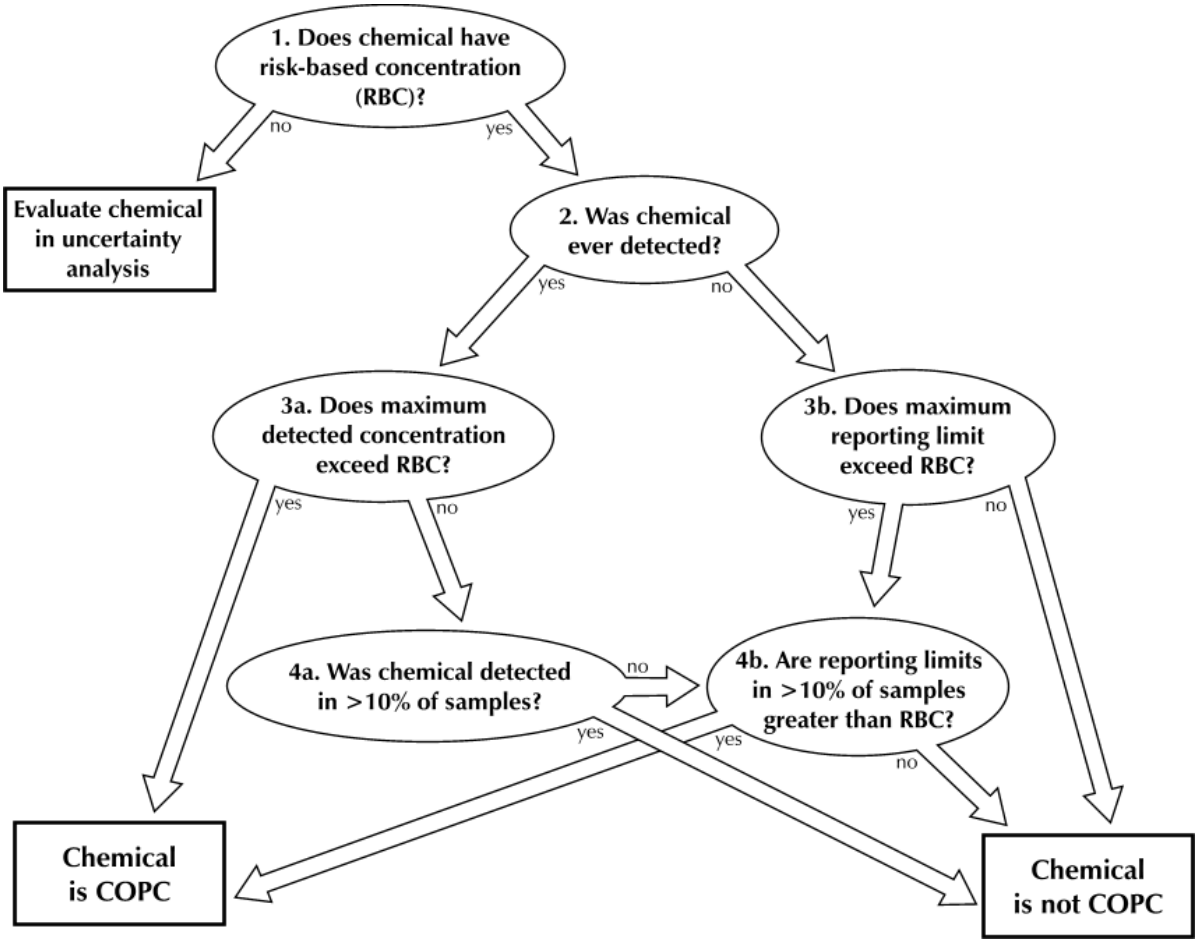


Figure B.3-2. COPC identification flowchart

Some chemicals (e.g., cPAHs and polychlorinated dibenzodioxins and dibenzofurans) were evaluated as groups, rather than individual compounds, using the TEQ approaches described in Section B.2.2.4 and Table B.2-5.

Screening was conducted separately for intertidal sediment exposure (i.e., beach play scenario and clamming scenario), intertidal and subtidal sediment exposure (i.e., netfishing scenarios), and seafood consumption (i.e., seafood consumption scenario). Specific analytical steps for evaluating background concentrations are described below in the media-specific sections. Tables describing the occurrence and selection of COPCs are provided in Attachment 1.

B.3.3.1 Sediment

EPA has not developed RBCs specifically for sediment, but soil RBCs are generally applicable for scenarios that include incidental ingestion and dermal contact with sediment. The Washington State Model Toxics Control Act (MTCA) contains residential soil RBCs, but they are higher (i.e., less protective) than those derived by EPA Region 9 because of different exposure parameter assumptions. Consequently, EPA RBCs were used instead of MTCA RBCs. Thus, RBCs[14] developed by EPA Region 9 (2004a) were selected for this HHRA. They are widely used for screening at many locations throughout the country.

EPA Region 9 (2004a) provides soil RBCs intended to be protective of human health risk in screening both industrial and residential scenarios. The equations used to calculate the RBCs incorporate exposure via ingestion, skin contact, and inhalation simultaneously. Region 9 RBCs for chemicals with non-carcinogenic effects were decreased by a factor of 10 to account for the target hazard quotients (HQs) of 0.1 used in screening by EPA Region 10. Both residential and industrial RBCs were used in the screening. Residential RBCs were applied to the beach play and clamming scenarios; industrial RBCs were applied to the netfishing scenario.

Tables in Attachment 1 compare the maximum sediment concentrations for each chemical with the applicable RBC[15] and include summary statistics, such as detection frequency, minimum detected concentration, and range of RLs. For the netfishing scenario, data for subtidal and intertidal sediments were combined in the screening because nets may come in contact with sediments at both water depths. Only intertidal sediment chemistry data were screened for the beach play and clamming scenarios. As a simplifying and health-protective approach, all intertidal data were used for

[14] EPA Region 9 uses the term preliminary remediation goals (PRGs) for RBCs.

[15] In some cases, surrogate RBCs were used if an RBC was not available for a particular COPC. For example, mercury concentrations were compared to the RBC for methyl mercury, chromium concentrations were compared to the RBC for hexavalent chromium, and thallium concentrations were compared to the RBC for thallium and compounds. All surrogate RBCs used are identified in the tables in Attachment 1.

screening for the beach play and clamming scenarios, rather than just sediment data from more localized areas where activities may more likely occur.

The COPCs for the three sediment exposure scenarios are identified in Table B.3-3, which is excerpted from Tables 1 and 2 of Attachment 1. Most of the COPCs were analyzed in hundreds of sediment samples. Two COPCs that were analyzed much less frequently are PCB congeners and dioxins/furans. The locations where these chemicals were analyzed are shown on Maps B.3-3 and B.3-4, respectively.

Table B.3-3. Identification of COPCs for sediment exposure scenarios

Chemical[a]	Netfishing Scenario		Beach Play and Clamming Scenarios	
	COPC?	Rationale	COPC?	Rationale
Detected Chemicals				
Inorganic				
Aluminum	yes	maximum detection > RBC	yes	maximum detection > RBC
Antimony	yes	maximum detection > RBC	yes	maximum detection > RBC
Arsenic	yes	maximum detection > RBC	yes	maximum detection > RBC
Barium	yes	maximum detection < RBC	yes	maximum detection > RBC
Cadmium	yes	maximum detection > RBC	yes	maximum detection > RBC
Chromium	yes	maximum detection > RBC	yes	maximum detection > RBC
Copper	yes	maximum detection > RBC	yes	maximum detection > RBC
Iron	yes	maximum detection > RBC	yes	maximum detection > RBC
Lead	yes	maximum detection > RBC	yes	maximum detection > RBC
Manganese	yes	maximum detection > RBC	yes	maximum detection > RBC
Mercury	no	maximum detection < RBC	yes	maximum detection > RBC
Molybdenum	no	maximum detection < RBC	yes	maximum detection > RBC
Silver	no	maximum detection < RBC	yes	maximum detection > RBC
Thallium	yes	maximum detection > RBC	yes	maximum detection > RBC
Vanadium	yes	maximum detection > RBC	yes	maximum detection > RBC
Zinc	no	maximum detection < RBC	yes	maximum detection > RBC
Organic				
cPAHs	yes	maximum detection > RBC	yes	maximum detection > RBC
Total DDTs	no	maximum detection < RBC	yes	maximum detection > RBC
Dieldrin	yes	maximum detection > RBC	yes	maximum detection > RBC
Dioxin/furan TEQ	yes	maximum detection > RBC	yes	maximum detection > RBC
Total PCBs	yes	maximum detection > RBC	yes	maximum detection > RBC
PCB TEQ	yes	maximum detection > RBC	yes	maximum detection > RBC
Toxaphene	yes	maximum detection > RBC	yes	maximum detection > RBC
Undetected Chemicals				
4,6-Dinitro-o-cresol	no	1 of 989 reporting limits > RBC	yes	105 of 378 reporting limits > RBC
Benzidine	yes	314 of 484 reporting limits > RBC	yes	6 of 6 reporting limits > RBC
Bis(2-chloroethyl) ether	no	55 of 1,038 reporting limits > RBC	yes	48 of 378 reporting limits > RBC

Chemical[a]	Netfishing Scenario		Beach Play and Clamming Scenarios	
	COPC?	Rationale	COPC?	Rationale
n-Nitrosodimethylamine	yes	10 of 10 reporting limits > RBC	yes	203 of 203 reporting limits > RBC
n-Nitroso-di-n-propylamine	no	100 of 1,038 reporting limits > RBC	yes	108 of 378 reporting limits > RBC

[a] Only those chemicals identified as a COPC for tissue or sediment are listed; the complete list of screened chemicals is in Attachment 1.

cPAH – carcinogenic polycyclic aromatic hydrocarbon

COPC – chemical of potential concern

PCB – polychlorinated biphenyl

RBC – risk-based concentration

TEQ – toxic equivalent

The COPC screening process for the netfishing scenarios identified a total of 20 COPCs (Table B.3-3). RLs were generally lower than RBCs, with a few exceptions. Eighteen of the twenty chemicals were detected at least once and had a maximum detected concentration greater than the RBC. Although benzidine and n-nitrosodimethylamine were never detected, they were identified as COPCs because over 10% of RLs exceeded the applicable RBC (see Table B.3-3). However, given the uncertainty surrounding these data, risk estimates for these two undetected chemicals are discussed only in the uncertainty analysis (Section B.6). The full list of comparisons of RLs to RBCs is provided in Table 1 of Attachment 1. Table 1 also presents the results from RBC screening for the 323 other chemicals analyzed in subtidal and intertidal surface sediment that were not selected as COPCs. Many of these chemicals were not selected as COPCs because of the lack of toxicity data. These chemicals are qualitatively evaluated in the uncertainty analysis (Section B.6).

A total of 28 COPCs were identified for the beach play and clamming scenarios (Table B.3-3). Twenty-three of these chemicals were screened in as COPCs because the maximum detected concentration was greater than the RBC for that chemical. Although never detected, five chemicals (4,6-dinitro-o-cresol, benzidine, bis(2-chloroethyl) ether, n-nitrosodimethylamine, and n-nitroso-di-n-propylamine) were identified as COPCs because over 10% of the RLs exceeded the applicable RBC. However, given the uncertainty surrounding these data, risk estimates for these five undetected chemicals are discussed only in the uncertainty analysis (Section B.6). The greater number of COPCs for the beach play and clamming scenarios compared to the netfishing scenario reflects the use of residential-based RBCs for the beach play and clamming scenarios. The residential-based RBCs are lower than the industrial-based RBCs, and hence there are more chemical exceedances, because they assume more frequent and longer exposures compared to industrial exposures. The full list of comparisons of RLs to RBCs is provided in Table 2 of Attachment 1. Table 2 of Attachment 1 also presents the screening for the 297 other chemicals analyzed in intertidal surface sediment that were not selected as COPCs.

EPA Superfund guidance (EPA 1989, 2002d, e) includes provisions for distinguishing site-related contamination from naturally occurring or other non-site-related chemical concentrations. Because metals and trace elements occur naturally in sediments in the absence of any human influence, an additional screen against data from background areas was performed. Specific areas that represent background conditions have not been sampled for this project for any metals or trace elements other than arsenic (see Section B.5.5). In the absence of background samples collected for this project, sediment chemistry data for metals from non-urban areas were compiled from the joint Ecology/Puget Sound Ambient Monitoring Program (PSAMP) for central Puget Sound (NOAA and Ecology 2000) for comparison purposes. Background for this screening process was defined as areas not influenced by human activities. As such, urban areas sampled under PSAMP, such as Elliott Bay and Sinclair Inlet, were excluded from the dataset used to estimate background concentrations. Tables 1 and 2 in Attachment 1 describe the locations included in the background concentration calculation.

The mean concentrations for each metal or trace element in LDW surface sediments were compared to the mean concentration for the 52 samples collected from central Puget Sound. For all chemicals except nickel, the LDW mean concentration was much higher than the central Puget Sound mean concentration (see Tables 1 and 2 in Attachment 1); therefore, no formal statistical testing was conducted. The mean concentrations for nickel (27 mg/kg dw in the PSAMP database and 28 mg/kg dw in the LDW) were not significantly different from each other, according to both parametric and non-parametric t-tests.[16] Therefore, nickel was not retained as a COPC. The background comparison step described here is different than the use of background data described in MTCA, which includes the calculation of upper percentiles (e.g., 80th or 90th percentile) to characterize background. The MTCA approach is intended for application during the selection of cleanup levels. All the other metal and trace element COPCs shown in Table B.3-3 were retained.

All COPCs identified in Table B.3-3 are quantitatively evaluated in this HHRA. Risk estimates for undetected chemicals that were identified as COPCs based solely on RLs and screening criteria are discussed in the uncertainty analysis (Section B.6).

B.3.3.2 Tissue

COPCs in fish and shellfish tissue were identified by comparing maximum concentrations against RBCs for fish tissue developed by EPA Region 3 (EPA 2005b). Fish tissue RBCs are not available from other EPA regions, including EPA Region 9 (EPA 2004a), or from MTCA. Exposure factors for fish RBCs from EPA Region 3

[16] Two different transformations were applied to the nickel sediment data: logarithmic and Rankit. The Rankit transformation is used to convert non-normal data into normal data by converting each data value to the Z-score of its cumulative percentile in the dataset. Rankit transformed data can be used in parametric tests to compute a non-parametric test equivalent.

include: target HQ = 1, target excess cancer risk = 10^{-6}, body weight = 70 kg, exposure frequency = 350 days per year, exposure duration = 30 yr, and fish ingestion rate = 54 g/day (EPA 2005b). These exposure factors are consistent with Region 10 guidance for performing risk assessments (EPA 1996a), with the exception of the target HQ. Region 10 recommends a target HQ of 0.1 to account for cumulative effects from multiple chemicals and pathways. Region 3 RBCs for chemicals with noncarcinogenic effects were therefore decreased by a factor of 10 to be consistent with guidance from EPA Region 10.

In addition to the modification described above for target HQ, the Region 3 RBCs for both carcinogenic and non-carcinogenic endpoints were modified to account for site-specific tribal exposure assumption differences in consumption rate (98 g vs. 54 g; see Section B.3.4.1), exposure frequency (365 days vs. 350 days), body weight (81.8 kg vs. 70 kg), and exposure duration (70 years vs. 30 years) that were required by EPA Region 10 (EPA 2005a, 2007b). As a result of these site-specific modifications, the Region 3 RBCs based on a carcinogenic endpoint were multiplied by 0.26 for use in this HHRA.[17] The Region 3 RBCs based on non-carcinogenic endpoints were multiplied by 0.64 after application of the 0.1 factor described above for the HQ modification. The difference between the correction factors for carcinogenic and non-carcinogenic endpoints results because the RBC equations for carcinogenic and non-carcinogenic endpoints have a different denominator (i.e., the averaging time for non-carcinogenic effects is the same as the exposure frequency and duration), and consequently these modifications had less impact on the non-cancer RBCs.

Table 3 in Attachment 1 compares the maximum concentration for each chemical analyzed in the tissue samples with the applicable RBC and includes summary statistics such as detection frequency. COPC screening was performed using the combined tissue dataset summarized in Table 3 in Attachment 1 rather than by tissue type (e.g., benthic fish fillets, crab edible meat). In other words, a single detected concentration greater than the applicable RBC could designate a COPC, regardless of tissue type. The COPCs for the seafood consumption scenarios are identified in Table B.3-4, which is excerpted from Table 3 of Attachment 1.

[17] Using the ratios of site-specific exposure factors to default exposure factors used in the Region 3 RBC equation, the correction factor of 0.26 was derived by the following equation: (81.8/70 kg)/[(365/350 days/yr) × (70/30 yrs) × (98/54 g/day)]. The correction factor of 0.64 was derived by the following equation: [(81.8/70 kg) × (25,550/10,500 days)]/(365/350 days/yr) × (70/30 yrs) × (98/54 g/day).

Table B.3-4. Identification of COPCs for the seafood consumption scenario

CHEMICAL	RATIONALE
Detected chemicals	
Inorganic	
Antimony	maximum detection > RBC
Arsenic (inorganic)	maximum detection > RBC
Cadmium	maximum detection > RBC
Chromium	maximum detection > RBC
Copper	maximum detection > RBC
Lead	no RBC available; EPA recommends use of alternate toxicity evaluation method (see Section B.3.4.4 for more information regarding lead models)
Mercury	maximum detection > RBC
Methylmercury	maximum detection > RBC
Nickel	maximum detection > RBC
Tributyltin as ion	maximum detection > RBC
Vanadium	maximum detection > RBC
Zinc	maximum detection > RBC
Organic	
4-Methylphenol	maximum detection > RBC
Aldrin	maximum detection > RBC
alpha-BHC	maximum detection > RBC
beta-BHC	maximum detection > RBC
Bis(2-ethylhexyl) phthalate	maximum detection > RBC
Butyl benzyl phthalate	maximum detection > RBC
cPAHs	maximum detection > RBC
Carbazole	maximum detection > RBC
Total chlordane	maximum detection > RBC
Total DDTs	maximum detection > RBC
Dieldrin	maximum detection > RBC
Endrin	maximum detection > RBC
Endrin aldehyde	maximum detection > RBC
gamma-BHC	maximum detection > RBC
Heptachlor	maximum detection > RBC
Heptachlor epoxide	maximum detection > RBC
Hexachlorobenzene	maximum detection > RBC
PCB TEQ	maximum detection > RBC
Total PCBs	maximum detection > RBC
Pentachlorophenol	maximum detection > RBC

CHEMICAL	RATIONALE
Undetected chemicals	
1,2-Diphenylhydrazine	35 of 35 reporting limits > RBC
1,3-Dichlorobenzene	79 of 145 reporting limits > RBC
1,4-Dichlorobenzene	110 of 145 reporting limits > RBC
2,4,6-Trichlorophenol	140 of 145 reporting limits > RBC
2,4-Dichlorophenol	79 of 145 reporting limits > RBC
2,4-Dinitrophenol	95 of 130 reporting limits > RBC
2,4-Dinitrotoluene	96 of 145 reporting limits > RBC
2,6-Dinitrotoluene	93 of 145 reporting limits > RBC
2-Chlorophenol	79 of 145 reporting limits > RBC
3,3'-Dichlorobenzidine	112 of 112 reporting limits > RBC
3-Nitroaniline	121 of 124 reporting limits > RBC
4,6-Dinitro-o-cresol	145 of 145 reporting limits > RBC
4-Chloroaniline	82 of 113 reporting limits > RBC
4-Nitroaniline	130 of 133 reporting limits > RBC
Aniline	110 of 132 reporting limits > RBC
Benzidine	77 of 77 reporting limits > RBC
Bis(2-chloroethyl) ether	145 of 145 reporting limits > RBC
Bis(2-chloroisopropyl) ether	142 of 145 reporting limits > RBC
Hexachlorobutadiene	145 of 145 reporting limits > RBC
Hexachlorocyclopentadiene	110 of 142 reporting limits > RBC
Hexachloroethane	82 of 144 reporting limits > RBC
Nitrobenzene	96 of 145 reporting limits > RBC
n-Nitroso-di-n-propylamine[a]	78 of 145 reporting limits > RBC
n-Nitrosodimethylamine	145 of 145 reporting limits > RBC
n-Nitrosodiphenylamine	79 of 145 reporting limits > RBC
Toxaphene	130 of 130 reporting limits > RBC
Chemicals not analyzed in tissue[b]	
Dioxin/furan TEQ	detected chemical identified as COPC in sediment; also identified as a potential bioaccumulative compound by EPA (2000a)

[a] One composite sample of whole-body English sole contained a detected concentration of 270 µg/kg ww. However, this result was qualified as JN (estimated concentration, tentative identification). Given the uncertain quantification for this single result (all other results were undetected), the risks for this chemical will be discussed in the uncertainty analysis (Section B.6).

[b] No other chemical analyzed in sediment but not analyzed in tissue was designated as a detected COPC and identified by EPA (2000a) as a potential bioaccumulative compound.

BHC – benzene hexachloride
COPC – chemical of potential concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
EPA – US Environmental Protection Agency
RBC – risk-based concentration
TEQ – toxic equivalent

Fifty-nine COPCs were identified for the seafood consumption scenario (Table B.3-4). All COPCs identified in Table B.3-4, except dioxin/furan TEQs, are evaluated quantitatively in this HHRA. The risks for dioxin/furan TEQs are discussed qualitatively. The risks for chemicals that were never detected but had maximum RLs that were greater than their applicable RBC are evaluated in the uncertainty analysis (Section B.6). Table 3 of Attachment 1 presents the screening for 296 chemicals analyzed in tissue samples that were not identified as COPCs.

In addition to COPCs identified from analyses of tissue samples from the LDW, chemicals that were analyzed in sediment but never analyzed in tissue were also evaluated to select COPCs. Approximately 200 chemicals were analyzed in LDW sediment but were never analyzed in tissue samples. These chemicals are listed in Table 4 of Attachment 1. Over 120 of these chemicals were detected in sediment, and five of the chemicals were identified as sediment COPCs. However, none of these sediment COPCs, other than dioxin/furan TEQs, were identified by EPA (2000a) as potential bioaccumulative compounds. Therefore, other than dioxin/furan TEQs, none of the chemicals listed in Table 4 of Attachment 1 were identified as COPCs for tissue.

Many of the chemicals detected in LDW tissue samples have also been detected in tissue samples collected from background or reference areas. English sole have been collected from many locations throughout Puget Sound since 1989 as part of PSAMP (West et al. 2001), but no Puget Sound-wide monitoring program exists for the other seafood species included in this HHRA. Chemistry data from English sole fillets collected in non-urban areas were compiled as part of this HHRA for comparison to LDW data. Average concentrations computed from these data, including a list of the non-urban areas, are presented in Table 3 in Attachment 1 for informational purposes. No such comparisons were possible for other species evaluated in this HHRA because of the lack of adequate background data. Average concentrations, calculated using one-half the RL for non-detect results, were calculated for eight chemicals (total PCBs, arsenic, benzoic acid, benzyl alcohol, bis(2-ethylhexyl) phthalate, copper, mercury, and total DDTs) detected in more than 10% of the English sole fillet samples from non-urban areas. PSAMP English sole sampling stations were sampled with different frequencies and with varying numbers of fish per composite. Averages were computed using all data from individual samples and did not consider adjustment for these factors. The average concentrations of mercury, copper, and bis(2-ethylhexyl) phthalate in the background samples were similar to the average LDW concentrations. Average concentrations of total PCBs and total DDTs in LDW English sole fillets were more than 10 times higher than average background concentrations. Benzoic acid and benzyl alcohol were not identified as COPCs. Additional discussion on background concentrations of arsenic, which are available for all the target species, is provided in Section B.5.5.

B.3.4 SELECTION OF EXPOSURE PARAMETERS AND CALCULATION OF CHRONIC DAILY INTAKE

Exposure to COPCs in sediment or fish and shellfish is expressed as the CDI, which is the estimated daily chemical dose for an individual occurring over the exposure duration for each scenario. Two routes of exposure are relevant: ingestion and dermal contact. The CDI for ingestion is calculated as:

$$CDI_o = \frac{EPC \times IR \times FI \times EF \times ED \times CF}{BW \times AT}$$ **Equation 3-1**

Where:

CDI_o = chronic daily intake from oral exposure route (mg/kg-day)
EPC = chemical-specific exposure point concentration (mg/kg)
IR = ingestion rate (g/day)
FI = fractional intake of media derived from contaminated source (unitless)
EF = exposure frequency (days per year)
ED = exposure duration (years)
CF = conversion factor (kg/g)
BW = body weight (kg)
AT = averaging time (days), equivalent to the ED for non-carcinogenic COPCs and 70 years for carcinogenic COPCs

The CDI for dermal exposure[18] is calculated as:

$$CDI_d = \frac{EPC \times ABS \times SA \times AF \times FI \times EF \times ED \times CF}{BW \times AT}$$ **Equation 3-2**

Where:

CDI_d = chronic daily intake from dermal exposure route (mg/kg-day)
EPC = chemical-specific exposure point concentration (mg/kg)
ABS = dermal absorption factor (unitless)
SA = skin surface area exposed (cm^2)
AF = sediment to skin adherence factor by event (mg/cm^2-event)
FI = fractional intake of media derived from contaminated source (unitless)
EF = exposure frequency (events/year)
ED = exposure duration (years)
CF = conversion factor (kg/mg)

[18] Although chronic daily intake technically refers to oral exposure only, this term is also used in the HHRA to refer to dermal exposure, which is technically an absorbed dose. For this HHRA, the adjustment between orally administered doses and dermally administered doses was made by adjusting the oral toxicological benchmarks, as appropriate, according to EPA guidance (2004d) (see Section B.3.4.2 for additional details).

BW = body weight (kg)
AT = averaging time (days)

The exposure scenarios quantified in this HHRA are summarized in Tables B.3-5 (seafood ingestion) and B.3-6 (sediment exposure). These tables include key exposure parameters, so that the scenarios can be compared to each other, and references to more detailed tables (Tables B.3-7 through B.3-26), in which all exposure parameters for each scenario are given. Following the presentation of these scenario-specific tables, additional discussion is provided for seafood consumption rates (Section B.3.4.1), dermal absorption factors (Section B.3.4.2), and EPCs (Section B.3.4.3) because the derivation of values for these parameters requires a more detailed explanation than can be given in table notes.

Table B.3-5. Summary of seafood ingestion scenarios

Scenario	Ingestion Rate (IR) (g/day)					Exposure Duration (years)	Location of Scenario-Specific Details
	Pelagic Fish	Benthic Fish	Crab	Other Shellfish	Total		
Adult tribal RME (Tulalip data)	8.1	7.5	43.4	38.5	97.5	70	Table B.3-7
Adult tribal CT (Tulalip data)	1.3	1.2	6.6	5.9	15.0	30	Table B.3-8
Child tribal RME (Tulalip data)	3.2	3.0	17.4	15.4	39.0	6	Table B.3-9
Child tribal CT (Tulalip data)	0.52	0.48	2.64	2.34	6.0	6	Table B-3-10
Adult tribal (Suquamish data)	56	29.1	54.8	443.6	583.5	70	Table B.3-11
Adult API – RME	4.9	2.4	10.6	33.6	51.5	30	Table B.3-12
Adult API – CT	0.5	0.24	1.1	3.5	5.3	9	Table B-3-13
Adult one meal per month[a]	7.5	7.5	7.5	7.5	na	30	Table B.3-14

[a] Adult one-meal-per-month consumption was evaluated by individual seafood categories independently to reflect different fishing and consumption practices.

API – Asian and Pacific Islander
CT – central tendency
IR – ingestion rate
na – not applicable
RME – reasonable maximum exposure

Table B.3-6. Summary of sediment exposure scenarios

Scenario	Incidental Sediment IR (g/day)	Exposure Frequency (days/yr)	Exposure Duration (years)	Skin Surface Area Exposed (cm^2)	Location of Scenario-Specific Details	
					Incidental Ingestion	Dermal
Netfishing RME	0.05	119	44	3,600	Table B.3-15	Table B.3-16
Netfishing CT	0.05	63	29	3,600	Table B.3-17	Table B.3-18
Beach play RME[a]	0.2	65	6	varies with age	Table B.3-19	Table B.3-20
Clamming 7 days per year	0.1	7	30	6,040	Table B.3-21	Table B.3-22
Tribal clamming RME	0.1	120	64	6,040	Table B.3-23	Table B.3-24
Tribal clamming 183 days per year	0.1	183	70	6,040	Table B.3-25	Table B.3-26

[a] For the beach play RME scenarios, the river was divided into eight sections to assess risks associated with different parts of the river. Children are evaluated from birth through 6 years of age.

CT – central tendency

RME – reasonable maximum exposure

IR – ingestion rate

Table B.3-7. Daily intake calculations – seafood ingestion, adult tribal RME scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m) + (EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg ww	Table B.3-39	Section B.3.4.3
IR-p	ingestion rate – pelagic fish	g/day	8.1	Section B.3.4.1
IR-b	ingestion rate – benthic fish	g/day	7.5	Section B.3.4.1
IR-bwb	ingestion rate – benthic fish, whole body	g/day	0	Section B.3.4.1
IR-c	ingestion rate – crabs, edible meat	g/day	33	Section B.3.4.1
IR-cwb	ingestion rate – crabs, whole body	g/day	10.4	Section B.3.4.1
IR-m	ingestion rate – mussels	g/day	0.82	Section B.3.4.1
IR-cl	ingestion rate – clams	g/day	37.7	Section B.3.4.1
FI	fractional intake derived from source	unitless	1[a]	EPA (2007b)
EF	exposure frequency	days/yr	365[b]	EPA (1991a)
ED-a	exposure duration – adult	years	70	EPA (2005a)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight-adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	25,550	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] A fractional intake derived from source of 1 was directed by EPA (2007b).

[b] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

EPA – US Environmental Protection Agency
na – not applicable
RME – reasonable maximum exposure
ww – wet weight

Table B.3-8. Daily intake calculations – seafood ingestion, adult tribal CT scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m) + (EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg	Table B.3-39[a]	Section B.3.4.3
IR-p	ingestion rate – pelagic fish	g/day	1.3	Section B.3.4.1
IR-b	ingestion rate – benthic fish	g/day	1.2	Section B.3.4.1
IR-bwb	ingestion rate – benthic fish, whole body	g/day	0	Section B.3.4.1
IR-c	ingestion rate – crabs, edible meat	g/day	5.0	Section B.3.4.1
IR-cwb	ingestion rate – crabs, whole body	g/day	1.6	Section B.3.4.1
IR-m	ingestion rate – mussels	g/day	0.10	Section B.3.4.1
IR-cl	ingestion rate – clams	g/day	5.8	Section B.3.4.1
FI	fractional intake derived from source	unitless	1[b]	EPA (2007b)
EF	exposure frequency	days/yr	365[c]	EPA (1991a)
ED-a	exposure duration – adult	years	30	EPA (EPA 1997)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	10,950	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] EPCs for CT scenarios are based on mean concentrations, in contrast to the EPCs for the RME scenarios, which are based on 95% UCLs on mean concentrations.

[b] A fractional intake derived from source of 1 was directed by EPA (2007b).

[c] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

CT – central tendency
EPA – US Environmental Protection Agency
na – not applicable
RME – reasonable maximum exposure
UCL – upper confidence limit
ww – wet weight

Table B.3-9. Daily intake calculations – seafood ingestion, child tribal RME scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion

Intake equation/model name: Chronic Daily Intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m)+(EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-ct × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg ww	Table B.3-39	Section B.3.4.3
IR-p	ingestion rate – pelagic fish	g/day	3.2	Section B.3.4.1
IR-b	ingestion rate – benthic fish	g/day	3.0	Section B.3.4.1
IR-bwb	ingestion rate – benthic fish, whole body	g/day	0	Section B.3.4.1
IR-c	ingestion rate – crabs, edible meat	g/day	13.2	Section B.3.4.1
IR-cwb	ingestion rate – crabs, whole body	g/day	4.2	Section B.3.4.1
IR-m	ingestion rate – mussels	g/day	0.33	Section B.3.4.1
IR – cl	ingestion rate – clams	g/day	15.1	Section B.3.4.1
FI	fractional intake derived from source	unitless	1[b]	EPA (2007b)
EF	exposure frequency	days/yr	365[a]	EPA (1991a)
ED-c	exposure duration – child	years	6	EPA (1991a)
CF	conversion factor	kg/g	0.001	na
BW-ct	body weight – child Tulalip	kg	15.2	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	2,190	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

[b] A fractional intake derived from source of 1 was directed by EPA (2007b).

EPA – US Environmental Protection Agency

na – not applicable

RME – reasonable maximum exposure

Table B.3-10. Daily intake calculations – seafood ingestion, child tribal CT scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion

Intake equation/model name: Chronic Daily Intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m)+(EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-ct × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
IR-p	ingestion rate – pelagic fish	g/day	0.52	Section B.3.4.1
IR-b	ingestion rate – benthic fish	g/day	0.48	Section B.3.4.1
IR-bwb	ingestion rate – benthic fish, whole body	g/day	0	Section B.3.4.1
IR-c	ingestion rate – crabs, edible meat	g/day	2.0	Section B.3.4.1
IR-cwb	ingestion rate – crabs, whole body	g/day	0.64	Section B.3.4.1
IR-m	ingestion rate – mussels	g/day	0.040	Section B.3.4.1
IR – cl	ingestion rate – clams	g/day	2.3	Section B.3.4.1
FI	fractional intake derived from source	unitless	1[b]	EPA (2007b) [b]
EF	exposure frequency	days/yr	365[c]	EPA (1991a)
ED-c	exposure duration – child	years	6	EPA (1991a)
CF	conversion factor	kg/g	0.001	na
BW-ct	body weight – child Tulalip	kg	15.2	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	2,190	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] EPCs for CT scenarios are based on mean concentrations, in contrast to the EPCs for the RME scenarios, which are based on 95% UCLs on mean concentrations.

[b] A fractional intake derived from source of 1 was directed by EPA (2007b).

[c] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

CT – central tendency
UCL – upper confidence limit
EPA – US Environmental Protection Agency
ww – wet weight
na – not applicable

Table B.3-11. Daily intake calculations – seafood ingestion, adult tribal scenario based on Suquamish data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m)+(EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg ww	Table B.3-39	Section B.3.4.3
IR-p	ingestion rate – pelagic fish	g/day	56	Section B.3.4.1
IR-b	ingestion rate – benthic fish	g/day	25.9	Section B.3.4.1
IR-bwb	ingestion rate – benthic fish, whole body	g/day	3.2	Section B.3.4.1
IR-c	ingestion rate – crabs, edible meat	g/day	41.6	Section B.3.4.1
IR-cwb	ingestion rate – crabs, whole body	g/day	13.2	Section B.3.4.1
IR-m	ingestion rate – mussels	g/day	5.0	Section B.3.4.1
IR-cl	ingestion rate – clams	g/day	438.6	Section B.3.4.1
FI	fractional intake derived from source	unitless	1[b]	EPA (2007b)
EF	exposure frequency	days/yr	365[a]	EPA (1991a)
ED-a	exposure duration – adult	years	70	EPA (2005a)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	79[c]	Suquamish Tribe (2000)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	25,550	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

[b] A fractional intake derived from source of 1 was directed by EPA (2007b).

[c] Average body weight based on information provided by the Suquamish Tribe.

EPA – US Environmental Protection Agency

na – not applicable

ww – wet weight

Table B.3-12. Daily intake calculations – seafood ingestion, adult API RME scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m)+(EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg ww	Table B.3-39	Section B.3.4.3
IR-p	ingestion rate – pelagic fish	g/day	4.9	Section B.3.4.1
IR-b	ingestion rate – benthic fish	g/day	2.0	Section B.3.4.1
IR-bwb	ingestion rate – benthic fish, whole body	g/day	0.39	Section B.3.4.1
IR-c	ingestion rate – crabs, edible meat	g/day	5.7	Section B.3.4.1
IR-cwb	ingestion rate – crabs, whole body	g/day	4.9	Section B.3.4.1
IR-m	ingestion rate – mussels	g/day	4.6	Section B.3.4.1
IR-cl	ingestion rate – clams	g/day	29.0	Section B.3.4.1
FI	fractional intake derived from source	unitless	1	Kissinger (2005)
EF	exposure frequency	days/yr	365[b]	EPA (1991a)
ED-a	exposure duration – adult	years	30	EPA (1989)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	63[c]	EPA (1999a)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	10,950	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] A fractional intake derived from source of 1 was directed by EPA (Kissinger 2005).

[b] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

[c] Average body weight for all surveyed individuals in API seafood consumption study in King County, as reported in EPA (1999a).

API – Asian and Pacific Islander

EPA – US Environmental Protection Agency

na – not applicable

RME – reasonable maximum exposure

ww – wet weight

Table B.3-13. Daily intake calculations – seafood ingestion, adult API CT scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m)+(EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg ww	Table B.3-39[a]	Section B.3.4.3
IR-p	ingestion rate – pelagic fish	g/day	0.5	Section B.3.4.1
IR-b	ingestion rate – benthic fish	g/day	0.2	Section B.3.4.1
IR-bwb	ingestion rate – benthic fish, whole body	g/day	0.04	Section B.3.4.1
IR-c	ingestion rate – crabs, edible meat	g/day	0.59	Section B.3.4.1
IR-cwb	ingestion rate – crabs, whole body	g/day	0.51	Section B.3.4.1
IR-m	ingestion rate – mussels	g/day	0.47	Section B.3.4.1
IR-cl	ingestion rate – clams	g/day	3.0	Section B.3.4.1
FI	fractional intake derived from source	unitless	1[b]	Kissinger (2005)
EF	exposure frequency	days/yr	365[c]	EPA (1991a)
ED-a	exposure duration – adult	years	9	EPA (1989)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	63[d]	EPA (1999a)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	3,285	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] EPCs for CT scenarios are based on mean concentrations, in contrast to the EPCs for the RME scenarios, which are based on 95% UCLs on mean concentrations.

[b] A fractional intake derived from source of 1 was directed by EPA (Kissinger 2005).

[c] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

[d] Average body weight for all surveyed individuals in API seafood consumption study in King County, as reported in EPA (1999a).

API – Asian and Pacific Islander
CT – central tendency
EPA – US Environmental Protection Agency
na – not applicable
UCL – upper confidence limit
ww – wet weight

Table B.3-14. Daily intake calculations – seafood ingestion, adult one-meal-per-month scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Exposure route: Ingestion

Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = [(EPC-p × IR-p) + (EPC-b × IR-b) + (EPC-bwb × IR-bwb) + (EPC-c × IR-c) + (EPC-cwb × IR-cwb) + (EPC-m × IR-m)+(EPC-cl × IR-cl)] × FI × EF × ED-a × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC-p	exposure point concentration in pelagic fish	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-b	exposure point concentration in benthic fish, fillet	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-bwb	exposure point concentration in benthic fish, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-c	exposure point concentration in crabs, edible meat	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cwb	exposure point concentration in crabs, whole body	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-m	exposure point concentration in mussels	mg/kg ww	Table B.3-39	Section B.3.4.3
EPC-cl	exposure point concentration in clams	mg/kg ww	Table B.3-39	Section B.3.4.3
IR-arp	ingestion rate – pelagic fish	g/day	7.5[a]	Section B.3.4.1
IR-arb	ingestion rate – benthic fish	g/day	7.5[a]	Section B.3.4.1
IR-arc	ingestion rate – crabs, edible meat	g/day	7.5[a]	Section B.3.4.1
IR-arcl	ingestion rate – clams	g/day	7.5[a]	Section B.3.4.1
FI	fractional intake derived from source	unitless	1	na
EF	exposure frequency	days/yr	365[b]	EPA (1991a)
ED-a	exposure duration – adult	years	30	EPA (1989)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	71.8	EPA (1997)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	10,950	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Adult one-meal-per-month consumption was evaluated by individual seafood categories independently to provide information to the public and risk managers on consumption of various potential types of fish and shellfish. Risks from adult one-meal-per-month consumption are divided into four scenarios that address risks individually for each of the four main seafood consumption categories (i.e., benthic fish, pelagic fish, clams, and crabs). Each scenario assumes that one 227 gram (8 oz.) meal is consumed per month, which equates to 7.5 g/day. Consumption of anadromous fish (e.g., salmon) is not considered based on the EPA recommendation that the site-related concentration term for salmon is zero for bioaccumulative contaminants (EPA 2005a).

[b] Default exposure frequency of 350 days/yr modified to 365 days/yr to account for the fact that seafood consumption rate estimates are based on 365 days/yr.

EPA – US Environmental Protection Agency

na – not applicable

ww – wet weight

Table B.3-15. Daily intake calculations – incidental sediment ingestion during netfishing, adult tribal RME scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Ingestion (incidental)
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × IR-s × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.3.4.3
IR-s	incidental ingestion rate	g/day	0.050	EPA (1991a)
FI	fractional intake derived from source	unitless	1[a]	na
EF	exposure frequency	days/yr	119[b]	na
ED	exposure duration	years	44[b]	na
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	16,060	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[b] Value recommended by EPA based on the length of the 2001 salmon season and on conversations with Muckleshoot Indian Tribe Assistant Harvest Manager regarding fishing frequency. This approach assumes that a fisher is present for each day of the fishing season. See Subappendix B.3 in Windward (2003b) for more details on the derivation of this value.

EPA – US Environmental Protection Agency

na – not applicable

RME – reasonable maximum exposure

dw – dry weight

Table B.3-16. Daily intake calculations – dermal contact with sediment during netfishing, adult tribal RME scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Dermal

Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × ABS × SA × AF × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
ABS	dermal absorption factor	unitless	Table B.3-34	Section B.2.4.2
SA	skin surface area exposed	cm^2	3,600[a]	EPA (1997)
AF	adherence factor by event	mg/cm^2-event	0.2	EPA (1999d)
FI	fractional intake derived from source	unitless	1[b]	na
EF	exposure frequency	events/yr	119[c]	na
ED	exposure duration	years	44[c]	na
CF	conversion factor	kg/mg	0.000001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	16,060	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Recommended surface area for commercial/industrial worker. Assumes that head, hands, and forearms are exposed. Selected value represents sum of 50th percentile surface areas for men (most netfishers are men) for these body parts; taken from Table 6-2 in EPA (1997). Given the higher body weight of individuals surveyed in Toy et al. (1996) compared to the general US population, the surface area values selected here for commercial/industrial workers may underestimate the surface area of tribal fishermen body parts. However, no conversion data are available at the present time.

[b] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[c] Value recommended by EPA based on conversation with Muckleshoot Indian Tribe Assistant Harvest Manager. See Subappendix B.3 in Windward (2003b) for more details on the derivation of this value.

Dw – dry weight

EPA – US Environmental Protection Agency

na – not applicable

RME – reasonable maximum exposure

Table B.3-17. Daily intake calculations – incidental sediment ingestion during netfishing, adult tribal CT scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Ingestion (incidental)
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × IR-s × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44[a]	Section B.2.4.3
IR-s	incidental ingestion rate	g/day	0.050	EPA (1991a)
FI	fractional intake derived from source	unitless	1[b]	na
EF	exposure frequency	days/yr	63[c]	na
ED	exposure duration	years	29[d]	na
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	10,585	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] EPCs for CT scenarios are based on mean concentrations, in contrast to the EPCs for the RME scenarios, which are based on 95% UCLs on mean concentrations.

[b] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[c] Value recommended by EPA based on conversation with Muckleshoot Indian Tribe Assistant Harvest Manager. Selected value is duration of coho fishing season (most individuals fish for coho). See Subappendix B.3 in Windward (2003b) for more details on the derivation of this value.

[d] Value recommended by EPA based on conversation with Muckleshoot Indian Tribe Assistant Harvest Manager. Selected value is EPA's best professional judgment assuming that fishing starts at age 16 and ends at age 45.

CT – central tendency

dw – dry weight

EPA – US Environmental Protection Agency

na – not applicable

UCL – upper confidence limit

Table B.3-18. Daily intake calculations – dermal contact with sediment during netfishing, adult tribal CT scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Dermal

Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × ABS × SA × AF × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44[a]	Section B.2.4.3
ABS	dermal absorption factor	unitless	Table B.3-34	Section B.2.4.2
SA	skin surface area exposed	cm^2	3,600[b]	EPA (1997)
AF	adherence factor by event	mg/cm^2-event	0.02[c]	EPA (2004d)
FI	fractional intake derived from source	unitless	1[d]	na
EF	exposure frequency	event/ year	63[e]	na
ED	exposure duration	years	29[f]	na
CF	conversion factor	kg/mg	0.000001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	10,585	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] EPCs for CT scenarios are based on mean concentrations, in contrast to the EPCs for the RME scenarios, which are based on 95% UCLs on mean concentrations.

[b] Recommended surface area for commercial/industrial worker. Assumes that head, hands, and forearms are exposed. Selected value represents sum of 50th percentile surface areas for men (most netfishers are men) for these body parts; taken from Table 6-2 in EPA (1997). Given the higher body weight of individuals surveyed in Toy et al. (1996) compared to the general US population, the surface area values selected here for commercial/industrial workers may underestimate the surface area of tribal fishermen body parts. However, no conversion data are available at the present time.

[c] Default value for CT industrial workers in Risk assessment guidance for Superfund (RAGS) Part E (EPA 2004d).

[d] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[e] Value recommended by EPA based on conversation with Muckleshoot Indian Tribe Assistant Harvest Manager. Selected value is duration of coho fishing season (most individuals fish for coho). See Subappendix B.3 in Windward (2003b) for more details on the derivation of this value.

[f] Value recommended by EPA based on conversation with Muckleshoot Indian Tribe Assistant Harvest Manager. Selected value is EPA's best professional judgment assuming that fishing starts at age 16 and ends at age 45.

CT – central tendency

dw – dry weight

EPA – US Environmental Protection Agency

na – not applicable

UCL – upper confidence limit

Table B.3-19. Daily intake calculations – incidental sediment ingestion during child beach play RME

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Ingestion (incidental)

Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = (EPC × IR-s × FI × EF × CF × 1/AT) × Γ (ED_i × 1/BW_i-c)

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
IR-s	incidental sediment ingestion rate	g/day	0.200	EPA (1997)
FI	fractional intake derived from source	unitless	1	na
EF	exposure frequency	days/yr	65[a]	Parametrix (2003)
ED_i	exposure duration – by age class	years	varies[b]	EPA (1991a)
CF	conversion factor	kg/g	0.001	na
BW_i-c	body weight – child	kg	varies[c]	EPA (2006a)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	2,190	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Based on 95th percentile for children from birth through 6 years old playing or digging in sand immediately adjacent to or in water at King County beach parks on Lake Union, Lake Washington, and Lake Sammamish (Parametrix 2003).

[b] Doses for six different age classes are calculated separately: < 1, 1 to 2, 2 to 3, 3 to 4, 4 to 5, and 5 to 6. Total exposure duration is 6 years, but duration for each year class is 1 year.

[c] Body weights for each age class are means for boys and girls combined (EPA 2006a).

Age class	BW_i (kg)	Age class	BW_i (kg)
< 1	9.1	3 to 4	15.3
1 to 2	11.3	4 to 5	17.4
2 to 3	13.3	5 to 6	19.7

dw – dry weight
EPA – US Environmental Protection Agency
na – not applicable

Table B.3-20. Daily intake calculations – dermal contact with sediment during child beach play RME

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Dermal
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = (EPC × ABS × AF × FI × EF × CF × 1/AT) × Γ (SA_i × ED_i × 1/BW_i-c)

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
ABS	dermal absorption factor	unitless	Table B.3-34	Section B.2.4.2
SA_i	skin surface area exposed – by age class	cm^2	varies[a]	EPA (1997)
AF	adherence factor by event	mg/cm^2-event	0.2	EPA (2004d)
FI	fractional intake derived from source	unitless	1	na
EF	exposure frequency	events/yr	65[b]	Parametrix (2003)
ED_i	exposure duration – by age class	years	varies[c]	EPA (1991a)
CF	conversion factor	kg/mg	0.000001	na
BW_i-c	body weight, child – by age class	kg	varies[d]	EPA (2006a)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	2,190	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Assumes that 35% of the total body surface area is exposed, roughly corresponding to an individual wearing a short-sleeve shirt and short pants but no shoes (EPA 1992a). Body surface area data taken from EPA (2006a) and correspond roughly to head, hands, lower arms, feet, and lower legs. Values shown below are means of the 50th percentile surface areas (total surface area × 0.35) between male and female children.

Age class	SA_i (cm^2)	Age class	SA_i (cm^2)	Age class	SA_i (cm^2)
< 1	1,330	2 to 3	2,069	4 to 5	2,515
1 to 2	1,750	3 to 4	2,298	5 to 6	2,751

[b] Based on 95th percentile for children from birth to 6 years old playing or digging in sand immediately adjacent to or in water at King County beach parks on Lake Union, Lake Washington, and Lake Sammamish (Parametrix 2003).

[c] Doses for six different age classes are calculated separately: : < 1, 1 to 2, 2 to 3, 3 to 4, 4 to 5, and 5 to 6. Total exposure duration is 6 years, but duration for each year class is 1 year.

Body weights for each age class are means for boys and girls combined (EPA 2006a).

Age class	BW_i (kg)	Age class	BW_i (kg)
< 1	9.1	3 to 4	15.3
1 to 2	11.3	4 to 5	17.4
2 to 3	13.3	5 to 6	19.7

dw – dry weight
EPA – US Environmental Protection Agency
na – not applicable

Table B.3-21. Daily intake calculations –incidental sediment ingestion during clamming, 7-day-per-year scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Ingestion (incidental)
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × IR-s × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
IR-s	incidental ingestion rate	g/day	0.1	EPA (1997)
FI	fractional intake derived from source	unitless	1[a]	na
EF	exposure frequency	days/yr	7[b]	na
ED	exposure duration	years	30	EPA (1989)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	71.8[c]	EPA (1997)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	10,950	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[b] Exposure frequency was assumed to be once per month during months when there is a daylight minus tide, based on NOAA tidal information (NOAA 2006) from 2004 through 2006.

[c] Mean body weight for male and female adults from Table 7-2 in EPA (1997).

dw – dry weight

EPA – US Environmental Protection Agency

na – not applicable

Table B.3-22. Daily intake calculations – dermal contact with sediment during clamming, 7-day-per-year scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Dermal

Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × ABS × SA × AF × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
ABS	dermal absorption factor	unitless	Table B.3-34	Section B.2.4.2
SA_i	skin surface area exposed	cm^2	6,040[a]	EPA (1997)
AF	adherence factor by event	mg/cm^2-event	0.2	EPA (2004d)
FI	fractional intake derived from source	unitless	1[b]	na
EF	exposure frequency	events/yr	7[c]	na
ED_i	exposure duration	years	30	EPA (1989)
CF	conversion factor	kg/mg	0.000001	na
BW-a	body weight – adult	kg	71.8[d]	EPA (1997)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	10,950	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Assumes that 39% of the total body surface area is exposed, roughly corresponding to a barefoot individual wearing a short-sleeve shirt and short pants (EPA 1992a). Body surface area data taken from Tables 6-2, 6-3 and 6-4 in EPA (1997) and corresponds to head, lower arms, hands, lower legs, and feet.

[b] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[c] Exposure frequency was assumed to be once per month during months when there is a daylight minus tide, based on NOAA tidal information (NOAA 2006) from 2004 through 2006.

[d] Mean body weight for male and female adults from Table 7-2 in EPA (1997).

dw – dry weight

EPA – US Environmental Protection Agency

na – not applicable

Table B.3-23. Daily intake calculations – incidental sediment ingestion during tribal clamming RME scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Ingestion (incidental)

Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × IR-s × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
IR-s	Incidental ingestion rate	g/day	0.1	EPA (1997)
FI	fractional intake derived from source	unitless	1[a]	na
EF	exposure frequency	days/yr	120[b]	Kissinger (2007c)
ED	exposure duration	years	64[c]	Kissinger (2007c)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	23,360	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[b] Exposure frequency determined by EPA to reflect tribal clamming patterns (Kissinger 2007c).

[c] Exposure duration determined by EPA to reflect tribal clamming patterns (Kissinger 2007c).

dw – dry weight

EPA – US Environmental Protection Agency

na – not applicable

Table B.3-24. Daily intake calculations – dermal contact with sediment during tribal clamming RME scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Dermal
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × ABS × SA × AF × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
ABS	dermal absorption factor	unitless	Table B.3-34	Section B.2.4.2
SA_i	skin surface area exposed	cm^2	6,040[a]	EPA (1997)
AF	adherence factor by event	mg/cm^2-event	0.2	EPA (2004d)
FI	fractional intake derived from source	unitless	1[b]	na
EF	exposure frequency	events/yr	120	Kissinger (2007c)
ED_i	exposure duration	years	64	Kissinger (2007c)
CF	conversion factor	kg/mg	0.000001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	23,360	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Assumes that 39% of the total body surface area is exposed, roughly corresponding to a barefoot individual wearing a short-sleeve shirt and short pants (EPA 1992a). Body surface area data taken from Tables 6-2, 6-3 and 6-4 in EPA (1997) and corresponds to head, lower arms, hands, lower legs, and feet.

[b] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[c] Exposure frequency determined by EPA to reflect tribal clamming patterns (Kissinger 2007c).

[d] Exposure duration determined by EPA to reflect tribal clamming patterns (Kissinger 2007c).

dw – dry weight

EPA – Environmental Protection Agency

na – not applicable

Table B.3-25. Daily intake calculations – incidental sediment ingestion during tribal clamming, 183-day-per-year scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Ingestion (incidental)
Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × IR-s × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
IR-s	Incidental ingestion rate	g/day	0.1	EPA (1997)
FI	fractional intake derived from source	unitless	1[a]	na
EF	exposure frequency	days/yr	183[b]	Kissinger (2007c)
ED	exposure duration	years	70[c]	Kissinger (2007c)
CF	conversion factor	kg/g	0.001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	25,550	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[b] Exposure frequency requested by Muckleshoot and Suquamish Tribes (Kissinger 2007c).

[c] Exposure duration requested by Muckleshoot and Suquamish Tribes (Kissinger 2007c).

dw – dry weight

EPA – US Environmental Protection Agency

na – not applicable

Table B.3-26. Daily intake calculations – dermal contact with sediment during tribal clamming, 183-day-per-year scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Exposure route: Dermal

Intake equation/model name: Chronic daily intake (CDI) (mg/kg-day) = EPC × ABS × SA × AF × FI × EF × ED × CF × 1/BW-a × 1/AT

Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference
EPC	exposure point concentration in sediment	mg/kg dw	Table B.3-44	Section B.2.4.3
ABS	dermal absorption factor	unitless	Table B.3-34	Section B.2.4.2
SA_i	skin surface area exposed	cm^2	6,040[a]	EPA (1997)
AF	adherence factor by event	mg/cm^2-event	0.2	EPA (2004d)
FI	fractional intake derived from source	unitless	1[b]	na
EF	exposure frequency	events/yr	183[c]	Kissinger (2007c)
ED_i	exposure duration	years	70[d]	Kissinger (2007c)
CF	conversion factor	kg/mg	0.000001	na
BW-a	body weight – adult	kg	81.8	Toy et al. (1996)
AT-C	averaging time – cancer	days	25,550	EPA (1989)
AT-N	averaging time – non-cancer	days	25,550	EPA (1989)

Source: Standard Table 4 in EPA (1998)

[a] Assumes that 39% of the total body surface area is exposed, roughly corresponding to a barefoot individual wearing a short-sleeve shirt and short pants (EPA 1992a). Body surface area data taken from Tables 6-2, 6-3 and 6-4 in EPA (1997) and corresponds to head, lower arms, hands, lower legs, and feet.

[b] Fractional intake of 1 used to be consistent with EPA direction for seafood consumption scenarios.

[c] Exposure frequency requested by Muckleshoot and Suquamish Tribes (Kissinger 2007c).

[d] Exposure duration requested by Muckleshoot and Suquamish Tribes (Kissinger 2007c).

dw – dry weight

EPA – Environmental Protection Agency

na – not applicable

B.3.4.1 Seafood ingestion rates

The primary documented use of the LDW as a fishery is the commercial salmon fishery operated by the Muckleshoot Indian Tribe. However, as indicated in Section B.2.1.2, because of the migratory nature of salmon, bioaccumulative chemicals found in adult salmon tissue do not likely represent the exposure of salmon to chemicals present in the LDW; and therefore, salmon consumption is not included in this risk assessment. The uncertainty analysis (Section B.6.1.2.2) includes a discussion of uncertainties in risk estimates resulting from not including consumption of salmon in the risk assessment.

Some information suggests that other relatively high fish-consuming populations may use the LDW for at least part of their fish collection (King County 1999b; EPA 1999a). A total of eight scenarios, including some RME and CT scenarios, were developed to represent a range of potential exposures via consumption of LDW seafood by different groups.

There are no surveys of tribal resources that specifically focus on the LDW. Based on recent regional tribal consumption surveys from Puget Sound (Toy et al. 1996; Suquamish Tribe 2000), the following five scenarios were developed: adult tribal scenarios (RME and CT) based on Tulalip data, child tribal scenarios (RME and CT) based on Tulalip data, and adult tribal scenario based on Suquamish data, as summarized in Section B.3.4.1.1. A review and interpretation of the two tribal consumption studies by EPA provides the basis for the tribal scenarios presented here (EPA 2007b). Uncertainty associated with the tribal adult ingestion rates is discussed further in Section B.6.1.2.2.

A seafood consumption survey prepared for the King County Water Quality Assessment (King County 1999b) verified that fish and crab were being harvested within and near the LDW. Specifically, seafood has been harvested from T-105 within the LDW and from the Spokane Street Bridge just north of the LDW adjacent to Harbor Island. The Spokane Street Bridge location was identified as the third most popular location for seafood harvest of the Elliott Bay and LDW locations included in the survey. Crabs were collected by more people than any other species. The number of individuals collecting sole was a third of the number of individuals who collected crabs. The King County survey also documented that a substantial fraction of Duwamish/Elliott Bay anglers are of Asian or Pacific Islander descent. Guidance for the development of API consumption scenarios (RME and CT) based on a recent King County survey (EPA 1999a) was also provided by EPA (Kissinger 2005).

Finally, to provide risk information for the general public, and to provide risk information on individual resource types, a scenario was developed for fishing that considered consumption of a single meal per month of pelagic fish, benthic fish, crabs, and clams. This approach is not based on any specific fish consumption survey and is instead intended to provide additional information for less frequent (i.e., one meal per

month) seafood consumers on a resource-by-resource basis. It can also be readily scaled to individual consumption rates.

B.3.4.1.1 Adult tribal seafood consumption scenarios based on Tulalip and Suquamish data

EPA Region 10 has developed tribal seafood consumption scenarios for application to CERCLA and RCRA sites in Puget Sound and the Strait of Georgia based on seafood consumption studies of the Tulalip Tribes and the Suquamish Tribe (EPA 2005a, 2007b). In these guidance documents, EPA specifies consumption rates for some tribal members for each type of seafood (i.e., seafood category). In EPA's decision chart for the identification of appropriate tribal seafood consumption rates, EPA identified the Tulalip Tribes consumption rate for application to the LDW (EPA 2005a). In comments on the draft HHRA, EPA (2006c) stated that "based on policy consideration, EPA is intending to use the Tulalip Tribes seafood consumption rate as the principal rate to compute health protective tribal seafood consumption risks." A scenario was also developed to represent the consumption rate for Tulalip tribal children.

The Muckleshoot and Suquamish Tribes requested that an exposure scenario developed by EPA using the Suquamish seafood consumption data also be included in the HHRA. EPA Region 10 has also provided guidance for the inclusion of this scenario, which "will assist in characterizing the range of seafood consumption risks"(EPA 2006c). EPA has stated that the results from the Suquamish consumption scenario will not be used to develop a list of risk drivers and associated RBTCs for the purpose of developing and evaluating remedial alternatives in the Feasibility Study.

The consumption rates in the guidance are based on seafood consumption surveys of the Tulalip Tribes (Toy et al. 1996) and the Suquamish Tribe (Suquamish Tribe 2000). Briefly, the 95th percentile of total seafood consumption from Puget Sound was attributed to different seafood categories (anadromous, bottom feeding, and pelagic fish, as well as shellfish) assuming the proportion of consumption in each category calculated for average consumption (including both consumers and non-consumers) also applied to the 95th percentile consumption of Puget Sound seafood. For example, the average consumption of anadromous fish divided by the sum of the averages of consumption of all seafood categories was 49.7%. Thus, it was assumed that 49.7% of the 95th percentile of total seafood consumed from Puget Sound by Tulalip Tribal members (194 g/day) was anadromous fish (96.4 g/day) (EPA 2007b). The same approach was applied for estimating the consumption of different seafood categories for the adult Tulalip CT scenario using the 50th percentile of total seafood consumed from Puget Sound (Hiltner 2007). Total quantities of non-anadromous seafood consumed for the tribal adult scenario based on Tulalip data were 97.5 g/day and 15 g/day for the RME and CT scenarios, respectively. Total non-anadromous seafood consumed for the tribal adult scenario based on Suquamish data was 583.5 g/day.

Table B.3-27 presents the tribal seafood consumption rates for different components of the market basket specified by EPA for application to the LDW. The last column discusses the presence and prevalence of each seafood group in the LDW. As agreed upon with EPA, consumption of anadromous fish was not included for LDW tribal exposure and risk estimates (EPA 2005a) because the bulk of the body burden of bioaccumulative contaminants in adult salmon is not thought to be obtained from the site. Because the site-related contaminant body burden is low, most risks associated with salmon consumption were deemed not to be site-related.

Table B.3-27. Seafood species consumed by Tulalip and Suquamish adults and LDW species used to represent consumed species

Seafood Category	Members	Grams per Day			Rationale for Inclusion/Exclusion and Representative Species Present in the LDW
		Adult Tulalip RME[a]	Adult Tulalip CT[b]	Adult Suquamish[c]	
Anadromous fish	Salmon	96.4	14.9	183.5	Consumption rate not used in this HHRA. Although adult salmon are common in LDW, EPA guidance (EPA 2005a) recommends against including them in the LDW HHRA because of the migratory behavior of salmon (see Section B.2.1.2).
Pelagic fish	including cod and perch	8.1	1.3	56	Perch are common in the LDW.
Benthic/ demersal fish	halibut, sole, rockfish, snappers	7.5	1.2	29.1	English sole and starry flounder are common in certain areas of the LDW, but other benthic and demersal fish, such as rockfish, are not common (see Table B.2-2).
Shellfish	bivalves, snails, shrimp, crabs	81.9	12.5	498.4	Some marine shellfish species (crabs, clams, and mussels) are present in parts of the LDW, although natural habitat conditions, such as salinity, limit the abundance of some of these species.

[a] From Table B-1 of EPA (2007b), 95th percentile of the total seafood consumption rate from Puget Sound = 194 g/day.

[b] Provided by EPA (Hiltner 2007); 50th percentile of total seafood consumption rate from Puget Sound = 29.9 g/day.

[c] From Table B-2 of EPA (2007b); 95th percentile of the total seafood consumption rate from Puget Sound = 766.8 g/day.

CT – central tendency

EPA – US Environmental Protection Agency

HHRA – human health risk assessment

LDW – Lower Duwamish Waterway

RME – reasonable maximum exposure

The consumption of different types of shellfish within the shellfish seafood category for the adult tribal RME scenario based on Tulalip data and adult tribal scenario based on Suquamish data was specified by EPA in the application of their framework to the

LDW (EPA 2005a). The species-specific information was used together with concentration data for that species (where available) in the market basket estimate. The same methodology was applied to develop the adult tribal CT scenario based on Tulalip data. Briefly, average consumption rates (for consumers and non-consumers) of clams, mussels, and crabs were calculated and used by EPA to develop concentration weighting factors that could be applied to the shellfish seafood category. Using the adult tribal RME clam consumption rate based on Tulalip data as an example, average clam consumption was 46% of the sum of averages of other shellfish consumed (clams, mussels, and crabs). This percentage was applied to the adult tribal shellfish consumption rate (81.9 g/day, 95th percentile of Puget Sound shellfish consumption) to generate a clam consumption rate of 37.7 g/day for the adult tribal RME scenario based on Tulalip data. Similar procedures were used to develop consumption rates for the adult tribal CT scenario based on Tulalip data and for the adult tribal scenario based on Suquamish data. Table B.3-28 presents the concentration weighting factors (as percentages) for clams, mussels, and crabs and the calculated consumption of each within the framework of the adult tribal RME and CT scenarios based on Tulalip data and adult tribal scenario based on Suquamish data.

The shellfish consumption rate was fractionated to develop ingestion rate-weighted concentrations when data on multiple species were available for the shellfish market basket fraction. Rates for individual shellfish market basket components should not be used outside this context. For example, if risks associated with consumption of a particular resource, such as crabs, were of interest, development of a 95th percentile consumer only crab consumption rate would be appropriate (i.e., the crab consumption rate provided here is part of a market basket representing the 95th percentile of total seafood consumption, but does not represent the 95th percentile of crab consumption).

Table B.3-28. Adult tribal consumption of shellfish (crabs, clams, and mussels) based on Tulalip and Suquamish data

Shellfish Type	Percentage of Total Shellfish Consumption	RME or 95TH Percentile Scenario Consumption Rate (g/day)[a]	CT Scenario Consumption Rate (g/day)
Adult tribal RME based on Tulalip data[b]			
Crabs	53	43	6.6
Clams[c]	46	38	5.8
Mussels	1	0.8	0.1
Adult tribal based on Suquamish data[d]			
Crabs	11	54.8	na
Clams[c]	88	438.6	na
Mussels	1	5.0	na

[a] The adult consumption rate is the product of the percentage of total consumption and the overall shellfish consumption rate for the Tulalip and Suquamish Tribes, as applicable. The rate based on the Tulalip Tribes

study (Toy et al. 1996) was determined by EPA to be the most appropriate for application to the LDW (EPA 2006c) and is therefore defined as the adult tribal RME scenario. The scenario based on Suquamish data is provided for estimation of upper bound risks and is not designated as an RME scenario.

[b] Tulalip Tribes 95th percentile total Puget Sound shellfish consumption = 81.9 g/day, consumption percentages from Table B-1of EPA (2007b). The Tulalip Tribes CT scenario for total Puget Sound seafood consumption was based on an ingestion rate of 29.9 g/day (Hiltner 2007).

[c] Includes Manila/littleneck clams, horse clams, butter clams, cockles, oysters, and scallops (EPA 2005a).

[d] Suquamish Tribe 95th percentile total Puget Sound shellfish consumption = 498.4 g/day, consumption percentages from Table B-2 of EPA (2007b).

CT – central tendency

EPA – US Environmental Protection Agency

na – not applicable

RME – reasonable maximum exposure

The EPA tribal seafood consumption framework did not provide specific guidance on the portions of seafood consumed (e.g., whole body vs. filleted fish) within a specific seafood category. Quantification of these portions allows refinement of risk estimates and reduction of uncertainty. For pelagic fish, clams, and mussels, only whole-body data were available (whole body, including the siphon but not the shell for mussels and clams) so it was not possible to consider the different types of tissue consumed for these seafood categories. For benthic fish from the LDW, both whole-body and fillet chemical concentration data were available. Similarly, for LDW crab, chemical concentration data for edible meat (i.e., muscle tissue) and estimates of whole-body chemical concentration data (based on edible meat and hepatopancreas) were also available. Information on the relative percentage of consumption of these seafood categories is available from the seafood consumptions surveys of the Tulalip Tribes (Toy et al. 1996) and the Suquamish Tribe (2000). The percentages for the tissue categories and mean consumption rates for whole-body crabs and whole-body benthic fish were used to calculate the consumption rates for each of the seafood tissue categories, as presented in Table B.3-29.

Table B.3-29. Portions of benthic fish and crab consumed – adult tribal RME and CT scenarios based on Tulalip data and adult tribal scenario based on Suquamish data

Seafood Category	Percentage of Consumption	RME Scenario or 95TH Percentile Consumption Rate (g/day)[a]	CT Scenario Consumption Rate (g/day)
Adult tribal RME scenario based on Tulalip data			
Crab, edible meat	76[b]	33	5.0
Crab, whole body	24[b]	10	1.6
Benthic fish, fillet	100[c]	7.5	1.2
Benthic fish, whole body	0[c]	0	0.0

Seafood Category	Percentage of Consumption	RME Scenario or 95TH Percentile Consumption Rate (g/day)[a]	CT Scenario Consumption Rate (g/day)
Adult tribal scenario based on Suquamish data			
Crab, edible meat	76[d]	41.6	na
Crab, whole body	24[d]	13.2	na
Benthic fish, fillet	89[d]	25.9	na
Benthic fish, whole body	11[d]	3.2	na

[a] Product of percentage of consumption and the consumption rate for total crab or benthic fish, from EPA framework (EPA 2005a); see Tables B.3-27 and B.3-28 of this document. The rate based on the Tulalip Tribes study (Toy et al. 1996) was determined by EPA to be the most appropriate for application to the LDW (EPA 2006c) and is therefore defined as the adult tribal RME scenario. The scenario based on Suquamish data is provided for estimation of upper-bound risks and is not designated as an RME scenario.

[b] Portions of crab consumed were not reported for Tulalip Tribes (Toy et al. 1996); values from the Suquamish Tribe (Suquamish Tribe 2000) were used as surrogates.

[c] No Tulalip Tribe respondents reported consumption of benthic whole-body fish (Toy et al. 1996).

[d] Values from the Suquamish Tribe (Suquamish Tribe 2000).

CT – central tendency

EPA – US Environmental Protection Agency

na – not applicable

RME – reasonable maximum exposure

B.3.4.1.2 Child tribal seafood consumption based on Tulalip data

EPA noted in their initial framework guidance document for selecting and using tribal fish and shellfish consumption rates for risk-based decisions (EPA 2007b) that child-specific rates appropriate for use in the framework are not available from the two Puget Sound studies (Toy et al. 1996; Suquamish Tribe 2000). The two consumption studies included adult-reported child seafood consumption for children under 5 years of age (Tulalip study, n = 21) and under 6 years of age (Suquamish study, n = 31). As discussed previously, the Tulalip Tribes study (Toy et al. 1996) was considered most relevant for the LDW. Thus, the child tribal exposure scenarios were developed based on data from the Tulalip Tribes consumption study. EPA specified that the total consumption rate for the child tribal RME scenario based on Tulalip data should be equal to 40% of the adult tribal RME consumption rate based on Tulalip data (EPA 2006c). The rationale provided by EPA (2007a) included concerns about the small number of children surveyed in the Tulalip Tribes study (i.e., low sample size) and the relatively low consumption rates reported as compared to other regional tribal fish and seafood consumption studies (CRITFC 1994; Toy et al. 1996) and national fish consumption studies (EPA 2002c). The 40% ratio is based on a comparison of child and adult fish and seafood consumption data from regional and national studies (EPA 2006c, 2007a). A child tribal CT scenario based on Tulalip data was also developed with a total seafood consumption rate equal to 40% of the adult tribal CT total seafood consumption rate based on Tulalip data (Hiltner 2007).

The limitations in sample size for estimating childhood consumption rates also limit these data for use in estimating the seafood categories consumed by children. Therefore, as agreed upon between the LDWG and EPA, the same percentages for consumption of the different seafood categories and portions used for the adult tribal scenario based on Tulalip data (Tables B.3-27 through B.3-29) were used for the child tribal scenarios (i.e., adult tribal RME and CT consumption rates based on Tulalip data for each seafood category and portion were multiplied by 40% to estimate child tribal RME and CT consumption rates based on Tulalip data) (Table B.3-30). Thus, no child-specific data from the Tulalip study, other than body weight, was used for the development of the child tribal exposure scenarios based on Tulalip data (Tables B.3-9 and B.3-10) (Toy et al. 1996). As with the adult tribal seafood consumption scenarios based on Tulalip data, consumption of anadromous fish was not included for LDW child tribal exposures and risk estimates based on Tulalip data (EPA 2005a), which consider only the consumption of resident seafood organisms. The total non-anadromous seafood consumed in the tribal child scenario based on Tulalip data was 38.6 g/day and 6.0 g/day for the RME and CT scenarios, respectively.

Seafood consumption rates based on the 95th percentile of seafood consumption for children reported in the Tulalip Tribes study (Toy et al. 1996) and associated risk estimates for consumption of resident LDW seafood are presented in the uncertainty analysis (Section B.6.1.2.3). Risk estimates for a child tribal scenario based on Suquamish data are also presented in the uncertainty analysis (Section B.6.1.2.3).

Table B.3-30. Rates of child tribal (RME and CT) seafood consumption based on Tulalip data associated with different seafood categories

Seafood Category	RME Scenario Consumption Rate (g/day)[a]	CT Scenario Consumption Rate (g/day)[b]
Anadromous fish[c]	38.6	6.0
Pelagic fish	3.2	0.52
Benthic fish, fillet	3.0	0.48
Benthic fish, whole body	0	0
Crab, edible meat	13.2	2.0
Crab, whole body	4.2	0.64
Clams[d]	15.1	2.3
Mussels	0.33	0.040

[a] Total consumption rate = 77.6 g/day. Total consumption rate and consumption rates for seafood categories calculated as 40% of the adult tribal RME consumption rates based on Tulalip data (Tables B.3-27 through B.3-29).

[b] Total consumption rate = 12 g/day. Total consumption rate and consumption rates for seafood categories calculated as 40% of the adult tribal CT consumption rates based on Tulalip data (Tables B.3-27 through B.3-29).

[c] Consumption rate not used in this HHRA.

[d] Includes Manila/littleneck clams, horse clams, butter clams, cockles, oysters, and scallops (EPA 2005a).

CT – central tendency

RME – reasonable maximum exposure

B.3.4.1.3 Adult API seafood consumption rates

A specific scenario was also developed for adult API consumption of LDW seafood. The API populations studied by EPA (1999a) may consume fish and shellfish collected from the LDW, but the survey did not include geographic distinctions to determine the fishing frequency in the LDW compared to other areas in King County over which the survey was based. However, information collected by WDFW enforcement personnel (Frame 2001) indicate that individuals of API ethnicity are more commonly encountered engaging in non-commercial fishing in the LDW than any other ethnic group. Several Puget Sound seafood consumption studies have documented a substantial number of API fishing in urban embayments (Landolt et al. 1985; McCallum 1985; Landolt et al. 1987). Although there is uncertainty regarding the degree of seafood consumption by any group within the LDW, this HHRA provides an estimate for the API population; this population may consume more seafood than does the general public.

The EPA study included 202 adult men and women from 20 different ethnic groups (Cambodian, Chinese, Filipino, Hmong, Japanese, Korean, Laotian, Mien, Samoan, and Vietnamese) (EPA 1999a). As in the adult tribal consumption rates based on Tulalip data, EPA provided guidance on the application of data from this study for deriving fish and shellfish consumption rates for risk assessment (Kissinger 2005). An approach similar to that used for the development of tribal rates was used for API consumption rate development. The raw data were used to estimate the 95th percentile of consumption by individuals reporting consumption of seafood caught in King County.

Unlike the tribal studies, however, where each individual respondent was weighted equally, the respondents in the API study were weighted to reflect their ethnic group's population in King County relative to their representation in the consumption study. For example, 20 of the study participants were Cambodian, representing 10% of the survey respondents (20/202). However, Cambodians make up only 3.91% of the total King County population of the 10 ethnic groups included in the study (EPA 1999a). Thus, Cambodians were over-represented in the survey relative to the populations of the other nine API groups in King County. To account for this over-representation, consumption data from each Cambodian respondent was weighted specifically to adjust for this difference (Kissinger 2005). The same was done for each respondent based on their ethnicity and the representation of their ethnicity in the study relative to the representation of their ethnicity in the King County API population.

In EPA's 2005 reanalysis of the 1999 API data, only data for individuals consuming seafood from King County were included; weights based on all participants in the survey were not developed. Weighting factors for King County consumers for various ethnic groups were a function of the percentage of that ethnic group as determined in the census and the number of individuals in that ethnic group that consumed seafood from King County. For example, the weighting factor for Cambodians was derived based on the fact that 11 out of 20 Cambodians consumed seafood harvested in King County, that the percentage of Cambodians in the 2000 US census for King County was 3.91%, and that there were 99 King County seafood consumers in the 1999 API study. The 95th percentile ingestion rate was developed from the consumer-only dataset of weighted ingestion rates.

The data were also adjusted to account for the fact that some shellfish consumption was reported on a cooked-weight basis, rather than on a raw-weight basis. Consumption of the following shellfish was recorded in terms of cooked weight: butter clams, cockles, crabs, geoducks, horse clams, *Macoma* clams, Manila/little neck clams, moon snails, and mussels (EPA 1999a). Consumption of soft-shell clams (*Mya arenaria*) was not recorded; it should be noted that soft-shell clams are the dominant clam present in the LDW. Two revised estimates of average (consumer and non-consumer) raw shellfish consumption were made by EPA, using 25% and 50% cooking loss correction factors for those shellfish species for which consumption was reported on a cooked-weight basis. The average of these two estimates was provided by EPA (Kissinger 2006a).[19] This approach for adjusting cooked weight is described in detail in the EPA guidance document for developing API consumption rates (Kissinger 2005). The recommended 95th percentile of total King County API seafood consumption in that document was 57.1 g/day (n=99, demographically weighted).

To apportion the total seafood consumption rate of 57.1 g/day into the different seafood categories, EPA calculated demographically weighted mean ingestion rates for each seafood category for individuals who consumed some seafood caught in King County. The demographically weighted mean ingestion rates were then used to derive the percentage of consumption of each seafood category (Table B.3-31). These percentages were then applied to the total consumption rate (57.1 g/day) to derive consumption rates for each seafood category (Table B.3-29). Anadromous fish were not included in the exposure scenario because of the lack of linkage between chemicals in LDW sediments and those found in adult salmon tissues, per EPA recommendation (EPA 2005a). To estimate the CT consumption rate for the API scenario, the 50th percentile of total King County API consumption (5.8 g/day) (Kissinger 2005) was multiplied by the percentage of consumption for the various seafood categories. Total non-anadromous seafood consumption for the API scenarios was 51.5 g/day and 5.3 g/day for the RME and CT scenarios, respectively.

[19] This calculation required access to the information beyond what was provided in the publicly available report (EPA 1999a).

Table B.3-31. Development of percentages and rates of adult API RME and CT seafood consumption associated with different seafood categories

Seafood Category	Percentage of Consumption[a]	RME Scenario Consumption Rate (g/day)[b]	CT Scenario Consumption Rate (g/day)[b]
Anadromous fish[c]	9.6	5.5	0.56
Pelagic fish	8.6	4.9[d]	0.5
Benthic fish	4.2	2.4[d]	0.24
Shellfish	77.5	44.2[d]	4.6

[a] Calculated from average consumption rates by seafood category for consumers of King County species as provided by EPA (Kissinger 2006a).

[b] For the RME scenario, the 95th percentile of total King County API seafood consumption, 57.1 g/day, (Kissinger 2005) was multiplied by the percentage of consumption for the various seafood categories. For the CT scenario, the 50th percentile of total King County API consumption,5.8 g/day (Kissinger 2005), was multiplied by the percentage of consumption for the various seafood categories.

[c] Consumption rate not used in this HHRA.

[d] Freshwater fish make up 8.3% of API seafood consumption. As requested by EPA, freshwater fish were apportioned into benthic fish, pelagic fish, and shellfish categories according to the respective consumption rates for those types of fish (EPA 2006c). This apportionment assumes that API consumers who catch and consume freshwater fish outside the LDW would instead catch and consume more marine species inside the LDW.

API – Asian and Pacific Islander

CT – central tendency

EPA – US Environmental Protection Agency

HHRA – human health risk assessment

RME – reasonable maximum exposure

To calculate the consumption of mussels, crabs, and clams for the API scenario, the same general approach was used as for the tribal consumption calculations. The average demographically weighted consumption of clams, mussels, and crabs for the API consumers of these shellfish species self-harvested only from King County (n = 99) was provided by EPA (Kissinger 2006a) and used to calculate the percentage of each shellfish type consumed (Table B.3-32) (Kissinger 2006a). This weighting factor was used with the estimate of the 95th percentile of King County API shellfish consumption (44.3 g/day, Table B.3-29) to calculate the consumption of clams, mussels, and crabs. As with the tribal consumption estimate, the crab consumption rates were apportioned among crab whole body and edible meat, and the benthic fish consumption rates were apportioned among benthic fish fillet and whole body (Table B.3-33) based on the reported consumption of these seafood tissue categories by API consumers.[20] This information was provided by EPA as demographically weighted average percentages of crab whole-body and crab edible-meat consumption by API members consuming at least some King County seafood (n = 96; 3 individuals did not

[20] Because of the low sample size, both self-harvesters and non-self-harvesters were used to estimate portions of crab and benthic fish consumed.

consume any crab) (Kissinger 2007a). Similarly, EPA provided the average demographically weighted percentages of whole-body versus fillet consumption by API members consuming at least some King County seafood (n = 99) (Kissinger 2007a). This latter information was used to apportion benthic fish consumption into benthic whole body and benthic fillet consumption.

Table B.3-32. API RME and CT consumption of shellfish (crabs, clams, and mussels)

Shellfish Type	Percentage of Total Shellfish Consumption[a]	RME Scenario Consumption Rate (g/day)[b,c]	CT Scenario Consumption Rate (g/day)[b,c]
Crabs	24.0	10.6	1.1
Clams[d]	65.6	29.0	3.0
Mussels	10.4	4.6	0.47

[a] Calculated from average consumption rates provided by EPA for API consumers of King County species (Kissinger 2006b).

[b] Product of percentage of total shellfish consumption (for each shellfish type)and total shellfish consumption (Table B.3-31).

[c] Consumption includes freshwater fish.

[d] Includes Manila/littleneck clams, horse clams, butter clams, cockles, oysters, and scallops.

API – Asian and Pacific Islander

CT – central tendency

EPA – US Environmental Protection Agency

RME – reasonable maximum exposure

Table B.3-33. Portions of benthic fish and crab consumed – adult API RME and CT scenarios

Seafood Category	Percentage of Consumption[a]	RME Scenario Consumption Rate (g/day)[b,c]	CT Scenario Consumption Rate (g/day)[b,c]
Crab, edible meat	53.3	5.7	0.59
Crab, whole body	46.7	4.9	0.51
Benthic fish, fillet	82.3	2.0	0.20
Benthic fish, whole body	17.7	0.39	0.04

[a] As provided by EPA for crab or fish (Kissinger 2007a) for API consumers of King County species.

[b] Percentage of consumption multiplied by total crab consumption (Table B.3-32) or total benthic fish consumption (Table B.3-31).

[c] Consumption includes freshwater fish.

API – Asian and Pacific Islander

CT – central tendency

EPA – US Environmental Protection Agency

RME – reasonable maximum exposure

B.3.4.1.4 Adult one-meal-per-month seafood consumption rates

Consumption rates for recreationally caught fish are not available for the LDW. Although there have been some creel studies conducted in the LDW area (Landolt et al. 1985; McCallum 1985), there has not been a comprehensive recreational fish consumption study for the LDW site or nearby areas of similar quality as the recent tribal studies (Toy et al. 1996; Suquamish Tribe 2000) and API studies (EPA 1999a). Recreational fishing is known to occur on the LDW, but the magnitude is uncertain. It is expected that current recreational consumption is likely to be low and potentially suppressed because of public awareness of chemical contamination in the LDW and WSDOH seafood consumption advisories for the LDW (WSDOH 2005).

In an effort to provide information that would allow site users to evaluate the risks associated with seafood consumption, four hypothetical scenarios were developed. To evaluate risks associated with consumption of various resources independently (i.e., in addition to the market basket approach applied for the tribal seafood consumption evaluation), the consumption of different seafood categories was evaluated independently for benthic fish (fillets), pelagic fish, clams, and crabs (edible meat). Each scenario assumed that consumption would average approximately one meal (227 g, per EPA (2000d) guidance) per month of a given seafood category, which equates to 7.5 g/day. Totaling the risks from each of these four scenarios provides an estimate of risk associated with four meals per month, one of each seafood category, although data to support this quantity and pattern of recreational consumption for current or future use are lacking. The one-meal-per-month seafood consumption scenario and the associated risk estimates are intended to serve as a tool for risk communication and are not intended to directly reflect actual recreational seafood consumption because these rates are highly uncertain and may currently be suppressed as a result of consumption advisories. The one-meal-per-month scenarios provide a basis for individuals to evaluate their own exposure using a method that is readily scaled to various seafood consumption levels. For example, if someone eats two meals per month of LDW crab and one meal per month of LDW pelagic fish, he or she could multiply the one-meal-per-month crab risk estimate by two and add the product to the one-meal-per-month pelagic fish risk estimate to approximate the risk associated with his or her own LDW seafood consumption.

As with the tribal and API scenarios and based on EPA recommendation, consumption of adult salmon from the LDW was excluded from the HHRA (EPA 2005a). Thus, although salmon have been identified as the most commonly sought species for recreational fishers in the LDW (King County 1999b), bioaccumulative chemical concentrations in adult salmon are believed to be largely attributable to uptake during their migrations far beyond the LDW, and thus most of the risks associated with consumption of adult salmon are not related to LDW sediments. Therefore, the adult one-meal-per-month exposure scenarios derived here do not address risks from the consumption of adult salmon from the LDW.

B.3.4.2 Sediment exposure parameters

Sediment exposure scenarios were developed in Section B.3.2 for clamming, netfishing, and beach play. All scenarios include exposures from dermal contact and incidental ingestion of sediment. Most of the exposure parameters relative to these exposure routes are provided in Tables B.3-15 to B.3-26. Two parameters that warrant additional discussion, dermal adherence factor and dermal absorption factor, are discussed below.

B.3.4.2.1 Dermal adherence factor

The potential for sediment to adhere to skin has not been well characterized. Data for adherence factors (AF) for marine sediments, such as those found in the LDW, are extremely limited. A range of adherence factors exist for various soil conditions, including wet soils. Kissel et al. (1996) showed that soil adherence typically increases with increasing moisture content. Although current EPA (2004d) guidelines address the increase in soil adherence factors associated with moisture present in soil or sediment, more recent research suggests that the actual marine sediment adherence factors may be higher than those derived by EPA for wet soil (Shoaf et al. 2005a, b). The level of adherence directly affects dermal exposure estimates. As sediment loading increases, the fraction of chemical that adheres to the skin and is available to be absorbed will remain constant until all of the skin is covered by a thin layer of soil (known as the mono-layer) (Duff and Kissel 1996). Once this mono-layer threshold is crossed, the fraction of chemical that can be absorbed will decrease, inasmuch as not all of the soil is in constant, direct contact with skin. Both the amount of soil required to form the mono-layer and the associated adherence capability of the soil depend on grain size. Generally, larger particles will have a lower adherence factor than smaller particles. However, as previously mentioned, wet marine sediments are generally expected to have higher adherence capabilities than similarly composed dry soil. For the purposes of this risk assessment, the EPA-recommended (2004d) value of 0.2 mg/cm^2-event for children playing in wet soil as a high-activity event is used in all risk calculations for the RME scenario. A lower adherence factor (0.02 mg/cm^2-event) is used for the netfishing CT scenario, as recommended by EPA (2004d). However, the effect on risk estimates from using higher soil adherence factors for wet soil and sediment suggested by Kissel et al. (1996) and Shoaf et al. (2005a, b) is further investigated in the uncertainty analysis (Section B.6).

B.3.4.2.2 Dermal absorption fraction

The dermal absorption fraction (ABS) refers to the fraction of the chemical in sediment applied to the skin surface that is absorbed into the bloodstream. Many studies have focused on this topic, but there is considerable uncertainty regarding chemical-specific values (EPA 1992a). EPA (2004d) has developed supplemental guidance for dermal risk assessment that provides ABS values for most of the organic COPCs identified in Table B.3-3, but provides ABS values for only two metal COPCs, arsenic and cadmium

(Table B.3-34). The guidance document states that speciation of inorganic substances is crucial to estimating dermal absorption and data are insufficient to derive default values for other inorganic substances. Older EPA guidance (EPA 2001b) on dermal absorption provided a general value of 0.01 for all metals, reflecting a generally low dermal absorption of metals. Because specific absorption values are not provided, the dermal absorption pathway was not evaluated quantitatively for metals without dermal absorption fractions. This approach is suggested in EPA (2004d), with values supplied in Exhibit 3-4 of that document. The potential health risks from dermal exposure to these metals is evaluated further in the uncertainty analysis, and includes a quantitative risk evaluation based on assumed absorption factors (Section B.6).

Table B.3-34. Dermal absorption fractions

CHEMICAL	ABS (unitless)	ORAL ABSORPTION ADJUSTMENT[a]
2,3,7,8-TCDD TEQ	0.03	none
4,6-Dinitro-o-cresol[b]	0.1	none
Aluminum	None	none
Antimony	None	RfD × 0.15
Arsenic	0.03	none
Barium	none	RfD × 0.07
Benzidine[b]	0.1	none
Bis(2-chloroethyl) ether[b]	0.1	none
Cadmium	0.001	RfD × 0.025 (diet and solids)
cPAHs	0.13	none
Chromium	none	RfD × 0.025
Copper	none	none
Total DDTs	0.03	none
Dieldrin[c]	0.1	none
Dioxin/furan TEQ	0.03	none
Iron	none	none
Lead	none	none
Manganese	none	RfD × 0.04
Mercury	none	RfD × 0.07
Molybdenum	none	none
n-Nitrosodimethylamine[b]	0.1	none
n-Nitroso-di-n-propylamine[b]	0.1	none
Total PCBs	0.14	none
PCB TEQ	0.14	none
Silver	none	RfD × 0.04
Thallium	none	none

CHEMICAL	ABS (unitless)	ORAL ABSORPTION ADJUSTMENT[a]
Toxaphene[c]	0.1	none
Vanadium	none	RfD × 0.026
Zinc	none	none

Source: RAGS Part E (EPA 2004d)

[a] The oral adjustment values are presented in Exhibit 4-1 of EPA (2004d).

[b] The ABS value for semivolatile organic compounds is 0.1, as recommended in EPA (2004d).

[c] The ABS value for these organochlorine pesticides is the default value for semivolatile organic compounds, as recommended in EPA (2004d).

ABS – dermal absorption fraction

cPAH – carcinogenic polycyclic aromatic hydrocarbon

EPA – US Environmental Protection Agency

PCB – polychlorinated biphenyl

RfD – reference dose (see Section B.4)

TCDD – tetrachlorodibenzo-*p*-dioxin

TEQ – toxic equivalent

The toxicological benchmarks presented in Section B.3.0 are based on orally administered doses, which are not necessarily equivalent to dermally absorbed doses because of incomplete oral and or dermal absorption. Although a summary of gastrointestinal absorption data for many chemicals is provided in Exhibit 4-1 of EPA (2004d), data are not available for all chemicals evaluated. In the case of organic chemicals evaluated in this HHRA, absorption via the oral route is greater than 50%. In these instances, EPA (2004d) recommends that no conversion of the oral toxicity value is needed. Thus, for this HHRA, a gastrointestinal absorption factor of 1 was used for organic chemicals (i.e., oral toxicological benchmarks were applied without modification).

Both reference doses (RfDs) and SLs become more potent when based on an absorbed, rather than ingested, dose. The oral absorption adjustment (see Table B.3-34) is intended to reflect the internal dose resulting in the observed effect to be consistent with estimation of the dermally absorbed exposure estimate. The potential for increased stringency of toxicity factors becomes apparent when the mode of action of these oral adjustment values is considered because the oral adjustment for RfDs is RfD × gastrointestional (GI) fraction absorbed, while the adjustment for SLs is SF/GI fraction absorbed. Currently, EPA does not recommend an absorption adjustment for any chemical with a carcinogenic mode of action.

In this assessment, cadmium was the only chemical with both a recommended dermal absorption factor and reduced oral absorption; thus, an adjustment to the cadmium RfD was made for analysis of the dermal exposure route. For cadmium, the adjustment factor shown in Table B.3-34 was applied to the oral RfD. The lower RfD for the internal dose (i.e., absorbed dose) reflects the incomplete absorption of the COPC in the oral studies used to generate the RfD. For other metals lacking an ABS factor, no dermal absorption was assumed for the risk characterization; and therefore, the RfD adjustment was not relevant. Alternative dermal absorption assumptions for

metal exposure through direct sediment contact are explored in the uncertainty analysis.

B.3.4.3 Exposure point concentrations

An EPC was calculated for each seafood consumption category and sediment exposure area. Figure B.3-3 shows the methods used to estimate EPCs based on the number of detected concentrations present in a given dataset.

No. of Detected Values		Method for Selecting EPC
0	→	Use one-half of the maximum reporting limit.
1 – 5	→	Select the higher of one-half the maximum reporting limit OR the maximum detected value.
6 or more	→	Use ProUCL 4.0, indicating detected and undetected values.

Figure B.3-3. Flowchart showing method for selecting EPC

A flowchart for selecting or calculating the appropriate EPC value is provided in Figure B.3-3. The primary consideration in this step was the number of detected values available for a particular chemical and exposure area. The ProUCL software used for this analysis allows detected and undetected values to be indicated and creates interpolated values for non-detects based on the perceived distribution of the detected concentrations. This method is an improvement over older versions of ProUCL, which had no provision for handling undetected values. Once any necessary interpolation is performed, the software conducts an analysis of the data to determine the most appropriate UCL and makes a recommendation.

In addition to the ProUCL EPCs used in this HHRA, a subset of EPCs were recalculated using MTCA-conforming methods as directed by Ecology (2007). Attachment 2 presents alternative EPCs for 31 seafood consumption scenario EPCs and 23 sediment exposure EPCs. These MTCA-conforming EPCs are then compared to the EPCs calculated by ProUCL to examine the effect of using MTCA data evaluation protocols.

As stated previously, the rationale for selecting EPCs was based largely on the detection frequency for each chemical. The approach to calculating EPCs that is outlined above represents the outcome of the combined efforts of LDWG and EPA (EPA 2006d, 2007a) to arrive at a method of calculating EPCs that would use all

available data, be statistically defensible where possible, and adopt health-protective policies for deriving EPCs when statistical approaches for computing 95% UCLs were not available. When fewer than six detected concentrations were available, the higher of either the maximum detected concentration or one-half the maximum RL was selected as the EPC. This approach was selected because EPA and Ecology have concluded that 95% UCLs on the mean (95% UCL) calculated from datasets with very few detected concentrations are not reliable enough for deriving EPCs. Chemical contamination datasets are often positively skewed. For such positively skewed datasets, the true mean is greater than the 50th percentile and can be substantially greater when skewness is large. When the number of samples used to characterize an exposure area is very small (e.g., n < 6), there is a significant probability that the maximum result among those few samples will be less than the true mean. Even when using an approach that assigns the maximum sample result as an EPC value, there is still a risk of underestimating exposures. This uncertainty is unavoidable when only a few samples are available to characterize an exposure area. For this risk assessment, the vast majority of tissue, clamming, and netfishing EPCs were developed from datasets with six or more samples. However, small sample sizes affected EPC development for several of the beach play areas (which included the smallest geographic areas evaluated for direct contact exposure). Details of the affected datasets and associated uncertainty are discussed in Section B.6.1.1.11. Nevertheless, the above approach was agreed upon with EPA and is intended to be a reasonable approach to estimate the EPC for small datasets.

Certain classes of compounds are comprised of individual compounds that have similar chemical structures as well as a common mechanism of toxicity. Exposure and toxicity are assessed for these classes on a group rather than on an individual compound basis. These compound groups include co-planar PCBs, chlorinated dioxins/furans and cPAHs. The methods for calculating totals (including PCB TEQ, dioxin/furan TEQ, and cPAH totals) on a sample-by-sample basis were previously presented in Section B.2.2.4 and briefly summarized here. The sum of the products of the concentration of each coplanar PCB and its TEF is called the PCB TEQ and is calculated on a per sample basis. Similarly, the sum of the products of each coplanar dioxin and furan and its TEF is the called the dioxin/furan TEQ and is also calculated on a per sample basis. The sum of the products of the concentration of each cPAH and its PEF is considered the cPAH total and is calculated on a per sample basis. Once the TEQs for PCBs, dioxin/furans, and total cPAHs are calculated on a per sample bias, the methods for calculating the EPC for each of those is the same as that for other chemicals. The methods for calculating the EPCs for tissue and sediment are described in detail in the following subsections.

B.3.4.3.1 Tissue

Based on the seafood consumption surveys summarized in Section B.3.4.1, seven consumption categories based on seafood types were identified. Table B.3-35 lists the species for which tissue data are included to develop EPCs for each of the seven categories.

Table B.3-35. Seafood consumption categories for developing EPCs

SEAFOOD CATEGORY	LDW SPECIES INCLUDED FOR TISSUE DATA
Benthic fish, fillet	English sole, starry flounder
Benthic fish, whole body	English sole, starry flounder
Pelagic fish	shiner surfperch, striped perch, pile perch
Crab, edible meat	Dungeness crab, slender crab, red rock crab
Crab, whole body	Dungeness crab, slender crab
Clams	eastern soft-shell clam
Mussels	bay mussel

EPC – exposure point concentration

LDW – Lower Duwamish Waterway

EPC values were determined for each seafood category as described in Section B.3.4.3. Summary statistics, the distribution type, and the UCL on the mean for chemical concentrations in tissue for all seafood consumption categories are presented in Tables B.3-36 through B.3-38. The tissue EPCs are summarized in Table B.3-39.

Table B.3-36. Exposure point concentrations and summary statistics for metals and trace elements in tissue

Chemical	Consumption Category	No. Detected/Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Antimony	benthic fish, fillet	3/11	0.0059	0.0036 J	0.02	½ maximum RL	0.010[b]
	benthic fish, whole body	24/24	0.0058	0.0111 J	na	Student's-t UCL	0.0068
	clams	14/14	0.05	0.252	na	Approximate Gamma UCL	0.090
	crab, edible meat	17/21	0.003	0.0037 J	0.02	95% Chebyshev (Mean, sd) UCL	0.0095
	crab, whole body	19/21	0.0033	0.0068 JM	0.01	95% KM (BCA) UCL	0.0037
	mussels	0/22	0.0086	nd	0.02	one-half maximum RL	0.010
	pelagic fish, whole body	25/29	0.0041	0.0079 J	0.02	95% KM (BCA) UCL	0.0039
Arsenic (inorganic)[d]	benthic fish, fillet	6/8	0.004	0.006 J	0.003	95% Chebyshev, pooled ½ RL	0.0062[c]
	benthic fish, whole body	8/8	0.056	0.09	na	Student's-t UCL	0.073
	clams	8/8	1.24	3.27	na	Student's-t UCL	2.0
	crab, edible meat	6/6	0.023	0.03	na	95% Chebyshev (Mean, sd) UCL	0.042
	crab, whole body	6/6	0.075	0.123 M	na	Student's-t UCL	0.11
	pelagic fish, whole body	8/10	0.057	0.16	0.01	95% KM (t) UCL	0.088
Cadmium	benthic fish, fillet	1/11	0.0026	0.0013 J	0.0079	one-half maximum RL	0.0040[b]
	benthic fish, whole body	24/24	0.0075	0.0151	na	Approximate Gamma UCL	0.0085
	clams	14/14	0.10	0.148	na	Student's-t UCL	0.11
	crab, edible meat	21/21	0.023	0.0444	na	Student's-t UCL	0.027
	crab, whole body	21/21	0.16	0.2951 M	na	Student's-t UCL	0.19
	mussels	22/22	0.49	0.84	na	Student's-t UCL	0.55
	pelagic fish, whole body	27/29	0.014	0.024	0.0046	95% KM (t) UCL	0.016
Chromium	benthic fish, fillet	2/11	0.053	0.062	0.12	maximum detect	0.062[b]
	benthic fish, whole body	21/24	0.4	3.74	0.14	95% KM (Chebyshev) UCL	1.0
	clams	14/14	0.67	1.32	na	Student's-t UCL	0.79
	crab, edible meat	2/21	0.05	0.16	0.11	maximum detect	0.16[b]
	crab, whole body	6/21	0.04	0.136 M	0.05	95% KM (Percentile Bootstrap) UCL	0.094[b]
	mussels	21/22	0.16	0.35	0.05	95% KM (BCA) UCL	0.19
	pelagic fish, whole body	26/29	0.2	0.45	0.13	95% KM (BCA) UCL	0.20

Table B.3-36, cont. Exposure point concentrations and summary statistics for metals and trace elements in tissue

Chemical	Consumption Category	No. Detected/Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Copper	benthic fish, fillet	17/17	0.61	1.39	na	95% Chebyshev, pooled RL	1.6[c]
	benthic fish, whole body	24/24	1.73	3.47	na	95% Chebyshev (Mean, sd) UCL	2.6
	clams	14/14	5.34	7.3	na	Student's-t UCL	6.0
	crab, edible meat	21/21	7.5	16	na	Student's-t UCL	8.5
	crab, whole body	21/21	14	24 M	na	Student's-t UCL	16
	mussels	22/22	1.2	1.7 J	na	Student's-t UCL	1.3
	pelagic fish, whole body	29/29	1.6	2.2	na	Student's-t UCL	1.7
Lead[e]	benthic fish, fillet	8/17	0.044	0.14	0.03	95% KM (Percentile Bootstrap) UCL	0.081
	benthic fish, whole body	24/24	0.35	0.95	na	Student's-t UCL	0.42
	clams	14/14	2.0	6.4	na	Approximate Gamma UCL	3.1
	crab, edible meat	21/21	0.045	0.24 J	na	95% Chebyshev (Mean, sd) UCL	0.11
	crab, whole body	21/21	0.067	0.22 JM	na	95% Chebyshev (Mean, sd) UCL	0.12
	mussels	22/22	0.41	0.72	na	Approximate Gamma UCL	0.49
	pelagic fish, whole body	29/29	0.11	0.26	na	Student's-t UCL	0.13
Mercury	benthic fish, fillet	23/23	0.041	0.083	na	95% Chebyshev, pooled RL	0.058[c]
	benthic fish, whole body	24/24	0.01	0.027	na	Student's-t UCL	0.020
	clams	14/14	0.02	0.022	na	Student's-t UCL	0.020
	crab, edible meat	25/25	0.057	0.11	na	Approximate Gamma UCL	0.064
	crab, whole body	21/21	0.046	0.097 M	na	Student's-t UCL	0.052
	mussels	21/21	0.013	0.023	na	Approximate Gamma UCL	0.014
	pelagic fish, whole body	31/31	0.033	0.088	na	Student's-t UCL	0.039
Nickel	benthic fish, fillet	8/11	0.026	0.079 J	0.02	95% Chebyshev, pooled ½ RL	0.063[c]
	benthic fish, whole body	24/24	0.29	2.06	na	95% Chebyshev (Mean, sd) UCL	0.63
	clams	14/14	0.597	1.09	na	Approximate Gamma UCL	0.69
	crab, edible meat	21/21	0.048	0.12	na	Approximate Gamma UCL	0.055
	crab, whole body	21/21	0.068	0.16 JM	na	Student's-t UCL	0.079
	mussels	22/22	0.15	0.42 J	na	Approximate Gamma UCL	0.18
	pelagic fish, whole body	29/29	0.37	0.545 J	na	95% Chebyshev (Mean, sd) UCL	0.47

Table B.3-36, cont. Exposure point concentrations and summary statistics for metals and trace elements in tissue

Chemical	Consumption Category	No. Detected/Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Tributyltin as ion	benthic fish, fillet	10/17	0.002	0.0057	0.002	95% Chebyshev, pooled one-half RL	0.0033[c]
	benthic fish, whole body	18/23	0.0057	0.015	0.0021	95% KM (Percentile Bootstrap) UCL	0.0074
	clams	14/14	0.32	0.66	na	Student's-t UCL	0.40
	crab, edible meat	9/25	0.0062	0.082	0.002	97.5% KM (Chebyshev) UCL	0.030[b]
	crab, whole body	15/21	0.0099	0.075 M	0.0008	97.5% KM (Chebyshev) UCL	0.036
	mussels	22/22	0.023	0.037	na	Student's-t UCL	0.026
	pelagic fish, whole body	31/31	0.051	0.18	na	95% Chebyshev (Mean, sd) UCL	0.081
Vanadium	benthic fish, fillet	0/8	0.1	nd	0.25	one-half maximum RL	0.13
	benthic fish, whole body	24/24	0.4	0.5	na	Student's-t UCL	0.40
	clams	14/14	1.3	2.65	na	Approximate Gamma UCL	1.5
	crab, edible meat	0/19	0.09	nd	0.21	one-half maximum RL	0.11
	crab, whole body	12/19	0.1	0.20 JM	0.11	95% KM (Percentile Bootstrap) UCL	0.17
	mussels	8/8	0.15	0.26	na	Student's-t UCL	0.19
	pelagic fish, whole body	22/26	0.4	1.23	0.25	95% KM (Chebyshev) UCL	0.62
Zinc	benthic fish, fillet	11/11	6.8	8.89	na	95% Chebyshev, pooled RL	8.6[c]
	benthic fish, whole body	24/24	12.9	16.1	na	Student's-t UCL	13
	clams	14/14	23.7	32.3	na	Student's-t UCL	26
	crab, edible meat	21/21	34	39.3	na	Student's-t UCL	36
	crab, whole body	21/21	31	37.3 M	na	Student's-t UCL	32
	mussels	22/22	30	44	na	Student's-t UCL	32
	pelagic fish, whole body	29/29	21	28	na	Student's-t UCL	22

[a] EPC statistics were calculated assuming the RL for undetected chemicals. All samples are composites of multiple individuals.

[b] EPC was calculated based on 50% or more undetected values. The uncertainty associated with the risk calculation based on this EPC is likely to be higher than the uncertainty associated with risk estimates based on detected concentrations, as discussed in Section B.6.

[c] Because of the availability of historical data for English sole from RM 0 to RM 1.5, the EPC was calculated as a weighted mean, rather than a mean of all data combined. Means were first calculated for each of the four tissue sampling areas, with the historical data for English sole included in Area 1. The mean for the EPC derivation was then calculated as the arithmetic average of the four tissue sampling area means. The upper 95% confidence limit on that mean was estimated using Chebyshev's nonparametric method with a pooled standard deviation from the four areas. Thus, no assumption is made of equal means or variances across tissue sampling areas.

Table B.3-36, cont. Exposure point concentrations and summary statistics for metals and trace elements in tissue

[d] No mussel data were available for this chemical. When calculating the CDI and risk values, the proportion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[e] Lead is included in this table because it was designated a COPC. However, risks associated with lead are evaluated separately, in Section B.3.4.4, using the Integrated Exposure Uptake Biokinetic Model (IEUBK) for lead exposure in children and the Adult Lead Model (ALM) for characterizing risks associated with adult lead exposure. Mean tissue concentrations are used for lead modeling rather than UCL values.

J qualifier – Analyte was positively identified and detected; however, concentration is an estimated value because the result is less than the quantitation limit or QC criteria were not met.

M qualifier – value is a weighted mean, as described in Table B.2-4.

BCA – bias-corrected accelerated

EPC – exposure point concentration

KM – Kaplan Meier method for calculating a UCL

na – not applicable

nd – not detected

RL – reporting limit

sd – standard deviation

t (t-distribution) – statistical method used to estimate the mean for a normally distributed set of samples

UCL – upper confidence limit on the mean

ww – wet weight

Table B.3-37. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Aldrin	benthic fish, fillet	0/17	0.0016	nd	0.0072	one-half maximum RL	0.0036
	benthic fish, whole body	1/24	0.0042	0.0062 JN	0.010	maximum detect	0.0062[b]
	clams	3/14	0.00059	0.0010 JN	0.0010	maximum detect	0.0010[b]
	crab, edible meat	0/19	0.0017	nd	0.0072	one-half maximum RL	0.0036
	crab, whole body	0/19	0.0009	nd	0.0036	one-half maximum RL	0.0018
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	1/26	0.001	0.0014 JN	0.0072	one-half maximum RL	0.0036[b]
alpha-BHC	benthic fish, fillet	1/17	0.0016	0.00038 JN	0.0072	one-half maximum RL	0.0036[b]
	benthic fish, whole body	0/24	0.004	nd	0.010	one-half maximum RL	0.0050
	clams	1/14	0.00049	0.00035N	0.0010	one-half maximum RL	0.00050[b]
	crab, edible meat	0/19	0.0017	nd	0.0072	one-half maximum RL	0.0036
	crab, whole body	3/19	0.001	0.00116 JNM	0.0036	one-half maximum RL	0.0018[b]
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	2/26	0.0012	0.00046 JN	0.0072	one-half maximum RL	0.0036[b]
beta-BHC	benthic fish, fillet	2/17	0.0016	0.0022 JN	0.0072	one-half maximum RL	0.0036[b]
	benthic fish, whole body	9/24	0.0046	0.0084 JN	0.010	95% KM (Percentile Bootstrap) UCL	0.0051[b]
	clams	10/14	0.0009	0.0019 JN	0.0011	95% KM (t) UCL	0.0012
	crab, edible meat	0/19	0.0021	nd	0.0082	one-half maximum RL	0.0041
	crab, whole body	0/19	0.0011	nd	0.0036	one-half maximum RL	0.0018
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	16/26	0.0057	0.015 JN	0.0072	95% KM (Percentile Bootstrap) UCL	0.0078

Table B.3-37, cont. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Total chlordane	benthic fish, fillet	11/17	0.0086	0.028 JN	0.00050	95% Chebyshev, pooled ½ RL	0.020[c]
	benthic fish, whole body	24/24	0.033	0.059 JN	na	Student's-t UCL	0.039
	clams	14/14	0.0021	0.0093 JN	na	95% Chebyshev (Mean, Sd) UCL	0.0047
	crab, edible meat	19/19	0.004	0.0063 JN	na	Student's-t UCL	0.0045
	crab, whole body	19/19	0.016	0.026 JNM	na	Approximate Gamma UCL	0.019
	mussels[d]	0/11	0.0034	nd	0.0067	one-half maximum RL	0.0034
	pelagic fish, whole body	26/26	0.031	0.33 JN	na	95% Chebyshev (Mean, Sd) UCL	0.084
Total DDTs	benthic fish, fillet	15/17	0.037	0.103 JN	0.0020	95% Chebyshev, pooled ½ RL	0.084[c]
	benthic fish, whole body	24/24	0.17	0.28 JN	na	Student's-t UCL	0.19
	clams	14/14	0.012	0.033 JN	na	Approximate Gamma UCL	0.015
	crab, edible meat	19/19	0.021	0.032 JN	na	Student's-t UCL	0.023
	crab, whole body	19/19	0.09	0.15 JNM	na	Student's-t UCL	0.11
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	26/26	0.17	1.02 JN	na	Approximate Gamma UCL	0.24
Dieldrin	benthic fish, fillet	0/17	0.0019	nd	0.0072	one-half maximum RL	0.0036
	benthic fish, whole body	0/24	0.004	nd	0.010	one-half maximum RL	0.0050
	clams	4/14	0.0024	0.005 JN	0.024	one-half maximum RL	0.012[b]
	crab, edible meat	1/19	0.0019	0.0013 JN	0.0072	one-half maximum RL	0.0036[b]
	crab, whole body	1/19	0.0017	0.0032 JNM	0.0078	one-half maximum RL	0.0039[b]
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	0/26	0.0015	nd	0.0072	one-half maximum RL	0.0036
Endrin	benthic fish, fillet	1/17	0.0016	0.0010 JN	0.0072	one-half maximum RL	0.0036[b]
	benthic fish, whole body	4/24	0.0044	0.014 JN	0.012	maximum detect	0.014[b]
	clams	11/14	0.00041	0.0016 JN	0.0010	95% KM (BCA) UCL	0.00056
	crab, edible meat	0/19	0.0017	nd	0.0072	one-half maximum RL	0.0036
	crab, whole body	0/19	0.0011	nd	0.0040	one-half maximum RL	0.0020
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	10/26	0.005	0.040 JN	0.072	95% KM (% Bootstrap) UCL	0.0067[b]

Table B.3-37, cont. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Endrin aldehyde	benthic fish, fillet	1/17	0.0021	0.0081 JN	0.0072	maximum detect	0.0081[b]
	benthic fish, whole body	2/24	0.0042	0.0071 JN	0.010	maximum detect	0.0071[b]
	clams	2/14	0.00059	0.00049 JN	0.0036	one-half maximum RL	0.0018[b]
	crab, edible meat	5/19	0.002	0.0028 JN	0.0072	one-half maximum RL	0.0036[b]
	crab, whole body	5/19	0.0016	0.0033 JNM	0.0037	maximum detect	0.0033[b]
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	3/26	0.0048	0.078 JN	0.0072	maximum detect	0.078[b]
gamma-BHC	benthic fish, fillet	0/17	0.0016	nd	0.0072	one-half maximum RL	0.0036
	benthic fish, whole body	2/24	0.0041	0.0043 JN	0.010	one-half maximum RL	0.0050[b]
	clams	3/14	0.00068	0.0025 JN	0.0010	maximum detect	0.0025[b]
	crab, edible meat	1/19	0.0018	0.0040 JN	0.0072	maximum detect	0.0040[b]
	crab, whole body	1/19	0.0012	0.0074 JNM	0.0036	maximum detect	0.0074[b]
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	7/26	0.0014	0.0051 JN	0.0072	95% KM (Percentile Bootstrap) UCL	0.0020[b]
Heptachlor	benthic fish, fillet	0/17	0.0016	nd	0.0072	one-half maximum RL	0.0036
	benthic fish, whole body	2/24	0.0042	0.0068 JN	0.010	maximum detect	0.0068[b]
	clams	0/14	0.0005	nd	0.0010	one-half maximum RL	0.00050
	crab, edible meat	0/19	0.0017	nd	0.0072	one-half maximum RL	0.0036
	crab, whole body	0/19	0.0009	nd	0.0036	one-half maximum RL	0.0018
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	1/26	0.0017	0.0097 JN	0.0072	maximum detect	0.0097[b]
Heptachlor epoxide	benthic fish, fillet	0/17	0.0017	nd	0.0072	one-half maximum RL	0.0036
	benthic fish, whole body	13/24	0.016	0.045 JN	0.010	95% KM (Percentile Bootstrap) UCL	0.025
	clams	5/14	0.00081	0.0015 JN	0.001	maximum detect	0.0015[b]
	crab, edible meat	15/19	0.0019	0.0030 JN	0.0072	95% KM (t) UCL	0.0021
	crab, whole body	15/19	0.0032	0.0055 JNM	0.004	95% KM (Percentile Bootstrap) UCL	0.0040
	mussels	0/11	0.00065	nd	0.0013	one-half maximum RL	0.00065
	pelagic fish, whole body	5/26	0.0026	0.010 JN	0.0072	maximum detect	0.010[b]

Table B.3-37, cont. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
PCB TEQ[e]	benthic fish, fillet	8/8	8.80×10^{-6}	1.41×10^{-5}	na	Student's-t UCL	1.17×10^{-5}
	benthic fish, whole body	8/8	1.59×10^{-5}	2.47×10^{-5}	na	Student's-t UCL	2.04×10^{-5}
	clams	8/8	1.48×10^{-6}	5.65×10^{-6} J	na	Approximate Gamma UCL	3.16×10^{-6}
	crab, edible meat	8/8	2.00×10^{-6}	2.93×10^{-6}	na	Student's-t UCL	2.41×10^{-6}
	crab, whole body	6/6	7.70×10^{-6}	1.16×10^{-5} M	na	Student's-t UCL	9.68×10^{-6}
	pelagic fish, whole body	11/11	1.99×10^{-5}	7.30×10^{-5} J	na	Approximate Gamma UCL	3.37×10^{-5}
Total PCBs	benthic fish, fillet	33/33	0.7	2.0	na	95% Chebyshev, pooled RL	1.2[c]
	benthic fish, whole body	45/45	2.2	4.7	na	Approximate Gamma UCL	2.6
	clams	14/14	0.14	0.58 J	na	99% Chebyshev (Mean, sd) UCL	0.60
	crab, edible meat	26/29	0.17	0.39 J	0.020	95% KM (t) UCL	0.20
	crab, whole body	25/25	0.89	1.9 JM	na	95% H-UCL	1.1
	mussels	18/22	0.034	0.060	0.013	95% KM (Percentile Bootstrap) UCL	0.041
	pelagic fish, whole body	53/53	1.7	18.4 J	na	95% H-UCL	1.9
Toxaphene[f]	benthic fish, fillet	0/17	0.09	nd	0.44	one-half maximum RL	**0.22**
	benthic fish, whole body	0/24	0.51	nd	1.8	one-half maximum RL	**0.90**
	clams	0/14	0.041	nd	0.25	one-half maximum RL	**0.13**
	crab, edible meat	0/19	0.092	nd	0.36	one-half maximum RL	**0.18**
	crab, whole body	0/19	0.11	nd	0.47	one-half maximum RL	**0.24**
	mussels	0/11	0.0065	nd	0.013	one-half maximum RL	**0.0065**
	pelagic fish, whole body	0/26	0.37	nd	4.8	one-half maximum RL	**2.4**

a EPC statistics were calculated assuming the RL for undetected chemicals. All samples are composites of multiple individuals.

b EPC was calculated based on 50% or more undetected values. The uncertainty associated with the risk calculation based on this EPC is likely to be higher than the uncertainty associated with risk estimates based on detected concentrations, as discussed in Section B.6.

f Because of the availability of historical data for English sole from RM 0-1.5, the EPC was calculated as a weighted mean, rather than a mean of all data combined. Means were first calculated for each of the four tissue sampling areas, with the historical data for English sole included in Area 1. The mean for the EPC derivation was then calculated as the arithmetic average of the four tissue sampling area means. The upper 95% confidence limit on that mean was estimated using Chebyshev's nonparametric method with a pooled standard deviation from the four areas. Thus, no assumption is made of equal means or variances across tissue sampling areas.

d Reported as chlordane in mussel samples, not total chlordane.

Table B.3-37, cont. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in tissue

[e] No mussel data were available for this chemical. When calculating the CDI and risk values, seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[f] COPC was never detected; therefore, risks associated with the EPC, shown in **bold** are evaluated in the uncertainty analysis (Section B.6).

J qualifier – Analyte was positively identified and detected; however, concentration is an estimated value because the result is less than the quantitation limit or QC criteria were not met.

N qualifier – tentative identification of the chemical

M qualifier – value is a weighted mean, as described in Table B.2-4.

BHC – benzene hexachloride

EPC – exposure point concentration

H-UCL – UCL based on Land's H-statistic

KM – Kaplan Meier method for calculating a UCL

na – not applicable

nd – not detected

PCB – polychlorinated biphenyl

RL – reporting limit

sd – standard deviation

t (t-distribution) – statistical method used to estimate the mean for a normally distributed set of samples

TEQ – toxic equivalent

UCL – upper confidence limit

ww – wet weight

Table B.3-38. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
1,2-Diphenylhydrazine[b]	benthic fish, fillet	0/6	0.014	nd	0.053	one-half maximum RL	**0.027**
	crab, edible meat	0/2	0.027	nd	0.053	one-half maximum RL	**0.027**
	crab, whole body	0/2	0.016	nd	0.031	one-half maximum RL	**0.016**
	mussels	0/22	0.026	nd	0.053	one-half maximum RL	**0.027**
	pelagic fish, whole body	0/3	0.040	nd	0.080	one-half maximum RL	**0.040**
1,3-Dichlorobenzene[b]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.23	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.02	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/22	0.0080	nd	0.016	one-half maximum RL	**0.0080**
	pelagic fish, whole body	0/29	0.12	nd	0.58	one-half maximum RL	**0.29**
1,4-Dichlorobenzene[b]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.23	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/22	0.0080	nd	0.016	one-half maximum RL	**0.0080**
	pelagic fish, whole body	0/29	0.12	nd	0.58	one-half maximum RL	**0.29**

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
2,4,6-Trichlorophenol[b]	benthic fish, fillet	0/14	0.79	nd	2.9	one-half maximum RL	**1.5**
	benthic fish, whole body	0/24	1.0	nd	2.9	one-half maximum RL	**1.5**
	clams	0/14	0.039	nd	0.080	one-half maximum RL	**0.040**
	crab, edible meat	0/21	1.0	nd	2.9	one-half maximum RL	**1.5**
	crab, whole body	0/21	0.55	nd	1.5	one-half maximum RL	**0.75**
	mussels	0/22	0.055	nd	0.11	one-half maximum RL	**0.055**
	pelagic fish, whole body	0/29	0.79	nd	15	one-half maximum RL	**7.5**
2,4-Dichlorophenol[b]	benthic fish, fillet	0/14	0.32	nd	1.2	one-half maximum RL	**0.60**
	benthic fish, whole body	0/24	0.47	nd	1.2	one-half maximum RL	**0.60**
	clams	0/14	0.039	nd	0.080	one-half maximum RL	**0.040**
	crab, edible meat	0/21	0.47	nd	1.2	one-half maximum RL	**0.60**
	crab, whole body	0/21	0.24	nd	0.60	one-half maximum RL	**0.30**
	mussels	0/22	0.013	nd	0.027	one-half maximum RL	**0.014**
	pelagic fish, whole body	0/29	0.25	nd	1.2	one-half maximum RL	**0.60**
2,4-Dinitrophenol[b]	benthic fish, fillet	0/14	3.2	nd	12	one-half maximum RL	**6.0**
	benthic fish, whole body	0/24	4.7	nd	12	one-half maximum RL	**6.0**
	clams	0/14	0.39	nd	0.80	one-half maximum RL	**0.40**
	crab, edible meat	0/21	4.7	nd	12	one-half maximum RL	**6.0**
	crab, whole body	0/21	2.4	nd	6.0	one-half maximum RL	**3.0**
	mussels	0/22	0.026	nd	0.053	one-half maximum RL	**0.027**
	pelagic fish, whole body	0/14	3.8	nd	29	one-half maximum RL	**15**

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
2,4-Dinitrotoluene[b]	benthic fish, fillet	0/14	0.36	nd	1.5	one-half maximum RL	**0.75**
	benthic fish, whole body	0/24	0.55	nd	1.5	one-half maximum RL	**0.75**
	clams	0/14	0.039	nd	0.080	one-half maximum RL	**0.040**
	crab, edible meat	0/21	0.72	nd	2.9	one-half maximum RL	**1.5**
	crab, whole body	0/21	0.41	nd	1.5	one-half maximum RL	**0.75**
	mussels	0/22	0.0055	nd	0.011	one-half maximum RL	**0.0055**
	pelagic fish, whole body	0/29	0.86	nd	15	one-half maximum RL	**7.5**
2,6-Dinitrotoluene[b]	benthic fish, fillet	0/14	0.36	nd	1.5	one-half maximum RL	**0.75**
	benthic fish, whole body	0/24	0.51	nd	1.5	one-half maximum RL	**0.75**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.68	nd	2.9	one-half maximum RL	**1.5**
	crab, whole body	0/21	0.38	nd	1.5	one-half maximum RL	**0.75**
	mussels	0/22	0.0055	nd	0.011	one-half maximum RL	**0.0055**
	pelagic fish, whole body	0/29	0.73	nd	15	one-half maximum RL	**7.5**
2-Chlorophenol[b]	benthic fish, fillet	0/14	0.33	nd	1.2	one-half maximum RL	**0.60**
	benthic fish, whole body	0/24	0.47	nd	1.2	one-half maximum RL	**0.60**
	clams	0/14	0.039	nd	0.080	one-half maximum RL	**0.040**
	crab, edible meat	0/21	0.47	nd	1.2	one-half maximum RL	**0.60**
	crab, whole body	0/21	0.24	nd	0.60	one-half maximum RL	**0.30**
	mussels	0/22	0.026	nd	0.053	one-half maximum RL	**0.027**
	pelagic fish, whole body	0/29	0.25	nd	1.2	one-half maximum RL	**0.60**
3,3'-Dichloro-benzidine[b,c]	benthic fish, fillet	0/8	14	nd	29	one-half maximum RL	**15**
	benthic fish, whole body	0/24	12	nd	29	one-half maximum RL	**15**
	clams	0/14	1.0	nd	2.0	one-half maximum RL	**1.0**
	crab, edible meat	0/21	11	nd	29	one-half maximum RL	**15**
	crab, whole body	0/19	6.7	nd	15	one-half maximum RL	**7.5**
	pelagic fish, whole body	0/26	6.8	nd	29	one-half maximum RL	**15**

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
3-Nitroaniline[b]	benthic fish, fillet	0/14	1.6	nd	5.8	one-half maximum RL	**2.9**
	benthic fish, whole body	0/24	2.3	nd	5.8	one-half maximum RL	**2.9**
	clams	0/14	0.20	nd	0.40	one-half maximum RL	**0.20**
	crab, edible meat	0/19	2.5	nd	5.8	one-half maximum RL	**2.9**
	crab, whole body	0/19	1.3	nd	2.9	one-half maximum RL	**1.5**
	mussels	0/8	0.055	nd	0.11	one-half maximum RL	**0.055**
	pelagic fish, whole body	0/26	2.0	nd	29	one-half maximum RL	**15**
4,6-Dinitro-o-cresol[b]	benthic fillet	0/14	1.6	nd	5.8	one-half maximum RL	**2.9**
	benthic fish, whole body	0/24	2.3	nd	5.8	one-half maximum RL	**2.9**
	clams	0/14	0.20	nd	0.40	one-half maximum RL	**0.20**
	crab, edible meat	0/21	2.2	nd	5.8	one-half maximum RL	**2.9**
	crab, whole body	0/21	1.2	nd	2.9	one-half maximum RL	**1.5**
	mussels	0/22	0.026	nd	0.053	one-half maximum RL	**0.027**
	pelagic fish, whole body	0/29	2.0	nd	29	one-half maximum RL	**15**
4-Chloroaniline[b,c]	benthic fish, fillet	0/11	1.0	nd	2.9	one-half maximum RL	**1.5**
	benthic fish, whole body	0/24	1.2	nd	2.9	one-half maximum RL	**1.5**
	clams	0/14	0.10	nd	0.20	one-half maximum RL	**0.10**
	crab, edible meat	0/19	1.2	nd	2.9	one-half maximum RL	**1.5**
	crab, whole body	0/19	0.67	nd	1.5	one-half maximum RL	**0.75**
	pelagic fish, whole body	0/26	0.68	nd	2.9	one-half maximum RL	**1.5**
4-Methylphenol	benthic fish, fillet	0/14	0.32	nd	1.2	one-half maximum RL	0.60
	benthic fish, whole body	0/24	0.47	nd	1.2	one-half maximum RL	0.60
	clams	7/14	0.032	0.041 J	0.080	95% KM (t) UCL	0.031[d]
	crab, edible meat	0/21	0.47	nd	1.2	one-half maximum RL	0.60
	crab, whole body	0/21	0.24	nd	0.60	one-half maximum RL	0.30
	mussels	0/22	0.013	nd	0.027	one-half maximum RL	0.014
	pelagic fish, whole body	1/29	0.28	1.5	1.2	maximum detect	1.5 [d]

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
4-Nitroaniline[b]	benthic fish, fillet	0/14	0.84	nd	2.9	one-half maximum RL	**1.5**
	benthic fish, whole body	0/24	1.3	nd	2.9	one-half maximum RL	**1.5**
	clams	0/14	0.10	nd	0.20	one-half maximum RL	**0.10**
	crab, edible meat	0/19	1.7	nd	5.8	one-half maximum RL	**2.9**
	crab, whole body	0/19	0.94	nd	2.9	one-half maximum RL	**1.5**
	mussels	0/14	0.055	nd	0.11	one-half maximum RL	**0.055**
	pelagic fish, whole body	0/29	1.5	nd	29	one-half maximum RL	**15**
Aniline[b]	benthic fish, fillet	0/11	4.1	nd	12	one-half maximum RL	**6.0**
	benthic fish, whole body	0/24	4.7	nd	12	one-half maximum RL	**6.0**
	clams	0/14	0.39	nd	0.80	one-half maximum RL	**0.40**
	crab, edible meat	0/19	5.1	nd	12	one-half maximum RL	**6.0**
	crab, whole body	0/19	2.7	nd	6.0	one-half maximum RL	**3.0**
	mussels	0/19	0.027	nd	0.053	one-half maximum RL	**0.027**
	pelagic fish, whole body	0/26	2.7	nd	12	one-half maximum RL	**6.0**
Benzidine[b,c]	benthic fish, fillet	0/1	25	nd	50	one-half maximum RL	**25**
	benthic fish, whole body	0/11	26	nd	72	one-half maximum RL	**36**
	clams	0/14	2.5	nd	5.0	one-half maximum RL	**2.5**
	crab, edible meat	0/14	29	nd	72	one-half maximum RL	**36**
	crab, whole body	0/13	15	nd	36	one-half maximum RL	**18**
	pelagic fish, whole body	0/24	17	nd	72	one-half maximum RL	**36**
Bis(2-chloroethyl) ether[b]	benthic fish, fillet	0/14	0.17	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.25	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.32	nd	1.2	one-half maximum RL	**0.60**
	crab, whole body	0/21	0.17	nd	0.60	one-half maximum RL	**0.30**
	mussels	0/22	0.008	nd	0.016	one-half maximum RL	**0.0080**
	pelagic fish, whole body	0/29	0.16	nd	0.58	one-half maximum RL	**0.29**

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Bis(2-chloroisopropyl) ether[b]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.23	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/22	0.026	nd	0.053	one-half maximum RL	**0.027**
	pelagic fish, whole body	0/29	0.13	nd	0.58	one-half maximum RL	**0.29**
Bis(2-ethylhexyl) phthalate	benthic fish, fillet	2/14	0.19	1.3 J	0.13	maximum detect	1.3[d]
	benthic fish, whole body	0/24	0.67	nd	3.6	one-half maximum RL	1.8
	clams	10/14	0.14	0.22 J	0.50	95% KM (Percentile Bootstrap) UCL	0.13
	crab, edible meat	0/21	0.050	nd	0.26	one-half maximum RL	0.13
	crab, whole body	3/21	0.040	0.08 JM	0.10	maximum detect	0.080[d]
	mussels	2/22	0.017	0.19	0.016	maximum detect	0.19[d]
	pelagic fish, whole body	5/29	0.74	2.1 J	3.6	maximum detect	2.1[d]
Butyl benzyl phthalate	benthic fish, fillet	0/14	0.32	nd	1.2	one-half maximum RL	0.60
	benthic fish, whole body	3/24	0.47	0.65	1.2	maximum detect	0.65[d]
	clams	0/14	0.020	nd	0.040	one-half maximum RL	0.020
	crab, edible meat	0/21	0.42	nd	1.2	one-half maximum RL	0.60
	crab, whole body	10/21	0.70	1.7 M	0.60	95% KM (Percentile Bootstrap) UCL	1.2[d]
	mussels	0/22	0.0080	nd	0.016	one-half maximum RL	0.0080
	pelagic fish, whole body	7/26	0.50	1.4	1.2	95% KM (Percentile Bootstrap) UCL	0.84[d]
Carbazole	benthic fish, fillet	0/14	0.78	nd	2.9	one-half maximum RL	1.5
	benthic fish, whole body	0/24	1.2	nd	2.9	one-half maximum RL	1.5
	clams	0/14	0.10	nd	0.20	one-half maximum RL	0.10
	crab, edible meat	0/21	1.1	nd	2.9	one-half maximum RL	1.5
	crab, whole body	0/21	0.61	nd	1.5	one-half maximum RL	0.75
	mussels	0/22	0.013	nd	0.027	one-half maximum RL	0.014

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
	pelagic fish, whole body	2/29	1.2	14	2.9	maximum detect	14[d]
cPAHs[c,e]	benthic fish, fillet	5/8	0.00039	0.00064 J	0.00045	maximum detect	0.00064
	benthic fish, whole body	21/24	0.0014	0.0028 J	0.00045	95% KM (Chebyshev) UCL	0.0023
	clams	14/14	0.015	0.044	na	approximate Gamma UCL	0.020
	crab, edible meat	8/19	0.00044	0.00084 J	0.00065	95% KM (t) UCL	0.00065[d]
	crab, whole body	19/19	0.00075	0.0024 JM	na	95% modified-t UCL	0.00092
	pelagic fish, whole body	26/26	0.00078	0.0022	na	95% modified-t UCL	0.00095
Hexachlorobenzene	benthic fish, fillet	1/14	0.0055	0.0011 JN	0.018	one-half maximum RL	0.0090[d]
	benthic fish, whole body	4/24	0.0045	0.0066 JN	0.010	maximum detect	0.0066[d]
	clams	9/14	0.00066	0.0010 JN	0.0010	95% KM (t) UCL	0.00086
	crab, edible meat	1/21	0.0023	0.00093 JN	0.016	one-half maximum RL	0.0080[d]
	crab, whole body	4/21	0.0020	0.0060 JNM	0.0092	maximum detect	0.0060[d]
	mussels	0/22	0.0080	nd	0.016	one-half maximum RL	0.0080
	pelagic fish, whole body	1/29	0.0025	0.0041 JN	0.024	one-half maximum RL	0.012[d]
Hexachlorobutadiene[b]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.23	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/22	0.014	nd	0.027	one-half maximum RL	**0.014**
	pelagic fish, whole body	0/29	0.12	nd	0.58	one-half maximum RL	**0.29**

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Hexachloro-cyclopentadiene[b]	benthic fish, fillet	0/11	25	nd	72	one-half maximum RL	**36**
	benthic fish, whole body	0/24	31	nd	72	one-half maximum RL	**36**
	clams	0/14	2.5	nd	5.0	one-half maximum RL	**2.5**
	crab, edible meat	0/21	28	nd	72	one-half maximum RL	**36**
	crab, whole body	0/21	15	nd	36	one-half maximum RL	**18**
	mussels	0/22	0.014	nd	0.027	one-half maximum RL	**0.014**
	pelagic fish, whole body	0/29	25	nd	360	one-half maximum RL	**180**
Hexachloroethane[b]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.23	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/21	0.014	nd	0.027	one-half maximum RL	**0.014**
	pelagic fish, whole body	0/29	0.12	nd	0.58	one-half maximum RL	**0.29**
Nitrobenzene[b]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.23	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/22	0.014	nd	0.027	one-half maximum RL	**0.014**
	pelagic fish, whole body	0/29	0.12	nd	0.58	one-half maximum RL	**0.29**

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
n-Nitroso-dimethylamine[b]	benthic fish, fillet	0/14	0.94	nd	5.7	one-half maximum RL	**2.9**
	benthic fish, whole body	0/24	1.5	nd	5.8	one-half maximum RL	**2.9**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	2.5	nd	12	one-half maximum RL	**6.0**
	crab, whole body	0/21	1.4	nd	6.0	one-half maximum RL	**3.0**
	mussels	0/22	0.055	nd	0.11	one-half maximum RL	**0.055**
	pelagic fish, whole body	0/29	0.90	nd	5.8	one-half maximum RL	**2.9**
n-Nitroso-di-n-propylamine[b,f]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	1/24	0.23	0.27 JN	0.58	one-half maximum RL	0.29[d]
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/22	0.014	nd	0.027	one-half maximum RL	**0.014**
	pelagic fish, whole body	0/29	0.13	nd	0.58	one-half maximum RL	**0.29**
n-Nitroso-diphenylamine[b]	benthic fish, fillet	0/14	0.16	nd	0.58	one-half maximum RL	**0.29**
	benthic fish, whole body	0/24	0.23	nd	0.58	one-half maximum RL	**0.29**
	clams	0/14	0.020	nd	0.040	one-half maximum RL	**0.020**
	crab, edible meat	0/21	0.22	nd	0.58	one-half maximum RL	**0.29**
	crab, whole body	0/21	0.12	nd	0.29	one-half maximum RL	**0.15**
	mussels	0/22	0.014	nd	0.027	one-half maximum RL	**0.014**
	pelagic fish, whole body	0/29	0.18	nd	2.9	one-half maximum RL	**1.5**

Table B.3-38, cont. Exposure point concentrations and summary statistics for SVOCs in tissue

Chemical	Seafood Category	No. Detected/ Total No. of Samples	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Pentachlorophenol	benthic fish, fillet	0/14	0.32	nd	5.8	one-half maximum RL	2.9
	benthic fish, whole body	6/24	0.61	1.6 J	2.9	99% KM (Chebyshev) UCL	0.78[d]
	clams	0/14	0.20	nd	0.40	one-half maximum RL	0.20
	crab, edible meat	0/21	0.044	nd	0.58	one-half maximum RL	0.29
	crab, whole body	0/21	0.016	nd	0.20	one-half maximum RL	0.10
	mussels	0/22	0.014	nd	0.027	one-half maximum RL	0.014
	pelagic fish, whole body	2/29	0.63	2.4 J	2.9	maximum detect	2.4[d]

[a] EPC statistics were calculated assuming the RL for undetected chemicals. All samples are composites of multiple individuals.

[b] COPC was never detected; therefore, risks associated with the EPC shown in **bold** are evaluated in the uncertainty analysis (Section B.6).

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[d] EPC was calculated based on 50% or more undetected values. The uncertainty associated with the risk calculation based on this EPC is likely to be higher than the uncertainty associated with risk estimates based on detected concentrations, as discussed in Section B.6.

[e] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization section of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[f] One composite sample of whole-body English sole contained a detected concentration of 0.27 mg/kg ww. However, this result was qualified as JN (estimated concentration, tentative identification). Given the uncertain quantification for this single result (all other results were undetected), the risks for this chemical will be discussed in the uncertainty analysis (Section B.6).

J qualifier – Analyte was positively identified and detected; however, concentration is an estimated value because the result is less than the quantitation limit or QC criteria were not met.

M qualifier – value is a weighted mean, as described in Table B.2-4.

N qualifier – tentative identification of the chemical

cPAH – carcinogenic polycyclic aromatic hydrocarbon

EPC – exposure point concentration

KM – Kaplan Meier method for calculating a UCL

na – not applicable (no analytical data for that chemical in that seafood category)

nd – not detected

RL – reporting limit

SVOC – semivolatile organic compound

t (t-distribution) – statistical method used to calculate the mean for a normally distributed set of samples

UCL – upper confidence limit

ww – wet weight

Table B.3-39. Summary of tissue EPCs by seafood category

Chemical	EPC (mg/kg ww)[a]						
	Benthic Fish, Fillet	Benthic Fish, Whole Body	Clams	Crab, Edible Meat	Crab, Whole Body	Mussels	Pelagic Fish, Whole Body
Metals and trace elements							
Antimony	0.010[b]	0.0068	0.090	0.0095	0.0037	0.010	0.0039
Arsenic (inorganic)	0.0062[c]	0.073	2.0	0.042	0.11	na[d]	0.088
Cadmium	0.0040[b]	0.0085	0.11	0.027	0.19	0.55	0.016
Chromium	0.062[b]	1.0	0.79	0.16[b]	0.094[b]	0.19	0.20
Copper	1.6[c]	2.6	6.0	8.5	16	1.3	1.7
Lead[e]	0.081	0.42	3.1	0.11	0.12	0.49	0.13
Mercury	0.058[c]	0.020	0.020	0.064	0.052	0.014	0.039
Nickel	0.063[c]	0.63	0.69	0.055	0.079	0.18	0.47
Tributyltin as ion	0.0033[c]	0.0074	0.40	0.030[b]	0.036	0.026	0.081
Vanadium	0.13	0.40	1.5	0.11	0.17	0.19	0.62
Zinc	8.6[c]	13	26	36	32	32	22
Organochlorine pesticides and PCBs							
Aldrin	0.0036	0.0062[b]	0.0010[b]	0.0036	0.0018	0.00065	0.0036[b]
alpha-BHC	0.0036[b]	0.0050	0.00050[b]	0.0036	0.0018[b]	0.00065	0.0036[b]
beta-BHC	0.0036[b]	0.0051[b]	0.0012	0.0041	0.0018	0.00065	0.0078
Total chlordane	0.020[c]	0.039	0.0047	0.0045	0.019	0.0034[f]	0.084
Total DDTs	0.084[c]	0.19	0.015	0.023	0.11	0.00065	0.24
Dieldrin	0.0036	0.0050	0.012[b]	0.0036[b]	0.0039[b]	0.00065	0.0036
Endrin	0.0036[b]	0.014[b]	0.00056	0.0036	0.0020	0.00065	0.0067[b]
Endrin aldehyde	0.0081[b]	0.0071[b]	0.0018[b]	0.0036[b]	0.0033[b]	0.00065	0.078[b]
gamma-BHC	0.0036	0.0050[b]	0.0025[b]	0.0040[b]	0.0074[b]	0.00065	0.0020[b]
Heptachlor	0.0036	0.0068[b]	0.00050	0.0036	0.0018	0.00065	0.0097[b]
Heptachlor epoxide	0.0036	0.025	0.0015[b]	0.0021	0.0040	0.00065	0.010[b]
PCB TEQ	1.16×10^{-5}	2.04×10^{-5}	3.16×10^{-6}	2.41×10^{-6}	9.68×10^{-6}	na[d]	3.37×10^{-5}
Total PCBs	1.2[c]	2.6	0.60	0.20	1.1	0.041	1.9
Toxaphene[g]	0.22	0.90	0.13	0.18	0.24	0.0065	2.4
SVOCs							
1,2-Diphenylhydrazine[g]	0.027	na	na	0.027	0.016	0.027	0.040
1,3-Dichlorobenzene[g]	0.29	0.29	0.020	0.29	0.15	0.0080	0.29
1,4-Dichlorobenzene[g]	0.29	0.29	0.020	0.29	0.15	0.0080	0.29
2,4,6-Trichlorophenol[g]	1.5	1.5	0.040	1.5	0.75	0.055	7.5
2,4-Dichlorophenol[g]	0.60	0.60	0.040	0.60	0.30	0.014	0.60

Chemical	EPC (mg/kg ww)[a]						
	Benthic Fish, Fillet	Benthic Fish, Whole Body	Clams	Crab, Edible Meat	Crab, Whole Body	Mussels	Pelagic Fish, Whole Body
2,4-Dinitrophenol[g]	6.0	6.0	0.40	6.0	3.0	0.027	15
2,4-Dinitrotoluene[g]	0.75	0.75	0.040	1.5	0.75	0.0055	7.5
2,6-Dinitrotoluene[g]	0.75	0.75	0.020	1.5	0.75	0.0055	7.5
2-Chlorophenol[g]	0.60	0.60	0.040	0.60	0.30	0.027	0.60
3,3'-Dichlorobenzidine[g]	15	15	1.0	15	7.5	na[d]	15
3-Nitroaniline[g]	2.9	2.9	0.20	2.9	1.5	0.055	15
4,6-Dinitro-o-cresol[g]	2.9	2.9	0.20	2.9	1.5	0.027	15
4-Chloroaniline[g]	1.5	1.5	0.10	1.5	0.75	na[d]	1.5
4-Methylphenol	0.60	0.60	0.031[b]	0.60	0.30	0.014	1.5 [b]
4-Nitroaniline[g]	1.5	1.5	0.10	2.9	1.5	0.055	15
Aniline[g]	6.0	6.0	0.40	6.0	3.0	0.027	6.0
Benzidine[g]	25	36	2.5	36	18	na[d]	36
Bis(2-chloroethyl)ether[g]	0.29	0.29	0.020	0.60	0.30	0.0080	0.29
Bis(2-chloroisopropyl)ether[g]	0.29	0.29	0.020	0.29	0.15	0.027	0.29
Bis(2-ethylhexyl)phthalate	1.3[b]	1.8	0.13	0.13	0.080[b]	0.19[b]	2.1[b]
Butyl benzyl phthalate	0.60	0.65[b]	0.020	0.60	1.2[b]	0.0080	0.84[b]
Carbazole	1.5	1.5	0.10	1.5	0.75	0.014	14[b]
cPAHs[h]	0.015	0.0023	0.020	0.00065[b]	0.00092	na[d]	0.00095
Hexachlorobenzene	0.0090[b]	0.0066[b]	0.00086	0.0080[b]	0.0060[b]	0.0080[b]	0.012[b]
Hexachlorobutadiene[g]	0.29	0.29	0.020	0.29	0.15	0.014	0.29
Hexachlorocyclopentadiene[g]	36	36	2.5	36	18	0.014	180
Hexachloroethane[g]	0.29	0.29	0.020	0.29	0.15	0.014	0.29
Nitrobenzene[g]	0.29	0.29	0.020	0.29	0.15	0.014	0.29
n-Nitrosodimethylamine[g]	2.9	2.9	0.020	6.0	3.0	0.055	2.9
n-Nitroso-di-n-propylamine[g,i]	0.29	0.29 [b]	0.020	0.29	0.15	0.014	0.29
n-Nitrosodiphenylamine[g]	0.29	0.29	0.020	0.29	0.15	0.014	1.5
Pentachlorophenol	2.9	0.78[b]	0.20	0.29	0.10	0.014	2.4[b]

[a] EPC statistics were calculated according to data reduction procedures described in Section B.3.4.3.

[b] EPC was calculated based on 50% or more undetected values. The uncertainty associated with the risk calculation based on this EPC is likely to be higher than the uncertainty associated with risk estimates based on detected concentrations, as discussed in Section B.6.

[c] Because of the availability of historical data for English sole from RM 0-1.5, the EPC was calculated as a weighted mean, rather than a mean of all data combined. Means were first calculated for each of the four tissue sampling areas, with the historical data for English sole included in Area 1. The mean for the EPC derivation was then calculated as the arithmetic average of the four tissue sampling area means. The upper 95% confidence limit on that mean was estimated using Chebyshev's nonparametric method with a pooled standard deviation from the four areas. Thus, no assumption is made of equal means or variances across tissue sampling areas.

[d] No mussel data were available for this chemical. When calculating the CDI and risk values, seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[e] Lead is included in this table because it was designated a COPC. However, risks associated with lead are evaluated separately, in Section B.3.4.4, using the Integrated Exposure Uptake Biokinetic Model (IEUBK) for lead exposure in children and the Adult Lead Model (ALM) for characterizing risks associated with adult lead exposure. UCL values are presented in this summary table, however, mean tissue concentrations are used for lead modeling rather than UCL values. Thus, the values presented here are for informational purposes only.

[f] Reported as chlordane in mussel samples, not total chlordane.

[g] COPC was never detected; therefore, risks associated with the EPC shown in **bold** are evaluated in the uncertainty analysis (Section B.6).

[h] cPAH concentrations are given in terms of benzo(a) pyrene equivalents. Data used in the risk characterization section of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[i] One composite sample of whole-body English sole contained a detected concentration of 0.27 mg/kg ww. However, this result was qualified as JN (estimated concentration, tentative identification). Given the uncertain quantification for this single result (all other results were undetected), the risks for this chemical are discussed in the uncertainty analysis (Section B.6).

BHC – benzene hexachloride

CDI – chronic daily intake

COPC – chemical of potential concern

EPC – exposure point concentration

na – not applicable; no analytical data for that chemical in that seafood category

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

RL – reporting limit

SVOC – semivolatile organic compound

TEQ – toxic equivalent

ww – wet weight

EPCs for chemicals in consumption categories with all undetected values (i.e., never detected in that tissue) were set equal to one-half the maximum RL. Risk estimates for chemicals that were never detected in any tissue from the LDW, but had RLs greater than the screening level, are presented in the uncertainty analysis (Section B.6). For chemicals detected at least once, risks are presented in the risk characterization section (Section B.5). A summary of LDW tissue chemistry data is provided in Attachment 1 of the HHRA.

B.3.4.3.2 Sediment

As described in Section B.3.2, different exposure areas were defined for each of the direct sediment exposure scenarios. Table B.3-40 identifies those exposure areas and references the maps where those exposure areas are shown. A potential source of bias exists in the tribal clamming RME scenario (120 days per year) and tribal clamming 183-day-per-year scenario, which were designed to include those areas of the LDW that may be reached by boat as well as from shore. These scenarios, unlike the 7-day-per-year clamming scenario, addressed risks associated with sediment contact in regions of the waterway such as Boeing Plant 2, Slip 4, and T-117, which are known

areas of contamination and therefore have a higher sampling density than many other areas of the LDW. This unequal sampling density between the two tribal scenarios and the 7-day-per-year scenario should be noted. Futhermore, as noted in Section B.2.3.1.1, an EPC based on a simple arithmetic mean, in which all concentrations are given equal weight in the computation of the mean, may overestimate exposure because more highly contaminated areas have been sampled more intensively than less contaminated areas. This spatial bias is most acute for PCBs. An alternate method for calculating a spatially weighted EPC for PCBs is evaluated in Section B.6.1.1.12.

Table B.3-40. Exposure areas for direct sediment exposure scenarios

SCENARIO	EXPOSURE AREA	MAP
Netfishing	All subtidal and intertidal areas (RM 0 – RM 6.0)	Map B.2-1
Clamming	clamming 7 days per year only from shoreline (RM 0.1 – RM 4.9) tribal clamming (RME or 183 days per year) from shoreline or boat (RM 0.1 – RM 4.9)	Map B.3-2
Beach play RME	Area 1 (RM 0.1 – RM 0.3) Area 2 (RM 0.5 – RM 1.0) Area 3 (RM 0.5 – RM 1.0) Area 4 (RM 2.0 – RM 2.4) Area 5 (RM 2.5 – RM 3.4) Area 6 (RM 2.7 – RM 2.8) Area 7 (RM 4.2 – RM 4.6) Area 8 (RM 4.6 – RM 5.0)	Map B.3-1

RM – river mile

EPCs were determined for all COPCs for each dermal and incidental ingestion sediment exposure scenario using ProUCL 4.0, as described in Section B.3.4.3. Summary statistics, the distribution type, and the UCL on the mean for chemical concentrations in sediment for all dermal exposure scenarios are presented in Tables B.3-41 through B.3-43. The sediment EPCs are summarized in Table B.3-44.

Table B.3-41. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Aluminum	beach play RME, area 1	No samples in this area were analyzed for this chemical					
	beach play RME, area 2	No samples in this area were analyzed for this chemical					
	beach play RME, area 3	6/6	8,400	15,100	na	95% Students-t UCL	12,000
	beach play RME, area 4	3/3	16,000	21,000	na	maximum detect	21,000
	beach play RME, area 5	7/7	14,000	15,000	na	95% Students-t UCL	15,000
	beach play RME, area 6	No samples in this area were analyzed for this chemical					
	beach play RME, area 7	3/3	17,000	23,000	na	maximum detect	23,000
	beach play RME, area 8	5/5	15,000	17,000	na	maximum detect	17,000
	tribal clamming – RME and 183 days per year	107/107	17,000	110,000	na	95% Modified-t UCL	20,000
	clamming – 7 days per year	33/33	13,000	23,000	na	95% Students-t UCL	15,000
	netfishing	455/455	20,000	110,000	na	95% Modified-t UCL	19,000
Antimony	beach play RME, area 1	2/4	0.4	1.05 J	0.3	maximum detect	1.1[a]
	beach play RME, area 2	3/5	0.8	2.28 J	0.4	maximum detect	2.3
	beach play RME, area 3	2/9	2	5.2 J	4.2	maximum detect	5.2[a]
	beach play RME, area 4	5/10	2	6.0 J	10	maximum detect	6.0[a]
	beach play RME, area 5	3/22	2	5.0 J	10	maximum detect	5.0[a]
	beach play RME, area 6	0/1	0.2	nd	0.3	one-half maximum RL	0.15
	beach play RME, area 7	1/9	2	0.09 J	10	one-half maximum RL	5.0[a]
	beach play RME, area 8	2/11	3	7.0 J	10	maximum detect	7.0[a]
	tribal clamming – RME and 183 days per year	52/159	5	110 J	31	95% KM (Chebyshev) UCL	8.2[a]
	clamming – 7 days per year	24/89	2	7.0 J	10	95% KM (t) UCL	1.3[a]

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
	netfishing	139/553	4	122 J	31	95% KM (BCA) UCL	3.4[a]
Arsenic	beach play RME, area 1	4/4	6.5	14.9	na	maximum detect	15
	beach play RME, area 2	5/5	12.1	20.7	na	maximum detect	21
	beach play RME, area 3	6/9	8.5	18	6.6	95% KM (Percentile Bootstrap) UCL	13
	beach play RME, area 4	10/10	8.2	17.3	na	95% Students-t UCL	11
	beach play RME, area 5	22/22	8.1	11.8	na	95% Students-t UCL	8.9
	beach play RME, area 6	1/1	9.8	9.8	na	maximum detect	9.8
	beach play RME, area 7	9/9	8.9	14	na	95% Students-t UCL	11
	beach play RME, area 8	11/11	8.7	15.6	na	95% Students-t UCL	10
	tribal clamming – RME and 183 days per year	254/275	20	1,100	31	95% KM (BCA) UCL	27
	clamming –7 days per year	100/103	8.8	20.7	6.6	95% KM (Percentile Bootstrap) UCL	9.5
	netfishing	755/817	20	1,100	31	95% KM (BCA) UCL	21
Barium	beach play RME, area 1	No samples in this area were analyzed for this chemical					
	beach play RME, area 2	No samples in this area were analyzed for this chemical					
	beach play RME, area 3	5/5	31.4	75.5	na	maximum detect	76
	beach play RME, area 4	3/3	59	81	na	maximum detect	81
	beach play RME, area 5	7/7	39	44	na	95% Students-t UCL	42
	beach play RME, area 6	No samples in this area were analyzed for this chemical					
	beach play RME, area 7	3/3	53	72	na	maximum detect	72
	beach play RME, area 8	5/5	49	58	na	maximum detect	58
	tribal clamming – RME and 183 days per year	106/106	140	3,500	na	95% Chebyshev (Mean, sd) UCL	310
	clamming – 7 days year	32/32	46	89	na	95% Students-t UCL	52
	netfishing	418/418	130	7,400	na	95% Chebyshev (Mean, sd) UCL	230

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Cadmium	beach play RME, area 1	2/4	0.1	0.066	0.3	one-half maximum RL	0.15[a]
	beach play RME, area 2	4/5	0.4	1	0.3	maximum detect	1.0
	beach play RME, area 3	5/9	0.8	2	0.42	maximum detect	2.0
	beach play RME, area 4	6/10	0.5	2.0 J	0.3	95% KM (Percentile Bootstrap) UCL	0.94
	beach play RME, area 5	11/22	0.2	0.4	0.7	95% KM (t) UCL	0.2[a]
	beach play RME, area 6	0/1	0.2	nd	0.3	one-half maximum RL	0.15
	beach play RME, area 7	5/9	0.2	0.8	0.5	maximum detect	0.80
	beach play RME, area 8	5/11	0.1	0.18	0.4	one-half maximum RL	0.20[a]
	tribal clamming – RME and 183 days per year	179/268	2	120	2.5	95% KM (Chebyshev) UCL	4.8
	clamming – 7 days per year	65/103	0.4	2.2	0.7	95% KM (BCA) UCL	0.42
	netfishing	565/800	1	120	2.5	95% KM (BCA) UCL	1.5
Chromium	beach play RME, area 1	4/4	15.5	20.8	na	maximum detect	21
	beach play RME, area 2	5/5	27	48	na	maximum detect	48
	beach play RME, area 3	9/9	27	48.4	na	95% Students-t UCL	38
	beach play RME, area 4	10/10	34	122 J	na	95% Chebyshev (Mean, sd) UCL	82
	beach play RME, area 5	22/22	23	61 J	na	95% Modified-t UCL	28
	beach play RME, area 6	1/1	23.4	23.4	na	maximum detect	23
	beach play RME, area 7	9/9	23	28	na	95% Students-t UCL	25
	beach play RME, area 8	11/11	21	26	na	95% Students-t UCL	23
	tribal clamming – RME and 183 days per year	275/275	50	1,100 J	na	95% Chebyshev (Mean, sd) UCL	81
	clamming – 7 days per year	103/103	26	122 J	na	95% Modified-t UCL	28
	netfishing	814/814	40	1,100 J	na	95% Chebyshev (Mean, sd) UCL	51

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Copper	beach play RME, area 1	4/4	33.4	50.2	na	maximum detect	50
	beach play RME, area 2	5/5	80.5	172 J	na	maximum detect	170
	beach play RME, area 3	9/9	52	133	na	95% Students-t UCL	79
	beach play RME, area 4	10/10	47	117	na	95% Approximate Gamma UCL	74
	beach play RME, area 5	22/22	43	180	na	95% Chebyshev (Mean, sd) UCL	74
	beach play RME, area 6	1/1	35	35	na	maximum detect	35
	beach play RME, area 7	9/9	32	49.4	na	95% Students-t UCL	38
	beach play RME, area 8	11/11	29	46.7	na	95% Students-t UCL	34
	tribal clamming – RME and 183 days per year	275/275	200	12,000 J	na	95% Chebyshev (Mean, sd) UCL	450
	clamming – 7 days per year	103/103	44	180	na	95% H-UCL	49
	netfishing	817/817	100	12,000 J	na	95% Chebyshev (Mean, sd) UCL	200
Iron	beach play RME, area 1	No samples in this area were analyzed for this chemical					
	beach play RME, area 2	No samples in this area were analyzed for this chemical					
	beach play RME, area 3	6/6	13,000	22,100	na	95% Students-t UCL	17,000
	beach play RME, area 4	3/3	25,000	32,000	na	maximum detect	32,000
	beach play RME, area 5	7/7	23,000	29,000	na	95% Students-t UCL	26,000
	beach play RME, area 6	No samples in this area were analyzed for this chemical					
	beach play RME, area 7	3/3	26,000	31,000	na	maximum detect	31,000
	beach play RME, area 8	5/5	23,000	26,000	na	maximum detect	26,000
	tribal clamming – RME and 183 days per year	107/107	29,000	160,000	na	95% Modified-t UCL	33,000
	clamming – 7 days per year	33/33	21,000	46,000 J	na	95% Students-t UCL	24,000
	netfishing	453/453	30,000	160,000	na	95% Modified-t UCL	29,000

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Lead[b]	beach play RME, area 1	4/4	30	71	na	maximum detect	71
	beach play RME, area 2	5/5	150	400	na	maximum detect	400
	beach play RME, area 3	9/9	81	210 J	na	95% Students-t UCL	130
	beach play RME, area 4	10/10	100	615	na	95% Approximate Gamma UCL	300
	beach play RME, area 5	22/22	32	70 J	na	95% Approximate Gamma UCL	37
	beach play RME, area 6	1/1	24	24	na	maximum detect	24
	beach play RME, area 7	9/9	14	19	na	95% Students-t UCL	16
	beach play RME, area 8	11/11	24	95	na	95% Approximate Gamma UCL	37
	tribal clamming – RME and 183 days per year	275/275	200	23,000	na	97.5% Chebyshev (Mean, Sd) UCL	780
	clamming – 7 days per year	103/103	50	615	na	95% H-UCL	60
	netfishing	817/817	100	23,000	na	97.5% Chebyshev (Mean, Sd) UCL	300
Manganese	beach play RME, area 1	No samples in this area were analyzed for this chemical					
	beach play RME, area 2	No samples in this area were analyzed for this chemical					
	beach play RME, area 3	6/6	164	310	na	95% Students-t UCL	240
	beach play RME, area 4	3/3	240	280	na	maximum detect	280
	beach play RME, area 5	7/7	260	300	na	95% Students-t UCL	280
	beach play RME, area 6	No samples in this area were analyzed for this chemical					
	beach play RME, area 7	3/3	380	430	na	maximum detect	430
	beach play RME, area 8	5/5	430	780	na	maximum detect	780
	tribal clamming – RME and 183 days per year	107/107	440	3,300	na	95% Chebyshev (Mean, Sd) UCL	650
	clamming – 7 days per year	33/33	270	780	na	95% Approximate Gamma UCL	310
	netfishing	450/450	340	3,300	na	95% Modified-t UCL	360

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Mercury	beach play RME, area 1	3/4	0.08	0.17	0.05	maximum detect	0.17
	beach play RME, area 2	4/5	0.2	0.63	0.05	maximum detect	0.63
	beach play RME, area 3	4/8	0.1	0.31	0.03	maximum detect	0.31[a]
	beach play RME, area 4	8/10	0.5	2.46	0.07	95% KM (Chebyshev) UCL	1.7
	beach play RME, area 5	14/22	0.08	0.23	0.1	95% KM (Percentile Bootstrap) UCL	0.11
	beach play RME, area 6	1/1	0.09	0.09	na	maximum detect	0.090
	beach play RME, area 7	7/9	0.08	0.16	0.1	95% KM (t) UCL	0.11
	beach play RME, area 8	8/11	0.09	0.21	0.1	95% KM (t) UCL	0.12
	tribal clamming – RME and 183 days per year	218/272	0.2	4.6 J	0.1	95% KM (BCA) UCL	0.23
	clamming – 7 days per year	78/103	0.1	2.46	0.1	95% KM (BCA) UCL	0.20
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					
Molybdenum	beach play RME, area 1	4/4	0.9	1.8	na	maximum detect	1.8
	beach play RME, area 2	5/5	1	3	na	maximum detect	3.0
	beach play RME, area 3	3/7	2	5.8	2.8	maximum detect	5.8 [a]
	beach play RME, area 4	7/7	2	5.1	na	95% Approximate Gamma UCL	3.2
	beach play RME, area 5	15/15	2	4	na	95% Approximate Gamma UCL	2.0
	beach play RME, area 6	1/1	1.5	1.5	na	maximum detect	1.5
	beach play RME, area 7	6/6	1	1.4	na	95% Students-t UCL	1.3
	beach play RME, area 8	6/6	1	2	na	95% Students-t UCL	1.9
	tribal clamming – RME and 183 days per year	93/97	3	49	2.8	95% KM (BCA) UCL	3.8
	clamming – 7 days per year	56/60	1	5.8	2.8	95% KM (Chebyshev) UCL	2.1
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Silver	beach play RME, area 1	1/4	0.1	0.04	0.4	one-half maximum RL	0.20[a]
	beach play RME, area 2	2/5	0.2	0.094	1	one-half maximum RL	0.50[a]
	beach play RME, area 3	4/9	0.4	0.796	0.6	maximum detect	0.80[a]
	beach play RME, area 4	7/10	0.5	1.7	0.4	95% KM (BCA) UCL	0.79
	beach play RME, area 5	9/22	0.2	0.18	1	95% KM (t) UCL	0.15[a]
	beach play RME, area 6	0/1	0.2	nd	0.4	one-half maximum RL	0.20
	beach play RME, area 7	4/9	0.2	0.21	0.7	one-half maximum RL	0.35[a]
	beach play RME, area 8	6/11	0.2	0.13 J	0.6	95% KM (t) UCL	0.12
	tribal clamming – RME and 183 days per year	143/265	2	270	5	95% KM (Chebyshev) UCL	6.7
	clamming – 7 days per year	55/103	0.3	5.7	1	95% KM (Chebyshev) UCL	0.57
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					
Thallium	beach play RME, area 1	1/4	0.08	0.036	0.3	one-half maximum RL	0.15[a]
	beach play RME, area 2	2/5	0.1	0.054	0.4	one-half maximum RL	0.20[a]
	beach play RME, area 3	0/9	10	nd	34	one-half maximum RL	17
	beach play RME, area 4	6/10	0.1	0.11 J	0.3	95% KM (t) UCL	0.092
	beach play RME, area 5	9/22	0.1	0.07	0.4	95% KM (t) UCL	0.062[a]
	beach play RME, area 6	0/1	0.2	nd	0.3	one-half maximum RL	0.15
	beach play RME, area 7	4/9	0.1	0.08	0.5	one-half maximum RL	0.25[a]
	beach play RME, area 8	6/11	0.1	0.07	0.4	95% KM (t) UCL	0.060
	tribal clamming – RME and 183 days per year	83/190	3	30	34	97.5% KM (Chebyshev) UCL	3.6[a]
	clamming – 7 days per year	42/89	1	0.18	34	95% KM (t) UCL	0.071[a]
	netfishing	325/638	3	32 J	53	97.5% KM (Chebyshev) UCL	2.4

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

Chemical	Scenario	No. Detected/ Total No. of Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Vanadium	beach play RME, area 1	4/4	45.8	47.2	na	maximum detect	47
	beach play RME, area 2	5/5	46	66.7	na	maximum detect	67
	beach play RME, area 3	1/1	49.8	49.8	na	maximum detect	50
	beach play RME, area 4	10/10	51	71	na	95% Students-t UCL	57
	beach play RME, area 5	22/22	53	68.7	na	95% Students-t UCL	55
	beach play RME, area 6	1/1	48.7	48.7	na	maximum detect	49
	beach play RME, area 7	9/9	58	69	na	95% Students-t UCL	64
	beach play RME, area 8	11/11	53	65.4	na	95% Students-t UCL	57
	tribal clamming – RME and 183 days per year	192/192	55	87	na	95% Student's-t UCL	56
	clamming – 7 days per year	81/81	51	71	na	95% Students-t UCL	53
	netfishing	557/557	59	150	na	95% Student's-t UCL	60
Zinc	beach play RME, area 1	4/4	72.6	142 J	na	maximum detect	140
	beach play RME, area 2	5/5	234	435 J	na	maximum detect	440
	beach play RME, area 3	9/9	170	480 J	na	95% Approximate Gamma UCL	350
	beach play RME, area 4	10/10	140	417	na	95% Approximate Gamma UCL	240
	beach play RME, area 5	22/22	92	246 J	na	95% Modified-t UCL	110
	beach play RME, area 6	1/1	91.7	91.7	na	maximum detect	92
	beach play RME, area 7	9/9	73	99	na	95% Students-t UCL	84
	beach play RME, area 8	11/11	83	211	na	95% Modified t-UCL	110
	tribal clamming – RME and 183 days per year	275/275	270	9,700	na	95% Chebyshev (Mean, sd) UCL	480
	clamming – 7 days per year	103/103	110	480 J	na	95% Chebyshev (Mean, sd) UCL	140
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					

Table B.3-41, cont. Exposure point concentrations and summary statistics for metals and trace elements in sediment

[a] EPC was calculated based on 50% or more undetected values. The uncertainty associated with the risk calculation based on this EPC is likely to be higher than the uncertainty associated with risk estimates based on detected concentrations, as discussed in the uncertainty analysis (Section B.6).

[b] Lead is included in this table because it was designated a COPC. However, risks associated with lead are evaluated separately, in Section B.3.4.4, using the Integrated Exposure Uptake Biokinetic Model (IEUBK) for lead exposure in children and the Adult Lead Model (ALM) for characterizing risks associated with adult lead exposure. Mean sediment concentrations are used for lead modeling rather than UCL values.

EPC statistics were calculated assuming the RL for undetected values.

J qualifier – Analyte was positively identified and detected; however, the given concentration is an estimated value because the result is less than the quantitation limit or QC criteria were not met.

BCA – bias-corrected accelerated

COPC – chemical of potential concern

dw – dry weight

EPC – exposure point concentration

H-UCL – UCL based on Land's H-statistic

KM – Kaplan Meier method for calculating a UCL

na – not applicable

RBC – risk-based concentration

RL – reporting limit

sd – standard deviation

t (t-distribution) – statistical method used to calculate the mean for a normally distributed set of samples

UCL – upper confidence limit

Table B.3-42. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in sediment

Chemical	Scenario	No. Detected/ Total No. Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Total DDTs	beach play RME, area 1	2/3	0.0018	0.00090 JN	0.0074	one-half maximum RL	0.0037
	beach play RME, area 2	2/2	0.0097	0.0133 J	na	maximum detect	0.013
	beach play RME, area 3	2/6	0.0051	0.021	0.0036	maximum detect	0.021[a]
	beach play RME, area 4	5/8	0.095	0.17	0.80	one-half maximum RL	0.40
	beach play RME, area 5	2/6	0.0085	0.035 J	0.013	maximum detect	0.035[a]
	beach play RME, area 6	0/1	1.0×10^{-3}	nd	1.0×10^{-3}	one-half maximum RL	0.00050
	beach play RME, area 7	1/2	0.0019	0.0027 J	0.002	maximum detect	0.0027[a]
	beach play RME, area 8	1/2	0.0046	0.0082 J	0.002	maximum detect	0.0082[a]
	tribal clamming – RME and 183 days per year	34/63	0.065	2.9 J	0.80	97.5% KM (Chebyshev) UCL	0.35
	clamming – 7 days per year	20/40	0.023	0.17	0.80	95% KM (BCA) UCL	0.022[a]
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					
Dieldrin	beach play RME, area 1	0/3	0.00075	nd	0.0025	one-half maximum RL	0.0013
	beach play RME, area 2	0/2	0.00049	nd	0.001	one-half maximum RL	0.00050
	beach play RME, area 3	1/6	0.0025	0.01	0.0036	maximum detect	0.010[a]
	beach play RME, area 4	2/8	0.006	0.017 J	0.034	maximum detect	0.017[a]
	beach play RME, area 5	0/6	0.0011	nd	0.0052	one-half maximum RL	0.0026
	beach play RME, area 6	0/1	0.001	nd	0.002	one-half maximum RL	0.0010
	beach play RME, area 7	0/2	0.00075	nd	0.002	one-half maximum RL	0.0010
	beach play RME, area 8	1/2	0.0017	0.0023	0.002	maximum detect	0.0023[a]
	tribal clamming – RME and 183 days per year	6/63	0.0071	0.28	0.091	95% KM (t) UCL	0.013[a]
	clamming – 7 days per year	4/40	0.0022	0.017 J	0.034	maximum detect	0.017[a]

Table B.3-42, cont. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in sediment

Chemical	Scenario	No. Detected/ Total No. Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
	netfishing	9/197	0.0035	0.28	0.091	95% KM (t) UCL	0.0045[a]
PCB TEQ	beach play RME, area 1	1/1	9.08×10^{-8}	9.08×10^{-8} J	na	maximum detect	9.08×10^{-8}
	beach play RME, area 2	1/1	6.69×10^{-6}	6.69×10^{-6} J	na	maximum detect	6.69×10^{-6}
	beach play RME, area 3	No samples in this area were analyzed for this chemical					
	beach play RME, area 4	4/4	5.15×10^{-5}	2.04×10^{-4}	na	maximum detect	2.04×10^{-4}
	beach play RME, area 5	3/3	1.09×10^{-6}	2.51×10^{-6} J	na	maximum detect	2.51×10^{-6}
	beach play RME, area 6	1/1	5.37×10^{-6}	5.37×10^{-6} J	na	maximum detect	5.37×10^{-6}
	beach play RME, area 7	2/2	4.71×10^{-7}	5.65×10^{-7}	na	maximum detect	5.65×10^{-7}
	beach play RME, area 8	2/2	1.19×10^{-6}	1.89×10^{-6}	na	maximum detect	1.89×10^{-6}
	tribal clamming – RME and 183 days per year	30/30	6.73×10^{-5}	1.38×10^{-3}	na	97.5% Chebyshev (MVUE) UCL	1.84×10^{-4}
	clamming – 7 days per year	18/18	1.44×10^{-5}	2.04×10^{-4}	na	99% Chebyshev (MVUE) UCL	4.19×10^{-5}
	netfishing	48/48	4.59×10^{-5}	1.38×10^{-3}	na	95% Chebyshev (MVUE) UCL	7.18×10^{-5}
Total PCBs	beach play RME, area 1	3/5	0.029	0.119	0.020	maximum detect	0.12
	beach play RME, area 2	6/7	0.1	0.29	0.020	95% KM (t) UCL	0.18
	beach play RME, area 3	11/14	0.089	0.42 J	0.017	95% KM (Chebyshev) UCL	0.24
	beach play RME, area 4	12/12	2.8	23	na	95% Adjusted Gamma UCL	11
	beach play RME, area 5	31/32	0.1	0.66	0.020	95% KM (Chebyshev) UCL	0.19
	beach play RME, area 6	2/2	0.54	0.97	na	maximum detect	0.97
	beach play RME, area 7	10/14	0.063	0.34	0.040	97.5% KM (Chebyshev) UCL	0.23
	beach play RME, area 8	12/18	0.056	0.52	0.040	97.5% KM (Chebyshev) UCL	0.23
	tribal clamming – RME and 183 days per year	415/440	2.0	110	0.040	97.5% KM (Chebyshev) UCL	4.0[b]
	clamming – 7 days per year	142/161	0.43	23	0.040	97.5% KM (Chebyshev) UCL	1.5
	netfishing	1205/1291	1.0	220	0.05	97.5% KM (Chebyshev) UCL	2.5[b]

Table B.3-42, cont. Exposure point concentrations and summary statistics for organochlorine pesticides and PCBs in sediment

Chemical	Scenario	No. Detected/ Total No. Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
Toxaphene	beach play RME, area 1	0/3	0.033	nd	0.098	one-half maximum RL	0.049
	beach play RME, area 2	0/2	0.025	nd	0.05	one-half maximum RL	0.025
	beach play RME, area 3	0/6	0.023	nd	0.18	one-half maximum RL	0.090
	beach play RME, area 4	0/8	0.2	nd	1.7	one-half maximum RL	0.85
	beach play RME, area 5	1/6	0.089	0.34 J	0.099	maximum detect	0.34[a]
	beach play RME, area 6	0/1	0.049	nd	0.097	one-half maximum RL	0.049
	beach play RME, area 7	0/2	0.037	nd	0.098	one-half maximum RL	0.049
	beach play RME, area 8	0/2	0.037	nd	0.097	one-half maximum RL	0.049
	tribal clamming – RME and 183 days per year	2/61	0.27	6.3 J	4.3	maximum detect	6.3[a]
	clamming – 7 days per year	1/40	0.077	0.34 J	1.7	one-half maximum RL	0.85[a]
	netfishing	2/195	0.11	6.3 J	4.3	maximum detect	6.3[a]

[a] EPC was calculated based on 50% or more undetected values. The uncertainty associated with the risk calculation based on this EPC is likely to be higher than the uncertainty associated with risk estimates based on detected concentrations, as discussed in Section B.6.

[b] The EPCs shown for total PCBs (and all other COPCs) are based on arithmetic UCLs. A spatially weighted UCL on the SWAC may more accurately reflect the true exposure. A method for calculating a spatially weighted UCL on the SWAC for PCBs in presented in Section B.6.1.1.12. SWAC UCL EPCs, based on UCLs on the spatially weighted means, are 0.47 mg/kg dw for netfishing and 0.90 mg/kg dw for tribal clamming. The SWAC UCL EPCs are presented here for comparison purposes but were not used for risk estimates in the risk characterization section.

EPC statistics were calculated assuming the RL for undetected values.

J qualifier – Analyte was positively identified and detected; however, concentration is an estimated value because the result is less than the quantitation limit or QC criteria were not met.

BCA – bias-corrected accelerated

COPC – chemical of potential concern

dw – dry weight

EPC – exposure point concentration

KM – Kaplan Meier method for calculating a UCL

MVUE – minimum-variance unbiased eliminator

na – not applicable

nd – not detected

PCB – polychlorinated biphenyl

RBC – risk based concentration

RL – reporting limit

SWAC – spatially weighted average concentration

t (t-distribution) – statistical method used to calculate the mean for a normally distributed set of samples

TEQ – toxic equivalent

UCL – upper confidence limit

Table B.3-43. Exposure point concentrations and summary statistics for SVOCs and dioxins/furans in sediment

Chemical	Scenario	No Detected/ Total No. Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
4,6-Dinitro-o-cresol [a]	beach play RME, area 1	0/4	0.086	nd	0.2	one-half maximum RL	**0.10**
	beach play RME, area 2	0/5	0.14	nd	0.5	one-half maximum RL	**0.25**
	beach play RME, area 3	0/9	0.044	nd	0.2	one-half maximum RL	**0.10**
	beach play RME, area 4	0/10	0.27	nd	2.9	one-half maximum RL	**1.5**
	beach play RME, area 5	0/22	0.18	nd	0.99	one-half maximum RL	**0.50**
	beach play RME, area 6	0/1	0.29	nd	0.58	one-half maximum RL	**0.29**
	beach play RME, area 7	0/9	0.12	nd	0.59	one-half maximum RL	**0.30**
	beach play RME, area 8	0/11	0.12	nd	0.59	one-half maximum RL	**0.30**
	tribal clamming – RME and 183 days per year	0/230	0.2	nd	2.9	one-half maximum RL	**1.5**
	clamming – 7 days per year	0/92	0.15	nd	2.9	one-half maximum RL	**1.5**
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					
Benzidine [a]	beach play RME, area 1	No samples in this area were analyzed for this chemical					
	beach play RME, area 2	No samples in this area were analyzed for this chemical					
	beach play RME, area 3	0/1	0.55	nd	1.1	one-half maximum RL	**0.55**
	beach play RME, area 4	No samples in this area were analyzed for this chemical					
	beach play RME, area 5	No samples in this area were analyzed for this chemical					
	beach play RME, area 6	No samples in this area were analyzed for this chemical					
	beach play RME, area 7	No samples in this area were analyzed for this chemical					
	beach play RME, area 8	No samples in this area were analyzed for this chemical					
	tribal clamming – RME and 183 days per year	0/2	0.63	nd	1.4	one-half maximum RL	**0.70**
	clamming – 7 days per	0/1	0.55	nd	1.1	one-half maximum RL	**0.55**

Table B.3-43, cont. Exposure point concentrations and summary statistics for SVOCs and dioxins/furans in sediment

Chemical	Scenario	No Detected/ Total No. Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
	year						
	netfishing	0/7	0.54	nd	1.7	one-half maximum RL	**0.85**
bis(2-chloroethyl) ether [a]	beach play RME, area 1	0/4	0.0086	nd	0.02	one-half maximum RL	**0.010**
	beach play RME, area 2	0/5	0.014	nd	0.05	one-half maximum RL	**0.025**
	beach play RME, area 3	0/9	0.011	nd	0.027	one-half maximum RL	**0.014**
	beach play RME, area 4	0/10	0.03	nd	0.29	one-half maximum RL	**0.15**
	beach play RME, area 5	0/22	0.021	nd	0.099	one-half maximum RL	**0.050**
	beach play RME, area 6	0/1	0.029	nd	0.058	one-half maximum RL	**0.029**
	beach play RME, area 7	0/9	0.015	nd	0.059	one-half maximum RL	**0.030**
	beach play RME, area 8	0/11	0.016	nd	0.059	one-half maximum RL	**0.030**
	tribal clamming – RME and 183 days per year	0/230	0.04	nd	0.29	one-half maximum RL	**0.15**
	clamming – 7 days per year	0/92	0.019	nd	0.29	one-half maximum RL	**0.15**
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					
cPAHs[b]	beach play RME, area 1	3/4	0.33	1.20	0.0091	maximum detect	1.2
	beach play RME, area 2	5/5	0.7	3.0	na	maximum detect	3.0
	beach play RME, area 3	7/9	0.66	2.9 J	0.036	95% KM (Chebyshev) UCL	2.1
	beach play RME, area 4	9/10	0.2	0.75 J	0.0091	97.5% KM (Chebyshev) UCL	0.73
	beach play RME, area 5	22/22	0.21	1.00 J	na	95% Chebyshev (MVUE) UCL	0.41
	beach play RME, area 6	1/1	0.44	0.44	na	maximum detect	0.44
	beach play RME, area 7	8/9	0.077	0.15	0.0094	95% KM (t) UCL	0.11
	beach play RME, area 8	11/11	0.23	0.62	na	95% Students-t UCL	0.32
	tribal clamming – RME and 183 days per year	255/264	0.5	11	0.11	95% KM (Chebyshev) UCL	0.77
	clamming – 7 days per	97/103	0.27	3	0.036	95% KM (Chebyshev) UCL	0.48

Table B.3-43, cont. Exposure point concentrations and summary statistics for SVOCs and dioxins/furans in sediment

Chemical	Scenario	No Detected/ Total No. Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
	year						
	netfishing	749/793	0.5	11	0.13	95% KM (Chebyshev) UCL	0.57
Dioxin/furan TEQ	beach play RME, area 1	No samples in this area were analyzed for this chemical					
	beach play RME, area 2	No samples in this area were analyzed for this chemical					
	beach play RME, area 3	No samples in this area were analyzed for this chemical					
	beach play RME, area 4	1/1	4.12×10^{-4}	4.12×10^{-4} J	na	maximum detect	4.12×10^{-4}
	beach play RME, area 5	1/1	2.20×10^{-6}	2.20×10^{-6} J	na	maximum detect	2.20×10^{-6}
	beach play RME, area 6	No samples in this area were analyzed for this chemical					
	beach play RME, area 7	1/1	1.70×10^{-6}	1.70×10^{-6}	na	maximum detect	1.70×10^{-6}
	beach play RME, area 8	No samples in this area were analyzed for this chemical					
	tribal clamming – RME and 183 days per year	11/11	3.40×10^{-4}	2.10×10^{-3} J	na	95% Adjusted Gamma UCL	1.42×10^{-3}
	clamming – 7 days per year	6/6	7.50×10^{-5}	4.12×10^{-4} J	na	99% Chebyshev (MVUE) UCL	3.65×10^{-4}
	netfishing	43/43	1.00×10^{-4}	2.10×10^{-3} J	na	99% Chebyshev (Mean, sd) UCL	6.10×10^{-4}
n-Nitroso-dimethylamine [a]	beach play RME, area 1	0/4	0.025	nd	0.1	one-half maximum RL	**0.050**
	beach play RME, area 2	0/5	0.031	nd	0.13	one-half maximum RL	**0.065**
	beach play RME, area 3	0/9	0.073	nd	0.18	one-half maximum RL	**0.090**
	beach play RME, area 4	0/7	0.04	nd	0.29	one-half maximum RL	**0.15**
	beach play RME, area 5	0/15	0.018	nd	0.1	one-half maximum RL	**0.050**
	beach play RME, area 6	0/1	0.016	nd	0.032	one-half maximum RL	**0.016**
	beach play RME, area 7	0/6	0.016	nd	0.033	one-half maximum RL	**0.017**
	beach play RME, area 8	0/6	0.016	nd	0.033	one-half maximum RL	**0.017**
	tribal clamming – RME and 183 days per year	0/99	0.055	nd	1	one-half maximum RL	**0.50**
	clamming – 7 days per	0/62	0.034	nd	0.5	one-half maximum RL	**0.25**

Table B.3-43, cont. Exposure point concentrations and summary statistics for SVOCs and dioxins/furans in sediment

Chemical	Scenario	No Detected/ Total No. Samples	Concentration (mg/kg dw)			Statistic Used	EPC Value (mg/kg dw)
			Mean Value	Maximum Detection	Maximum RL		
	year						
	netfishing	0/295	0.068	nd	1.8	one-half maximum RL	**0.90**
n-Nitroso-di-n-propylamine [a]	beach play RME, area 1	0/4	0.012	nd	0.033	one-half maximum RL	**0.017**
	beach play RME, area 2	0/5	0.027	nd	0.13	one-half maximum RL	**0.065**
	beach play RME, area 3	0/9	0.021	nd	0.071	one-half maximum RL	**0.036**
	beach play RME, area 4	0/10	0.083	nd	1.4	one-half maximum RL	**0.70**
	beach play RME, area 5	0/22	0.017	nd	0.04	one-half maximum RL	**0.020**
	beach play RME, area 6	0/1	0.016	nd	0.032	one-half maximum RL	**0.016**
	beach play RME, area 7	0/9	0.016	nd	0.04	one-half maximum RL	**0.020**
	beach play RME, area 8	0/11	0.017	nd	0.04	one-half maximum RL	**0.020**
	tribal clamming – RME and 183 days per year	0/230	0.05	nd	1.4	one-half maximum RL	**0.70**
	clamming – 7 days per year	0/92	0.025	nd	1.4	one-half maximum RL	**0.70**
	netfishing	Not a COPC in this scenario because the maximum detection did not exceed the RBC used for this scenario.					

[a] Chemical was never detected; therefore risks from chemicals shown in **bold** are evaluated in the uncertainty analysis (Section B.6).

[b] cPAH concentrations are given in terms of benzo(a)pyrene equivalents.

J qualifier – Analyte was positively identified and detected; however, the concentration is an estimated value because the result is less than the quantitation limit or QC criteria were not met.

COPC – chemical of potential concern

dw – dry weight

EPC – exposure point concentration

KM – Kaplan-Meier method for calculating a UCL

MVUE – minimum-variance biased eliminators

na – not available

nd – not detected

RBC – risk based concentration

RL – reporting limit

sd – standard deviation

SVOC – semivolatile organic compound

t (t-distribution) – statistical method used to calculate the mean for a normally distributed set of samples

UCL – upper confidence limit

Table B.3-44. Summary of exposure point concentrations in sediment by exposure scenario

Chemical	EPC Value (mg/kg dw)										
	Beach Play RME Area 1	Beach Play RME Area 2	Beach Play RME Area 3	Beach Play RME Area 4	Beach Play RME Area 5	Beach Play RME Area 6	Beach Play RME Area 7	Beach Play RME Area 8	Tribal Clamming – RME and 183 Days per Year	Clamming – 7 Days per Year	Netfishing
Metals and trace elements											
Aluminum	nd	nd	12,000	21,000	15,000	nd	23,000	17,000	20,000	15,000	19,000
Antimony	1.1[a]	2.3	5.2[a]	6.0[a]	5.0[a]	0.15	5.0[a]	7.0[a]	8.2[a]	1.3[a]	3.4[a]
Arsenic	15	21	13	11	8.9	9.8	11	10	27	9.5	21
Barium	nd	nd	76	81	42	nd	72	58	310	52	230
Cadmium	0.15[a]	1.0	2.0	0.94	0.23 [a]	0.15	0.80	0.20[a]	4.8	0.42	1.5
Chromium	21	48	38	82	28	23	25	23	81	28	51
Copper	50	170	79	74	74	35	38	34	450	49	200
Iron	nd	nd	17,000	32,000	26,000	nd	31,000	26,000	33,000	24,000	29,000
Lead[b]	71	400	130	300	37	24	16	37	780	60	300
Manganese	nd	nd	240	280	280	nd	430	780	650	310	360
Mercury	0.17	0.63	0.31[a]	1.7	0.11	0.090	0.11	0.12	0.23	0.20	na
Molybdenum	1.8	3.0	5.8[a]	3.2	2.0	1.5	1.3	1.9	3.8	2.1	na
Silver	0.20[a]	0.50[a]	0.80[a]	0.79	0.15[a]	0.20	0.35[a]	0.12	6.7	0.57	na
Thallium	0.15[a]	0.20[a]	17	0.092	0.062[a]	0.15	0.25[a]	0.060	3.6[a]	0.071[a]	2.4
Vanadium	47	67	50	57	55	49	64	57	56	53	60
Zinc	140	440	350	240	110	92	84	110	480	140	na
Organochlorine pesticides and PCBs											
Total DDTs	0.0037	0.013	0.021[a]	0.40	0.035[a]	0.00050	0.0027[a]	0.0082[a]	0.35	0.022[a]	na
Dieldrin	0.0013	0.00050	0.010[a]	0.017[a]	0.0026	0.0010	0.0010	0.0023[a]	0.013[a]	0.017[a]	0.0045[a]
PCB TEQ	9.08×10^{-8}	6.69×10^{-6}	nd	2.04×10^{-4}	2.51×10^{-6}	5.37×10^{-6}	5.65×10^{-7}	1.89×10^{-6}	1.84×10^{-4}	4.19×10^{-5}	7.18×10^{-5}

Table B.3-44, cont. Summary of exposure point concentrations in sediment by exposure scenario

Chemical	EPC Value (mg/kg dw)										
	Beach Play RME Area 1	Beach Play RME Area 2	Beach Play RME Area 3	Beach Play RME Area 4	Beach Play RME Area 5	Beach Play RME Area 6	Beach Play RME Area 7	Beach Play RME Area 8	Tribal Clamming – RME and 183 Days per Year	Clamming – 7 Days per Year	Netfishing
Total PCBs	0.12	0.18	0.24	11	0.19	0.97	0.23	0.23	4.0	1.5	2.5
Toxaphene	0.049	0.025	0.090	0.85	0.34[a]	0.049	0.049	0.049	6.3[a]	0.85[a]	6.3[a]
SVOCs and dioxins/furans											
4,6-Dinitro-o-cresol[d]	0.10	0.25	0.10	1.5	0.50	0.29	0.30	0.30	1.5	1.5	na
Benzidine[d]	nd	nd	0.55	nd	nd	nd	nd	nd	0.70	0.55	0.85
bis(2-chloroethyl)ether[d]	0.010	0.025	0.014	0.15	0.050	0.029	0.030	0.030	0.15	0.15	na
cPAHs[e]	1.2	3.0	2.1	0.73	0.41	0.44	0.11	0.32	0.77	0.48	0.57
Dioxin/furan TEQ	nd	nd	nd	4.12×10^{-4}	2.20×10^{-6}	nd	1.70×10^{-6}	nd	1.42×10^{-3}	3.65×10^{-4}	6.10×10^{-4}
n-Nitroso-dimethylamine[d]	0.050	0.065	0.090	0.15	0.050	0.016	0.017	0.017	0.50	0.25	0.90
n-Nitroso-di-n-propylamine[d]	0.017	0.065	0.036	0.70	0.020	0.016	0.020	0.020	0.70	0.70	na

[a] EPC was calculated based on 50% or more undetected values. The uncertainty associated with the risk calculation based on this EPC is likely to be higher than the uncertainty associated with risk estimates based on detected concentrations, as discussed in Section B.6.

[b] Lead is included in this table because it was designated a COPC. However, risks associated with lead are evaluated separately, in Section B.3.4.4, using the Integrated Exposure Uptake Biokinetic Model (IEUBK) for lead exposure in children and the Adult Lead Model (ALM) for characterizing risks associated with adult lead exposure. UCL values are presented in this summary table, however, mean sediment concentrations are used for lead modeling rather than UCL values. Thus, the values presented here are only for informational purposes.

[c] Chemical is not a COPC in this scenario.

[d] COPC was never detected; therefore, risks from chemicals shown in **bold** are evaluated in the uncertainty analysis (Section B.6).

[e] cPAH concentrations are given in terms of benzo(a)pyrene equivalents.

COPC – chemical of potential concern

dw – dry weight

EPC – exposure point concentration

na – not applicable (chemical was not a COPC for this scenario)

nd – no data available for this exposure area

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

SVOC – semivolatile organic compound

TEQ – toxic equivalent

B.3.4.4 Lead modeling

Risk estimates from lead exposure were not made using the equations presented in Section B.3.4. Instead, risks were estimated using the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) (EPA 1994) and the Adult Lead Model (ALM) (EPA 2003c). The parameterization of each model is discussed in separate sections below.

B.3.4.4.1 Children (IEUBK)

The IEUBK model (Version 1.0 for Windows) predicts blood-lead concentrations for children exposed to lead in their environment. The model requires input such as relevant absorption parameters and intake and exposure rates. The model then calculates and recalculates a complex set of equations to estimate the potential concentration of lead in the blood for a hypothetical population of children (aged 6 months to 7 years).

Default input parameters exist in the model for lead intake via air, drinking water, and diet. The IEUBK model allows for alternate dietary data to be used if data are available. If site-specific data are available, they are used to calculate the lead concentration for the alternate dietary source and the percentage of total dietary input that is represented by the alternate dietary source. The alternate dietary data are added to the other source data to derive a combined intake from all sources. For this HHRA, all default parameters recommended for use in the model by EPA were maintained except for alternate dietary source and soil lead concentrations. The default values for diet vary from 2.60 to 3.16 µg/day. These values are used to determine dietary lead exposure, unless data describing an alternate dietary source are entered. The alternate sources may include data for fish from fishing, home grown fruits and vegetables, and game animals from hunting. The model requires input on both the concentration of lead in the alternate dietary sources as well as the proportion of total dietary intake these categories represent (the default concentration for all replacement foods = 0 mg/kg, default percentage of all food consumed = 0%). For the LDW, only the fish from fishing category was adjusted, because data for other food-borne sources of lead were not available. Table B.3-45 presents the alternate food source lead concentration due to fish from fishing as well as the proportion of dietary intake represented by fish.

Site-specific data were used to adjust the pre-set sediment lead concentration of 200 µg/g, which represents a "plausible value for urban soil lead concentration"(EPA 2002f). Exposure to lead in soil was calculated as a time weighted average in accordance with child direct sediment exposure scenarios. On days a child visited the site, all of their lead exposure was assumed to come from the site, while on days when the site was not visited, the pre-set value of 200 µg/g was used. Using a weighted average allowed for a better estimate of true soil exposure. It should be noted that the pre-set value of 200 µg/g was above the lead concentration of measured site specific

data. Indoor, or household dust lead concentrations were calculated according to model guidelines based on the pre-set value of 200 µg/g soil lead (EPA 2002f). This value represents the concentration of lead immediately outside the home (compared to sediment exposure at the site which is further away from the home). Input values which replaced these three default settings are presented in Table B.3-45.

Table B.3-45. Input parameters for IEUBK lead model

Parameter	Value	Unit	Exposure Frequency
Sediment/soil concentration	167 (Area 7)	mg/kg dw	65 days per year
	191 (Area 2)	mg/kg dw	65 days per year
Alternate food source concentration[a]	0.42	µg Pb/g	365 days per year
Alternate food source fraction[b]	12	%	na

[a] Alternate food source concentration was derived as a single value for all seafood categories by weighting the concentration in each seafood category by the amount of that category that is consumed. This calculation used the seafood tissue EPCs (Table B.3-39) and the median child seafood consumption rates for each category. The alternate food source concentration was determined by summing the product of the mean EPC × ingestion rate for each seafood category and then dividing that total by the sum of the ingestion rates for each seafood category (see Table B.3-45 for calculations). Median values for ingestion rates were used per IEUBK model use guidelines (EPA 1994).

[b] 12 g/day (average amount of Puget Sound seafood consumed per day)/98.05 g/day (total meat consumed per day) (EPA 2006c).

dw – dry weight

EPC – exposure point concentration

IEUBK – Integrated Exposure Uptake Biokinetic Model for Lead in Children

na – not applicable

Pb – lead

ww – wet weight

The values described in Table B.3-45 are based on available data for LDW sediment and fish tissue.

Lead EPCs were calculated for intertidal sediments for each of the beach play RME scenarios. Beach play RME – Area 2 had the highest EPC, and beach play RME – Area 7 had the lowest EPC. Specifically, lead exposure was assumed to occur on each of the 65 days specified by the exposure frequency for the beach play scenario. In each of the beach play RME scenario evaluations, exposure to soils at 200 mg/kg dw was also assumed to occur on the days when sediment exposures did not occur (i.e., 300 days per year). A time-weighted average EPC was calculated for each of the intertidal exposure areas by multiplying the number of days exposed to either default soil lead or LDW sediment and dividing the sum of those by 365 days. The results of these calculations are presented in Table B.3-46.

The risks calculated using default values along with these site-specific data may not fully reflect the extent of lead exposure to children living in areas surrounding the LDW because of the lack of site-specific data for lead in household dust, water, and residential soils in the neighborhoods surrounding the LDW. Data describing the

potential lead exposures of children via these exposure pathways in the South Park and Georgetown neighborhoods were insufficient to fully parameterize the model for soil lead concentration both because the required data were not readily available, and because the focus of this HHRA is to assess the risks to people from contact with LDW sediment rather than to assess other residential exposures, the default values recommended by EPA in the IEUBK model manual were used. These values included an assumed 200 mg/kg dw pre-set soil concentration, which was intended to reflect an anthropogenic geometric mean concentration in urban areas. Actual site-specific lead concentrations in LDW-area soils are not known and could be higher or lower than this assumed value.

Table B.3-46. Sediment lead values and time-weighted average EPCs

EPC Type	Mean EPCs (mg/kg dw)							
	Area 1	Area 2	Area 3	Area 4	Area 5	Area 6	Area 7	Area 8
Mean sediment lead concentration from beach play areas	30	150	81	100	32	24	14	24
Time-weighted average lead concentrations based on sediment and soil exposures[a]	170	191	179	182	170	169	167	169

[a] Derived from sediment lead values:
$[(Pb_{sed} \times EF_{sed}) + (Pb_{soil} \times EF_{soil})/(EF_{sed} + EF_{soil})]$, where Pb_{sed} = mean sediment lead concentration (mg/kg dw)
EF_{sed} = beach play RME exposure frequency, 65 days/yr
Pb_{soil} = average default soil Pb concentration, 200 mg/kg dw
EF_{soil} = soil exposure frequency, calculated by subtraction from default exposure frequency ($EF_{sed} + EF_{soil} = 365$), value is 300 days/yr.

dw – dry weight

EPC – exposure point concentration

Pb – lead

Both a high-end and low-end time weighted average sediment EPC were used in the model to illustrate the range of risks. It should be noted that even the highest calculated lead EPC based on LDW sediment exposure is less than the IEUBK model default value of 200 mg/kg dw.

Alternate dietary data from the child tribal scenario based on Tulalip data for the consumption of fish and shellfish were included in the model as described in Table B.3-47. The IEUBK model applies average or CT estimates for all terms (EPA 1994). For seafood consumption rates, the median child seafood consumption rate was identified based on 40% of the median adult tribal seafood consumption rate based on Tulalip data of 29.9 g/day (EPA 2006c). Furthermore, the percentage of the alternate food source (fish) of its food group (all meat) was set at 12% (Table B.3-47). The results of the IEUBK model runs are presented in Section B.5.4. In order to calculate the average food lead concentration in the variety of fish consumed by tribal children, the median ingestion rate was multiplied by the mean lead concentration for each seafood category. The sum of the results of this calculation were then divided by the total

ingestion rate to get the average lead concentration for LDW fish. Table B.3-47 presents the details of this calculation.

Table B.3-47. Median ingestion rate and mean exposure point concentration by seafood category for tribal children based on Tulalip data

Seafood Category	Median Ingestion Rate (g/day)	Mean Lead Concentration (mg/kg ww)	Ingestion Rate × Mean Lead Concentration (µg/day)
Anadromous fish[a]	5.96	0.04[b]	0.2384
Pelagic fish	0.52	0.11	0.06
Benthic fish, fillet	0.48	0.04	0.019
Benthic fish, whole body	0	0.35	0
Crab, edible meat	2.00	0.045	0.09
Crab, whole body	0.64	0.067	0.04
Mussels, edible meat	0.04	0.41	0.02
Clams, whole body	2.32	1.96	4.55
Total	12	-	5.0

Note: Alternate food source concentration of lead = 5.0 µg/day/12 g/day = 0.42 µg Pb/g, as shown in Table B.3-45.

[a] As directed by EPA, anadromous fish were included in the seafood consumption rate for children in the IEUBK model. This model is intended to quantify the cumulative exposure to lead for children living along the LDW, regardless of source. There are dietary sources other than seafood that may contain lead, but there are no site-specific data to quantify the exposure so the default food lead concentration was used as a surrogate for all other food-borne sources of lead exposure.

[b] maximum detected concentration from PSAMP database (n = 36) (West et al. 2001). All but one result was a non-detect at 0.02 or 0.03 mg/kg ww.

Pb – lead

PSAMP – Puget Sound Ambient Monitoring Program

ww – wet weight

B.3.4.4.2 Adults (ALM)

The ALM is based on protecting the developing fetus of a pregnant woman, the most sensitive subpopulation affected by adult lead exposure. The model incorporates exposure to soil that is more representative of older children and adults than young children. Accordingly, EPA has used this model to estimate soil lead cleanup levels for sites at which the likely exposed population would be older children or adults. Although the model was developed to assess soil exposures, it has been applied in the LDW, in agreement with EPA Region 10, to evaluate exposure to lead in both sediments and in fish and shellfish. Adjustments were made to the model to account for fish intake (EPA 2007c). Specifically, Kissinger (2002) provided a revised algorithm that incorporates an exposure term for seafood consumption. This approach provides a way to evaluate cumulative exposure to lead in the LDW from both dermal soil contact and seafood ingestion.

The ALM applied for the LDW estimates an average blood lead level in adults based on additional exposure (above a baseline level) to lead in sediments, seafood, and air. An estimated fetal blood lead level is then calculated from the estimated adult blood lead levels (Equation 3-3). The contribution of lead from air at the LDW site was considered negligible because blood lead levels are much less sensitive to passive re-entrainment of lead from soil in air. The equation is thus:

$$PbB_{adult,central} = \frac{PbB_0 + BKSF \times FI \times ((PB_s \times IR_s \times AF_s \times EF_s) + (PB_f \times IR_f \times AF_f \times EF_f))}{AT}$$ **Equation 3-3**

where $PbB_{adult,central}$ is the geometric mean blood lead level (µg/dL) in exposed adults. The definition and parameterization of the other variables in the equation above are provided in Table B.3-48. A summary of the data used to determine mean EPC values is presented in Table B.3-49. This information replicates what is presented in EPC Tables B.3-36 and B.3-41. It is presented again here for the convenience of the reader. The same group of samples was used to determine these mean EPCs as was used for generation of EPCs for other chemicals.

Table B.3-48. Input parameters for ALM

PARAMETER	DESCRIPTION	VALUE	UNITS
PbB_0	adult baseline (geometric mean) blood lead level	1.7[a]	µg/dL
BKSF	biokinetic slope factor	0.4 (EPA default)	µg/dL per µg/day
FI	fractional intake	1	unitless
IR_s	sediment ingestion rate – beach play RME and netfishing	50 (EPA default)[b]	mg/day
IR_s	sediment ingestion rate – clamming	100 (EPA default)[b]	mg/day
IR_f	seafood ingestion rate	15[c]	g/day
Pb_s	mean lead concentration in sediment – beach play RME scenario	150[d]	mg/kg dw
Pb_s	mean lead concentration in sediment – tribal clamming RME	200	mg/kg dw
Pb_s	mean lead concentration in sediment – netfishing	100	mg/kg dw
EF_s	exposure frequency for tribal clamming RME	120	days/yr
EF_s	exposure frequency for beach play RME	65	days/yr
EF_s	exposure frequency for netfishing	119	days/yr
Pb_f	lead concentration in seafood	0.80[e]	mg/kg ww
EF_f	exposure frequency for seafood consumption	365	days/yr
AF_s	gastrointestinal absorbance fraction for lead in sediment	0.12 (EPA default for soil)[f]	unitless
AF_f	gastrointestinal absorbance fraction for lead in tissue	0.12[g]	unitless
AT	averaging time	365	days

[a] Because communities bordering the LDW include a sizable Mexican-American population, the average baseline blood lead level of Mexican-American women in the US was used (EPA 2002a).

[b] Although EPA has not developed default exposure assumptions for sediments, a conservative assumption was applied that assumes sediment consumption would be equivalent to 100% of the assumed soil and dust intake on each day an individual visited the LDW.

[c] Median Puget Sound seafood consumption rate (Hiltner 2007). The median resident fish consumption rate was developed by taking the median Tulalip Tribes' fish consumption rate for all species harvested from Puget Sound, 29.9 g/day, and adjusting it to represent consumption of resident species only. See Section B.3.4.1.1 for an explanation of this adjustment.

[d] The selected median sediment lead concentration is from the Area 2 scenario. All the other beach play RME scenarios had lower median lead concentrations (see Table B.3-49).

[e] Lead concentration in seafood equals the sum of (median lead concentration calculated by ProUCL × ingestion rate) for each seafood category/total IR (see Table B.3-50).

[f] Gastrointestinal absorption fraction for lead in sediment (EPA 2003c).

[g] Gastrointestinal absorption fraction for lead in tissue (EPA 2007c).

ALM – Adult Lead Model

dw – dry weight

EPA – US Environmental Protection Agency

LDW – Lower Duwamish Waterway

ww – wet weight

Table B.3-49. Summary of lead data and mean EPC values

Seafood Category or Exposure Scenario	Detection Frequency	Mean EPC (mg/kg)	Maximum Detect (mg/kg)	Maximum RL (mg/kg)	95TH UCL EPC (mg/kg)
Tissue					
Benthic fish, fillet	8/17	0.044	0.14	0.03	0.081
Benthic fish, whole body	24/24	0.35	0.95	na	0.42
Clams	14/14	2.0	6.4	na	3.1
Crab, edible meat	21/21	0.045	0.24 J	na	0.11
Crab, whole body	21/21	0.067	0.22 JM	na	0.12
Mussels	22/22	0.41	0.72	na	0.49
Pelagic fish, whole body	29/29	0.11	0.26	na	0.13
Sediment					
Beach play RME, area 1	4/4	30	71	na	71
Beach play RME, area 2	5/5	150	400	na	400
Beach play RME, area 3	9/9	81	210 J	na	130
Beach play RME, area 4	10/10	100	615	na	390
Beach play RME, area 5	22/22	32	70 J	na	37
Beach play RME, area 6	1/1	24	24	na	24
Beach play RME, area 7	9/9	14	19	na	16
Beach play RME, area 8	11/11	24	95	na	37
Tribal clamming – (RME & 183 days/yr)	275/275	200	23,000	na	780
Clamming – 7 days per year	103/103	50	615	na	68
Netfishing	817/817	100	23,000	na	300

EPC – exposure point concentration
RL – reporting limit

Lead EPC values were calculated for tissue and sediment based on the 95th UCL as was done for all other chemicals evaluated in the HHRA. However, because the ALM guidelines (EPA 2003c) recommend using mean sediment and tissue values for calculating risks from lead exposure, mean values were calculated and applied in the ALM (Table B.3-49). Median ingestion rates were calculated and used in the ALM to assess risks from the ingestion of fish tissue containing lead. In order to illustrate the range of risks to adults exposed to lead in the LDW, the beach play area with the highest average sediment value as well as the two adult RME sediment exposure scenarios were evaluated. The selected scenarios were beach play RME at Area 2, tribal clamming RME, and netfishing, with mean lead values of 150, 200, and 100 mg/kg dw, respectively. Low-end clamming (7 days per year) and the other beach play RME scenarios were not evaluated using the ALM because the mean sediment concentrations were lower for those scenarios, ranging from 14 to 100 mg/kg dw, which are largely consistent with background concentrations of lead in soil in Washington State. Table B.3-49 presents the mean sediment concentrations as well as the EPC values based on the 95th UCL for both tissue and sediment data.

The adult tribal CT ingestion rate based on Tulalip data (Hiltner 2007) was used in the lead model because EPA guidance calls for use of median ingestion rates in the ALM. See Section B.3.4.1.1 for additional discussion of these ingestion rates. The adult Tulalip CT ingestion rates were combined with the mean lead concentrations for each seafood category to calculate a weighted average lead concentration for all seafood, as shown in Table B.3-50. Anadromous fish consumption was not specifically addressed in the tissue lead calculations because it was considered to be part of baseline dietary exposure, which is included in the baseline blood lead level.

Table B.3-50. Calculation of median lead concentrations in tissue

Seafood Category	Mean Sediment Concentration (mg/kg ww)	Median Ingestion Rate (g/day)	Median Ingestion Rate × Mean Sediment Concentration
Benthic fish, fillet	0.04	1.20	0.048
Benthic fish, whole body	0.35	0	0
Clams	1.96	5.8	11
Crab, edible meat	0.045	5.0	0.23
Crab, whole body	0.067	1.6	0.11
Mussels	0.41	0.10	0.04
Pelagic fish, whole body	0.11	1.30	0.14
Total seafood ingestion rate		**15.0**	

Seafood Category	Mean Sediment Concentration (mg/kg ww)	Median Ingestion Rate (g/day)	Median Ingestion Rate × Mean Sediment Concentration
Sum of product of ingestion rates × median concentrations			**11.9**
Median lead concentration in seafood = 11.9/15.0 = 0.80[a]			

[a] Lead concentration in seafood equals the sum of (mean sediment concentration × median ingestion rate) for each seafood category/total median ingestion rate for all seafood categories. Mean sediment concentrations were calculated from the same set of samples used to calculate EPCs for other chemicals.

EPC – exposure point concentration

ww – wet weight

The model output includes both CT (geometric mean) and 95th percentile fetal blood lead levels. The 95th percentile fetal blood lead level is calculated using Equation 3-4:

$$PbB_{fetal95} = PbB_{adult,central} \times GSD_{i,adult}^{1.645} \times R_{fetal/maternal}$$ **Equation 3-4**

Where:

$PbB_{fetal95}$	=	95th percentile fetal blood lead level (µg/dL)
$PbB_{adult,central}$	=	central estimate of maternal adult blood lead concentration
$GSD_{i,adult}$	=	geometric standard deviation of the blood lead distribution
1.645	=	95th percentile value for the Student's t distribution
$R_{fetal/maternal}$	=	proportionality constant between fetal and maternal blood lead concentration

The geometric standard deviation (GSD) is an estimation of variation in blood lead levels around the geometric mean. It is used to estimate upper percentile blood lead levels for an individual and provide a health-protective estimate of the probability of an individual exceeding a given blood lead level (target risk goal). In accordance with EPA (2002a), a GSD of 2.29 was applied to this model. Fetal blood lead levels were predicted based on the EPA assumption that fetal blood lead levels at birth are 90% of the maternal blood lead level. A 10 µg/dL blood lead level for a fetus is associated with a 11.1 µg/dL blood lead level for the mother according to EPA (2003c). The probablility of exceeding the 10-µg/dL blood lead threshold for an individual was calculated using the following mathematical function in Microsoft® Excel®:

$$P_{exceedance} = 1 - Normdist(Ln(Pb_{target}/Pb_{central} \times R_{fetal/maternal})) / Ln(GSD))$$ **Equation 3-5**

Where:

Pb_{target}	=	child threshold blood lead level (in this application, 10 µg/dL)
$Pb_{central}$	=	child central tendency blood lead estimate
$R_{fetal/maternal}$	=	proportionality constant between fetal and maternal blood lead concentration
GSD	=	geometric standard deviation of the blood lead distribution

B.3.5 CHRONIC DAILY INTAKE RATES

CDI rates represent the estimated daily chemical dose for an individual averaged over the exposure duration for each scenario. Separate CDIs are calculated for chemicals with carcinogenic and non-carcinogenic effects because the averaging time over which the doses are calculated are different.

Tables 1 through 7 in Attachment 3 present the results of CDI calculations performed using Equations 3-1 and 3-2 and the exposure parameters given in Tables B.3-7 through B.3-26. The CDI results are used in the risk characterization and uncertainty analysis (Sections B.5 and B.6, respectively). Risk estimates for COPCs that were never detected are presented in the uncertainty analysis.

B.4 Toxicity Assessment

The toxicity assessment is an evaluation of each chemical's potential to cause health effects based on available toxicological information.

Quantitative estimates of toxicity potential have been developed by EPA and other agencies. EPA (2003b) has developed a hierarchical order of toxicity values for use in human health risk assessments that was applied for development of toxicity values for COPCs for this risk assessment:

- **Tier 1 –** EPA's Integrated Risk Information System (IRIS) database
- **Tier 2 –** EPA's Provisional Peer-Reviewed Toxicity Values (PPRTVs), Office of Research and Development/National Center for Environmental Assessment
- **Tier 3 –** Other toxicity values. Tier 3 includes additional EPA and non-EPA sources of toxicity information. Priority is given to those sources of information that are the most current, the basis for which is transparent and publicly available, and which have been peer reviewed. Sources include EPA regional offices, EPA Health Effects Assessment Summary Tables (HEAST) values, California EPA, and Agency for Toxic Substance and Disease Registry (ATSDR) minimal risk levels.

Chemicals may be quantitatively evaluated on the basis of their non-carcinogenic and/or carcinogenic potential. The toxicity values used for evaluating exposure to chemicals with non-carcinogenic and carcinogenic effects are called the RfD and SF, respectively.

The RfD is an estimate, with uncertainty spanning perhaps an order of magnitude or greater, of the daily exposure to the human population, including sensitive sub-populations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. In developing toxicity values for non-cancer effects, EPA reviews available data to identify the most sensitive endpoint and population (i.e., the effects that occur at the lowest concentration). These available data include effects on children

and other sensitive subpopulations. Chemicals may have additional adverse effects that occur at higher exposure levels.

The SF represents a plausible upper-bound estimate of the probability of a carcinogenic response per unit intake of a chemical over a lifetime. EPA has recently updated their guidance for carcinogen risk assessment to emphasize consideration of mode of action (e.g., mutagenesis) in the development of SFs (EPA 2005c). Generally, the SF is based on a dose-response curve using available carcinogenic data for a given chemical. Mathematical models are used to extrapolate from high experimental doses to the low doses expected for human contact in the environment. The selection of the mathematical model for dose extrapolation (e.g., linear or nonlinear) should be informed by the mode of action of the chemical (EPA 2005c).

The toxicity values used in this HHRA are summarized in Tables B.4-1 (non-cancer) and B.4-2 (cancer). The toxicological endpoints, as given in IRIS, that were used to establish the RfDs are presented in Table B.4-3. Many chemicals may have adverse effects that are not included in Table B.4-3 because they occur at higher doses than the effects upon which the RfDs were based. For some chemicals for which the RfD was from a source other than IRIS, ATSDR was used to identify the toxicological endpoints. For example, although not identified as a critical effect for the development of the RfD for PCBs (or PCB Aroclors) in IRIS, nervous system effects, particularly neurodevelopmental effects, are well-documented across a range of PCB exposure levels (ATSDR 2000; Longnecker et al. 2003). Therefore, PCBs were included in the evaluation of non-cancer hazards associated with the neurological endpoint in the risk characterization. Additional discussion of uncertainties associated with the RfDs used for risk characterization is provided in Section B.6.2.1.

The pharmacokinetics, acute toxicity, chronic toxicity, and potential carcinogenicity of each COPC are discussed in further detail in Attachment 4. The discussion of toxic effects in Attachment 4 includes many different exposure routes, some of which are not relevant to environmental exposure within the LDW, such as occupational inhalation exposure. The exposure routes are included only for completeness. This information was obtained primarily from:

- EPA's IRIS database; (www.epa.gov/iris)
- EPA's 1997 values contained in the HEAST
- Toxicological profiles presented in EPA's *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories* (EPA 2000d)
- EPA's Office of Ground Water and Drinking Water (OGWDW; www.epa.gov/OGWDW/hfacts.html)
- The ATSDR ToxFAQs (www.atsdr.cdc.gov/toxfaq.html)
- Hazardous Substance Data Bank (HSDB; toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB)

Toxicity Assessment for PCB TEQ, dioxin/furan TEQ, and cPAHs

As previously noted in Sections B.2.2.4 and B.3.4.3, certain classes of compounds are comprised of individual compounds that have similar chemical structures as well as a common mechanism of toxicity. Toxicity assessment for these classes is done on a group, rather than on an individual compound basis. These compound groups include co-planar PCB congeners, dioxins and furans, and cPAHs. The specific chemicals in each group were presented in Table B.2-5.

The toxicity of co-planar PCBs and dioxin and furans is assessed here using a toxic equivalency approach. Each congener is assigned a TEF describing the toxicity of that congener relative to the toxicity of the reference compound, 2,3,7,8-TCDD. A congener that is assumed to be equal in toxicity to 2,3,7,8-TCDD would have a TEF of 1.0. A congener that is assumed to be half as toxic as 2,3,7,8-TCDD would have a TEF of 0.5, and so on.

There have been several efforts to develop TCDD TEFs for dioxins/furans and PCBs having 2,3,7,8-TCDD-like toxicity (EPA 2000b). The most recent effort occurred at an expert meeting organized by the World Health Organization (WHO) in 2005 (Van den Berg et al. 2006). The WHO effort examined a number of lines of evidence to develop a consensus based list of TEFs. Table B.2-5 provides the WHO 2005 TEFs for PCBs and dioxins and furans.

As discussed in Section B.2.2.4, the sum of the products of the concentration of each coplanar PCB and its TEF is called the PCB TEQ and is calculated on a per sample basis. Similarly, the sum of the products of each coplanar dioxin and furan and its TEF is the called the dioxin/furan TEQ and is also calculated on a per sample basis. The excess cancer risk posed by PCB TEQ and dioxin/furan TEQ is then determined by multiplying the TEQ CDI by the SL for the reference compound, 2,3,7,8-TCDD (see Table B.4-2). In this document, PCB TEQ and dioxin/furan TEQ exposure and risk estimates are presented separately.

The toxicity of multiple cPAHs may be evaluated using the relative potency approach. This approach involves comparison of the cancer causing ability of a particular cPAH to a reference compound, benzo[a]pyrene (BaP), by means of a potency equivalency factor (PEF). A cPAH with a PEF of 1.0 is assumed to be as effective as BaP in inducing cancer. A cPAH with a PEF of 0.5 would be assumed to be half as effective as BaP in inducing cancer, and so on.

PEFs for cPAHs have been developed by the California Environmental Protection Agency (California EPA 1994) based on various toxic endpoints. EPA has also developed relative potency factors (RPFs, similar to PEFs) for these cPAHs (EPA 1993). It was felt that the California EPA cPAH PEFs had better documentation than EPA RPF values and were consequently used in the LDW HHRA.

As discussed in Section B.2.2.4, the sum of the products of the concentration of each cPAH and its PEF is considered the cPAH TEQ and is calculated on a per sample basis. The excess cancer risk posed by all cPAHs is then computed by multiplying the cPAH (i.e., total BaP equivalents) CDI by the BaP SL (Table B.4-2).

Table B.4-1. Non-cancer toxicity data (oral) for chemicals of potential concern

Chemical[a]	Chemical Class	Oral RfD (mg/kg-day)	Critical Effect	Uncertainty Factor	RfD Source	Source Date[b]	Notes
1,3-Dichlorobenzene	SVOC	0.003[c]	na	na	NCEA	4/7/2006	
1,4-Dichlorobenzene	SVOC	0.03	na	na	NCEA	4/7/2006	
2,4,6-Trichlorophenol	SVOC	0.0001	na	na	NCEA	10/20/2004	
2,4-Dichlorophenol	SVOC	0.003	decreased delayed hypersensitivity response	100	IRIS	3/8/2006	
2,4-Dinitrophenol	SVOC	0.002	cataract formation	1,000	IRIS	3/8/2006	
2,4-Dinitrotoluene	SVOC	0.002	neurotoxicity, Heinz bodies and biliary tract hyperplasia	100	IRIS	3/8/2006	
2,6-Dinitrotoluene	SVOC	0.001	na	na	PPRTV	4/7/2006	
2-Chlorophenol	SVOC	0.005	reproductive effects	1,000	IRIS	3/8/2006	
3-Nitroaniline	SVOC	0.0003	na	na	EPA (2004a)	10/20/2004	PPRTV toxicity values for this chemical have been retired but are available in EPA's Region 9 RBC table.
4,6-Dinitro-o-cresol	SVOC	0.0001	na	na	EPA (2004a)	10/20/2004	PPRTV toxicity values for this chemical have been retired but are available in EPA's Region 9 RBC table.
4-Chloroaniline	SVOC	0.004	non-neoplastic lesions of splenic capsule	3,000	IRIS	3/8/2006	
4-Methylphenol	SVOC	0.005	na	na	HEAST	4/7/2006	
4-Nitroaniline	SVOC	0.003	na	na	EPA (2004a)	10/20/2004	PPRTV toxicity values for this chemical have been retired but are available in EPA's Region 9 RBC table.
Aldrin	pesticide	0.00003	liver toxicity	1,000	IRIS	3/8/2006	
alpha-BHC	pesticide	0.0005	na	na	NCEA	10/20/2004	
Aluminum	metal	1	na	na	PPRTV	10/20/2004	
Aniline	pesticide	0.007	na	na	PPRTV	4/7/2006	
Antimony	metal	0.0004	longevity, blood glucose, and cholesterol	1,000	IRIS	3/8/2006	

Table B.4-1, cont. Non-cancer toxicity data (oral) for chemicals of potential concern

Chemical[a]	Chemical Class	Oral RfD (mg/kg-day)	Critical Effect	Uncertainty Factor	RfD Source	Source Date[b]	Notes
Arsenic	metal	0.0003	hyperpigmentation, keratosis, and possible vascular complications	3	IRIS	3/8/2006	surrogate = inorganic arsenic
Barium	metal	0.2	nephropathy	300	IRIS	4/6/2006	
Benzidine	SVOC	0.003	brain cell vacuolization; liver cell alterations in females	1,000	IRIS	3/8/2006	
beta-BHC	pesticide	0.0002	na	na	NCEA	10/20/2004	
Bis(2-chloroisopropyl) ether	SVOC	0.04	decrease in hemoglobin and possible erythrocyte destruction	1,000	IRIS	3/8/2006	
Bis(2-ethylhexyl) phthalate	phthalate	0.02	increased relative liver weight	1,000	IRIS	3/8/2006	
Butyl benzyl phthalate	phthalate	0.2	significantly increased liver-to-body weight and liver-to-brain weight ratios	1,000	IRIS	3/8/2006	
Cadmium	metal	0.001	significant proteinuria	10	IRIS	3/8/2006	cadmium RfD for food was used for this risk assessment
Total chlordane	pesticide	0.0005	hepatic necrosis	300	IRIS	3/8/2006	surrogate = chlordane (technical); total includes alpha-chlordane, gamma-chlordane, and chlordane samples
Chromium	metal	0.003	none reported	300	IRIS	3/8/2006	surrogate = hexavalent chromium
Copper	metal	0.04	na	na	HEAST	4/7/2006	
Total DDTs	pesticide	0.0005	liver lesions	100	IRIS	3/8/2006	surrogate = 4,4'-DDT; total includes isomers of DDD, DDE, and DDT
Dieldrin	pesticide	0.00005	liver lesions	100	IRIS	3/8/2006	
Endrin	pesticide	0.0003	mild histological lesions in liver, occasional convulsions	100	IRIS	3/8/2006	
Endrin aldehyde	pesticide	0.0003	mild histological lesions in liver, occasional convulsions	100	IRIS	3/8/2006	surrogate = endrin
gamma-BHC	pesticide	0.0003	liver and kidney toxicity	1,000	IRIS	3/8/2006	
Heptachlor	pesticide	0.0005	liver weight increases in males	300	IRIS	3/8/2006	
Heptachlor epoxide	pesticide	0.000013	increased liver-to-body weight ratio in both males and females	1,000	IRIS	3/8/2006	
Hexachlorobenzene	SVOC	0.0008	liver effects	100	IRIS	3/8/2006	
Hexachlorobutadiene	SVOC	0.0002	na	na	HEAST	4/7/2006	

Table B.4-1, cont. Non-cancer toxicity data (oral) for chemicals of potential concern

Chemical[a]	Chemical Class	Oral RfD (mg/kg-day)	Critical Effect	Uncertainty Factor	RfD Source	Source Date[b]	Notes
Hexachlorocyclopentadiene	SVOC	0.006	chronic irritation	1,000	IRIS	3/8/2006	
Hexachloroethane	SVOC	0.001	atrophy and degeneration of renal tubules	1,000	IRIS	3/8/2006	
Iron	metal	0.3	na	na	NCEA	4/7/2006	
Manganese	metal	0.14	central nervous system effects (other effects include impairment of neurobehavioral function)	1	IRIS	4/6/2006	RfD selected is applicable to exposures via ingestion of food
Mercury	metal	0.0001	developmental neuropsychological impairment	10	IRIS	3/8/2006	surrogate = methylmercury
Molybdenum	metal	0.005	increased uric acid levels	30	IRIS	4/6/2006	
Nickel	metal	0.02	decreased body and organ weights	300	IRIS	3/8/2006	
Nitrobenzene	SVOC	0.0005	hematologic, adrenal, renal and hepatic lesions	10,000	IRIS	3/8/2006	
n-Nitrosodimethylamine	SVOC	0.000008	na	na	PPRTV	10/20/2004	
n-Nitrosodiphenylamine	SVOC	0.02	na	na	PPRTV	10/20/2004	
Total PCBs	PCB	0.00002	ocular exudate, inflamed and prominent Meibomian glands, distorted nail growth, decreased antibody response	300	IRIS	3/8/2006	surrogate = Aroclor 1254, the lowest and most protective RfD available for PCBs in IRIS; total includes Aroclors 1016, 1221, 1232, 1242, 1248, 1254, and 1260
Pentachlorophenol	SVOC	0.03	liver and kidney pathology	100	IRIS	3/8/2006	
Silver	metal	0.005	argyria	3	IRIS	3/8/2006	
Thallium	metal	0.00007	increased levels of SGOT and LDH	3000	IRIS	5/15/2006	surrogate = by conversion from thallium chloride according to IRIS
Tributyltin as ion	organo-metal	0.00015	immunosuppression	100	IRIS	3/8/2006	surrogate = by conversion from tributyltin oxide (multiply by 0.49)
Vanadium	metal	0.001	na	na	NCEA	4/7/2006	
Zinc	metal	0.3	decreases in erythrocyte Cu, Zn-superoxide dismutase activity in healthy adults	3	IRIS	3/8/2006	

Table B.4-1, cont. Non-cancer toxicity data (oral) for chemicals of potential concern

[a] Chemicals for which no RfD was available were excluded from this table. These chemicals include 1,2-diphenylhydrazine, 3,3-dichlorobenzidine, bis(2-chloroethyl) ether, carbazole, n-nitroso-di-n-propylamine, cPAHs, and toxaphene.

[b] The IRIS date is the date the database was searched; the HEAST, NCEA, and PPRTV dates are the dates that the EPA Region 3 or Region 9 RBC tables (the sources of the HEAST, NCEA, and PPRTV values) were updated. When available, the more recently updated Region 3 RBC table was used (updated on April 7, 2006), rather than the Region 9 RBC table, which was updated on October 20, 2004.

[c] RfD of 0.03 in EPA Region 4 RBC table is based on an error in an NCEA paper establishing this RfD. Corrected RfD from EPA Region 3 RBC table has been used in this risk assessment.

BHC – benzene hexachloride

cPAH – carcinogenic polycyclic aromatic hydrocarbon

Cu – copper

EPA – US Environmental Protection Agency

HEAST – Health Effects Assessment Summary Tables

IRIS – Integrated Risk Information System

LDH – lactate dehydrogenase

na – not available (no RfD has been developed for this chemical)

NCEA – National Center for Exposure Assessment (EPA; provisional RfDs)

PCB – polychlorinated biphenyl

PPRTV – Provisional Peer-Reviewed Toxicity Values (EPA)

RBC – risk-based concentration

RfD – reference dose

SGOT – serum glutamic oxaloacetic transaminase

SVOC – semivolatile organic compound

Zn – zinc

Table B.4-2. Cancer toxicity data (oral/dermal) for chemicals of potential concern

Chemical[a]	Parameter Classification	Oral Cancer Slope Factor (mg/kg-day)$^{-1}$	Cancer Description Guideline[b]	Source	Source Date[c]	Notes
1,2-Diphenylhydrazine	SVOC	0.8	B2	IRIS	3/8/2006	
1,4-Dichlorobenzene	SVOC	0.024	C (IARC)	HEAST	4/7/2006	
2,4,6-Trichlorophenol	SVOC	0.011	B2	IRIS	3/8/2006	
3,3'-Dichlorobenzidine	SVOC	0.45	B2	IRIS	3/8/2006	
3-Nitroaniline	SVOC	0.021	na	EPA (2004a)	10/20/2004	PPRTV toxicity values for this chemical have been retired but are available in EPA's Region 9 RBC table.
4-Methylphenol	SVOC	na	C	IRIS	10/11/06	
4-Nitroaniline	SVOC	0.021	na	EPA (2004a)	10/20/2004	PPRTV toxicity values for this chemical have been retired but are available in EPA's Region 9 RBC table.
Aldrin	pesticide	17	B2	IRIS	3/8/2006	
alpha-BHC	pesticide	6.3	B2	IRIS	3/8/2006	
Aniline	pesticide	0.0057	B2	IRIS	3/8/2006	
Arsenic	metal	1.5	A	IRIS	3/8/2006	surrogate = inorganic arsenic
Benzidine	SVOC	230	A	IRIS	3/8/2006	
beta-BHC	pesticide	1.8	C	IRIS	3/8/2006	
Bis(2-chloroethyl) ether	SVOC	1.1	B2	IRIS	3/8/2006	
Bis(2-chloroisopropyl) ether	SVOC	0.07	D (IARC)	HEAST	4/7/2006	
Bis(2-ethylhexyl) phthalate	phthalate	0.014	B2	IRIS	3/8/2006	
Butyl benzyl phthalate	phthalate	na	C	IRIS	10/11/2006	
Carbazole	SVOC	0.02	D (IARC)	EPA (2004a)	10/20/2004	
cPAHs	PAH	7.3	B2	IRIS	3/8/2006	slope factor based onbenzo(a)pyrene
Total chlordane	pesticide	0.35	B2	IRIS	3/8/2006	surrogate = chlordane (technical); total includes alpha-chlordane, gamma-chlordane, and chlordane samples

Table B.4-2, cont. Cancer toxicity data (oral/dermal) for chemicals of potential concern

Chemical[a]	Parameter Classification	Oral Cancer Slope Factor (mg/kg-day)$^{-1}$	Cancer Description Guideline[b]	Source	Source Date[c]	Notes
Total DDTs	pesticide	0.34	B2	IRIS	3/8/2006	surrogate = 4,4'-DDT; total includes isomers of DDD, DDE, and DDT
Dioxin/furan TEQ	dioxins	150,000	A (IARC)	HEAST	4/7/2006	slope factor based on 2,3,7,8-TCDD
Dieldrin	pesticide	16	B2	IRIS	3/8/2006	
gamma-BHC	pesticide	1.3	C (IARC)	HEAST	4/7/2006	
Heptachlor	pesticide	4.5	B2	IRIS	3/8/2006	
Heptachlor epoxide	pesticide	9.1	B2	IRIS	3/8/2006	
Hexachlorobenzene	SVOC	1.6	B2	IRIS	3/8/2006	
Hexachlorobutadiene	SVOC	0.078	C	IRIS	3/8/2006	
Hexachloroethane	SVOC	0.014	C	IRIS	3/8/2006	
Mercury	metal	na	C	IRIS	10/11/2006	surrogate = methylmercury
n-Nitrosodimethylamine	SVOC	51	B2	IRIS	3/8/2006	
n-Nitroso-di-n-propylamine	SVOC	7	B2	IRIS	3/8/2006	
n-Nitrosodiphenylamine	SVOC	0.0049	B2	IRIS	3/8/2006	
PCB TEQ	PCB	150,000	B2	HEAST	4/7/2006	slope factor based on 2,3,7,8-TCDD
Total PCBs	PCB	2	B2	IRIS	3/8/2006	upper-bound slope factor used for this risk estimate, total includes Aroclors 1016, 1221, 1232, 1242, 1248, 1254, and 1260
Pentachlorophenol	SVOC	0.12	B2	IRIS	3/8/2006	
Toxaphene	pesticide	1.1	B2	IRIS	3/8/2006	

[a] Chemicals included in this table are either Class A, B, or C chemicals with regard to their cancer-causing potential (see below) or are class D chemicals with available cancer slope factors. Cadmium and chromium, although known carcinogens, are excluded from this table because they are carcinogens only via the inhalation pathway.

[b] A = known human carcinogen; B1 = probable human carcinogen (based on limited evidence of carcinogenicity in humans); B2 = probable human carcinogen (sufficient evidence in animals and inadequate or no evidence in humans); C = possible human carcinogen (limited evidence from animal studies and inadequate or no data in humans); D = not classifiable as to human carcinogenicity.

[c] The IRIS date is the date that the database was searched; the HEAST, NCEA, and PPRTV dates are the dates that the EPA Region 3 or Region 9 RBC tables (the sources of the HEAST, NCEA, and PPRTV values) were updated. When available, the more recently updated Region 3 RBC table was used (updated on April 7, 2006), as opposed to the Region 9 RBC table, which was updated on October 20, 2004.

Table B.4-2, cont. Cancer toxicity data (oral/dermal) for chemicals of potential concern

BHC – benzene hexachloride
cPAH – carcinogenic polycyclic aromatic hydrocarbon
EPA – US Environmental Protection Agency
HEAST – Health Effects Assessment Summary Tables
IARC – International Agency for Research on Cancer
IRIS – Integrated Risk Information System
na – not applicable
NCEA – National Center for Exposure Assessment (EPA)
OSHA – Occupational Safety and Health Administration
PCB – polychlorinated biphenyl
PPRTV – Provisional Peer-Reviewed Toxicity Values (EPA)
RBC – risk based concentration
SVOC – semivolatile organic compound
TEQ – toxic equivalent

Table B.4-3. Toxicological endpoints for COPCs with non-carcinogenic effects

Chemical	Endpoint										
	Kidney	Liver	Development	Cardiovascular System	Endocrine System	Hematologic System	Immune System	Nervous System	Skin	Eyes	Reproductive System
1,2-Diphenylhydrazine[a]											
1,3-Dichlorobenzene[b]											
1,4-Dichlorobenzene[b]											
2,4,6-Trichlorophenol[b]											
2,4-Dichlorophenol							X				
2,4-Dinitrophenol										X	
2,4-Dinitrotoluene		X				X		X			
2,6-Dinitrotoluene[b]											
2-Chlorophenol											X
3,3'-Dichlorobenzidine[a]											
3-Nitroaniline[b]											
4,6-Dinitro-o-cresol[b]											
4-Chloroaniline							X				
4-Methylphenol[c]	X	X						X	X		
4-Nitroaniline[b]											
Aldrin		X									
alpha-BHC[d]		X									
Aluminum[b]											
Aniline[b]											
Antimony					X	X					
Arsenic				X					X		
Barium	X										
Benzidine		X									
beta-BHC[d]		X									
Bis(2-chloroethyl) ether[a]											

Table B.4-3, cont. Toxicological endpoints for COPCs with non-carcinogenic effects

Chemical	Endpoint										
	Kidney	Liver	Development	Cardiovascular System	Endocrine System	Hematologic System	Immune System	Nervous System	Skin	Eyes	Reproductive System
Bis(2-chloroisopropyl) ether						X					
Bis(2-ethylhexyl) phthalate		X									
Butyl benzyl phthalate		X									
Cadmium	X										
Carbazole[a]											
cPAHs[a]											
Total chlordane		X									
Chromium (as hexavalent chromium)											
Copper[e]	X	X									
Total DDTs		X									
Dieldrin		X									
Endrin		X									
Endrin aldehyde (as endrin)		X									
gamma-BHC	X	X									
Heptachlor		X									
Heptachlor epoxide		X									
Hexachlorobenzene		X									
Hexachlorobutadiene[b]											
Hexachlorocyclopentadiene											
Hexachloroethane	X										
Iron[b]											
Lead[a]											
Manganese								X			
Mercury (as methylmercury)			X					X			
Molybdenum	X							X			
Nickel											
Nitrobenzene	X	X			X	X					

Table B.4-3, cont. Toxicological endpoints for COPCs with non-carcinogenic effects

Chemical	Endpoint										
	Kidney	Liver	Development	Cardiovascular System	Endocrine System	Hematologic System	Immune System	Nervous System	Skin	Eyes	Reproductive System
n-Nitrosodimethylamine[b]											
n-Nitroso-di-n-propylamine[a]											
n-Nitrosodiphenylamine[b]											
Total PCBs (based on Aroclor 1254)[f]			X				X	X[g]			
Pentachlorophenol	X	X									
Silver									X		
Thallium						X					
Toxaphene[a]											
Tributyltin as ion (by conversion from tributyltin oxide)							X				
Vanadium[h]				X							
Zinc						X					

Note: Each of these chemicals will likely have additional toxic effect endpoints at exposures above the reference dose. The toxicological profile section of the HHRA (Attachment 4) may be consulted by readers desiring more information on toxic effect endpoints. ATSDR toxicological profiles are an excellent resource for this information.

[a] No RfD is available for this chemical.

[b] The RfD for this chemical is from a source other than IRIS, and thus no information is available regarding the endpoints used to calculate the RfD.

[c] The RfD for this chemical is from a source other than IRIS. The endpoints for 4-methylphenol were identified using ATSDR (2006).

[d] The RfD for this chemical is from a source other than IRIS. The endpoint for alpha-BHC and beta-BHC was identified using ATSDR (2005c).

[e] The RfD for this chemical is from a source other than IRIS. The endpoints for copper were identified using ATSDR (2004).

[f] PCB effects on skin (chloracne) are well-documented, but these are associated with acute exposures at levels much higher than the RfD (ATSDR 2000).

[g] Nervous system effects for PCBs were not identified in IRIS for development of the RfD, but such effects, particularly neurodevelopmental effects, are well-documented (ATSDR 2000).

[h] The RfD for this chemical is from a source other than IRIS. The endpoint for vanadium was identified using ATSDR (1995).

X – Indicates that the IRIS RfD for a particular chemical was calculated for the identified endpoint.

BHC – benzene hexachloride

COPC – chemical of potential concern

PAH – polycyclic aromatic hydrocarbon

PCB – polychlorinated hydrocarbon

B.5 Risk Characterization

This section presents risk estimates for all exposure scenarios presented in Section B.3 based on the toxicity information presented in Section B.4. The equations used to calculate the risk estimates are presented, followed by the calculation results. These estimates are useful for characterizing the risks to people who could be exposed to chemicals found in LDW seafood and sediment, as well as for identifying COCs, which are defined here as chemicals with an excess cancer risk estimate greater than 1×10^{-6} or a non-cancer HQ greater than 1 for any RME scenario, and of those chemicals, identifying those with the greatest contributions to risk estimates.

Risks have been evaluated for various scenarios that reflect different assumed degrees of potential exposure. RME is the highest exposure that is reasonably expected to occur at a site. EPA generally uses RME and associated risks to evaluate remedial actions at a site (EPA 1989). RME by definition likely overestimates exposure for many individuals. In characterizing uncertainty in exposure and risks, it is useful to examine CT exposures and risks (National Research Council 1994). CT risk estimates are intended to reflect average exposures. CT exposures and risks are not favored in decision-making because they will underestimate exposure and risk for a substantial number of individuals (EPA 1989). Another method of examining risk and exposure is to look at risks associated with some unit of exposure that a member of the public can use to estimate risks associated with their individual behavior. This last method was used to characterize seafood consumption risk on an individual basis, with the unit of exposure being one meal per month.

B.5.1 Risk Estimate Calculations

Carcinogenic risks and non-carcinogenic health effects were evaluated separately because of fundamental differences in their critical toxicity values. Equations for each type of effect are presented in separate subsections that follow.

B.5.1.1 Carcinogenic risks

For chemicals with carcinogenic effects, EPA guidance (1989) is based on the theory that the risk of cancer is proportional to the dose, with the assumption that there is no threshold. In other words, this approach follows from the assumption that there is never a zero probability of cancer risk when exposed to these chemicals at any concentration. For relatively low probabilities (i.e., below 1 in 100), carcinogenic risks are calculated by multiplying the estimated exposure level (the chronic daily intake [CDI]) by the cancer SF for each chemical.[21]

[21] In cases where excess cancer risk estimates exceeded 1 in 100, the exponential version of the risk equation was used, as per EPA guidance (1989).

$$\text{Risk} = \text{CDI} \times \text{SF}$$ **Equation 5-1**

Where:

Risk	=	estimated chemical-specific individual excess[22] lifetime excess cancer risk (unitless)
CDI	=	chemical-specific chronic daily intake (mg/kg-day)
SF	=	route- and chemical-specific carcinogenic SF $(\text{mg/kg-day})^{-1}$

Cancer risk is expressed as a lifetime excess cancer risk. This concept assumes that the risk of cancer from a given chemical is in "excess" of the background risk of developing cancer (i.e., approximately 41% of men and women born today will be diagnosed with cancer during their lifetimes, according to the National Cancer Institute (NCI 2006).[23]

EPA has recently provided additional guidance for children's carcinogenic risk assessment (EPA 2005e), and Region 10 has provided instructions on implementation of this guidance to the LDW (EPA 2006c). For cPAHs, which have been identified as having a mutagenic mode of action, dose estimates are adjusted upwards in the risk calculation in the following manner to account for potential greater susceptibility of children from 0 to 2 and from 2 to6 years of age compared with older children and adults:

$$\text{cPAH risk ages 0 to 6} = \left[\left(\text{dose cPAH}_{\text{overall}} \times 2/6\right) \times 10 + \left(\text{dose cPAH}_{\text{overall}} \times 4/6\right) \times 3\right] \times \text{cPAH SF}$$ **Equation 5-2**

For all exposure routes (i.e., ingestion of seafood or sediment and dermal contact with sediment), this dose adjustment has been made in the final risk calculation rather than an adjustment to exposures or to carcinogenic potency. Implementation of this approach results in approximately a five-fold increase in the cancer risk estimate for cPAHs for children and is based on the assumption that toxicity of carcinogens with a mutagenic mode of action could be greater for young children than for older children or adults.

Excess cancer risks are probabilities expressed in scientific notation. For example, 1×10^{-5} is equivalent to 0.00001 or 1 in 100,000. Excess cancer risks are presented with only one significant figure to acknowledge the uncertainty in the underlying cancer

---

[22] Excess cancer risk refers to risks associated with site-specific exposure, above and beyond risks associated with exposure from all other causes, including exposure to carcinogenic sources outside the site.

[23] The American Cancer Society indicates that approximately one of every two men and one of every three women will get some form of cancer during their lifetimes (http://www.cancer.org/docroot/CRI/content/CRI_2_4_1x_Who_gets_cancer.asp?sitearea=).

SFs, per EPA guidance (1989), and in the exposure assumptions underlying the calculations.

B.5.1.2 Non-carcinogenic health effects

Chemicals with non-carcinogenic health effects are believed not to be toxic below a certain threshold because of protective mechanisms in the body (EPA 1989); a critical chemical dose must be exceeded before health effects are observed. The potential for non-carcinogenic health effects is represented by the ratio of a chemical's exposure level and the route-specific RfD, and is expressed as a hazard quotient (HQ):

$$HQ = CDI/RfD \qquad \textbf{Equation 5-3}$$

Where:

HQ	=	estimated chemical-specific hazard quotient (unitless)
CDI	=	chemical-specific chronic daily intake (mg/kg-day)
RfD	=	route- and chemical-specific reference dose (mg/kg-day)

The HQ is recommended by EPA as a way to quantify the potential for non-carcinogenic health effects (EPA 1989). HQs are not risk probabilities; the likelihood of an adverse effect does not usually increase linearly with the calculated value. An HQ greater than 1 indicates potential adverse health effects from the chemical exposure, although the same HQ may not equate to the same magnitude of adverse health effects for all chemicals. HQ interpretation considers the shape and slope of the dose-response curve in the area of observation, the magnitude of uncertainty and modifying factors to the RfD, and the confidence assigned to the RfD by EPA.

HQs for individual COPCs with similar toxicological effects may be summed to yield an effect-specific hazard index (HI) (EPA 1989). The effect-specific HI is an expression of the additivity of non-carcinogenic health effects. An effect-specific HI can be calculated by summing HQs for chemicals with similar toxicological effects (e.g., immunotoxicity), as described in Table B.4-3. If the sum of all HQs is less than 1, no effect-specific HIs are calculated because they would also not exceed 1.

B.5.1.3 Acceptable risk thresholds

The CERCLA and MTCA rules have in common the goal of protecting human health. However, the acceptable risk thresholds differ between the two rules. CERCLA risk thresholds are given as a range and MTCA risk thresholds are given as point estimates for individual chemical and pathway risks and as a point estimate for cumulative risks. Additional details on the differences in risk thresholds between the two rules are described below.

CERCLA risk thresholds are discussed in the National Contingency Plan (NCP; 40 CFR 300). In assessing the carcinogenic risks posed by a site, EPA established an excess cancer risk of 1×10^{-6} (1 chance in 1 million) as a "point of departure" for establishing risk thresholds of concern. Where the cumulative excess cancer risk to an

individual based on the RME for current and future land use is less than 1×10^{-4} (1 chance in 10,000) and the non-carcinogenic HI (see Section B.5.1.2) is less than 1, action generally is not warranted unless there are adverse environmental impacts (EPA 1991b). Cumulative excess cancer risks between 10^{-6} and 10^{-4} may or may not be considered acceptable, depending on site-specific factors such as the potential for exposure, technical limitations of remediation, and data uncertainties.

MTCA risk thresholds are discussed in WAC 173-340-700 (Ecology 2001a). For Method B (universal method), cleanup levels for individual carcinogens are based on the upper bound of the estimated excess lifetime cancer risk of 1×10^{-6}. For individual non-carcinogenic substances, cleanup levels are set at concentrations that are anticipated to result in no acute or chronic toxic effects on human health (that is, an HQ of 1 or less). Considering multiple hazardous substances and multiple exposure pathways, the total excess lifetime cancer risk at the site may not exceed 1×10^{-5}, with risks for individual hazardous substances in each exposure pathway not to exceed 1×10^{-6} and levels for non-carcinogens cannot exceed the point at which a hazardous substance may cause illness in humans (i.e., HI cannot exceed 1).

As noted in Section B.1, this HHRA was developed based on site-specific risk assessment methodology consistent with EPA guidance and with the LDW work plan (Windward 2004d), Phase 1 HHRA (Windward 2003b), and subsequent agency comments. As such, it did not follow specific MTCA protocols necessary for the development of cleanup levels under WAC 173-240-708. Although risk thresholds (i.e., cancer risk estimates exceeding 1×10^{-6} or an HI greater than 1), are the same within CERCLA and MTCA, the exposure scenarios that are evaluated in MTCA have not been specifically addressed in this HHRA. It should be noted that many of the MTCA exposure scenarios are either not appropriate or specific for assessment of sediment contaminant risks. Nevertheless, the scenarios in this HHRA have been evaluated by Ecology and found to be sufficiently protective to comply with the MTCA risk assessment framework (Ecology 2007). Therefore, chemicals with cancer risk estimates exceeding 1×10^{-6} or an HI greater than 1 for any RME scenario (see Section B.5.1.1) will be referred to as COCs in accordance with CERCLA.

B.5.2 RISK CHARACTERIZATION FORMAT

Excess cancer risks and HQs are presented according to the format recommended in EPA (1998). The primary purpose of the HHRA is to characterize risks from site-related exposures in support of risk management decisions and remedial options. Risk estimates also provide information to the public about what their health risks may be from engaging in different activities associated with the LDW (e.g., beach play and consumption of LDW seafood). Therefore, risks are characterized and quantified for chemicals detected in LDW sediment or seafood.

A number of COPCs that were never detected but had RLs that exceeded RBCs have been identified for each exposure pathway and medium. Hypothetical risk estimates

from these undetected COPCs are quantified and discussed in the uncertainty analysis (Section B.6). Risks estimates attributable to these undetected chemicals have very high uncertainty, and thus are not considered appropriate for identifying locations where remediation might be warranted.

Excess cancer risks are summed for all chemicals within each exposure scenario. Exposure scenarios in which the same receptor is exposed via multiple pathways simultaneously were addressed by summing the RME estimates for those pathways. This approach was applied to all direct sediment exposure scenarios that involved both dermal absorption and incidental sediment ingestion. In addition, excess cancer risk estimates were summed across some potentially related scenarios (e.g., netfishing and seafood consumption). For some combinations of scenarios, the highest RME pathway risk estimate may be several orders of magnitude higher than the other scenarios. The resulting risk estimate for the combination of multiple scenarios may then differ only slightly or not at all from the risk estimate for the RME scenario alone.

In this section, CDIs are presented with two significant figures, while excess cancer risks and HQs are presented with only one significant figure. The former reflects the accuracy of the CDI equations, and the latter reflects the accuracy of the cancer SFs and reference doses, as per the EPA IRIS database. Sums of excess cancer risk estimates are reported with one significant figure as well. For example, the sum of excess risk estimates of 2×10^{-4} and 3×10^{-5} would be reported as 2×10^{-4}, not 2.3×10^{-4}. Hazard indices (HIs, sums of HQs) are presented with one significant figure if they are less than 1, or to the nearest integer if they are greater than 1. This is to allow the reader to follow summations. For example, HQs of 4 and 10 would be summed to an HI of 14, not 10. However, HQs of 0.01 and 0.001 would be summed to an HI of 0.01, not 0.011.

Risks associated with surface water recreation, although not explicitly estimated in this HHRA, were also considered as part of the cumulative risk evaluation. Risk estimates calculated by King County (1999d) for highly exposed adult and child swimmers were added to the risk estimate sums described above for several scenarios (e.g., the seafood consumption and netfishing scenarios).

B.5.3 RISK CHARACTERIZATION RESULTS

This section presents the results for each exposure scenario: seafood consumption (Section B.5.3.1), netfishing (Section B.5.3.2), beach play RME (Section B.5.3.3), and clamming (Section B.5.3.4). Excess cancer risks and HQs for the various exposure scenarios are presented in tables, as appropriate, in these subsections. Additional scenarios and associated risk estimates for dog walkers and habitat restoration workers are also discussed in the uncertainty analysis (Section B.6.1.9).

Detection frequency was quite variable across chemicals and media. Low detection frequency affects the confidence in the consistent presence of a chemical in a particular medium as well as the ability to generate a UCL on the mean. Each risk estimate for

seafood consumption scenarios that included a market basket of seafood categories is based on consumption of five to seven seafood categories (not all scenarios included consumption of whole-body benthic fish and mussel samples were not analyzed for all COPCs). Many chemicals detected in tissue and sediment samples, particularly pesticides, were JN-qualified based on the tentative nature of their identification and quantification (as discussed in Section B.2.2). To distinguish these risk estimates, which carry a higher degree of uncertainty than risk estimates based on more definitive identification and quantification, the JN-qualified chemicals are grouped separately in the lower portion of the risk tables for both seafood consumption and direct sediment exposure scenarios. For cancer risk estimates, subtotals both including and excluding these tentatively identified chemicals are provided. In addition, for the seafood consumption scenarios, some chemicals were not detected in many of the seafood categories (e.g., crab whole body, mussels) included in the risk calculations. The contribution to the total risk estimate from seafood categories for which there were no detected concentrations sometimes exceeded 50% because of the use of RLs in deriving the EPC term. For these chemicals, the risk estimates are footnoted. Many of these chemicals were also JN-qualified pesticides, indicating greater uncertainty in the resultant risk estimates. The uncertainties related to analytical identification and detection limit issues are discussed in greater detail in the uncertainty analysis (Section B.6).

PCBs were found to be associated with high risks for seafood consumption exposure scenarios. In examining the risks posed by PCBs, it is important to understand issues stemming from the use of commercial PCB mixture toxicity information to describe the toxicity of environmental PCB mixtures. PCBs consist of 209 individual congeners. Aroclors are commercial mixtures of PCB congeners that contain a large number of individual congeners. The different Aroclors contain many of the same congeners and vary mostly in terms of the relative abundance of specific congeners. After a commercial mixture is released into the environment, the original congener composition of the commercial PCB mixture changes over time through various processes (e.g., partitioning between environmental media, chemical transformation, and preferential bioaccumulation) (Cogliano 1998). The assessment of cancer risks for environmental PCB mixtures is complicated in that carcinogenicity data are available for commercial but not environmental mixtures. Consequently, the carcinogenicity of commercial mixtures must be used to estimate the toxicity of environmental mixtures that may have a different congener composition than the Aroclors used to develop the carcinogenicity data. Cancer risks for environmental PCB mixtures may be estimated on the basis of either: 1) commercial Aroclor toxicity (hereafter referred to as total PCB risks), or 2) the toxicity of specific components of Aroclor mixtures (i.e., co-planar PCB congeners that have a mode of toxicity similar to that of dioxin [hereafter referred to as PCB TEQ risks]). Total PCB cancer risks are computed by multiplying the total PCB CDI by the SF for PCBs (as Aroclors). As discussed in Section B.4, after making appropriate adjustments for the TEFs for the individual dioxin-like PCB congeners,

PCB TEQ cancer risks are computed by multiplying the PCB TEQ CDI by the dioxin SF.

Challenges exist in using total PCB and PCB TEQ cancer risk estimates to represent the true risks posed by environmental PCB mixtures. Descriptions of the cancer risks posed by environmental PCB mixtures are bounded on the low end by use of total PCB or PCB TEQ cancer risk estimates and bounded on the high end by adding total PCB and PCB TEQ cancer risk estimates together. There are issues with both of these approaches. Environmental processes (e.g., bioaccumulation) may increase levels of more highly chlorinated and potentially more toxic congeners (e.g., co-planar PCBs with dioxin-like toxicity) relative to those found in commercial PCB mixtures (EPA 1996b). Hence, using either total PCB or PCB TEQ cancer risk estimates alone to describe overall environmental PCB cancer risks may underestimate the true risk posed by an environmental PCB mixture. However, adding total PCB and PCB TEQ cancer risks may overestimate the true risk posed by an environmental PCB mixture. Co-planar PCBs were present in the commercial mixtures used to derive the Aroclor SF; hence, there is a likely potential for "double counting" the risk posed by the co-planar PCBs when adding total PCB and PCB TEQ cancer risks.

EPA's approach to characterizing PCB cancer risks has varied based on the availability of congener analyses at specific sites, the PCBs that were released into the environment (i.e., which specific Aroclor was released), and the site-specific environmental processes. EPA guidance suggests approaches for combining total PCB and PCB TEQ risks in the *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures* (EPA 2000e) and in P*CBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures* (EPA 1996b). These guidance documents suggest an approach roughly equivalent to adding total PCB and PCB TEQ risks. The Hudson and Housatonic Superfund site risk assessments presented total PCB and PCB TEQ risks separately. In the case of the Hudson River PCB assessment, the risks were not combined, and a qualitative discussion of PCB toxicity was presented in the risk characterization (TAMS and Gradient 2000). In EPA Region 10's evaluation of risks posed by PCBs in Columbia River fish (EPA 2002b), Aroclor and PCB TEQ risks were added together (EPA 2002b). The uncertainty section of the HHRA (Section B.6) should be consulted for a more complete discussion of the issues associated with the use of total PCB and PCB TEQ risks in characterizing the overall risks posed by environmental PCB mixtures.

EPA, Ecology, and LDWG agreed to present total PCB and PCB TEQ risks separately in the LDW HHRA. Two different total excess cancer risk estimates are provided in the tables that follow: one includes all COPC risks except those for PCB TEQ (i.e., includes risks from total PCBs and all other COPCs); the other includes all COPCs, plus the PCB TEQ and excludes total PCBs. As shown in summary tables in Section B.5.6, whether total PCB and PCB TEQ excess cancer risks were considered separately or summed, the risks would be unacceptable. The uncertainty associated

with PCB risk characterization as well as alternative options for consideration of total risk, including both total PCBs (as Aroclors) and PCB TEQ estimates, are discussed in the uncertainty analysis (Section B.6).The adult one-meal-per-month risk estimates may be used to inform the public about the risks that might occur if they were to consume specific seafood type(s) from the LDW with a particular frequency. It should be noted that one-meal-per-month risk estimates are not meant to describe the actual behavior of any group that may consume seafood from the LDW (e.g., recreational anglers). These risk estimates can be adjusted to account for specific patterns of higher or lower consumption and consumption of multiple seafood categories. For example, if someone eats two meals per month of LDW crab and one meal per month of LDW pelagic fish, he or she could multiply the one-meal-per-month crab risk estimate by two and add the product to the one-meal-per-month pelagic fish risk estimate to approximate the risk associated with his or her own LDW seafood consumption. It is important to recognize the assumptions of the one-meal-per-month scenarios, however (see Table B.3-14), including the assumption that LDW seafood is consumed at this rate for 30 years. For exposure durations less than 30 years, risks might be overestimated. Similarly, if the exposure duration is more than 30 years, risks might be underestimated. As with other seafood consumption risk estimates presented in this section, it was assumed for the one-meal-per-month scenario that the concentrations of chemicals in tissue (i.e., the EPCs summarized in Table B.3-39) do not change over time.

B.5.3.1 Seafood consumption

B.5.3.1.1 Excess cancer risk estimates

Total upper bound excess cancer estimates for seafood consumption significantly exceeded 1×10^{-6} for each of the scenarios evaluated (Tables B.5-1 through B.5-8), regardless of the PCB summation approach (i.e., the inclusion of total PCBs or PCB TEQ in the sum). The highest cancer risk estimates were for the adult tribal scenario based on Suquamish data (3×10^{-2}) (Table B.5-5), followed by the adult tribal RME scenario based on Tulalip data ($\leq 3 \times 10^{-3}$, Table B.5-1), the adult API RME scenario (1×10^{-3},Table B.5-6), and the child tribal RME scenario based on Tulalip data ($\leq 8 \times 10\text{-}^{4}$, Table B.5-3). The lowest risk estimates were for the adult one-meal-per-month crab consumption scenario (4×10^{-5}) (Table B.5-8).

Total excess cancer risk estimates for all the adult one-meal-per-month seafood consumption scenarios (representing one meal per month of the indicated seafood categories; Table B.5-8) and the CT scenarios (adult tribal scenario based on Tulalip data, child tribal scenario based on Tulalip data, and adult API scenario) (Tables B.5-2, B.5-4, and B.5-7, respectively) were one or more orders of magnitude lower than those for the adult tribal RME scenario based on Tulalip data (Table B.5-1). For the adult one-meal-per-month scenarios, total excess cancer risks estimates were highest for pelagic fish and clam consumption and lowest for crab consumption. PCBs were the dominant chemical contributor to the excess cancer risks for benthic fish, crab, and

pelagic fish one-meal-per-month consumption, and arsenic was the dominant contributor for clam one-meal-per-month consumption.

The differences in risk estimates across these scenarios reflect the differences in the overall rates of seafood consumed and differences in the relative consumption rates for various seafood categories within each scenario, which resulted in dissimilar estimates of chemical intakes among the scenarios. Differences in body weight and exposure duration across scenarios also contributed to the different risk estimates.

Table B.5-1. Excess cancer risk estimates for the adult tribal RME seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Excess Cancer Risk
Arsenic[b, c]	Table B.3-34	9.7×10^{-4}	1.5	1×10^{-3}
Bis(2-ethylhexyl) phthalate	Table B.3-36	4.5×10^{-4}	0.014	6×10^{-6}
cPAHs[b, d]	Table B.3-36	9.8×10^{-6}	7.3	7×10^{-5}
Dioxin/furan TEQ[e]	na	na	na	na
PCB TEQ[b]	Table B.3-35	8.1×10^{-9}	150,000	1×10^{-3}
Total PCBs	Table B.3-35	8.0×10^{-4}	2	2×10^{-3}
Pentachlorophenol[e]	Table B.3-36	7.3×10^{-4}	0.12	9×10^{-5}
			Subtotal excluding PCB TEQ	**3×10^{-3}**
			Subtotal excluding total PCBs	**2×10^{-3}**
Tentatively identified chemicals (JN-qualified)				
Aldrin[f]	Table B.3-35	2.8×10^{-6}	17	5×10^{-5}
alpha-BHC[f]	Table B.3-35	2.6×10^{-6}	6.3	2×10^{-5}
beta-BHC[f]	Table B.3-35	3.5×10^{-6}	1.8	6×10^{-6}
Carbazole	Table B.3-36	2.3×10^{-3}	0.02	5×10^{-5}
Total chlordane	Table B.3-35	1.7×10^{-5}	0.35	6×10^{-6}
Total DDTs	Table B.3-35	6.2×10^{-5}	0.34	2×10^{-5}
Dieldrin	Table B.3-35	8.2×10^{-6}	16	1×10^{-4}
gamma-BHC	Table B.3-35	4.2×10^{-6}	1.3	6×10^{-6}
Heptachlor[f]	Table B.3-35	3.2×10^{-6}	4.5	1×10^{-5}
Heptachlor epoxide	Table B.3-35	3.4×10^{-6}	9.1	3×10^{-5}
Hexachlorobenzene	Table B.3-36	6.5×10^{-6}	1.6	1×10^{-5}
			Subtotal	**3×10^{-4}**
Total excess cancer risk (excluding PCB TEQ)				**3×10^{-3}**
Total excess cancer risk (excluding total PCBs)				**2×10^{-3}**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for cPAH concentrations are from only 2004; historical data were not used because of high reporting limits. Risks related to 2004 and historical cPAH data are analyzed in the uncertainty analysis (Section B.6).

[e] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

[f] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

Table B.5-2. Excess cancer risk estimates for the adult tribal CT seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk
Arsenic[b, c]	Table B.3-34	4.1×10^{-5}	1.5	6×10^{-5}
Bis(2-ethylhexyl) phthalate	Table B.3-36	1.2×10^{-5}	0.014	2×10^{-7}
cPAHs[b, d]	Table B.3-36	5.1×10^{-7}	7.3	4×10^{-6}
Dioxin/furan TEQ[e]	na	na	na	na
PCB TEQ[b]	Table B.3-35	3.8×10^{-10}	150,000	6×10^{-5}
Total PCBs	Table B.3-35	3.2×10^{-5}	2	6×10^{-5}
Pentachlorophenol[f]	Table B.3-36	1.4×10^{-5}	0.12	2×10^{-6}
			Subtotal excluding PCB TEQ	**1×10^{-4}**
			Subtotal excluding total PCBs	**1×10^{-4}**
Tentatively identified chemicals (JN-qualified)				
Aldrin[f]	Table B.3-35	8.7×10^{-8}	17	1×10^{-6}
alpha-BHC[f]	Table B.3-35	8.6×10^{-8}	6.3	5×10^{-7}
beta-BHC[f]	Table B.3-35	1.4×10^{-7}	1.8	3×10^{-7}
Carbazole	Table B.3-36	5.0×10^{-5}	0.02	1×10^{-6}
Total chlordane	Table B.3-35	5.7×10^{-7}	0.35	2×10^{-7}
Total DDTs	Table B.3-35	3.1×10^{-6}	0.34	1×10^{-6}
Dieldrin	Table B.3-35	1.6×10^{-7}	16	3×10^{-6}
gamma-BHC	Table B.3-35	9.8×10^{-8}	1.3	1×10^{-7}
Heptachlor[f]	Table B.3-35	8.9×10^{-8}	4.5	4×10^{-7}
Heptachlor epoxide	Table B.3-35	1.3×10^{-7}	9.1	1×10^{-6}
Hexachlorobenzene	Table B.3-36	1.5×10^{-7}	1.6	2×10^{-7}
			Subtotal	**9×10^{-6}**
Total excess cancer risk (excluding PCB TEQ)				**1×10^{-4}**
Total excess cancer risk (excluding total PCBs)				**1×10^{-4}**

[a] The values used for EPCs in the CT scenario were based on the mean concentration for each seafood category. These EPCs were calculated using one-half the RL for non-detects (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for cPAH concentrations are from only 2004; historical data were not used because of high reporting limits. Risks related to 2004 and historical cPAH data are analyzed in the uncertainty analysis (Section B.6).

[e] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans

[f] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

Table B.5-3. Excess cancer risk estimates for the child tribal RME seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor (kg/mg-day)$^{-1}$	Excess Cancer Risk
Arsenic[b, c]	Table B.3-34	1.8×10^{-4}	1.5	3×10^{-4}
Bis(2-ethylhexyl) phthalate	Table B.3-36	8.3×10^{-5}	0.014	1×10^{-6}
cPAHs[b, d, e]	Table B.3-36	1.8×10^{-6}	7.3	7×10^{-5}
Dioxin/furan TEQ[f]	na	na	na	na
PCB TEQ[b]	Table B.3-35	1.5×10^{-9}	150,000	2×10^{-4}
Total PCBs	Table B.3-35	1.5×10^{-4}	2	3×10^{-4}
Pentachlorophenol[f]	Table B.3-36	1.3×10^{-4}	0.12	2×10^{-5}
			Subtotal excluding PCB TEQ	**7×10^{-4}**
			Subtotal excluding total PCBs	**6×10^{-4}**
Tentatively identified chemicals (JN-qualified)				
Aldrin[g]	Table B.3-35	5.2×10^{-7}	17	9×10^{-6}
alpha-BHC[g]	Table B.3-35	4.8×10^{-7}	6.3	3×10^{-6}
beta-BHC[g]	Table B.3-35	6.5×10^{-7}	1.8	1×10^{-6}
Carbazole	Table B.3-36	4.2×10^{-4}	0.02	8×10^{-6}
Total chlordane	Table B.3-35	3.1×10^{-6}	0.35	1×10^{-6}
Total DDTs	Table B.3-35	1.1×10^{-5}	0.34	4×10^{-6}
Dieldrin	Table B.3-35	1.5×10^{-6}	16	2×10^{-5}
gamma-BHC	Table B.3-35	7.8×10^{-7}	1.3	1×10^{-6}
Heptachlor	Table B.3-35	5.9×10^{-7}	4.5	3×10^{-6}
Heptachlor epoxide[g]	Table B.3-35	6.2×10^{-7}	9.1	6×10^{-6}
Hexachlorobenzene	Table B.3-36	1.2×10^{-6}	1.6	2×10^{-6}
			Subtotal	**6×10^{-5}**
Total excess cancer risk (excluding PCB TEQ)				**8×10^{-4}**
Total excess cancer risk (excluding total PCBs)				**7×10^{-4}**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic

[d] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for carcinogenic PAHs is based on dose adjustments across the 0-to-6-year age range of children. See section B.5.1 for more information.

[e] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for cPAH concentrations are from only 2004; historical data were not used because of high reporting limits. Risks related to 2004 and historical cPAH data are analyzed in the uncertainty analysis (Section B.6).

[f] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

[g] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

Table B.5-4. Excess cancer risk estimates for the child tribal CT seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk
Arsenic[b,c]	Table B.3-34	1.8×10^{-5}	1.5	3×10^{-5}
Bis(2-ethylhexyl) phthalate	Table B.3-36	5.2×10^{-6}	0.014	7×10^{-8}
cPAHs[b,d,e]	Table B.3-36	2.1×10^{-7}	7.3	8×10^{-6}
Dioxin/furan TEQ[f]	na	na	na	na
PCB TEQ[b]	Table B.3-35	1.5×10^{-10}	150,000	2×10^{-5}
Total PCBs	Table B.3-35	1.4×10^{-5}	2	3×10^{-5}
Pentachlorophenol[g]	Table B.3-36	5.9×10^{-6}	0.12	7×10^{-7}
			Subtotal excluding PCB TEQ	**7×10^{-5}**
			Subtotal excluding total PCBs	**6×10^{-5}**
Tentatively identified chemicals (JN-qualified)				
Aldrin[g]	Table B.3-35	3.7×10^{-8}	17	6×10^{-7}
alpha-BHC[g]	Table B.3-35	3.7×10^{-8}	6.3	2×10^{-7}
beta-BHC[g]	Table B.3-35	6.0×10^{-8}	1.8	1×10^{-7}
Carbazole	Table B.3-36	2.2×10^{-5}	0.02	4×10^{-7}
Total chlordane	Table B.3-35	2.5×10^{-7}	0.35	9×10^{-8}
Total DDTs	Table B.3-35	1.3×10^{-6}	0.34	4×10^{-7}
Dieldrin	Table B.3-35	6.9×10^{-8}	16	1×10^{-6}
gamma-BHC	Table B.3-35	4.2×10^{-8}	1.3	5×10^{-8}
Heptachlor[g]	Table B.3-35	3.8×10^{-8}	4.5	2×10^{-7}
Heptachlor epoxide	Table B.3-35	5.6×10^{-8}	9.1	5×10^{-7}
Hexachlorobenzene	Table B.3-36	6.6×10^{-8}	1.6	1×10^{-7}
			Subtotal	**4×10^{-6}**
Total excess cancer risk (excluding PCB TEQ)				**7×10^{-5}**
Total excess cancer risk (excluding total PCBs)				**6×10^{-5}**

[a] The values used for EPCs in the CT scenario were based on the mean concentration for each seafood category. These EPCs were calculated using one-half the RL for non-detects (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for carcinogenic PAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[e] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for cPAH concentrations are from only 2004; historical data were not used because of high reporting limits. Risks related to 2004 and historical cPAH data are analyzed in the uncertainty analysis (Section B.6).

[f] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

[g] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

Table B.5-5. Excess cancer risk estimates for the adult tribal seafood consumption scenario based on Suquamish data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor Age: Adult

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk
Arsenic[b, c, d]	Table B.3-34	1.1×10^{-2}	1.5	2×10^{-2}
Bis(2-ethylhexyl) phthalate	Table B.3-36	2.8×10^{-3}	0.014	4×10^{-5}
cPAHs[b, e]	Table B.3-36	1.1×10^{-4}	7.3	8×10^{-4}
Dioxin/furan TEQ[f]	na	na	na	na
PCB TEQ[b]	Table B.3-35	4.9×10^{-8}	150,000	7×10^{-3}
Total PCBs[d]	Table B.3-35	5.5×10^{-3}	2	1×10^{-2}
Pentachlorophenol[g]	Table B.3-36	4.0×10^{-3}	0.12	5×10^{-4}
			Subtotal excluding PCB TEQ	**3×10^{-2}**
			Subtotal excluding total PCBs	**3×10^{-2}**
Tentatively identified chemicals (JN-qualified)				
Aldrin	Table B.3-35	1.2×10^{-5}	17	2×10^{-4}
alpha-BHC	Table B.3-35	8.9×10^{-6}	6.3	6×10^{-5}
beta-BHC	Table B.3-35	1.6×10^{-5}	1.8	3×10^{-5}
Carbazole	Table B.3-36	1.2×10^{-2}	0.02	2×10^{-4}
Total chlordane	Table B.3-35	1.0×10^{-4}	0.35	3×10^{-5}
Total DDTs	Table B.3-35	3.2×10^{-4}	0.34	1×10^{-4}
Dieldrin	Table B.3-35	7.3×10^{-5}	16	1×10^{-3}
gamma-BHC	Table B.3-35	2.0×10^{-5}	1.3	3×10^{-5}
Heptachlor	Table B.3-35	1.3×10^{-5}	4.5	6×10^{-5}
Heptachlor epoxide	Table B.3-35	1.9×10^{-5}	9.1	2×10^{-4}
Hexachlorobenzene	Table B.3-36	2.2×10^{-5}	1.6	4×10^{-5}
			Subtotal	**2×10^{-3}**
Total excess cancer risk (excluding PCB TEQ)				**3×10^{-2}**
Total excess cancer risk (excluding total PCBs)				**3×10^{-2}**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic

[d] Because the excess cancer risk is greater than or equal to 0.01 (1×10^{-2}), risk was calculated using the exponential equation in EPA (1989).

[e] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for cPAH concentrations are from only 2004; historical data were not used because of high reporting limits. Risks related to 2004 and historical cPAH data are analyzed in the uncertainty analysis (Section B.6).

[f] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

[g] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

Table B.5-6. Excess cancer risks for the API RME seafood consumption scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Asian and Pacific Islander fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Excess Cancer Risk
Arsenic[b, c]	Table B.3-34	4.4×10^{-4}	1.5	7×10^{-4}
Bis(2-ethylhexyl) phthalate	Table B.3-36	1.3×10^{-4}	0.014	2×10^{-6}
cPAHs[b, d]	Table B.3-36	4.4×10^{-6}	7.3	3×10^{-5}
Dioxin/furan TEQ[e]	na	na	na	na
PCB TEQ[b]	Table B.3-35	2.6×10^{-9}	150,000	4×10^{-4}
Total PCBs	Table B.3-35	2.5×10^{-4}	2	5×10^{-4}
Pentachlorophenol	Table B.3-36	1.8×10^{-4}	0.12	2×10^{-5}
			Subtotal excluding PCB TEQ	**1×10^{-3}**
			Subtotal excluding total PCBs	**1×10^{-3}**
Tentatively identified chemicals (JN-qualified)				
Aldrin	Table B.3-35	6.0×10^{-7}	17	1×10^{-5}
alpha-BHC	Table B.3-35	5.0×10^{-7}	6.3	3×10^{-6}
beta-BHC	Table B.3-35	8.0×10^{-7}	1.8	1×10^{-6}
Carbazole	Table B.3-36	5.9×10^{-4}	0.02	1×10^{-5}
Total chlordane	Table B.3-35	5.0×10^{-6}	0.35	2×10^{-6}
Total DDTs	Table B.3-35	1.7×10^{-5}	0.34	6×10^{-6}
Dieldrin	Table B.3-35	2.8×10^{-6}	16	5×10^{-5}
gamma-BHC	Table B.3-35	1.0×10^{-6}	1.3	1×10^{-6}
Heptachlor	Table B.3-35	7.1×10^{-7}	4.5	3×10^{-6}
Heptachlor epoxide	Table B.3-35	9.8×10^{-7}	9.1	9×10^{-6}
Hexachlorobenzene	Table B.3-36	1.5×10^{-6}	1.6	2×10^{-6}
			Subtotal	**1×10^{-4}**
Total excess cancer risk (excluding PCB TEQ)				**1×10^{-3}**
Total excess cancer risk (excluding total PCBs)				**1×10^{-3}**

[a] An EPC for each seafood category was calculated in the exposure section (Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[e] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

API – Asian and Pacific Islander

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

Table B.5-7. Excess cancer risks for the API CT seafood consumption scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Asian and Pacific Islander fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk
Arsenic[b, c]	Table B.3-34	8.5×10^{-6}	1.5	1×10^{-5}
Bis(2-ethylhexyl) phthalate	Table B.3-36	1.9×10^{-6}	0.014	3×10^{-8}
cPAHs[b, d]	Table B.3-36	1.0×10^{-7}	7.3	8×10^{-7}
Dioxin/furan TEQ[e]	na	na	na	na
PCB TEQ[b]	Table B.3-35	5.0×10^{-11}	150,000	7×10^{-6}
Total PCBs	Table B.3-35	4.2×10^{-6}	2	8×10^{-6}
Pentachlorophenol	Table B.3-36	2.1×10^{-6}	0.12	3×10^{-7}
			Subtotal excluding PCB TEQ	**2×10^{-5}**
			Subtotal excluding total PCBs	**2×10^{-5}**
Tentatively identified chemicals (JN-qualified)				
Aldrin	Table B.3-35	9.2×10^{-9}	17	2×10^{-7}
alpha-BHC	Table B.3-35	8.9×10^{-9}	6.3	6×10^{-8}
beta-BHC	Table B.3-35	1.7×10^{-8}	1.8	3×10^{-8}
Carbazole	Table B.3-36	4.2×10^{-6}	0.02	8×10^{-8}
Total Chlordane	Table B.3-35	7.9×10^{-8}	0.35	3×10^{-8}
Total DDTs	Table B.3-35	4.0×10^{-7}	0.34	1×10^{-7}
Dieldrin	Table B.3-35	2.2×10^{-8}	16	4×10^{-7}
gamma-BHC	Table B.3-35	1.1×10^{-8}	1.3	1×10^{-8}
Heptachlor	Table B.3-35	9.4×10^{-9}	4.5	4×10^{-8}
Heptachlor epoxide	Table B.3-35	1.6×10^{-8}	9.1	1×10^{-7}
Hexachlorobenzene	Table B.3-36	2.2×10^{-8}	1.6	3×10^{-8}
			Subtotal	**1×10^{-6}**
Total excess cancer risk (excluding PCB TEQ)				**2×10^{-5}**
Total excess cancer risk (excluding total PCBs)				**2×10^{-5}**

[a] The values used for EPCs in the CT scenario were based on the mean concentration for each seafood category. These EPCs were calculated using one-half the RL for non-detects (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[e] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

API – Asian and Pacific Islander

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

Table B.5-8. Excess cancer risk estimates associated with the consumption of one-meal-per-month of seafood by adults

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Adult one-meal-per-month fish and shellfish consumers
Receptor age: Adult

Chemical	Seafood Category	EPC (mg/kg ww)	Cancer CDI (mg/kg-day)	Cancer Slope Factor (mg/kg-day)	Excess Cancer Risk
Arsenic[a]	benthic fish	0.0062	2.8×10^{-7}	1.5	4×10^{-7}
	clam	2.0	9.0×10^{-5}	1.5	1×10^{-4}
	crab edible meat	0.042	1.9×10^{-6}	1.5	3×10^{-6}
	pelagic fish	0.086	3.9×10^{-6}	1.5	6×10^{-6}
Bis(2-ethylhexyl) phthalate	benthic fish	1.3	5.8×10^{-5}	0.014	8×10^{-7}
	clam	0.13	5.8×10^{-6}	0.014	8×10^{-8}
	crab edible meat[b]	0.13	5.8×10^{-6}	0.014	8×10^{-8}
	pelagic fish	2.1	9.4×10^{-5}	0.014	1×10^{-6}
cPAHs[c]	benthic fish	0.0064	2.9×10^{-8}	7.3	2×10^{-7}
	clam	0.02	9.0×10^{-7}	7.3	7×10^{-6}
	crab edible meat	0.00065	2.9×10^{-8}	7.3	2×10^{-7}
	pelagic fish	0.00095	4.3×10^{-8}	7.3	3×10^{-7}
Dioxin/furan TEQ[d]	benthic fish	na	na	na	na
	clam	na	na	na	na
	crab edible meat	na	na	na	na
	pelagic fish	na	na	na	na
PCB TEQ	benthic fish	1.17×10^{-5}	5.2×10^{-10}	150,000	8×10^{-5}
	clam	3.16×10^{-6}	1.4×10^{-10}	150,000	2×10^{-5}
	crab edible meat	2.41×10^{-6}	1.1×10^{-10}	150,000	2×10^{-5}
	pelagic fish	3.37×10^{-5}	1.5×10^{-9}	150,000	2×10^{-4}
Total PCBs	benthic fish	1.2	5.4×10^{-5}	2	1×10^{-4}
	clam	0.6	2.7×10^{-5}	2	5×10^{-5}
	crab edible meat	0.20	9.0×10^{-6}	2	2×10^{-5}
	pelagic fish	1.9	8.5×10^{-5}	2	2×10^{-4}
Pentachlorophenol	benthic fish[b]	2.9	1.3×10^{-4}	0.12	2×10^{-5}
	clam[b]	0.2	9.0×10^{-6}	0.12	1×10^{-6}
	crab edible meat[b]	0.29	1.3×10^{-5}	0.12	2×10^{-6}
	pelagic fish	2.4	1.1×10^{-4}	0.12	1×10^{-5}
Subtotal excluding PCB TEQ				**benthic fish**	**1×10^{-4}**
				clam	**2×10^{-4}**
				crab edible meat	**3×10^{-5}**

Chemical	Seafood Category	EPC (mg/kg ww)	Cancer CDI (mg/kg-day)	Cancer Slope Factor (mg/kg-day)	Excess Cancer Risk
				pelagic fish	**2×10^{-4}**
Subtotal excluding Total PCBs				**benthic fish**	**1×10^{-4}**
				clam	**1×10^{-4}**
				crab edible meat	**3×10^{-5}**
				pelagic fish	**2×10^{-4}**
Tentatively identified chemicals (JN-qualified)					
Aldrin	benthic fish[b]	0.0036	1.6×10^{-7}	17	3×10^{-6}
	clam[b]	0.0010	4.5×10^{-8}	17	8×10^{-7}
	crab edible meat[b]	0.0036	1.6×10^{-7}	17	3×10^{-6}
	pelagic fish	0.0036	1.6×10^{-7}	17	3×10^{-6}
alpha-BHC	benthic fish	0.0036	1.6×10^{-7}	6.3	1×10^{-6}
	clam	0.00050	2.2×10^{-8}	6.3	1×10^{-7}
	crab edible meat[b]	0.0036	1.6×10^{-7}	6.3	1×10^{-6}
	pelagic fish	0.0036	1.6×10^{-7}	6.3	1×10^{-6}
beta-BHC	benthic fish	0.0036	1.6×10^{-7}	1.8	3×10^{-7}
	clam	0.00050	5.4×10^{-8}	1.8	1×10^{-7}
	crab edible meat[b]	0.0036	1.8×10^{-7}	1.8	3×10^{-7}
	pelagic fish	0.0036	3.5×10^{-7}	1.8	6×10^{-7}
Carbazole	benthic fish[b]	1.5	6.7×10^{-5}	0.02	1×10^{-6}
	clam[b]	0.10	4.5×10^{-6}	0.02	9×10^{-8}
	crab edible meat[b]	1.5	6.7×10^{-5}	0.02	1×10^{-6}
	pelagic fish	14	6.3×10^{-4}	0.02	1×10^{-5}
Total chlordane	benthic fish	0.020	9.0×10^{-7}	0.35	3×10^{-7}
	clam	0.0047	2.1×10^{-7}	0.35	7×10^{-8}
	crab edible meat	0.0045	2.0×10^{-7}	0.35	7×10^{-8}
	pelagic fish	0.084	3.8×10^{-6}	0.35	1×10^{-6}
Total DDTs	benthic fish	0.084	3.8×10^{-6}	0.34	1×10^{-6}
	clam	0.015	6.7×10^{-7}	0.34	2×10^{-7}
	crab edible meat	0.023	1.0×10^{-6}	0.34	4×10^{-7}
	pelagic fish	0.24	1.1×10^{-5}	0.34	4×10^{-6}
Dieldrin	benthic fish[b]	0.0036	1.6×10^{-7}	16	3×10^{-6}
	clam	0.012	5.4×10^{-7}	16	9×10^{-6}
	crab edible meat	0.0036	1.6×10^{-7}	16	3×10^{-6}
	pelagic fish[a]	0.0036	1.6×10^{-7}	16	3×10^{-6}
gamma-BHC	benthic fish[a]	0.0036	1.6×10^{-7}	1.3	2×10^{-7}
	clam	0.0025	1.1×10^{-7}	1.3	1×10^{-7}
	crab edible meat	0.0040	1.8×10^{-7}	1.3	2×10^{-7}
	pelagic fish	0.0020	9.0×10^{-8}	1.3	1×10^{-7}

Chemical	Seafood Category	EPC (mg/kg ww)	Cancer CDI (mg/kg-day)	Cancer Slope Factor (mg/kg-day)	Excess Cancer Risk
Heptachlor	benthic fish[b]	0.0036	1.6×10^{-7}	4.5	7×10^{-7}
	clam[b]	0.00050	2.2×10^{-8}	4.5	1×10^{-7}
	crab edible meat[b]	0.0036	1.6×10^{-7}	4.5	7×10^{-7}
	pelagic fish	0.0097	4.3×10^{-7}	4.5	2×10^{-6}
Heptachlor epoxide	benthic fish[b]	0.0036	1.6×10^{-7}	9.1	1×10^{-6}
	clam	0.0015	6.7×10^{-8}	9.1	6×10^{-7}
	crab edible meat	0.0021	9.4×10^{-8}	9.1	9×10^{-7}
	pelagic fish	0.0010	4.5×10^{-7}	9.1	4×10^{-6}
Hexachlorobenzene	benthic fish	0.0090	4.0×10^{-7}	1.6	6×10^{-7}
	clam	0.00086	3.8×10^{-8}	1.6	6×10^{-8}
	crab edible meat	0.0080	3.6×10^{-7}	1.6	6×10^{-7}
	pelagic fish	0.012	5.4×10^{-7}	1.6	9×10^{-7}
Subtotal				**benthic fish**	**1×10^{-5}**
				clam	**1×10^{-5}**
				crab edible meat	**1×10^{-5}**
				pelagic fish	**3×10^{-5}**
Total excess cancer risk (excluding PCB TEQ)				**benthic fish**	**1×10^{-4}**
				clam	**2×10^{-4}**
				crab edible meat	**4×10^{-5}**
				pelagic fish	**2×10^{-4}**
Total excess cancer risk (excluding total PCBs)				**benthic fish**	**1×10^{-4}**
				clam	**1×10^{-4}**
				crab edible meat	**4×10^{-5}**
				pelagic fish	**2×10^{-4}**

[a] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[b] No detected values in this seafood category. CDI and risk estimate are based on one-half the maximum reporting limit.

[c] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[d] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

ww – wet weight

The majority of the total excess cancer risk for all the seafood consumption scenarios is attributable to a combination of PCBs (either total PCBs or PCB TEQ) and inorganic arsenic. For most scenarios, estimates of total excess cancer risk were similar regardless of the summation approach applied to PCBs (i.e., whether based on inclusion of PCB TEQ or total PCBs in the sum). For all scenarios, the total PCBs excess cancer risk estimate was equal to or greater than the PCB TEQ excess cancer risk, but differences were not more than two-fold for all scenarios except the adult one-meal-per-month clam scenario. For the adult tribal RME seafood consumption scenario based on the Tulalip Tribes' data, PCB TEQ risks were 1×10^{-3}, while total PCBs risks were 2×10^{-3}. Because PCB congeners were not analyzed in mussels (as described in Section B.3.4.3), for the calculation of PCB TEQ risks for consumption scenarios that included a market basket of seafood categories, the consumption of mussels was apportioned to other seafood categories. Because mussel consumption made up only a small percentage of total seafood consumption in these scenarios, this reapportioning had little impact on total risk estimates.

Tissue data for dioxins/furans were not collected. The calculated risk results do not include the unknown contribution to exposure and risks from dioxins/furans. Dioxins/furans are nevertheless assumed to contribute risks and are identified as risk drivers (see Section B.7).

All chemicals evaluated in the risk characterization for the adult tribal RME scenario based on Tulalip data were designated COCs based on excess cancer risk estimates greater than 1×10^{-6}, including chemicals that were detected infrequently as well as those with high detection frequencies such as arsenic, total PCBs, and PCB TEQ. For the child tribal RME scenario based on Tulalip data and adult API RME scenario, most but not all COPCs were identified as COCs. For the adult and child tribal CT scenarios based on Tulalip data, many fewer chemicals (5 and 4, respectively) were exceeded 1×10^{-6} excess cancer risk. For the adult API CT scenario, only arsenic, total PCBs, and PCB TEQ had excess cancer risk estimates that exceeded 1×10^{-6}. Inclusion of the JN-qualified chemicals did not change the total risk estimates for most scenarios, with the exception of the adult one-meal-per-month crab scenario which had the lowest risk estimates. As indicated in the footnotes to the tables for all scenarios that included a market basket of seafood categories, the majority of the risk for some of the JN-qualified chemicals was attributable to use of one-half the maximum RL as the EPC for some seafood categories. For the adult one-meal-per-month consumption scenarios, risk associated with many of the JN-qualified chemicals was related to the use of one-half the maximum RL for seafood categories for which there were no detected values. One-half the maximum RL was used as the EPC in a seafood category if there were no detected concentrations for that category. The uncertainty associated with risk estimates for JN-qualified and infrequently detected chemicals is discussed in the uncertainty analysis (Section B.6).

B.5.3.1.2 Non-cancer hazard estimates

Unacceptable non-cancer hazards (HIs > 1) were predicted for all seafood consumption scenarios. Effect-specific HIs were calculated for cardiovascular, developmental, hematologic, immunological, kidney, liver, neurological and dermal effects, as described in Section B.5.1.2. The chemicals associated with each endpoint are identified in the footnotes of Tables B.5-9 to B.5-16.

The developmental, immunological, and neurological HIs exceeded 1 for all scenarios except the adult one-meal-per-month – crab scenario, primarily because of PCBs. These HIs were relatively higher for the adult tribal scenario based on Suquamish data, the adult and child tribal RME scenarios based on Tulalip data, and API RME scenario than for the API CT and adult one-meal-per-month scenarios. Specifically, HIs of 41 were estimated for developmental, immunological, and neurological effects for the adult tribal RME scenario based on Tulalip data (Table B.5-9), and HIs of 87 or more were estimated for these three effects for the child tribal RME scenario based on Tulalip data (Table B.5-11). The adult tribal scenario based on Suquamish data had HIs that exceeded 275 for developmental, immunological, and neurological effects (Table B.5-13). The adult API RME HIs were 29 for developmental and neurological and 30 for immunological effects (Table B.5-14). In addition, the HIs for cardiovascular, hematological, kidney, liver, and dermal endpoints exceeded 1 for the adult tribal scenario based on Suquamish data (Table B.5-13). With the exception of the hematological and kidney endpoints, the HIs for these same effects exceeded 1 for one or more of the RME scenarios (but not the CT scenarios). All effects-specific HIs for adult one-meal-per-month scenarios were less than or equal to 10 (Table B.5-16). For all scenarios, the majority of the total developmental, neurological, and immunological HIs (> 80 to 90%) was attributable to PCBs, although arsenic had an HQ greater than 1 for four scenarios and TBT and vanadium had HQs greater than 1 for two scenarios. Two additional chemicals had HQs greater than 1 only for the adult tribal scenario based on Suquamish data. Only PCBs had an HQ >1 for the adult one-meal-per-month seafood consumption scenarios.

Table B.5-9. Non-cancer hazard estimates for the adult tribal RME seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
4-Methylphenol[b]	Table B.3-36	5.0×10^{-4}	0.005	0.1
Antimony	Table B.3-34	4.7×10^{-5}	0.0004	0.1
Arsenic[c, d]	Table B.3-34	9.7×10^{-4}	0.0003	3
Bis(2-ethylhexyl) phthalate	Table B.3-36	4.5×10^{-4}	0.02	0.02
Butyl benzyl phthalate[b]	Table B.3-36	5.4×10^{-4}	0.2	0.003
Cadmium	Table B.3-34	9.3×10^{-5}	0.001	0.09
Chromium	Table B.3-34	4.7×10^{-4}	0.003	0.2
Copper	Table B.3-34	8.6×10^{-3}	0.04	0.2
Mercury	Table B.3-34	5.1×10^{-5}	0.0001	0.5
Nickel	Table B.3-34	4.0×10^{-4}	0.02	0.02
Total PCBs	Table B.3-35	8.0×10^{-4}	0.00002	40
Pentachlorophenol[b]	Table B.3-36	7.3×10^{-4}	0.03	0.02
TBT (as ion)	Table B.3-34	2.1×10^{-4}	0.00015	1
Vanadium	Table B.3-34	8.3×10^{-4}	0.001	0.8
Zinc	Table B.3-34	3.4×10^{-2}	0.3	0.1
			Subtotal	46
Tentatively identified chemicals (JN-qualified)				
Aldrin[b]	Table B.3-35	2.8×10^{-6}	0.00003	0.09
alpha-BHC[b]	Table B.3-35	2.6×10^{-6}	0.0005	0.005
beta-BHC[b]	Table B.3-35	3.5×10^{-6}	0.0002	0.02
Total chlordane	Table B.3-35	1.7×10^{-5}	0.0005	0.03
Total DDTs	Table B.3-35	6.2×10^{-5}	0.0005	0.1
Dieldrin	Table B.3-35	8.2×10^{-6}	0.00005	0.2
Endrin[b]	Table B.3-35	3.0×10^{-6}	0.0003	0.01
Endrin aldehyde	Table B.3-35	1.1×10^{-5}	0.0003	0.04
gamma-BHC	Table B.3-35	4.2×10^{-6}	0.0003	0.01
Heptachlor[b]	Table B.3-35	3.2×10^{-6}	0.0005	0.006
Heptachlor epoxide	Table B.3-35	3.4×10^{-6}	0.000013	0.3
Hexachlorobenzene	Table B.3-36	6.5×10^{-6}	0.0008	0.008
			Subtotal	**0.8**
Hazard indices by effect:				
Hazard Index for cardiovascular endpoint[e]				**4**
Hazard Index for developmental endpoint[f]				**41**

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Hazard Index for hematologic endpoint[g]				**0.2**
Hazard Index for immunological endpoint[h]				**41**
Hazard index for kidney endpoint[i]				**0.4**
Hazard index for liver endpoint[j]				**1**
Hazard index for neurological endpoint[k]				**41**
Hazard index for dermal endpoint[l]				**3**
Total hazard index across all exposure routes/pathways[m]				**47**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 to B.3-36).

[b] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[d] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[e] Cardiovascular endpoint is for arsenic and vanadium.

[f] Developmental endpoint is for : PCBs and mercury.

[g] Hematologic endpoint is for antimony and zinc.

[h] Immunological endpoint is for PCBs and TBT.

[I] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[j] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[k] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[l] Dermal endpoint is for 4-methylphenol and arsenic.

[m] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Table B.5-10. Non-cancer hazard estimates for the adult tribal CT seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
4-Methylphenol[b]	Table B.3-36	4.5×10^{-5}	0.005	0.009
Antimony	Table B.3-34	4.0×10^{-6}	0.0004	0.01
Arsenic[c, d]	Table B.3-34	9.7×10^{-5}	0.0003	0.3
Bis(2-ethylhexyl) phthalate	Table B.3-36	2.8×10^{-5}	0.02	0.001
Butyl benzyl phthalate[b]	Table B.3-36	5.4×10^{-5}	0.2	0.0003
Cadmium	Table B.3-34	1.2×10^{-5}	0.001	0.01
Chromium	Table B.3-34	5.5×10^{-5}	0.003	0.02
Copper	Table B.3-34	1.1×10^{-3}	0.04	0.03
Mercury	Table B.3-34	6.9×10^{-6}	0.0001	0.07
Nickel	Table B.3-34	5.3×10^{-5}	0.02	0.003
Total PCBs	Table B.3-35	7.5×10^{-5}	0.00002	4
Pentachlorophenol[b]	Table B.3-36	3.2×10^{-5}	0.03	0.001
TBT (as ion)	Table B.3-34	2.4×10^{-5}	0.00015	0.2
Vanadium	Table B.3-34	1.1×10^{-4}	0.001	0.1
Zinc	Table B.3-34	4.8×10^{-3}	0.3	0.02
			Subtotal	**5**
Tentatively identified chemicals (JN-qualified)				
Aldrin[b]	Table B.3-35	2.0×10^{-7}	0.00003	0.007
alpha-BHC[b]	Table B.3-35	2.0×10^{-7}	0.0005	0.0004
beta-BHC[b]	Table B.3-35	3.3×10^{-7}	0.0002	0.002
Total chlordane	Table B.3-35	1.3×10^{-6}	0.0005	0.003
Total DDTs	Table B.3-35	7.1×10^{-6}	0.0005	0.01
Dieldrin	Table B.3-35	3.7×10^{-7}	0.00005	0.007
Endrin[b]	Table B.3-35	2.6×10^{-7}	0.0003	0.0009
Endrin aldehyde	Table B.3-35	3.0×10^{-7}	0.0003	0.001
gamma-BHC	Table B.3-35	2.3×10^{-7}	0.0003	0.0008
Heptachlor[b]	Table B.3-35	2.1×10^{-7}	0.0005	0.0004
Heptachlor epoxide	Table B.3-35	3.0×10^{-7}	0.000013	0.02
Hexachlorobenzene	Table B.3-36	3.6×10^{-7}	0.0008	0.0004
			Subtotal	**0.05**
Hazard indices by effect:				
Hazard Index for cardiovascular endpoint[e]				**0.4**
Hazard Index for developmental endpoint[f]				**4**
Hazard Index for hematologic endpoint[g]				**0.03**

CHEMICAL	EPC (mg/kg ww)[a]	NON-CANCER CDI (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT
Hazard Index for immunological endpoint[h]				**4**
Hazard index for kidney endpoint[i]				**0.05**
Hazard index for liver endpoint[j]				**0.1**
Hazard index for neurological endpoint[k]				**4**
Hazard index for dermal endpoint[l]				**0.3**
Total hazard index across all exposure routes/pathways[m]				**5**

[a] The values used for EPCs in the CT scenario were based on the mean concentration for each seafood category. These EPCs were calculated using one-half the RL for non-detects (see Tables B.3-34 through B.3-36).

[b] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[d] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[e] Cardiovascular endpoint is for arsenic and vanadium.

[f] Developmental endpoint is for PCBs and mercury.

[g] Hematologic endpoint is for antimony and zinc.

[h] Immunological endpoint is for PCBs and TBT.

[I] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[j] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[k] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[l] Dermal endpoint is for 4-methylphenol and arsenic.

[m] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Table B.5-11. Non-cancer hazard estimates for the child tribal RME seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (kg/mg-day)	Hazard Quotient
4-Methylphenol[b]	Table B.3-36	1.1×10^{-3}	0.005	0.2
Antimony	Table B.3-34	1.0×10^{-4}	0.0004	0.3
Arsenic[c, d]	Table B.3-34	2.1×10^{-3}	0.0003	7
Bis(2-ethylhexyl) phthalate	Table B.3-36	9.7×10^{-4}	0.02	0.05
Butyl benzyl phthalate[b]	Table B.3-36	1.2×10^{-3}	0.2	0.006
Cadmium	Table B.3-34	2.0×10^{-4}	0.001	0.2
Chromium	Table B.3-34	1.0×10^{-3}	0.003	0.3
Copper	Table B.3-34	1.8×10^{-2}	0.04	0.5
Mercury	Table B.3-34	1.1×10^{-4}	0.0001	1
Nickel	Table B.3-34	8.7×10^{-4}	0.02	0.04
Total PCBs	Table B.3-35	1.7×10^{-3}	0.00002	86
Pentachlorophenol[b]	Table B.3-36	1.6×10^{-3}	0.03	0.05
TBT (as ion)	Table B.3-34	4.5×10^{-4}	0.00015	3
Vanadium	Table B.3-34	1.8×10^{-3}	0.001	2
Zinc	Table B.3-34	7.3×10^{-2}	0.3	0.2
			Subtotal	**101**
Tentatively identified chemicals (JN-qualified)				
Aldrin[b]	Table B.3-35	6.1×10^{-6}	0.00003	0.2
alpha-BHC[b]	Table B.3-35	5.6×10^{-6}	0.0005	0.01
beta-BHC[b]	Table B.3-35	7.6×10^{-6}	0.0002	0.04
Total chlordane	Table B.3-35	3.6×10^{-5}	0.0005	0.07
Total DDTs	Table B.3-35	1.3×10^{-4}	0.0005	0.3
Dieldrin	Table B.3-35	1.8×10^{-5}	0.00005	0.4
Endrin[b]	Table B.3-35	6.4×10^{-6}	0.0003	0.02
Endrin aldehyde	Table B.3-35	2.4×10^{-5}	0.0003	0.08
gamma-BHC	Table B.3-35	9.1×10^{-6}	0.0003	0.03
Heptachlor[b]	Table B.3-35	6.9×10^{-6}	0.0005	0.01
Heptachlor epoxide	Table B.3-35	7.3×10^{-6}	0.000013	0.6
Hexachlorobenzene	Table B.3-36	1.4×10^{-5}	0.0008	0.02
			Subtotal	**2**
Hazard indices by effect:				
Hazard Index for cardiovascular endpoint[e]				**9**
Hazard Index for developmental endpoint[f]				**87**
Hazard Index for hematologic endpoint[g]				**0.5**
Hazard Index for immunological endpoint[h]				**89**

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (kg/mg-day)	Hazard Quotient
Hazard index for kidney endpoint[i]				**1**
Hazard index for liver endpoint[j]				**3**
Hazard index for neurological endpoint[k]				**87**
Hazard index for dermal endpoint[l]				**7**
Total hazard index across all exposure routes/pathways[m]				**103**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 to B.3-36).

[b] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[e] Cardiovascular endpoint is for arsenic and vanadium.

[f] Developmental endpoint is for PCBs and mercury.

[g] Hematologic endpoint is for antimony and zinc.

[h] Immunological endpoint is for PCBs and TBT.

[I] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[j] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[k] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[l] Dermal endpoint is for 4-methylphenol and arsenic

[m] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, His were calculated for individual endpoints.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Table B.5-12. Non-cancer hazard estimates for the child tribal CT seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
4-Methylphenol[b]	Table B.3-36	9.7×10^{-5}	0.005	0.02
Antimony	Table B.3-35	8.5×10^{-6}	0.0004	0.02
Arsenic[c, d]	Table B.3-35	2.1×10^{-4}	0.0003	0.7
Bis(2-ethylhexyl) phthalate	Table B.3-36	6.1×10^{-5}	0.02	0.003
Butyl benzyl phthalate[b]	Table B.3-36	1.2×10^{-4}	0.2	0.0006
Cadmium	Table B.3-34	2.7×10^{-5}	0.001	0.03
Chromium	Table B.3-34	1.2×10^{-4}	0.003	0.04
Copper	Table B.3-34	2.5×10^{-3}	0.04	0.06
Mercury	Table B.3-34	1.5×10^{-5}	0.0001	0.1
Nickel	Table B.3-34	1.1×10^{-4}	0.02	0.006
Total PCBs	Table B.3-35	1.6×10^{-4}	0.00002	8
Pentachlorophenol[b]	Table B.3-36	6.9×10^{-5}	0.03	0.002
TBT (as ion)	Table B.3-34	5.2×10^{-5}	0.00015	0.3
Vanadium	Table B.3-34	2.3×10^{-4}	0.001	0.2
Zinc	Table B.3-34	1.0×10^{-2}	0.3	0.03
			Subtotal	**10**
Tentatively identified chemicals (JN-qualified)				
Aldrin[b]	Table B.3-35	4.4×10^{-7}	0.00003	0.01
alpha-BHC[b]	Table B.3-35	4.3×10^{-7}	0.0005	0.0009
beta-BHC[b]	Table B.3-35	7.0×10^{-7}	0.0002	0.004
Total chlordane	Table B.3-35	2.9×10^{-6}	0.0005	0.006
Total DDTs	Table B.3-35	1.5×10^{-5}	0.0005	0.03
Dieldrin	Table B.3-35	8.0×10^{-7}	0.00005	0.02
Endrin[b]	Table B.3-35	5.6×10^{-7}	0.0003	0.002
Endrin aldehyde	Table B.3-35	6.5×10^{-7}	0.0003	0.002
gamma-BHC	Table B.3-35	4.9×10^{-7}	0.0003	0.002
Heptachlor[b]	Table B.3-35	4.5×10^{-7}	0.0005	0.0009
Heptachlor epoxide	Table B.3-35	6.5×10^{-7}	0.000013	0.05
Hexachlorobenzene	Table B.3-36	7.7×10^{-7}	0.0008	0.001
			Subtotal	**0.1**
Hazard indices by effect:				
Hazard Index for cardiovascular endpoint[e]				**0.9**
Hazard Index for developmental endpoint[f]				**8**
Hazard Index for hematologic endpoint[g]				**0.05**

CHEMICAL	EPC (mg/kg ww)[a]	NON-CANCER CDI (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT
Hazard Index for immunological endpoint[h]				**8**
Hazard index for kidney endpoint[i]				**0.1**
Hazard index for liver endpoint[j]				**0.3**
Hazard index for neurological endpoint[k]				**8**
Hazard index for dermal endpoint[l]				**0.7**
Total hazard index across all exposure routes/pathways[m]				**10**

[a] The values used for EPCs in the CT scenario were based on the mean concentration for each seafood category. These EPCs were calculated using ½ the RL for non-detects (see Tables B.3-34 through B.3-36).

[b] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[d] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[e] Cardiovascular endpoint is for arsenic and vanadium.

[f] Developmental endpoint is for PCBs and mercury.

[g] Hematologic endpoint is for antimony and zinc.

[h] Immunological endpoint is for PCBs and TBT.

[I] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[j] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[k] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[l] Dermal endpoint is for 4-methylphenol and arsenic.

[m] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

Table B.5-13. Non-cancer hazard estimates for the adult tribal seafood consumption scenario based on Suquamish data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
4-Methylphenol	Table B.3-36	1.8×10^{-3}	0.005	0.4
Antimony	Table B.3-34	5.1×10^{-4}	0.0004	1
Arsenic[b,c]	Table B.3-34	1.1×10^{-2}	0.0003	38
Bis(2-ethylhexyl) phthalate	Table B.3-36	2.8×10^{-3}	0.02	0.1
Butyl benzyl phthalate	Table B.3-36	1.4×10^{-3}	0.2	0.007
Cadmium	Table B.3-34	7.0×10^{-4}	0.001	0.7
Chromium	Table B.3-34	4.7×10^{-3}	0.003	2
Copper	Table B.3-34	4.2×10^{-2}	0.04	1
Mercury	Table B.3-34	2.0×10^{-4}	0.0001	2
Nickel	Table B.3-34	4.3×10^{-3}	0.02	0.2
Total PCBs	Table B.3-35	5.5×10^{-3}	0.00002	274
Pentachlorophenol[d]	Table B.3-36	4.0×10^{-3}	0.03	0.1
TBT (as ion)	Table B.3-34	2.3×10^{-3}	0.00015	15
Vanadium	Table B.3-34	8.9×10^{-3}	0.001	9
Zinc	Table B.3-34	1.9×10^{-1}	0.3	0.6
			Subtotal	**344**
Tentatively identified chemicals (JN-qualified)				
Aldrin	Table B.3-35	1.2×10^{-5}	0.00003	0.4
alpha-BHC	Table B.3-35	8.9×10^{-6}	0.0005	0.02
beta-BHC	Table B.3-35	1.6×10^{-5}	0.0002	0.08
Total chlordane	Table B.3-35	1.0×10^{-4}	0.0005	0.2
Total DDTs	Table B.3-35	3.2×10^{-4}	0.0005	0.6
Dieldrin	Table B.3-35	7.3×10^{-5}	0.00005	1
Endrin	Table B.3-35	1.2×10^{-5}	0.0003	0.04
Endrin aldehyde	Table B.3-35	7.1×10^{-5}	0.0003	0.2
gamma-BHC	Table B.3-35	2.0×10^{-5}	0.0003	0.07
Heptachlor	Table B.3-35	1.3×10^{-5}	0.0005	0.03
Heptachlor epoxide	Table B.3-35	1.9×10^{-5}	0.000013	1
Hexachlorobenzene	Table B.3-36	2.2×10^{-5}	0.0008	0.03
			Subtotal	**4**
Hazard indices by effect:				
Hazard Index for cardiovascular endpoint[e]				**47**
Hazard Index for developmental endpoint[f]				**276**

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Hazard Index for hematologic endpoint[g]				**2**
Hazard Index for immunological endpoint[h]				**289**
Hazard index for kidney endpoint[i]				**2**
Hazard index for liver endpoint[j]				**7**
Hazard index for neurological endpoint[k]				**276**
Hazard index for dermal endpoint[l]				**38**
Total hazard index across all exposure routes/pathways[m]				**348**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 to B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[e] Cardiovascular endpoint is for arsenic and vanadium.

[f] Developmental endpoint is for PCBs and mercury.

[g] Hematologic endpoint is for antimony.

[h] Immunological endpoint is for PCBs and TBT.

[I] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[j] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[k] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[l] Dermal endpoint is for 4-methylphenol and arsenic.

[m] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, His were calculated for individual endpoints.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Table B.5-14. Non-cancer hazard estimates for the API RME seafood consumption scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Asian and Pacific Islander fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
4-Methylphenol	Table B.3-36	2.3×10^{-4}	0.005	0.05
Antimony	Table B.3-34	4.4×10^{-5}	0.0004	0.1
Arsenic[b, c]	Table B.3-34	1.0×10^{-3}	0.0003	3
Bis(2-ethylhexyl) phthalate	Table B.3-36	3.1×10^{-4}	0.02	0.02
Butyl benzyl phthalate	Table B.3-36	2.5×10^{-4}	0.2	0.001
Cadmium	Table B.3-34	1.1×10^{-4}	0.001	0.1
Chromium	Table B.3-34	4.2×10^{-4}	0.003	0.1
Copper	Table B.3-34	5.1×10^{-3}	0.04	0.1
Mercury	Table B.3-34	2.5×10^{-5}	0.0001	0.3
Nickel	Table B.3-34	3.8×10^{-4}	0.02	0.02
Total PCBs	Table B.3-35	5.8×10^{-4}	0.00002	29
Pentachlorophenol	Table B.3-36	4.1×10^{-4}	0.03	0.01
TBT (as ion)	Table B.3-34	2.0×10^{-4}	0.00015	1
Vanadium	Table B.3-34	7.8×10^{-4}	0.001	0.8
Zinc	Table B.3-34	2.2×10^{-2}	0.3	0.07
			Subtotal	**35**
Tentatively identified chemicals (JN-qualified)				
Aldrin	Table B.3-35	1.4×10^{-6}	0.00003	0.05
alpha-BHC	Table B.3-35	1.2×10^{-6}	0.0005	0.002
beta-BHC	Table B.3-35	1.9×10^{-6}	0.0002	0.009
Total chlordane	Table B.3-35	1.2×10^{-5}	0.0005	0.02
Total DDTs	Table B.3-35	4.0×10^{-5}	0.0005	0.08
Dieldrin	Table B.3-35	6.6×10^{-6}	0.00005	0.1
Endrin	Table B.3-35	1.5×10^{-6}	0.0003	0.005
Endrin aldehyde	Table B.3-35	7.8×10^{-6}	0.0003	0.03
gamma-BHC	Table B.3-35	2.4×10^{-6}	0.0003	0.008
Heptachlor	Table B.3-35	1.7×10^{-6}	0.0005	0.003
Heptachlor epoxide	Table B.3-35	2.3×10^{-6}	0.000013	0.2
Hexachlorobenzene	Table B.3-36	3.4×10^{-6}	0.0008	0.004
			Subtotal	**0.5**

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Hazard indices by effect:				
Hazard Index for cardiovascular endpoint[d]				**4**
Hazard Index for developmental endpoint[e]				**29**
Hazard Index for hematologic endpoint[f]				**0.2**
Hazard Index for immunological endpoint[g]				**30**
Hazard index for kidney endpoint[h]				**0.3**
Hazard index for liver endpoint[i]				**0.8**
Hazard index for neurological endpoint[j]				**29**
Hazard index for dermal endpoint[k]				**3**
Total hazard index across all exposure routes/pathways[l]				**35**

[a] An EPC for each seafood category was calculated in the exposure section (Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] Cardiovascular endpoint is for arsenic and vanadium.

[e] Developmental endpoint is for PCBs and mercury.

[f] Hematologic endpoint is for antimony and zinc.

[g] Immunological endpoint is for PCBs and TBT.

[h] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[i] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[j] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[k] Dermal endpoint is for 4-methylphenol and arsenic.

[l] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

API – Asian and Pacific Islander

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Table B.5-15. Non-cancer hazard estimates for the API CT seafood consumption scenario

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Asian and Pacific Islander fish and shellfish consumers
Receptor age: Adult

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
4-Methylphenol	Table B.3-36	1.2×10^{-5}	0.005	0.002
Antimony	Table B.3-34	2.5×10^{-6}	0.0004	0.006
Arsenic[b, c]	Table B.3-34	6.6×10^{-5}	0.0003	0.2
Bis(2-ethylhexyl) phthalate	Table B.3-36	1.4×10^{-5}	0.02	0.0007
Butyl benzyl phthalate	Table B.3-36	1.6×10^{-5}	0.2	0.00008
Cadmium	Table B.3-34	1.0×10^{-5}	0.001	0.01
Chromium	Table B.3-34	3.6×10^{-5}	0.003	0.01
Copper	Table B.3-34	4.6×10^{-4}	0.04	0.01
Mercury	Table B.3-34	2.3×10^{-6}	0.0001	0.02
Nickel	Table B.3-34	3.4×10^{-5}	0.02	0.002
Total PCBs	Table B.3-35	3.3×10^{-5}	0.00002	2
Pentachlorophenol	Table B.3-36	1.7×10^{-5}	0.03	0.0006
TBT (as ion)	Table B.3-34	1.6×10^{-5}	0.00015	0.1
Vanadium	Table B.3-34	6.8×10^{-5}	0.001	0.07
Zinc	Table B.3-34	2.1×10^{-3}	0.3	0.007
			Subtotal	**2**
Tentatively identified chemicals (JN-qualified)				
Aldrin	Table B.3-35	7.2×10^{-8}	0.00003	0.002
alpha-BHC	Table B.3-35	6.9×10^{-8}	0.0005	0.0001
beta-BHC	Table B.3-35	1.3×10^{-7}	0.0002	0.0006
Total chlordane	Table B.3-35	6.1×10^{-7}	0.0005	0.001
Total DDTs	Table B.3-35	3.1×10^{-6}	0.0005	0.006
Dieldrin	Table B.3-35	1.7×10^{-7}	0.00005	0.003
Endrin	Table B.3-35	9.7×10^{-8}	0.0003	0.0003
Endrin aldehyde	Table B.3-35	1.1×10^{-7}	0.0003	0.0004
gamma-BHC	Table B.3-35	8.3×10^{-8}	0.0003	0.0003
Heptachlor	Table B.3-35	7.3×10^{-8}	0.0005	0.0001
Heptachlor epoxide	Table B.3-35	1.2×10^{-7}	0.000013	0.009
Hexachlorobenzene	Table B.3-36	1.7×10^{-7}	0.0008	0.0002
			Subtotal	**0.02**

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Hazard indices by effect:				
Hazard Index for cardiovascular endpoint[d]				**0.3**
Hazard Index for developmental endpoint[e]				**2**
Hazard Index for hematologic endpoint[f]				**0.01**
Hazard Index for immunological endpoint[g]				**2**
Hazard index for kidney endpoint[h]				**0.02**
Hazard index for liver endpoint[i]				**0.05**
Hazard index for neurological endpoint[j]				**2**
Hazard index for dermal endpoint[k]				**0.2**
Total hazard index across all exposure routes/pathways[l]				**2**

[a] The values used for EPCs in the CT scenario were based on the mean concentration for each seafood category. These EPCs were calculated using one-half the RL for non-detects (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] Cardiovascular endpoint is for arsenic and vanadium.

[e] Developmental endpoint is for PCBs and mercury.

[f] Hematologic endpoint is for antimony and zinc.

[g] Immunological endpoint is for: PCBs and TBT.

[h] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[i] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[j] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[k] Dermal endpoint is for 4-methylphenol and arsenic.

[l] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

API – Asian and Pacific Islander

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Table B.5-16. Non-cancer hazard estimates associated with the consumption of one meal per month of seafood by adults

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Adults consuming one meal per month of fish or shellfish
Receptor age: Adult

Chemical	Seafood Category	EPC (mg/kg ww)	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
4-Methylphenol	benthic fish[a]	0.60	6.3×10^{-5}	0.005	0.01
	clam[a]	0.031	3.2×10^{-6}	0.005	0.0006
	crab edible meat[a]	0.60	6.3×10^{-5}	0.005	0.01
	pelagic fish	1.5	1.6×10^{-4}	0.005	0.03
Antimony	benthic fish	0.010	1.0×10^{-6}	0.0004	0.003
	clam	0.090	9.4×10^{-6}	0.0004	0.02
	crab edible meat	0.0095	9.9×10^{-7}	0.0004	0.002
	pelagic fish	0.0039	4.1×10^{-7}	0.0004	0.001
Arsenic[b]	benthic fish	0.0062	6.5×10^{-7}	0.0003	0.002
	clam	2.0	2.1×10^{-4}	0.0003	0.7
	crab edible meat	0.042	4.4×10^{-6}	0.0003	0.01
	pelagic fish	0.088	9.2×10^{-6}	0.0003	0.03
Bis(2-ethylhexyl) phthalate	benthic fish	1.3	1.4×10^{-4}	0.02	0.007
	clam	0.13	1.4×10^{-5}	0.02	0.0007
	crab edible meat[a]	0.13	1.4×10^{-5}	0.02	0.0007
	pelagic fish	2.1	2.2×10^{-4}	0.02	0.01
Butyl benzyl phthalate	benthic fish[a]	0.60	6.3×10^{-5}	0.2	0.0003
	clam[a]	0.020	2.1×10^{-6}	0.2	0.00001
	crab edible meat[a]	0.60	6.3×10^{-5}	0.2	0.0003
	pelagic fish	0.84	8.8×10^{-5}	0.2	0.0004
Cadmium	benthic fish	0.0040	4.2×10^{-7}	0.001	0.0004
	clam	0.11	1.1×10^{-5}	0.001	0.01
	crab edible meat	0.027	2.8×10^{-6}	0.001	0.003
	pelagic fish	0.016	1.7×10^{-6}	0.001	0.002
Chromium	benthic fish	0.062	6.5×10^{-6}	0.003	0.002
	clam	0.79	8.3×10^{-5}	0.003	0.03
	crab edible meat	0.16	1.7×10^{-5}	0.003	0.006
	pelagic fish	0.20	2.1×10^{-5}	0.003	0.007

Chemical	Seafood Category	EPC (mg/kg ww)	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Copper	benthic fish	1.6	1.7×10^{-4}	0.04	0.004
	clam	6.0	6.3×10^{-4}	0.04	0.02
	crab edible meat	8.5	8.9×10^{-4}	0.04	0.02
	pelagic fish	1.7	1.8×10^{-4}	0.04	0.004
Mercury	benthic fish	0.058	6.1×10^{-6}	0.0001	0.06
	clam	0.020	2.1×10^{-6}	0.0001	0.02
	crab edible meat	0.064	6.7×10^{-6}	0.0001	0.07
	pelagic fish	0.039	4.1×10^{-6}	0.0001	0.04
Nickel	benthic fish	0.063	6.6×10^{-6}	0.02	0.0003
	clam	0.69	7.2×10^{-5}	0.02	0.004
	crab edible meat	0.055	5.7×10^{-6}	0.02	0.0003
	pelagic fish	0.47	4.9×10^{-5}	0.02	0.002
Total PCBs	benthic fish	1.2	1.3×10^{-4}	2×10^{-5}	6
	clam	0.60	6.3×10^{-5}	2×10^{-5}	3
	crab edible meat	0.20	2.1×10^{-5}	2×10^{-5}	1
	pelagic fish	31.9	2.0×10^{-4}	2×10^{-5}	10
Pentachlorophenol	benthic fish[a]	2.9	3.0×10^{-4}	0.03	0.01
	clam[a]	0.20	2.1×10^{-5}	0.03	0.0007
	crab edible meat[a]	0.29	3.0×10^{-5}	0.03	0.001
	pelagic fish	2.4	2.5×10^{-4}	0.03	0.008
TBT (as ion)	benthic fish	0.0033	3.4×10^{-7}	0.00015	0.002
	clam	0.40	4.2×10^{-5}	0.00015	0.3
	crab edible meat	0.030	3.1×10^{-6}	0.00015	0.02
	pelagic fish	0.081	8.5×10^{-6}	0.00015	0.06
Vanadium	benthic fish[a]	0.13	1.4×10^{-5}	0.001	0.01
	clam	1.5	1.6×10^{-4}	0.001	0.2
	crab edible meat[a]	0.11	1.1×10^{-5}	0.001	0.01
	pelagic fish	0.62	6.5×10^{-5}	0.001	0.06
Zinc	benthic fish	8.6	9.0×10^{-4}	0.3	0.003
	clam	26	2.7×10^{-3}	0.3	0.009
	crab edible meat	36	3.8×10^{-3}	0.3	0.01
	pelagic fish	22	2.3×10^{-3}	0.3	0.008
Subtotal				**benthic fish**	**6**
				clam	**4**
				crab edible meat	**1**
				pelagic fish	**10**

Chemical	Seafood Category	EPC (mg/kg ww)	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Tentatively identified chemicals (JN-qualified)					
Aldrin	benthic fish[a]	0.0036	3.8×10^{-7}	3×10^{-5}	0.01
	clam	0.0010	1.0×10^{-7}	3×10^{-5}	0.003
	crab edible meat[a]	0.0036	3.8×10^{-7}	3×10^{-5}	0.01
	pelagic fish	0.0036	3.8×10^{-7}	3×10^{-5}	0.01
alpha-BHC	benthic fish	0.0036	3.8×10^{-7}	0.0005	0.0008
	clam	0.00050	5.2×10^{-8}	0.0005	0.0001
	crab edible meat[a]	0.0036	3.8×10^{-7}	0.0005	0.0008
	pelagic fish	0.0036	3.8×10^{-7}	0.0005	0.0008
beta-BHC	benthic fish	0.0036	3.8×10^{-7}	0.0002	0.002
	clam	0.0012	1.3×10^{-7}	0.0002	0.0006
	crab edible meat[a]	0.0041	4.3×10^{-7}	0.0002	0.002
	pelagic fish	0.0078	8.1×10^{-7}	0.0002	0.004
Total chlordane	benthic fish	0.020	2.1×10^{-6}	0.0005	0.004
	clam	0.0047	4.9×10^{-7}	0.0005	0.001
	crab edible meat	0.0045	4.7×10^{-7}	0.0005	0.0009
	pelagic fish	0.084	8.8×10^{-6}	0.0005	0.02
Total DDTs	benthic fish	0.084	8.8×10^{-6}	0.0005	0.02
	clam	0.015	1.6×10^{-6}	0.0005	0.003
	crab edible meat	0.023	2.4×10^{-6}	0.0005	0.005
	pelagic fish	0.24	2.5×10^{-5}	0.0005	0.05
Dieldrin	benthic fish[a]	0.0036	3.8×10^{-7}	5×10^{-5}	0.008
	clam	0.012	1.3×10^{-6}	5×10^{-5}	0.03
	crab edible meat	0.0036	3.8×10^{-7}	5×10^{-5}	0.008
	pelagic fish[a]	0.0036	3.8×10^{-7}	5×10^{-5}	0.008
Endrin	benthic fish	0.0036	3.8×10^{-7}	0.0003	0.001
	clam	0.00056	5.8×10^{-8}	0.0003	0.0002
	crab edible meat[a]	0.0036	3.8×10^{-7}	0.0003	0.001
	pelagic fish	0.0067	7.0×10^{-7}	0.0003	0.002
Endrin aldehyde	benthic fish	0.0081	8.5×10^{-7}	0.0003	0.003
	clam	0.0018	1.9×10^{-7}	0.0003	0.0006
	crab edible meat	0.0036	3.8×10^{-7}	0.0003	0.001
	pelagic fish	0.078	8.1×10^{-6}	0.0003	0.03

Chemical	Seafood Category	EPC (mg/kg ww)	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
gamma-BHC	benthic fish[a]	0.0036	3.8×10^{-7}	0.0003	0.001
	clam	0.0025	2.6×10^{-7}	0.0003	0.0009
	crab edible meat	0.0040	4.2×10^{-7}	0.0003	0.001
	pelagic fish	0.0020	2.1×10^{-7}	0.0003	0.0007
Heptachlor	benthic fish[a]	0.0036	3.8×10^{-7}	0.0005	0.0008
	clam[a]	0.00050	5.2×10^{-8}	0.0005	0.0001
	crab edible meat[a]	0.0036	3.8×10^{-7}	0.0005	0.0008
	pelagic fish	0.0097	1.0×10^{-6}	0.0005	0.002
Heptachlor epoxide	benthic fish[a]	0.0036	3.8×10^{-7}	1.3×10^{-5}	0.03
	clam	0.0015	1.6×10^{-7}	1.3×10^{-5}	0.01
	crab edible meat	0.0021	2.2×10^{-7}	1.3×10^{-5}	0.02
	pelagic fish	0.010	1.0×10^{-6}	1.3×10^{-5}	0.08
Hexachlorobenzene	benthic fish	0.0090	9.4×10^{-7}	0.0008	0.001
	clam	0.00086	9.0×10^{-8}	0.0008	0.0001
	crab edible meat	0.0080	8.4×10^{-7}	0.0008	0.001
	pelagic fish	0.012	1.3×10^{-6}	0.0008	0.002
Subtotal				**benthic fish**	**0.08**
				clam	**0.05**
				crab edible meat	**0.05**
				pelagic fish	**0.2**
Hazard indices by effect:					
Hazard index for cardiovascular endpoint[c]				**benthic fish**	**0.01**
				clam	**0.9**
				crab edible meat	**0.02**
				pelagic fish	**0.09**
Hazard index for developmental endpoint[d]				**benthic fish**	**6**
				clam	**3**
				crab edible meat	**1**
				pelagic fish	**10**
Hazard index for hematologic endpoint[e]				**benthic fish**	**0.006**
				clam	**0.03**
				crab edible meat	**0.01**
				pelagic fish	**0.009**
Hazard index for immunological endpoint[f]				**benthic fish**	**6**
				clam	**3**
				crab edible meat	**1**
				pelagic fish	**10**

Chemical	Seafood Category	EPC (mg/kg ww)	Non-Cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Hazard index for kidney endpoint[g]				**benthic fish**	**0.03**
				clam	**0.03**
				crab edible meat	**0.04**
				pelagic fish	**0.04**
Hazard index for liver endpoint[h]				**benthic fish**	**0.1**
				clam	**0.1**
				crab edible meat	**0.09**
				pelagic fish	**0.3**
Hazard index for neurological endpoint[i]				**benthic fish**	**6**
				clam	**3**
				crab edible meat	**1**
				pelagic fish	**10**
Hazard index for dermal endpoint[j]				**benthic fish**	**0.01**
				clam	**0.7**
				crab edible meat	**0.02**
				pelagic fish	**0.06**
Total hazard index across all exposure routes/pathways[k]				**benthic fish**	**6**
				clam	**4**
				crab edible meat	**1**
				pelagic fish	**10**

[a] No detected values in this seafood category. CDI and risk estimate are based on one-half the maximum reporting limit.

[b] Arsenic concentrations based on inorganic arsenic.

[c] Cardiovascular endpoint is for arsenic and vanadium.

[d] Developmental endpoint is for PCBs and mercury.

[e] Hematologic endpoint is for antimony and zinc.

[f] Immunological endpoint is for PCBs and TBT.

[g] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[h] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[i] Neurological endpoint is for 4-methylphenol, mercury and total PCBs.

[j] Dermal endpoint is for 4-methylphenol and arsenic.

[k] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

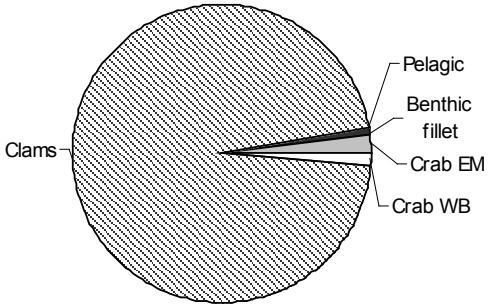
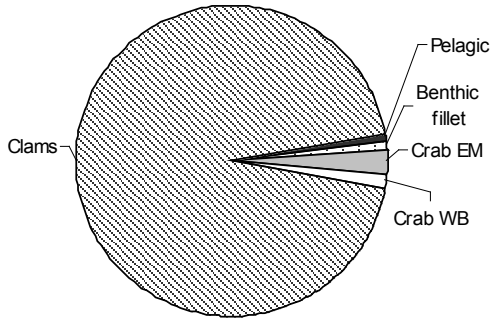
ww – wet weight

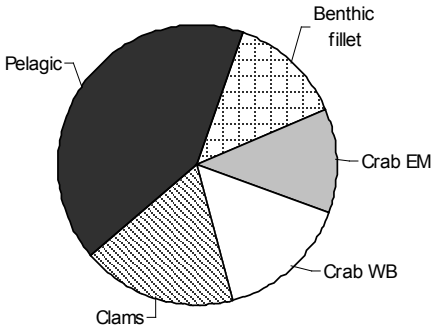
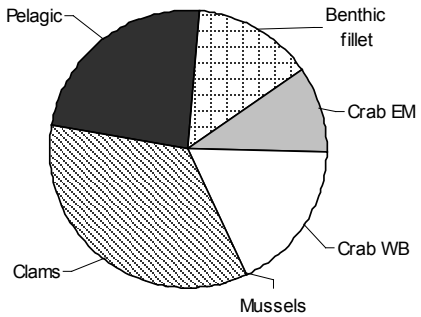
B.5.3.1.3 Risk estimates by seafood category for chemicals contributing the greatest amount to seafood consumption risk estimates

The previous sections summarized excess cancer risks and non-cancer hazards. This section discusses the specific chemicals and the seafood categories contributing most to these estimates. Chemicals were selected for this discussion based on the exceedance of risk thresholds and by their contribution to the total risk estimate. Arsenic, cPAHs, PCB TEQ, and total PCBs were determined to be the dominant contributors to both cancer and non-cancer risk estimates (Tables B.5-1 through B.5-16), with excess cancer risks for each of these chemicals greater than 1×10^{-6} for one or more seafood consumption scenarios. In addition, each of these chemicals also contributed 5% or more of the total excess cancer risks for one or more seafood consumption scenarios. Some of these chemicals also had HQs greater than 1 for one or more seafood consumption scenarios. These chemicals were among the most frequently detected chemicals in seafood, and their concentrations varied greatly across the seven seafood categories. For example, the total PCB EPCs ranged from 0.04 mg/kg ww for mussels to 2.6 mg/kg ww for whole body benthic fish. Similarly, consumption rates for different seafood categories also varied across scenarios. Together, these variations in chemical concentrations and consumption rates led to the ranges in risk estimates among the various seafood consumption scenarios. Although body weight and exposure duration assumptions also differed across scenarios, these differences had a smaller influence on risk estimates than seafood consumption rates.

The proportional contributions of each seafood category to risk estimates are presented in Tables B.5-17 to B.5-21 and discussed below. The adult and child tribal RME scenarios based on Tulalip data are shown together in Table B.5-17 because the apportionment of the market basket was done in the same way, meaning that the percent of total risk associated with each consumption category is the same. Likewise, the adult and child tribal CT scenarios based on Tulalip data are shown together in Table B.5-18. These summaries of the contributions of specific chemicals to seafood consumption risks do not include the unknown contributions from dioxins/furans, because tissue data were not collected for that class of chemicals.

Table B.5-17. Comparison of excess cancer risks and non-cancer hazards by seafood category for selected chemicals in the adult tribal RME and child tribal RME seafood consumption scenarios based on Tulalip data

Consumption Category	EPC (mg/kg ww)	Ingestion Rate (g/day)		Percent of Chemical's Risk	Excess Cancer Risk		Non-Cancer Hazard Quotient		Adult or Child Tribal RME (Tulalip data) Seafood Consumption Risk Expressed as Pie Chart[a]
		Adult	Child		Adult	Child	Adult	Child	
Arsenic[b]									
Pelagic	0.088	8.2	3.3	0.9%	1×10^{-5}	2×10^{-6}	0.03	0.06	
Benthic fillet	0.0062	7.6	3.0	0.06%	9×10^{-7}	2×10^{-7}	0.002	0.004	
Crab edible meat	0.042	33	13	1.8%	3×10^{-5}	5×10^{-6}	0.06	0.12	
Crab whole body	0.11	10	4.2	1.5%	2×10^{-5}	4×10^{-6}	0.05	0.10	
Clams	2.0	38	15	95.8%	1×10^{-3}	3×10^{-4}	3	7	
Total risk from arsenic					**1×10^{-3}**	**3×10^{-4}**	**3**	**7**	
cPAHs (2004 data only)[b,c,d]									
Pelagic	0.00095	8.2	3.3	1.0%	7×10^{-7}	7×10^{-7}	na	na	
Benthic fillet	0.00064	7.6	3.0	0.6%	4×10^{-7}	4×10^{-7}	na	na	
Crab edible meat	0.00065	33	13	2.7%	2×10^{-6}	2×10^{-6}	na	na	
Crab whole body	0.00092	10	4.2	1.2%	9×10^{-7}	8×10^{-7}	na	na	
Clams	0.020	38	15	94.5%	7×10^{-5}	7×10^{-5}	na	na	
Total risk from cPAHs					**7×10^{-5}**	**7×10^{-5}**	**na**	**na**	

Consumption Category	EPC (mg/kg ww)	Ingestion Rate (g/day)		Percent of Chemical's Risk	Excess Cancer Risk		Non-Cancer Hazard Quotient		Adult or Child Tribal RME (Tulalip data) Seafood Consumption Risk Expressed as Pie Chart[a]
		Adult	Child		Adult	Child	Adult	Child	
PCB TEQ[b]									
Pelagic	3.37×10^{-5}	8.2	3.3	41.4%	5×10^{-4}	9×10^{-5}	na	na	
Benthic fillet	1.17×10^{-5}	7.6	3.0	13.3%	2×10^{-4}	3×10^{-5}	na	na	
Crab edible meat	2.41×10^{-6}	33	13	12.0%	1×10^{-4}	3×10^{-5}	na	na	
Crab whole body	9.68×10^{-6}	10	4.2	15.3%	2×10^{-4}	3×10^{-5}	na	na	
Clams	3.16×10^{-6}	38.0	15	18.0%	2×10^{-4}	4×10^{-5}	na	na	
Total risk from PCB TEQ					**1×10^{-3}**	**2×10^{-4}**	**na**	**na**	
Total PCBs									
Pelagic	1.9	8.1	3.2	23.6%	4×10^{-4}	7×10^{-5}	9	20	
Benthic fillet	1.2	7.5	3.0	13.8%	2×10^{-4}	4×10^{-5}	6	12	
Crab edible meat	0.20	33	13	10.1%	2×10^{-4}	3×10^{-5}	4	9	
Crab whole body	1.1	10	4.2	17.6%	3×10^{-4}	5×10^{-5}	7	15	
Mussels	0.041	0.82	0.33	0.05%	8×10^{-7}	2×10^{-7}	0.02	0.04	
Clams	0.60	38	15	34.8%	6×10^{-4}	1×10^{-4}	14	30	
Total risk from total PCBs					**2×10^{-3}**	**3×10^{-4}**	**40**	**86**	

[a] Figures represent both cancer and non-cancer risks. Risk percentages are based on EPC and ingestion rates, meaning that the percentage of risk from each consumption category is the same for cancer and non-cancer risks.

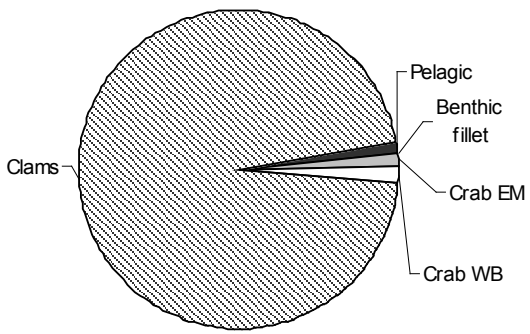
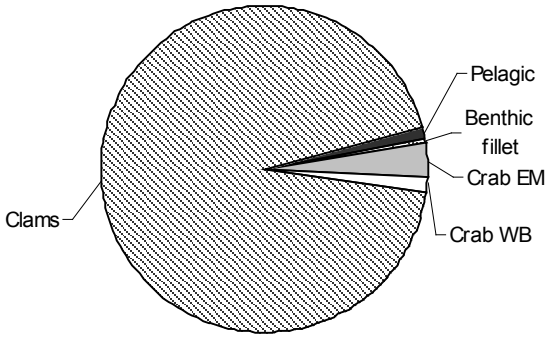
[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

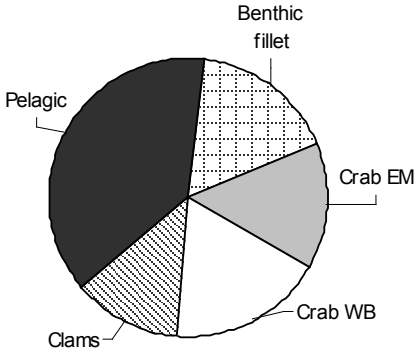
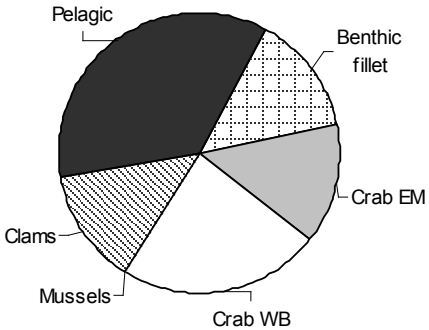
[c] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[d] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for children for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

EM – edible meat
EPC – exposure point concentration
na – not applicable
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent
WB – whole body

Table B.5-18. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult tribal CT and child tribal CT seafood consumption scenarios based on Tulalip data

Consumption Category	EPC[a] (mg/kg ww)	Ingestion Rate (g/day)		Percent of Chemical's Risk	Excess Cancer Risk		Non-Cancer Hazard Quotient		Adult or Child Tribal CT (Tulalip data) Seafood Consumption Risk Expressed as Pie Chart[b]
		Adult	Child		Adult	Child	Adult	Child	
Arsenic[c]									
Pelagic	0.057	1.4	0.55	1.0%	6×10^{-7}	3×10^{-7}	0.003	0.007	
Benthic fillet	0.0040	1.3	0.51	0.06%	4×10^{-8}	2×10^{-8}	0.0002	0.0004	
Crab edible meat	0.023	5.3	2.1	1.5%	1×10^{-6}	4×10^{-7}	0.00	0.01	
Crab whole body	0.075	1.7	0.68	1.6%	1×10^{-6}	4×10^{-7}	0.005	0.01	
Clams	1.2	6.1	2.4	95.8%	6×10^{-5}	3×10^{-5}	0.3	0.7	
Total risk from arsenic					**6×10^{-5}**	**3×10^{-5}**	**0.3**	**0.7**	
cPAHs (2004 data only)[c,d,e]									
Pelagic	0.00078	1.4	0.55	1.1%	4×10^{-8}	9×10^{-8}	na	na	
Benthic fillet	0.00050	1.3	0.51	0.6%	2×10^{-8}	5×10^{-8}	na	na	
Crab edible meat	0.000629	5.3	2.1	3.4%	1×10^{-7}	3×10^{-7}	na	na	
Crab whole body	0.00075	1.7	0.68	1.3%	5×10^{-8}	1×10^{-7}	na	na	
Clams	0.015	6.1	2.4	93.6%	4×10^{-6}	8×10^{-6}	na	na	
Total risk from cPAHs					**4×10^{-6}**	**8×10^{-6}**	**na**	**na**	

Consumption Category	EPC[a] (mg/kg ww)	Ingestion Rate (g/day)		Percent of Chemical's Risk	Excess Cancer Risk		Non-Cancer Hazard Quotient		Adult or Child Tribal CT (Tulalip data) Seafood Consumption Risk Expressed as Pie Chart[b]
		Adult	Child		Adult	Child	Adult	Child	
PCB TEQ[c]									
Pelagic	1.99×10^{-5}	1.4	0.55	37.9%	2×10^{-5}	9×10^{-6}	na	na	
Benthic fillet	9.50×10^{-6}	1.3	0.51	16.7%	9×10^{-6}	4×10^{-6}	na	na	
Crab edible meat	2.00×10^{-6}	5.3	2.1	14.7%	8×10^{-6}	4×10^{-6}	na	na	
Crab whole body	7.70×10^{-6}	1.7	0.68	18.1%	1×10^{-5}	4×10^{-6}	na	na	
Clams	1.48×10^{-6}	6.1	2.4	12.6%	7×10^{-6}	3×10^{-6}	na	na	
Total risk from PCB TEQ					**6×10^{-5}**	**2×10^{-5}**	**na**	**na**	
Total PCBs									
Pelagic	1.7	1.3	0.52	35.6%	2×10^{-5}	1×10^{-5}	1	3	
Benthic fillet	0.70	1.2	0.48	13.8%	9×10^{-6}	4×10^{-6}	0.5	1	
Crab edible meat	0.17	5.0	2.0	13.9%	9×10^{-6}	4×10^{-6}	0.5	1	
Crab whole body	0.89	1.6	0.64	23.3%	1×10^{-5}	6×10^{-6}	0.9	2	
Mussels	0.034	0.10	0.040	0.06%	4×10^{-8}	2×10^{-8}	0.002	0.004	
Clams	0.14	5.8	2.3	13.3%	9×10^{-6}	4×10^{-6}	0.5	1	
Total risk from total PCBs					**6×10^{-5}**	**3×10^{-5}**	**4**	**8**	

[a] EPC used for CT scenarios is mean value

[b] Figures represent both cancer and non-cancer risks. Risk percentages are based on EPC and ingestion rates, meaning that the percentage of risk from each consumption category is the same for cancer and non-cancer risks.

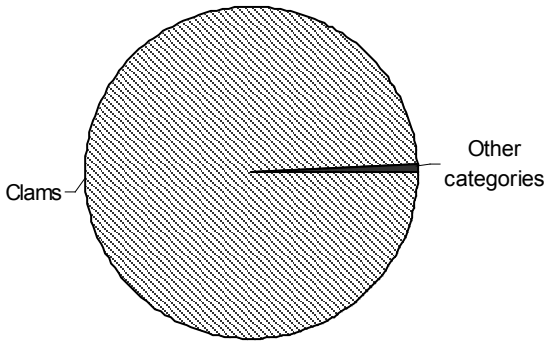
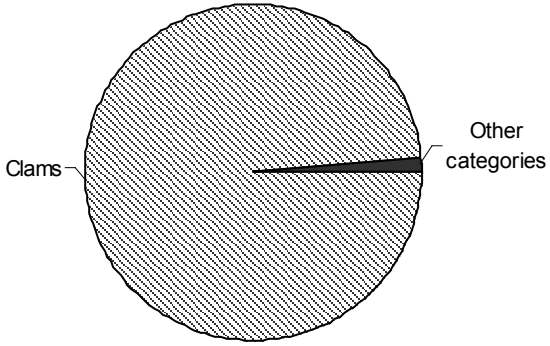
[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

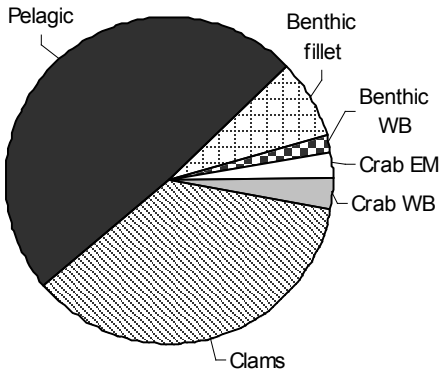
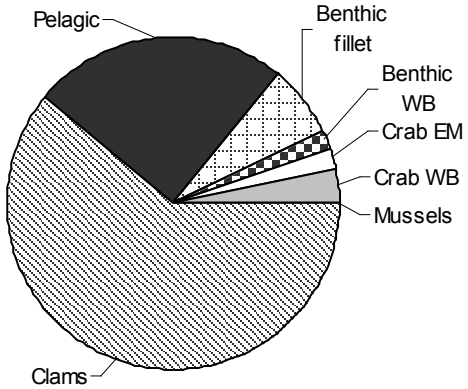
[d] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[e] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for children for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

EM – edible meat
EPC – exposure point concentration
CT – central tendency
na – not applicable
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
TEQ – toxic equivalent
WB – whole body

Table B.5-19. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult tribal seafood consumption scenario based on Suquamish data

Consumption Category	EPC (mg/kg ww)	Ingestion Rate (g/day)	Percent of Chemical's Risk	Excess Cancer Risk	Non-Cancer Hazard Quotient	Adult Tribal (Suquamish data) Seafood Consumption Risk Expressed as Pie Chart[a]
Arsenic[b]						
Pelagic	0.088	56.5	0.6%	9×10^{-5}	0.2	
Benthic fillet	0.0062	26.1	0.02%	3×10^{-6}	0.007	
Benthic whole body	0.073	3.2	0.03%	4×10^{-6}	0.01	
Crab edible meat	0.042	42.0	0.2%	3×10^{-5}	0.08	
Crab whole body	0.11	13.3	0.2%	3×10^{-5}	0.06	
Clams	2.0	442.4	99.0%	2×10^{-2}	37	
			Total risk from arsenic	**2×10^{-2}**	**38**	
cPAHs (2004 data only)[b,c]						
Pelagic	0.00095	56.5	0.6%	5×10^{-6}	na	
Benthic fillet	0.00064	26.1	0.2%	2×10^{-6}	na	
Benthic whole body	0.0023	3.2	0.08%	7×10^{-7}	na	
Crab edible meat	0.00065	42.0	0.3%	3×10^{-6}	na	
Crab whole body	0.00092	13.3	0.1%	1×10^{-6}	na	
Clams	0.020	442.4	98.7%	8×10^{-4}	na	
			Total risk from cPAHs	**8×10^{-4}**	**na**	

Consumption Category	EPC (mg/kg ww)	Ingestion Rate (g/day)	Percent of Chemical's Risk	Excess Cancer Risk	Non-Cancer Hazard Quotient	Adult Tribal (Suquamish data) Seafood Consumption Risk Expressed as Pie Chart[a]
PCB TEQ[b]						
Pelagic	3.37×10^{-5}	56.5	48.8%	4×10^{-3}	na	
Benthic fillet	1.17×10^{-5}	26.1	7.8%	6×10^{-4}	na	
Benthic whole body	2.04×10^{-5}	3.2	1.7%	1×10^{-4}	na	
Crab edible meat	2.41×10^{-6}	42.0	2.6%	2×10^{-4}	na	
Crab whole body	9.68×10^{-6}	13.3	3.3%	2×10^{-4}	na	
Clams	3.16×10^{-6}	442.4	35.8%	3×10^{-3}	na	
			Total risk from PCB TEQ	**7×10^{-3}**	**na**	
Total PCBs						
Pelagic	1.9	56.0	24.6%	3×10^{-3}	67	
Benthic fillet	1.2	25.9	7.2%	8×10^{-4}	20	
Benthic whole body	2.6	3.2	1.9%	2×10^{-4}	5	
Crab edible meat	0.20	41.6	1.9%	2×10^{-4}	5	
Crab whole body	1.1	13.2	3.4%	4×10^{-4}	9	
Mussels	0.041	5.0	0.05%	5×10^{-6}	0.1	
Clams	0.60	438.6	60.9%	7×10^{-3}	167	
			Total risk from total PCBs	**1×10^{-2}**	**274**	

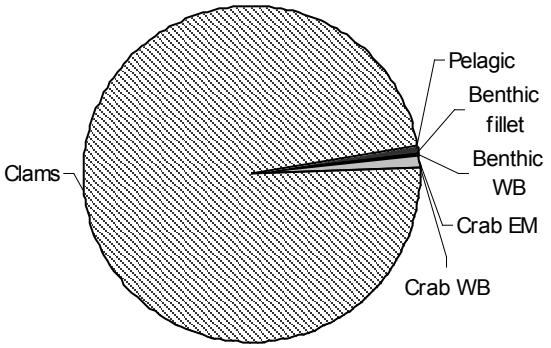
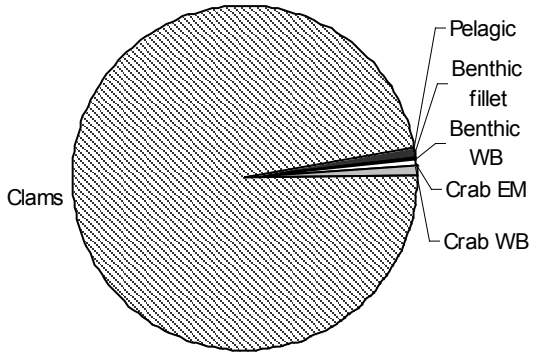
[a] Figures represent both cancer and non-cancer risks. Risk percentages are based on EPC and ingestion rates, meaning that the percentage of risk from each consumption category is the same for cancer and non-cancer risks.

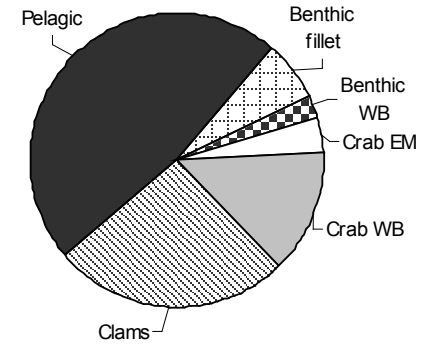
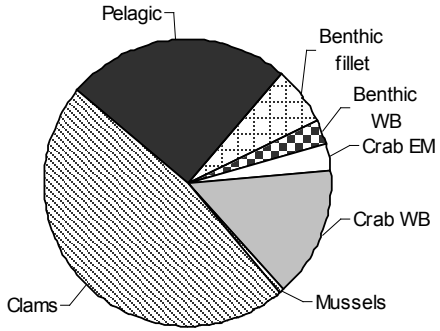
[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

EM – edible meat
EPC – exposure point concentration
na – not applicable
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
TEQ – toxic equivalent
WB – whole body

Table B.5-20. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult API RME seafood consumption scenario

Consumption Category	EPC (mg/kg ww)	Ingestion Rate (g/day)	Percent of Chemical's Risk	Excess Cancer Risk	Non-Cancer Hazard Quotient	Adult API RME Seafood Consumption Risk Expressed as Pie Chart[a]
Arsenic[b]						
Pelagic	0.088	5.4	0.7%	5×10^{-6}	0.03	
Benthic fillet	0.0062	2.2	0.02%	1×10^{-7}	0.0007	
Benthic whole body	0.073	0.4	0.05%	3×10^{-7}	0.002	
Crab edible meat	0.042	6.3	0.4%	3×10^{-6}	0.01	
Crab whole body	0.11	5.4	0.9%	6×10^{-6}	0.03	
Clams	2.0	32	97.9%	6×10^{-4}	3	
			Total risk from arsenic	**7×10^{-4}**	**3**	
cPAHs (2004 data only)[b,c]						
Pelagic	0.00095	5.4	0.8%	3×10^{-7}	na	
Benthic fillet	0.00064	2.2	0.2%	7×10^{-8}	na	
Benthic whole body	0.0023	0.4	0.2%	5×10^{-8}	na	
Crab edible meat	0.00065	6.3	0.6%	2×10^{-7}	na	
Crab whole body	0.00092	5.4	0.8%	2×10^{-7}	na	
Clams	0.020	32	97.5%	3×10^{-5}	na	
			Total risk from cPAHs	**3×10^{-5}**	**na**	

Consumption Category	EPC (mg/kg ww)	Ingestion Rate (g/day)	Percent of Chemical's Risk	Excess Cancer Risk	Non-Cancer Hazard Quotient	Adult API RME Seafood Consumption Risk Expressed as Pie Chart[a]
PCB TEQ[b]						
Pelagic	3.37×10^{-5}	5.4	47.3%	2×10^{-4}	na	
Benthic fillet	1.17×10^{-5}	2.2	6.7%	3×10^{-5}	na	
Benthic whole body	2.04×10^{-5}	0.4	2.3%	9×10^{-6}	na	
Crab edible meat	2.41×10^{-6}	6.3	3.9%	2×10^{-5}	na	
Crab whole body	9.68×10^{-6}	5.4	13.6%	5×10^{-5}	na	
Clams	3.16×10^{-6}	32	26.2%	1×10^{-4}	na	
			Total risk from PCB TEQ	**4×10^{-4}**	**na**	
Total PCBs						
Pelagic	1.9	4.9	25.3%	1×10^{-4}	7	
Benthic fillet	1.2	2.0	6.5%	3×10^{-5}	2	
Benthic whole body	2.6	0.4	2.8%	1×10^{-5}	0.8	
Crab edible meat	0.20	5.7	3.1%	2×10^{-5}	0.9	
Crab whole body	1.1	4.9	14.6%	7×10^{-5}	4	
Mussels	0.041	4.6	0.5%	3×10^{-6}	0.1	
Clams	0.60	29	47.2%	2×10^{-4}	14	
			Total risk from total PCBs	**5×10^{-4}**	**29**	

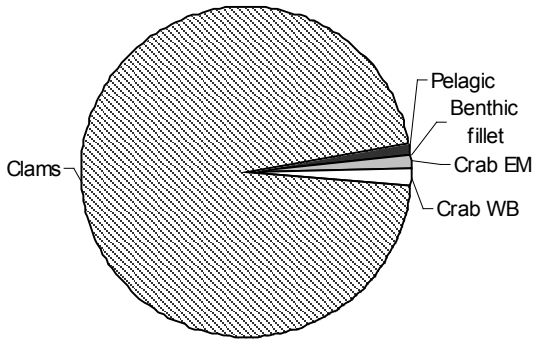
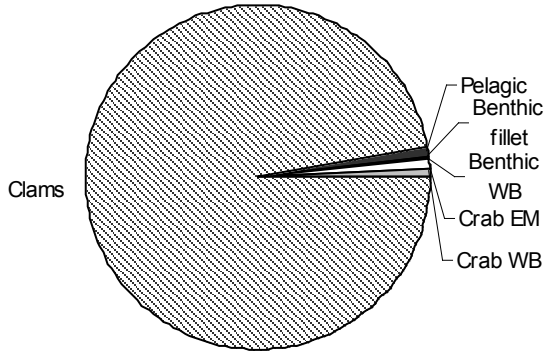
[a] Figures represent both cancer and non-cancer risks. Risk percentages are based on EPC and ingestion rates, meaning that the percentage of risk from each consumption category is the same for cancer and non-cancer risks.

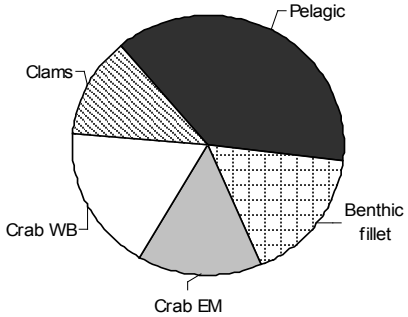
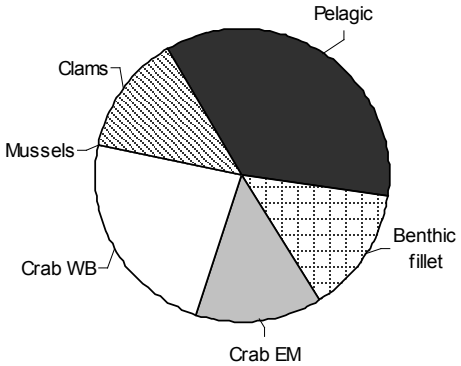
[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

API – Asian and Pacific Islander
EM – edible meat
EPC – exposure point concentration
na – not applicable
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent
WB – whole body

Table B.5-21. Comparison of excess cancer and non-cancer risks by consumption class for selected chemicals in the adult API CT seafood consumption scenario

Consumption Category	EPC[a] (mg/kg-ww)	Ingestion Rate (g/day)	Percent of Chemical's Risk	Excess Cancer Risk	Non-Cancer Hazard Quotient	Adult API CT Seafood Consumption Risk Expressed as Pie Chart[b]
Arsenic[c]						
Pelagic	0.057	0.55	0.8%	1×10^{-7}	0.002	
Benthic fillet	0.0040	0.23	0.02%	3×10^{-9}	0.00005	
Benthic whole body	0.056	0.04	0.06%	8×10^{-9}	0.0001	
Crab edible meat	0.023	0.65	0.4%	5×10^{-8}	0.0008	
Crab whole body	0.075	0.55	1.0%	1×10^{-7}	0.002	
Clams	1.2	3.3	97.8%	1×10^{-5}	0.2	
			Total risk from arsenic	**1×10^{-5}**	**0.2**	
cPAHs (2004 data only)[c,d]						
Pelagic	0.00078	0.55	0.9%	6×10^{-9}	na	
Benthic fillet	0.00050	0.23	0.2%	2×10^{-9}	na	
Benthic whole body	0.0014	0.04	0.1%	9×10^{-10}	na	
Crab edible meat	0.00063	0.65	0.4%	6×10^{-9}	na	
Crab whole body	0.00075	0.55	1.0%	6×10^{-9}	na	
Clams	0.015	3.3	97.2%	7×10^{-7}	na	
			Total risk from cPAHs	**8×10^{-7}**	**na**	

Consumption Category	EPC[a] (mg/kg-ww)	Ingestion Rate (g/day)	Percent of Chemical's Risk	Excess Cancer Risk	Non-Cancer Hazard Quotient	Adult API CT Seafood Consumption Risk Expressed as Pie Chart[b]
PCB TEQ[c]						
Pelagic	1.99×10^{-5}	0.55	45.4%	3×10^{-6}	na	
Benthic fillet	9.50×10^{-6}	0.23	8.8%	7×10^{-7}	na	
Benthic whole body	1.59×10^{-5}	0.04	2.9%	2×10^{-7}	na	
Crab edible meat	2.00×10^{-6}	0.65	5.3%	4×10^{-7}	na	
Crab whole body	7.70×10^{-6}	0.66	17.6%	1×10^{-6}	na	
Clams	1.48×10^{-6}	3.3	20.0%	1×10^{-6}	na	
Total risk from PCB TEQ				**7×10^{-6}**	**na**	
Total PCBs						
Pelagic	1.7	0.50	40.8%	3×10^{-6}	0.7	
Benthic fillet	0.70	0.20	6.8%	6×10^{-7}	0.1	
Benthic whole body	2.2	0.04	4.3%	4×10^{-7}	0.07	
Crab edible meat	0.17	0.59	4.9%	4×10^{-7}	0.08	
Crab whole body	0.89	0.51	22.0%	2×10^{-6}	0.4	
Mussels	0.034	0.47	0.8%	7×10^{-8}	0.01	
Clams	0.14	3.0	20.4%	2×10^{-6}	0.3	
Total risk from total PCBs				**8×10^{-6}**	**2**	

[a] EPC used for CT scenarios is mean value

[b] Figures represent both cancer and non-cancer risks. Risk percentages are based on EPC and ingestion rates, meaning that the percentage of risk from each consumption category is the same for cancer and non-cancer risks.

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[d] cPAH concentrations are presented as benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

API – Asian and Pacific Islander
CT – central tendency
EM – edible meat
EPC – exposure point concentration
na – not applicable
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
TEQ – toxic equivalent
WB – whole body

As shown in Table B.5-17, the vast majority of risk estimated for inorganic arsenic and cPAHs in the adult and child tribal RME scenarios based on Tulalip data was attributable to clam consumption. Clam consumption was responsible for most of the risk for arsenic and cPAHs for the other consumption scenarios that included a market basket of seafood categories as well (Tables B.5-17 to B.5-21). Inorganic arsenic and cPAH risks were mostly related to clam consumption because clams have higher concentrations of these chemicals and were assumed to be heavily consumed compared to other seafood categories. For inorganic arsenic, 96% of excess cancer risk estimates were attributed to the consumption of clams for the adult and child tribal RME scenarios based on Tulalip data (Table B.5-17). As discussed in Section B.5.5, a portion of the inorganic arsenic in the LDW, particularly for seafood categories other than clams, is related to sources outside the LDW, including general background sources.

For the adult and child tribal consumption scenarios (RME and CT) based on Tulalip data, pelagic fish contributed over one-third of the total PCB TEQ risk estimates, even though pelagic fish were a relatively small portion of consumption by mass (Tables B.5-17 and B.5-18). Crabs (both whole body and edible meat) and clams together contributed significantly to estimated PCB TEQ excess cancer risk. For total PCBs in the adult tribal scenarios based on Tulalip data, the same categories (i.e., pelagic fish, crab, and clam) were key contributors, although pelagic fish were less significant in the total PCB risk estimates than in PCB TEQ risk estimates. It should be noted that some of the differences in risk apportionment seen for total PCBs and PCB TEQs may result from the fact that different sets of samples were used to derive each of these risk estimates (see Section 6.1.1.8).

The relative contribution of the different seafood categories to risk associated with PCBs, cPAHs, and arsenic was similar for the adult tribal RME scenario based on Tulalip data, child tribal RME scenario based on Tulalip data, and adult API scenario (Tables B.5-17, B.5-18, B.5-20, and B.5-21). One notable difference between the tribal scenarios based on Tulalip data and API risk percentages was the increased consumption of whole-body tissue for the API population. Thus, the percentage of the total risk for each chemical from whole-body benthic fish tissue and whole-body crab tissue was higher in API scenarios.

However, there were significant differences between these scenarios and the adult tribal scenario based on Suquamish data (Table B.5-20). Over 98% of the estimated excess cancer risk for the adult tribal scenario based on Suquamish data for either arsenic or cPAHs was attributable to clam consumption, which constituted the majority of their seafood diet (438.6 g of 583.5 g total daily non-anadromous seafood consumption). The concentration of inorganic arsenic in clams was much higher than in other seafood categories (over 10 times higher than the next highest seafood category). In the case of excess cancer risks for either PCB TEQ or total PCBs, both clams and pelagic fish were significant contributors to the overall risk levels, each

making up 25% or more of the total risk. Although pelagic fish were a much smaller portion of the diet in the adult tribal scenario based on Suquamish data (56 g/day, as compared with clam consumption above), they were a major contributor to PCB risks because of their high PCB concentrations.

B.5.3.1.4. Influence of chemicals with low detection frequency on seafood risk estimates

Risk were estimated for chemicals detected in at least one tissue sample; however, many chemicals were detected in only one or a few of the several seafood categories included in each of the risk estimates. If a chemical was not detected in a seafood category, one-half the highest RL was selected as the EPC for that category. Where the RLs were high for undetected analytes, related risk estimates could be heavily influenced by the use of one-half the highest RL. Of the 30 COPCs detected in tissue, 19 were not detected in at least one of the seven seafood consumption categories. The different behaviors and feeding strategies of aquatic biota could result in COPCs being present in certain classes of aquatic biota and not in others. The presence of these COPCs in at least one seafood category could indicate that some risk could be associated with them, although if these COPCs were present, they were present at concentrations less than the RL.

To investigate whether RLs for COPCs with low detection frequencies might have influenced risk estimates for individual COPCs, the percent of each chemical's risk that was attributed to consumption categories with no detected values (i.e., the EPC is based on one-half the highest RL) was evaluated for several seafood consumption scenarios. For the adult and child tribal scenarios based on Tulalip data, greater than 50% of the risk for 7 of the 30 detected COPCs was derived from consumption categories in which a given COPC was not detected (see Table B.5-22). For the adult tribal scenario based on Suquamish data, one chemical had this designation (Table B.5-22), and two other chemicals[24] had over 40% of the risk estimates derived from categories with no detected concentrations. For the adult API scenarios (CT and RME), contributions from undetected seafood categories did not exceed 50% of the risk estimates for any chemicals evaluated in the risk characterization, but three chemicals[25] had over 40% of their risk estimates associated with seafood categories with no detects.

The chemicals identified in Table B.5-22 and were also identified with footnotes in the risk results tables for the tribal seafood consumption scenarios based on Tulalip and Suquamish data. Table B.5-22 also presents the seafood categories for which there were no detected values. Chemicals with no detected values for the seafood consumption category being evaluated were also identified with footnotes in the risk results tables for the one-meal-per-month scenarios. Crab edible meat and mussels had

[24] Butyl benzyl phthalate and heptachlor

[25] 4-methylphenol, heptachlor, and pentachlorophenol

undetected values for all of these chemicals, while pelagic fish had detected values for all these chemicals. The undetected chemicals were primarily pesticides. When pesticides were detected in tissues, they were mostly qualified as JN, indicating substantial uncertainty about whether the analyte is actually present in the tissue and its concentration. The uncertainties associated with infrequently detected pesticides and the JN-qualification are further discussed in the uncertainty section (B.6.1.1.3).

Table B.5-22. Summary for different seafood consumption scenarios of chemicals with greater than 50% of risk derived from seafood categories with no detected values

Chemical	Seafood Consumption Scenarios with Greater than 50% of Risk from Undetected Chemicals[a]		Seafood Consumption Categories with No Detected Values						
	Adult and Child Tribal RME and CT (Tulalip Data)	Adult Tribal (Suquamish Data)	Benthic Fillet	Benthic WB	Clams	Crab EM	Crab WB	Mussel	Pelagic
4-Methylphenol	x		x	x		x	x	x	
Aldrin	x		x			x	x	x	
alpha-BHC	x			x		x		x	
beta-BHC	x					x	x	x	
Butyl benzyl phthalate	x		x		x	x		x	
Endrin	x					x	x	x	
Heptachlor	x		x		x	x	x	x	
Pentachlorophenol	x	x	x		x	x	x	x	

[a] Greater than 50% of the risk in the indicated seafood consumption scenarios is derived from seafood consumption categories with no detected values. In these cases, one-half the maximum reporting limit was selected as the EPC.

BHC – benzene hexachloride

CT – central tendency

EM – edible meat

RME – reasonable maximum exposure

WB – whole body

RLs also influenced EPCs when there was a mixture of detected and undetected concentrations for samples within a seafood category. There were over 20 chemicals with at least one seafood category in which the chemical was detected in less than 50% of the samples. Risk estimates for dieldrin and hexachlorobenzene, in particular, appear to have been influenced by the RLs for undetected samples. Over 50% of the risk for these chemicals for the adult and child tribal RME scenarios based on Tulalip data, and for the adult API (RME and CT) scenarios was derived from seafood categories in which the EPC as well as the maximum RL were greater than the maximum detected concentration. This was also true for the risk estimates for alpha-benzene hexachloride (BHC) for the adult API (RME and CT) scenarios. For the same

three chemicals (alpha-BHC, dieldrin, and hexachlorobenzene) for the adult tribal scenario based on Suquamish data, over 70% of risk was from seafood categories where the maximum RL and EPC were greater than the maximum detected concentration.

B.5.3.2 Netfishing

Netfishing risks were estimated for the entire LDW for both the RME and CT scenarios. It is possible that individual tribal members would use smaller areas of the LDW, but there is no information available on areas actually fished. For example, individual Muckleshoot gillnetters may use the same portions of the LDW from fishing season to fishing season. The health risks associated with more localized use is discussed in the uncertainty section (B.6.3.3.4).

Overall, the risk estimates associated with netfishing were much lower than for seafood consumption. For the netfishing RME scenario, the total excess cancer risk for both the incidental sediment ingestion and dermal absorption exposure routes was 3×10^{-5} for the two calculated summations excluding either the PCB TEQ or total PCBs (see introduction to Section B.5.3) (Table B.5-23). The greatest contributor to the netfishing RME excess cancer risk was dioxin/furan TEQ, which accounted for approximately two-thirds of the total excess cancer risk. The majority of the risk attributed to dioxin/furan TEQ is associated with elevated concentrations in sediment at locations specifically targeted during the 2005 sampling because they were near suspected sources of dioxins/furans (Map B.3-4) (Windward 2005h). Because dioxins/furans are typically found as background contaminants in urban areas (EPA 2000c) including sediments in the greater Seattle area (see Section B.5.5), a portion of the risk attributed to dioxin/furan TEQ is likely related to sources outside the LDW. Potential sources of urban background concentrations of dioxins/furans in the LDW and Seattle areas, and associated cancer risks, are discussed in Section B.5.5.

The total excess cancer risk estimate for the combined exposure routes for the netfishing CT scenario was 5×10^{-6} when summed excluding PCB TEQ, and 6×10^{-6} when summed excluding total PCBs (Table B.5-24). Also, as was the case with the netfishing RME scenario, dioxin/furan TEQ was the greatest risk contributor for the netfishing CT scenario, contributing approximately half of the estimated total excess cancer risk. For the RME scenario, arsenic, dioxins/furans, PCB TEQ, total PCBs, and toxaphene exceeded the 1×10^{-6} excess cancer risk estimate. For the CT scenario, only dioxin/furans exceeded 1×10^{-6} excess cancer risk estimate (Tables B.5-23 and B.5-24).

Non-cancer hazards for the netfishing scenario were found to be below levels of concern, with the total HIs for both RME and CT scenarios substantially less than 1 (Tables B.5-25 and B.5-26). Effect-specific HIs were not calculated because the total HI across all effects was less than 1 (see Section B.5.1.2).

Table B.5-23. Excess cancer risk estimates for the netfishing RME scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Commercial fishermen
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	21	2.6×10^{-6}	1.1×10^{-6}	1.5	4×10^{-6}	2×10^{-6}	6×10^{-6}
cPAHs[b]	0.57	7.1×10^{-8}	1.3×10^{-7}	7.3	5×10^{-7}	1×10^{-6}	1×10^{-6}
Dioxin/furan TEQ	0.000610	7.6×10^{-11}	3.3×10^{-11}	150,000	1×10^{-5}	5×10^{-6}	2×10^{-5}
PCB TEQ	0.0000718	9.0×10^{-12}	1.8×10^{-11}	150,000	1×10^{-6}	3×10^{-6}	4×10^{-6}
Total PCBs	2.5[c]	3.1×10^{-7}	6.3×10^{-7}	2	6×10^{-7}	1×10^{-6}	2×10^{-6}
						Subtotal excluding PCB TEQ	**3×10^{-5}**
						Subtotal excluding Total PCBs	**3×10^{-5}**
Tentatively identified chemicals (JN-qualified)							
Dieldrin	0.0045	5.6×10^{-10}	8.1×10^{-10}	16	9×10^{-9}	1×10^{-8}	2×10^{-8}
Toxaphene	6.3	7.9×10^{-7}	1.1×10^{-6}	1.1	9×10^{-7}	1×10^{-6}	2×10^{-6}
						Subtotal	**2×10^{-6}**
Total risk (excluding PCB TEQ) across both exposure routes							**3×10^{-5}**
Total risk (excluding total PCBs) across both exposure routes							**3×10^{-5}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[c] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

CDI – chronic daily intake

cPAH – carcinogenic polycyclic aromatic hydrocarbon

dw – dry weight

EPC – exposure point concentration

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

SWAC – spatially weighted average concentration

TEQ – toxic equivalent

Table B.5-24. Excess cancer risk estimates for the netfishing CT scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Commercial fishermen
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	21	9.2×10^{-7}	4.0×10^{-8}	1.5	1×10^{-6}	6×10^{-8}	1×10^{-6}
cPAHs[b]	0.57	2.5×10^{-8}	4.7×10^{-9}	7.3	2×10^{-7}	3×10^{-8}	2×10^{-7}
Dioxin/furan TEQ	0.000610	2.7×10^{-11}	1.2×10^{-12}	150,000	4×10^{-6}	2×10^{-7}	4×10^{-6}
PCB TEQ	0.0000718	3.1×10^{-12}	6.3×10^{-13}	150,000	5×10^{-7}	9×10^{-8}	6×10^{-7}
Total PCBs	2.5[c]	1.1×10^{-7}	2.2×10^{-8}	2	2×10^{-7}	4×10^{-8}	3×10^{-7}
						Subtotal excluding PCB TEQ	**5×10^{-6}**
						Subtotal excluding Total PCBs	**6×10^{-6}**
Tentatively identified chemicals (JN-qualified)							
Dieldrin	0.0045	2.0×10^{-10}	2.8×10^{-11}	16	3×10^{-9}	5×10^{-10}	4×10^{-9}
Toxaphene	6.3	2.8×10^{-7}	4.0×10^{-8}	1.1	3×10^{-7}	4×10^{-8}	3×10^{-7}
						Subtotal	**3×10^{-7}**
Total risk (excluding PCB TEQ) across both exposure routes							**5×10^{-6}**
Total risk (excluding total PCBs) across both exposure routes							**6×10^{-6}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[c] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

CDI – chronic daily intake

cPAH – carcinogenic polycyclic aromatic hydrocarbon

CT – central tendency

dw – dry weight

EPC – exposure point concentration

PCB – polychlorinated biphenyl

SWAC – spatially weighted average concentration

TEQ – toxic equivalent

Table B.5-25. Non-cancer hazard estimates for the netfishing RME scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Commercial fishermen
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	19,000	3.8×10^{-3}	na[b]	1	0.004	na[b]	0.004
Antimony	3.4	6.8×10^{-7}	na[b]	0.0004	0.002	na[b]	0.002
Arsenic	21	4.2×10^{-6}	1.8×10^{-6}	0.0003	0.01	0.006	0.02
Barium	230	4.6×10^{-5}	na[b]	0.2	0.0002	na[b]	0.0002
Cadmium[c]	1.5	3.0×10^{-7}	4.3×10^{-9}	0.001	0.0003	0.0002	0.0005
Chromium	51	1.0×10^{-5}	na[b]	0.003	0.003	na[b]	0.003
Copper	200	4.0×10^{-5}	na[b]	0.04	0.001	na[b]	0.001
Iron	29,000	5.8×10^{-3}	na[b]	0.3	0.02	na[b]	0.02
Manganese	360	7.2×10^{-5}	na[b]	0.14	0.0005	na[b]	0.0005
Total PCBs	2.5[d]	5.0×10^{-7}	1.0×10^{-6}	0.00002	0.02	0.05	0.07
Thallium	2.4	4.8×10^{-7}	na[b]	0.00007	0.007	na[b]	0.007
Vanadium	60	1.2×10^{-5}	na[b]	0.001	0.01	na[b]	0.01
						Subtotal	**0.1**
Tentatively identified chemicals (JN-qualified)							
Dieldrin	0.0045	9.0×10^{-10}	1.3×10^{-9}	0.00005	0.00002	0.00003	0.00005
						Subtotal	**0.00005**
Total hazard index across both exposure routes[e]							**0.1**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure is based on an absorbed dose. The adjustment allows exposure and toxicity to be expressed in terms of absorbed dose (EPA 2004d).

[d] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

[e] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RfD – reference dose
RME – reasonable maximum exposure
SWAC – spatially weighted average concentration

Table B.5-26. Non-cancer hazard estimates for the netfishing CT scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Commercial fishermen
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	19,000	2.0×10^{-3}	na[b]	1	0.002	na[b]	0.002
Antimony	3.4	3.6×10^{-7}	na[b]	0.0004	0.0009	na[b]	0.0009
Arsenic	21	2.2×10^{-6}	9.6×10^{-8}	0.0003	0.007	0.0003	0.008
Barium	230	2.4×10^{-5}	na[b]	0.2	0.0001	na[b]	0.0001
Cadmium[c]	1.5	1.6×10^{-7}	2.3×10^{-10}	0.001	0.0002	0.000009	0.0002
Chromium	51	5.4×10^{-6}	na[b]	0.003	0.002	na[b]	0.002
Copper	200	2.1×10^{-5}	na[b]	0.04	0.0005	na[b]	0.0005
Iron	29,000	3.1×10^{-3}	na[b]	0.3	0.01	na[b]	0.01
Manganese	360	3.8×10^{-5}	na[b]	0.14	0.0003	na[b]	0.0003
Total PCBs	2.5[d]	2.6×10^{-7}	5.3×10^{-8}	0.00002	0.01	0.003	0.01
Thallium	2.4	2.5×10^{-7}	na[b]	0.00007	0.004	na[b]	0.004
Vanadium	60	6.3×10^{-6}	na[b]	0.001	0.006	na[b]	0.006
						Subtotal	**0.04**
Tentatively identified chemicals (JN-qualified)							
Dieldrin	0.0045	4.7×10^{-10}	6.8×10^{-11}	0.00005	0.000009	0.000001	0.00001
						Subtotal	0.00001
Total hazard index across both exposure routes[e]							**0.04**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure is based on an absorbed dose. The adjustment allows exposure and toxicity to be expressed in terms of absorbed dose (EPA 2004d).

[d] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

[e] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake
CT – central tendency
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RfD – reference dose
SWAC – spatially weighted average concentration

B.5.3.3 Beach play RME

Upper bound excess cancer risks and non-cancer hazards were estimated individually for the eight beach play intertidal exposure areas, as described in Section B.3.2.2. As with the seafood consumption and netfishing scenarios, total risk estimates were similar for summations that excluded either PCB TEQ or total PCBs. Total excess cancer risks (regardless of summation approach) were as follows for the specified area:

- Area 1 2×10^{-5} (Table B.5-27)
- Area 2 5×10^{-5} (Table B.5-28)
- Area 3 3×10^{-5} (Table B.5-29)
- Area 4 3×10^{-5} (Table B.5-30)
- Area 5 8×10^{-6} (Table B.5-31)
- Area 6 9×10^{-6} and 8×10^{-6}, excluding either the PCB TEQ or total PCB estimates, respectively (Table B.5-32)
- Area 7 5×10^{-5} (Table B.5-33)
- Area 8 7×10^{-6} (Table B.5-34)

Risk estimates for all beach play RME scenarios exceeded the total excess cancer risk threshold of 1×10^{-6}. The spatial coverage of sediment areas for many chemicals, particularly dioxins/furans, was limited for the beach play RME scenarios (Map B.3-1). Uncertainties in risk estimates related to spatial coverage of the sediment data are discussed in Section B.6.1.7. In addition, risk estimates specifically for the Duwamish Waterway Park are presented in Section B.6.3.3.2, and risk estimates for dog walkers and habitat restoration workers are presented in Section B.6.1.9.

Table B.5-27. Excess cancer risk estimates for the beach play RME Area 1 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	15	3.4×10^{-6}	2.0×10^{-7}	1.5	5×10^{-6}	3×10^{-7}	5×10^{-6}
cPAHs[b, c]	1.2	2.7×10^{-7}	7.1×10^{-8}	7.3	1×10^{-5}	3×10^{-6}	1×10^{-5}
Dioxin/furan TEQ[d]	na	na	na	na	na	na	na
PCB TEQ	9.08×10^{-8}	2.1×10^{-14}	5.8×10^{-15}	150,000	3×10^{-9}	9×10^{-10}	4×10^{-9}
Total PCBs	0.12	2.7×10^{-8}	7.6×10^{-9}	2	5×10^{-8}	2×10^{-8}	7×10^{-8}
						Subtotal excluding PCB TEQ	**2×10^{-5}**
						Subtotal excluding Total PCBs	**2×10^{-5}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0037	8.4×10^{-10}	5.0×10^{-11}	0.34	3×10^{-10}	2×10^{-11}	3×10^{-10}
Dieldrin	0.0013	3.0×10^{-10}	5.9×10^{-11}	16	5×10^{-9}	9×10^{-10}	6×10^{-9}
Toxaphene[e]	0.049	1.1×10^{-8}	2.2×10^{-9}	1.1	1×10^{-8}	2×10^{-9}	1×10^{-8}
						Subtotal	**2×10^{-8}**
Total risk (excluding PCB TEQ) across both exposure routes							**2×10^{-5}**
Total risk (excluding total PCBs) across both exposure routes							**2×10^{-5}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[d] No data exist for this chemical in this area

[e] Not detected in this area but included for completeness and comparison to other areas.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table B.5-28. Excess cancer risk estimates for the beach play RME Area 2 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	21	4.8×10^{-6}	2.9×10^{-7}	1.5	7×10^{-6}	4×10^{-7}	7×10^{-6}
cPAHs[b, c]	3	6.8×10^{-7}	1.8×10^{-7}	7.3	3×10^{-5}	7×10^{-6}	4×10^{-5}
Dioxin/furan TEQ[d]	na	na	na	na	na	na	na
PCB TEQ	6.69×10^{-6}	1.5×10^{-12}	4.2×10^{-13}	150,000	2×10^{-7}	6×10^{-8}	3×10^{-7}
Total PCBs	0.18	4.1×10^{-8}	1.1×10^{-8}	2	8×10^{-8}	2×10^{-8}	1×10^{-7}
Subtotal excluding PCB TEQ							**5×10^{-5}**
Subtotal excluding Total PCBs							**5×10^{-5}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.013	3.0×10^{-9}	1.8×10^{-10}	0.34	1×10^{-9}	6×10^{-11}	1×10^{-9}
Dieldrin	0.0005	1.1×10^{-10}	2.3×10^{-11}	16	2×10^{-9}	4×10^{-10}	2×10^{-9}
Toxaphene[e]	0.025	5.7×10^{-9}	1.1×10^{-9}	1.1	6×10^{-9}	1×10^{-9}	7×10^{-9}
Subtotal							**1×10^{-8}**
Total risk (excluding PCB TEQ) across both exposure routes							**5×10^{-5}**
Total risk (excluding total PCBs) across both exposure routes							**5×10^{-5}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[d] No data exist for this chemical in this area

[e] Not detected in this area but included for completeness and comparison to other areas.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table B.5-29. Excess cancer risk estimates for the beach play RME Area 3 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	13	3.0×10^{-6}	1.8×10^{-7}	1.5	4×10^{-6}	3×10^{-7}	4×10^{-6}
cPAHs[b, c]	2.1	4.8×10^{-7}	1.2×10^{-7}	7.3	2×10^{-5}	5×10^{-6}	3×10^{-5}
Dioxin/furan TEQ[d]	na	na	na	na	na	na	na
PCB TEQ[d]	na	na	na	na	na	na	na
Total PCBs	0.24	5.5×10^{-8}	1.5×10^{-8}	2	1×10^{-7}	3×10^{-8}	1×10^{-7}
						Subtotal excluding PCB TEQ	**3×10^{-5}**
						Subtotal excluding Total PCBs	**3×10^{-5}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.021	4.8×10^{-9}	2.9×10^{-10}	0.34	2×10^{-9}	1×10^{-10}	2×10^{-9}
Dieldrin	0.01	2.3×10^{-9}	4.5×10^{-10}	16	4×10^{-8}	7×10^{-9}	5×10^{-8}
Toxaphene[e]	0.09	2.0×10^{-8}	4.1×10^{-9}	1.1	2×10^{-8}	4×10^{-9}	2×10^{-8}
						Subtotal	**7×10^{-8}**
Total risk (excluding PCB TEQ) across both exposure routes							**3×10^{-5}**
Total risk (excluding total PCBs) across both exposure routes							**3×10^{-5}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[d] No data exist for this chemical in this area

[e] Not detected in this area but included for completeness and comparison to other areas.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table B.5-30. Excess cancer risk estimates for the beach play RME Area 4 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	11	2.5×10^{-6}	1.5×10^{-7}	1.5	4×10^{-6}	2×10^{-7}	4×10^{-6}
cPAHs[b, c]	0.73	1.7×10^{-7}	4.3×10^{-8}	7.3	6×10^{-6}	2×10^{-6}	8×10^{-6}
Dioxin/furan TEQ	4.12×10^{-4}	9.4×10^{-11}	5.6×10^{-12}	150,000	1×10^{-5}	8×10^{-7}	1×10^{-5}
PCB TEQ	2.04×10^{-4}	4.6×10^{-11}	1.3×10^{-11}	150,000	7×10^{-6}	2×10^{-6}	9×10^{-6}
Total PCBs	11	2.5×10^{-6}	7.0×10^{-7}	2	5×10^{-6}	1×10^{-6}	6×10^{-6}
Subtotal excluding PCB TEQ							**3×10^{-5}**
Subtotal excluding Total PCBs							**3×10^{-5}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.15	9.1×10^{-8}	5.4×10^{-9}	0.34	3×10^{-8}	2×10^{-9}	3×10^{-8}
Dieldrin	0.014	3.9×10^{-9}	7.7×10^{-10}	16	6×10^{-8}	1×10^{-8}	7×10^{-8}
Toxaphene[d]	0.24	1.9×10^{-7}	3.9×10^{-8}	1.1	2×10^{-7}	4×10^{-8}	2×10^{-7}
Subtotal							**3×10^{-7}**
Total risk (excluding PCB TEQ) across both exposure routes							**3×10^{-5}**
Total risk (excluding total PCBs) across both exposure routes							**3×10^{-5}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[d] Not detected in this area but included for completeness and comparison to other areas.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table B.5-31. Excess cancer risk estimates for the beach play RME Area 5 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	8.9	2.0×10^{-6}	1.2×10^{-7}	1.5	3×10^{-6}	2×10^{-7}	3×10^{-6}
cPAHs[b, c]	0.41	9.3×10^{-8}	2.4×10^{-8}	7.3	4×10^{-6}	9×10^{-7}	5×10^{-6}
Dioxin/furan TEQ	2.20×10^{-6}	5.0×10^{-13}	3.0×10^{-14}	150,000	8×10^{-8}	4×10^{-9}	8×10^{-8}
PCB TEQ	2.51×10^{-6}	5.7×10^{-13}	1.6×10^{-13}	150,000	9×10^{-8}	2×10^{-8}	1×10^{-7}
Total PCBs	0.19	4.3×10^{-8}	1.2×10^{-8}	2	9×10^{-8}	2×10^{-8}	1×10^{-7}
Subtotal excluding PCB TEQ							**8×10^{-6}**
Subtotal excluding Total PCBs							**8×10^{-6}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.035	8.0×10^{-9}	4.8×10^{-10}	0.34	3×10^{-9}	2×10^{-10}	3×10^{-9}
Dieldrin	0.0026	5.9×10^{-10}	1.2×10^{-10}	16	9×10^{-9}	2×10^{-9}	1×10^{-8}
Toxaphene	0.34	7.7×10^{-8}	1.5×10^{-8}	1.1	9×10^{-8}	2×10^{-8}	1×10^{-7}
Subtotal							**1×10^{-7}**
Total risk (excluding PCB TEQ) across both exposure routes							**8×10^{-6}**
Total risk (excluding total PCBs) across both exposure routes							**8×10^{-6}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table B.5-32. Excess cancer risk estimates for the beach play RME Area 6 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	9.8	2.2×10^{-6}	1.3×10^{-7}	1.5	3×10^{-6}	2×10^{-7}	3×10^{-6}
cPAHs[b, c]	0.44	1.0×10^{-7}	2.6×10^{-8}	7.3	4×10^{-6}	1×10^{-6}	5×10^{-6}
Dioxin/furan TEQ[d]	na	na	na	na	na	na	na
PCB TEQ	5.37×10^{-6}	1.2×10^{-12}	3.4×10^{-13}	150,000	2×10^{-7}	5×10^{-8}	3×10^{-7}
Total PCBs	.97	2.2×10^{-7}	6.2×10^{-8}	2	4×10^{-7}	1×10^{-7}	5×10^{-7}
						Subtotal excluding PCB TEQ	**9×10^{-6}**
						Subtotal excluding Total PCBs	**8×10^{-6}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0005	1.1×10^{-10}	6.8×10^{-12}	0.34	4×10^{-11}	2×10^{-12}	4×10^{-11}
Dieldrin	0.001	2.3×10^{-10}	4.5×10^{-11}	16	4×10^{-9}	7×10^{-10}	5×10^{-9}
Toxaphene[e]	0.049	1.1×10^{-8}	2.2×10^{-9}	1.1	1×10^{-8}	2×10^{-9}	1×10^{-8}
						Subtotal	**2×10^{-8}**
Total risk (excluding PCB TEQ) across both exposure routes							**9×10^{-6}**
Total risk (excluding total PCBs) across both exposure routes							**8×10^{-6}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[d] No data exist for this chemical in this area

[e] Not detected in this area but included for completeness and comparison to other areas.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table B.5-33. Excess cancer risk estimates for the beach play RME Area 7 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	11	2.5×10^{-6}	1.5×10^{-7}	1.5	4×10^{-6}	2×10^{-7}	4×10^{-6}
cPAHs[b, c]	0.11	2.5×10^{-8}	6.5×10^{-9}	7.3	1×10^{-6}	3×10^{-7}	1×10^{-6}
Dioxin/furan TEQ	1.70×10^{-6}	3.9×10^{-13}	2.3×10^{-14}	150,000	6×10^{-8}	3×10^{-9}	6×10^{-8}
PCB TEQ	5.65×10^{-7}	1.3×10^{-13}	3.6×10^{-14}	150,000	2×10^{-8}	5×10^{-9}	3×10^{-8}
Total PCBs	0.23	5.2×10^{-8}	1.5×10^{-8}	2	1×10^{-7}	3×10^{-8}	1×10^{-7}
						Subtotal excluding PCB TEQ	**5×10^{-6}**
						Subtotal excluding Total PCBs	**5×10^{-6}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0027	6.1×10^{-10}	3.7×10^{-11}	0.34	2×10^{-10}	1×10^{-11}	2×10^{-10}
Dieldrin	0.001	2.3×10^{-10}	4.5×10^{-11}	16	4×10^{-9}	7×10^{-10}	5×10^{-9}
Toxaphene[d]	0.049	1.1×10^{-8}	2.2×10^{-9}	1.1	1×10^{-8}	2×10^{-9}	1×10^{-8}
						Subtotal	**2×10^{-8}**
Total risk (excluding PCB TEQ) across both exposure routes							**5×10^{-6}**
Total risk (excluding total PCBs) across both exposure routes							**5×10^{-6}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[d] Not detected in this area but included for completeness and comparison to other areas.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table B.5-34. Excess cancer risk estimates for the beach play RME Area 8 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	10	2.3×10^{-6}	1.4×10^{-7}	1.5	3×10^{-6}	2×10^{-7}	3×10^{-6}
cPAHs[b, c]	0.32	7.3×10^{-8}	1.9×10^{-8}	7.3	3×10^{-6}	7×10^{-7}	4×10^{-6}
Dioxin/furan TEQ[d]	na	na	na	na	na	na	na
PCB TEQ	1.89×10^{-6}	4.3×10^{-13}	1.2×10^{-13}	150,000	6×10^{-8}	2×10^{-8}	8×10^{-8}
Total PCBs	0.23	5.2×10^{-8}	1.5×10^{-8}	2	1×10^{-7}	3×10^{-8}	1×10^{-7}
						Subtotal excluding PCB TEQ	**7×10^{-6}**
						Subtotal excluding Total PCBs	**7×10^{-6}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0082	1.9×10^{-9}	1.1×10^{-10}	0.34	6×10^{-10}	4×10^{-11}	6×10^{-10}
Dieldrin	0.0023	5.2×10^{-10}	1.0×10^{-10}	16	8×10^{-9}	2×10^{-9}	1×10^{-8}
Toxaphene[e]	0.049	1.1×10^{-8}	2.2×10^{-9}	1.1	1×10^{-8}	2×10^{-9}	1×10^{-8}
						Subtotal	**2×10^{-8}**
Total risk (excluding PCB TEQ) across both exposure routes							**7×10^{-6}**
Total risk (excluding total PCBs) across both exposure routes							**7×10^{-6}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[d] No data exist for this chemical in this area.

[e] Not detected in this area but included for completeness and comparison to other areas.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

For all of the beach play RME areas except Area 4, the majority (over 93%) of total excess cancer risk was attributable to cPAHs and arsenic. No results for dioxins/furans are available for five of the eight beach play areas, and only a single sample was analyzed for dioxins/furans in the other three areas. The limited data for this class of chemicals, compared to other chemicals, contributes relatively high uncertainty to the comparative contribution of dioxins/furans to risks. In beach play Area 4, the risks for the RME exposure scenario were spread primarily among arsenic, PCBs, carcinogenic PAHs, and dioxin/furan TEQ with the greatest contribution from the latter two chemical groups. The risk from arsenic and cPAHs was above the 1×10^{-6} excess cancer risk threshold in all areas assessed in the beach play RME scenarios.

The breakdown of chemicals contributing to the excess cancer risks associated with beach play RME Area 4 is different from that of other areas because a single sample is driving the EPC calculation for total PCBs, PCB TEQ, and dioxin/furan TEQ. As a result, the EPCs for these three chemicals in Area 4 are two orders of magnitude higher than the EPCs for the other areas. The implications for EPC calculations and risk estimates of a single or a few samples with high concentrations are discussed in the uncertainty analysis (Section B.6.1.1).

HQs were less than 1 for all chemicals for beach play RME scenarios at all beach play areas except for total PCBs at Area 4, where the HQ was 1 (Tables B.5-35 through B.5-42). Total His for all chemicals were less than 1 for all areas except beach play Areas 3 and 4, which had total His of 1 and 2, respectively. Endpoint-specific His were calculated only for Area 4 because total His for all other areas did not exceed 1. For Area 4, endpoint-specific His did not exceed 1.

Table B.5-35. Non-cancer hazard estimates for the beach play RME Area 1 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum[b]	na	na	na	1	na	na	na
Antimony	1.1	2.9×10^{-6}	na[c]	0.0004	0.007	na[c]	0.007
Arsenic	15.0	4.0×10^{-5}	2.4×10^{-6}	0.0003	0.1	0.008	0.1
Barium[b]	-	-	-	0.2	-	-	-
Cadmium[d]	0.15	4.0×10^{-7}	7.9×10^{-10}	0.001	0.0004	0.00003	0.0004
Chromium	21.0	5.6×10^{-5}	na[c]	0.003	0.02	na[c]	0.02
Copper	50.0	1.3×10^{-4}	na[c]	0.04	0.003	na[c]	0.003
Iron[b]	-	-	-	0.3	-	-	-
Manganese[b]	-	-	-	0.14	-	-	-
Mercury	0.17	4.5×10^{-7}	na[c]	0.0001	0.005	na[c]	0.005
Molybdenum	1.8	4.8×10^{-6}	na[c]	0.005	0.001	na[c]	0.001
Total PCBs	0.12	3.2×10^{-7}	8.9×10^{-8}	0.00002	0.02	0.004	0.02
Silver	0.20	5.3×10^{-7}	na[c]	0.005	0.0001	na[c]	0.0001
Thallium	0.15	4.0×10^{-7}	na[c]	0.00007	0.006	na[c]	0.006
Vanadium	47	1.2×10^{-4}	na[c]	0.001	0.1	na[c]	0.1
Zinc	140	3.7×10^{-4}	na[c]	0.3	0.001	na[c]	0.001
						Subtotal	**0.3**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0037	9.8×10^{-9}	5.9×10^{-10}	0.0005	0.00002	0.000001	0.00002
Dieldrin	0.0013	3.5×10^{-9}	6.9×10^{-10}	0.00005	0.00007	0.00001	0.00008
						Subtotal	**0.0001**
Total hazard index across both exposure routes[e]							**0.3**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No data exist for this chemical in this area

[c] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[d] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[e] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake

dw – dry weight

EPC – exposure point concentration

na – not available

PCB – polychlorinated biphenyl

RfD – reference dose

RME – reasonable maximum exposure

Table B.5-36. Non-cancer hazard estimates for the beach play RME Area 2 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum[b]	-	-	-	1	-	-	-
Antimony	2.3	6.1×10^{-6}	na[c]	0.0004	0.02	na[c]	0.02
Arsenic	21	5.6×10^{-5}	3.3×10^{-6}	0.0003	0.2	0.01	0.2
Barium[b]	-	-	-	0.2	-	-	-
Cadmium[d]	1	2.7×10^{-6}	5.3×10^{-9}	0.001	0.003	0.0002	0.003
Chromium	48	1.3×10^{-4}	na[c]	0.003	0.04	na[c]	0.04
Copper	170	4.5×10^{-4}	na[c]	0.04	0.01	na[c]	0.01
Iron[b]	-	-	-	0.3	-	-	-
Manganese[b]	-	-	-	0.1	-	-	-
Mercury	0.63	1.7×10^{-6}	na[c]	0.0001	0.02	na[c]	0.02
Molybdenum	3	8.0×10^{-6}	na[c]	0.005	0.002	na[c]	0.002
Total PCBs	0.18	4.8×10^{-7}	1.3×10^{-7}	0.00002	0.02	0.007	0.03
Silver	0.5	1.3×10^{-6}	na[c]	0.005	0.0003	na[c]	0.0003
Thallium	0.2	5.3×10^{-7}	na[c]	0.00007	0.008	na[c]	0.008
Vanadium	67	1.8×10^{-4}	na[c]	0.001	0.2	na[c]	0.2
Zinc	440	1.2×10^{-3}	na[c]	0.3	0.004	na[c]	0.004
						Subtotal	**0.5**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.013	3.5×10^{-8}	2.1×10^{-9}	0.0005	0.00007	0.000004	0.00007
Dieldrin	0.0005	1.3×10^{-9}	2.6×10^{-10}	0.00005	0.00003	0.000005	0.00003
						Subtotal	**0.0001**
Total hazard index across both exposure routes[e]							**0.5**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No data exist for this chemical in this area

[c] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[d] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[e] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake

dw – dry weight

EPC – exposure point concentration

na – not available

PCB – polychlorinated biphenyl

RfD – reference dose

RME – reasonable maximum exposure

Table B.5-37. Non-cancer hazard estimates for the beach play RME Area 3 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	12000	3.2×10^{-2}	na[b]	1	0.03	na[b]	0.03
Antimony	5.2	1.4×10^{-5}	na[b]	0.0004	0.03	na[b]	0.03
Arsenic	13	3.5×10^{-5}	2.1×10^{-6}	0.0003	0.1	0.007	0.1
Barium	76	2.0×10^{-4}	na[b]	0.2	0.001	na[b]	0.001
Cadmium[c]	2	5.3×10^{-6}	1.1×10^{-8}	0.001	0.005	0.0004	0.005
Chromium	38	1.0×10^{-4}	na[b]	0.003	0.03	na[b]	0.03
Copper	79	2.1×10^{-4}	na[b]	0.04	0.005	na[b]	0.005
Iron	17000	4.5×10^{-2}	na[b]	0.3	0.2	na[b]	0.2
Manganese	240	6.4×10^{-4}	na[b]	0.14	0.005	na[b]	0.005
Mercury	0.31	8.2×10^{-7}	na[b]	0.0001	0.008	na[b]	0.008
Molybdenum	5.8	1.5×10^{-5}	na[b]	0.005	0.003	na[b]	0.003
Total PCBs	0.24	6.4×10^{-7}	1.8×10^{-7}	0.00002	0.03	0.009	0.04
Silver	0.80	2.1×10^{-6}	na[b]	0.005	0.0004	na[b]	0.0004
Thallium	17	4.5×10^{-5}	na[b]	0.00007	0.6	na[b]	0.6
Vanadium	50	1.3×10^{-4}	na[b]	0.001	0.1	na[b]	0.1
Zinc	350	9.3×10^{-4}	na[b]	0.3	0.003	na[b]	0.003
						Subtotal	**1**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.021	5.6×10^{-8}	3.3×10^{-9}	0.0005	0.0001	0.000007	0.0001
Dieldrin	0.01	2.7×10^{-8}	5.3×10^{-9}	0.00005	0.0005	0.0001	0.0006
						Subtotal	**0.00007**
Total hazard index across both exposure routes[d]							**1**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to determine the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake

dw – dry weight

EPC – exposure point concentration

na – not available

PCB – polychlorinated biphenyl

RfD – reference dose

RME – reasonable maximum exposure

Table B.5-38. Non-cancer hazard estimates for the beach play RME Area 4 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	21000	5.6×10^{-2}	na[b]	1	0.06	na[b]	0.06
Antimony	6	1.6×10^{-5}	na[b]	0.0004	0.04	na[b]	0.04
Arsenic	11	2.9×10^{-5}	1.7×10^{-6}	0.0003	0.1	0.006	0.1
Barium	81	2.2×10^{-4}	na[b]	0.2	0.001	na[b]	0.001
Cadmium[c]	0.94	2.5×10^{-6}	5.0×10^{-9}	0.001	0.002	0.0002	0.002
Chromium	82	2.2×10^{-4}	na[b]	0.003	0.07	na[b]	0.07
Copper	74	2.0×10^{-4}	na[b]	0.04	0.005	na[b]	0.005
Iron	32000	8.5×10^{-2}	na[b]	0.3	0.3	na[b]	0.3
Manganese	280	7.4×10^{-4}	na[b]	0.14	0.005	na[b]	0.005
Mercury	1.7	4.5×10^{-6}	na[b]	0.0001	0.05	na[b]	0.05
Molybdenum	3.2	8.5×10^{-6}	na[b]	0.005	0.002	na[b]	0.002
Total PCBs	11	2.9×10^{-5}	8.1×10^{-6}	0.00002	1	0.4	1
Silver	0.79	2.1×10^{-6}	na[b]	0.005	0.0004	na[b]	0.0004
Thallium	0.092	2.4×10^{-7}	na[b]	0.00007	0.003	na[b]	0.003
Vanadium	57	1.5×10^{-4}	na[b]	0.001	0.2	na[b]	0.2
Zinc	240	6.4×10^{-4}	na[b]	0.3	0.002	na[b]	0.002
						Subtotal	**2**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.40	1.1×10^{-6}	6.3×10^{-8}	0.0005	0.002	0.0001	0.002
Dieldrin	0.017	4.5×10^{-8}	9.0×10^{-9}	0.00005	0.0009	0.0002	0.001
						Subtotal	**0.003**

Hazard indices by effect:	
Hazard Index for Cardiovascular Endpoint[d]	**0.3**
Hazard Index for Developmental Endpoint[e]	**1**
Hazard Index for Hematologic Endpoint[f]	**0.05**
Hazard Index for Immunological Endpoint[g]	**1**
Hazard index for Kidney Endpoint[h]	**0.01**
Hazard index for Liver Endpoint[i]	**0.008**
Hazard index for Neurological Endpoint[j]	**1**
Hazard index for Dermal Endpoint[k]	**0.1**
Total hazard index across both exposure routes[l]	**2**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] Cardiovascular endpoint is for arsenic and vanadium.

[e] Developmental endpoint is for total PCBs and mercury.

[f] Hematologic endpoint is for antimony, thallium, and zinc.

[g] Immunological endpoint is for total PCBs.

[h] Kidney endpoint is for barium, cadmium, copper, and molybdenum.

[i] Liver endpoint is for copper, total DDTs, and dieldrin.

[j] Neurological endpoint is for manganese, mercury, molybdenum, and total PCBs.

[k] Dermal endpoint is for arsenic and silver.

[l] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

CDI – chronic daily intake

dw – dry weight

EPC – exposure point concentration

na – not available

PCB – polychlorinated biphenyl

RfD – reference dose

RME – reasonable maximum exposure

Table B.5-39. Non-cancer hazard estimates for the beach play RME Area 5 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	15000	4.0×10^{-2}	na[b]	1	0.04	na[b]	0.04
Antimony	5	1.3×10^{-5}	na[b]	0.0004	0.03	na[b]	0.03
Arsenic	8.9	2.4×10^{-5}	1.4×10^{-6}	0.0003	0.08	0.005	0.09
Barium	42	1.1×10^{-4}	na[b]	0.2	0.0006	na[b]	0.0006
Cadmium[c]	0.23	6.1×10^{-7}	1.2×10^{-9}	0.001	0.0006	0.00005	0.0007
Chromium	28	7.4×10^{-5}	na[b]	0.003	0.02	na[b]	0.02
Copper	74	2.0×10^{-4}	na[b]	0.04	0.005	na[b]	0.005
Iron	26,000	6.9×10^{-2}	na[b]	0.3	0.2	na[b]	0.2
Manganese	280	7.4×10^{-4}	na[b]	0.14	0.005	na[b]	0.005
Mercury	0.11	2.9×10^{-7}	na[b]	0.0001	0.003	na[b]	0.003
Molybdenum	2.0	5.3×10^{-6}	na[b]	0.005	0.001	na[b]	0.001
Total PCBs	0.19	5.0×10^{-7}	1.4×10^{-7}	0.00002	0.03	0.007	0.04
Silver	0.15	4.0×10^{-7}	na[b]	0.005	0.00008	na[b]	0.00008
Thallium	0.062	1.6×10^{-7}	na[b]	0.00007	0.002	na[b]	0.002
Vanadium	55	1.5×10^{-4}	na[b]	0.001	0.1	na[b]	0.1
Zinc	110	2.9×10^{-4}	na[b]	0.3	0.001	na[b]	0.001
						Subtotal	**0.5**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.035	9.3×10^{-8}	5.6×10^{-9}	0.0005	0.0002	0.00001	0.0002
Dieldrin	0.0026	6.9×10^{-9}	1.4×10^{-9}	0.00005	0.0001	0.00003	0.0001
						Subtotal	**0.0003**
Total hazard index across both exposure routes[d]							**0.5**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake
dw – dry weight
EPC – exposure point concentration
na – not available
PCB – polychlorinated biphenyl
RfD – reference dose
RME – reasonable maximum exposure

Table B.5-40. Non-cancer hazard estimates for the beach play RME Area 6 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum[b]	-	-	-	1	-	-	-
Antimony	0.15	4.0×10^{-7}	na[c]	0.0004	0.001	na[c]	0.001
Arsenic	9.8	2.6×10^{-5}	1.6×10^{-6}	0.0003	0.09	0.005	0.1
Barium[b]	-	-	-	0.2	-	-	-
Cadmium[d]	0.15	4.0×10^{-7}	7.9×10^{-10}	0.001	0.0004	0.00003	0.0004
Chromium	23	6.1×10^{-5}	na[c]	0.003	0.02	na[c]	0.02
Copper	35	9.3×10^{-5}	na[c]	0.04	0.002	na[c]	0.002
Iron[b]	-	-	-	0.3	-	-	-
Manganese[b]	-	-	-	0.14	-	-	-
Mercury	0.09	2.4×10^{-7}	na[c]	0.0001	0.002	na[c]	0.002
Molybdenum	1.5	4.0×10^{-6}	na[c]	0.005	0.0008	na[c]	0.0008
Total PCBs	0.97	2.6×10^{-6}	7.2×10^{-7}	0.00002	0.1	0.04	0.1
Silver	0.2	5.3×10^{-7}	na[c]	0.005	0.0001	na[c]	0.0001
Thallium	0.15	4.0×10^{-7}	na[c]	0.00007	0.006	na[c]	0.006
Vanadium	49	1.3×10^{-4}	na[c]	0.001	0.1	na[c]	0.1
Zinc	92	2.4×10^{-4}	na[c]	0.3	0.0008	na[c]	0.0008
						Subtotal	**0.3**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0005	1.3×10^{-9}	7.9×10^{-11}	0.0005	0.000003	0.0000002	0.000003
Dieldrin	0.0010	2.7×10^{-9}	5.3×10^{-10}	0.00005	0.00005	0.00001	0.00006
						Subtotal	**0.00006**
Total hazard index across both exposure routes[e]							**0.3**

a All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

b No data exist for this chemical in this area.

c No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

d Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[e] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake
dw – dry weight
EPC – exposure point concentration
na – not available
PCB – polychlorinated biphenyl
RfD – reference dose
RME – reasonable maximum exposure

Table B.5-41. Non-cancer hazard estimates for the beach play RME Area 7 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	23,000	6.1×10^{-2}	na[b]	1	0.06	na[b]	0.06
Antimony	5	1.3×10^{-5}	na[b]	0.0004	0.03	na[b]	0.03
Arsenic	11	2.9×10^{-5}	1.7×10^{-6}	0.0003	0.1	0.006	0.1
Barium	72	1.9×10^{-4}	na[b]	0.2	0.001	na[b]	0.001
Cadmium[c]	0.8	2.1×10^{-6}	4.2×10^{-9}	0.001	0.002	0.0002	0.002
Chromium	25	6.6×10^{-5}	na[b]	0.003	0.02	na[b]	0.02
Copper	38	1.0×10^{-4}	na[b]	0.04	0.003	na[b]	0.003
Iron	31,000	8.2×10^{-2}	na[b]	0.3	0.3	na[b]	0.3
Manganese	430	1.1×10^{-3}	na[b]	0.14	0.008	na[b]	0.008
Mercury	0.11	2.9×10^{-7}	na[b]	0.0001	0.003	na[b]	0.003
Molybdenum	1.3	3.5×10^{-6}	na[b]	0.005	0.0007	na[b]	0.0007
Total PCBs	0.23	6.1×10^{-7}	1.7×10^{-7}	0.00002	0.03	0.009	0.04
Silver	0.35	9.3×10^{-7}	na[b]	0.005	0.0002	na[b]	0.0002
Thallium	0.25	6.6×10^{-7}	na[b]	0.00007	0.009	na[b]	0.009
Vanadium	64	1.7×10^{-4}	na[b]	0.001	0.2	na[b]	0.2
Zinc	84	2.2×10^{-4}	na[b]	0.3	0.0007	na[b]	0.0007
						Subtotal	**0.8**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0027	7.2×10^{-9}	4.3×10^{-10}	0.0005	0.00001	0.0000009	0.00002
Dieldrin	0.001	2.7×10^{-9}	5.3×10^{-10}	0.00005	0.00005	0.00001	0.00006
						Subtotal	**0.00008**
Total hazard index across both exposure routes[d]							**0.8**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake
dw – dry weight
EPC – exposure point concentration
na – not available
PCB – polychlorinated biphenyl
RfD – reference dose
RME – reasonable maximum exposure

Table B.5-42. Non-cancer hazard estimates for the beach play RME Area 8 scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents
Receptor age: Child

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	17,000	4.5×10^{-2}	na[b]	1	0.05	na[b]	0.05
Antimony	7	1.9×10^{-5}	na[b]	0.0004	0.05	na[b]	0.05
Arsenic	10	2.7×10^{-5}	1.6×10^{-6}	0.0003	0.09	0.005	0.1
Barium	58	1.5×10^{-4}	na[b]	0.2	0.0008	na[b]	0.0008
Cadmium[c]	0.2	5.3×10^{-7}	1.1×10^{-9}	0.001	0.0005	0.00004	0.0005
Chromium	23	6.1×10^{-5}	na[b]	0.003	0.02	na[b]	0.02
Copper	34	9.0×10^{-5}	na[b]	0.04	0.002	na[b]	0.002
Iron	26,000	6.9×10^{-2}	na[b]	0.3	0.2	na[b]	0.2
Manganese	780	2.1×10^{-3}	na[b]	0.14	0.01	na[b]	0.01
Mercury	0.12	3.2×10^{-7}	na[b]	0.0001	0.003	na[b]	0.003
Molybdenum	1.9	5.0×10^{-6}	na[b]	0.005	0.001	na[b]	0.001
Total PCBs	0.23	6.1×10^{-7}	1.7×10^{-7}	0.00002	0.03	0.009	0.04
Silver	0.12	3.2×10^{-7}	na[b]	0.005	0.00006	na[b]	0.00006
Thallium	0.06	1.6×10^{-7}	na[b]	0.00007	0.002	na[b]	0.002
Vanadium	57	1.5×10^{-4}	na[b]	0.001	0.2	na[b]	0.2
Zinc	110	2.9×10^{-4}	na[b]	0.3	0.001	na[b]	0.001
						Subtotal	**0.7**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.0082	2.2×10^{-8}	1.3×10^{-9}	0.0005	0.00004	0.000003	0.00005
Dieldrin	0.0023	6.1×10^{-9}	1.2×10^{-9}	0.00005	0.0001	0.00002	0.0001
						Subtotal	**0.0002**
Total hazard index across both exposure routes[d]							**0.7**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, His were not calculated for individual endpoints.

CDI – chronic daily intake
dw – dry weight
EPC – exposure point concentration
na – not available
PCB – polychlorinated biphenyl
RfD – reference dose
RME – reasonable maximum exposure

B.5.3.4 Clamming

Risks were estimated for three clamming scenarios, including a 7-day-per-year clamming scenario, a tribal clamming RME scenario (120 days per year), and a tribal clamming 183-day-per-year scenario, as described in Section B.3.2.3. The clamming scenarios presented in this section consist only of exposures to sediment via incidental ingestion and dermal contact. Consumption of clams is evaluated in the seafood consumption scenarios. The effect of summing risks from clamming with clam consumption is evaluated in Section B.5.3.5.

The total excess cancer risk estimates for the combined exposure routes (dermal absorption and incidental sediment ingestion) for the 7-day-per-year clamming scenario were 1×10^{-6}, regardless of the summation approach (i.e., the exclusion of PCB TEQ or total PCBs, respectively) (Table B.5-43).

Table B.5-43. Excess cancer risk estimates for the 7-day-per-year clamming scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents/occasional clam collectors
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	9.5	1.1×10^{-7}	3.9×10^{-8}	1.5	2×10^{-7}	6×10^{-8}	3×10^{-7}
cPAHs[b]	0.48	5.5×10^{-9}	8.6×10^{-9}	7.3	4×10^{-8}	6×10^{-8}	1×10^{-7}
Dioxin/furan TEQ	3.65×10^{-4}	4.2×10^{-12}	1.5×10^{-12}	150,000	6×10^{-7}	2×10^{-7}	8×10^{-7}
PCB TEQ	4.19×10^{-5}	4.8×10^{-13}	8.1×10^{-13}	150,000	7×10^{-8}	1×10^{-7}	2×10^{-7}
Total PCBs	1.5	1.7×10^{-8}	2.9×10^{-8}	2	3×10^{-8}	6×10^{-8}	9×10^{-8}
Subtotal excluding PCB TEQ							**1×10^{-6}**
Subtotal excluding Total PCBs							**1×10^{-6}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.022	2.5×10^{-10}	9.1×10^{-11}	0.34	9×10^{-11}	3×10^{-11}	1×10^{-10}
Dieldrin	0.017	1.9×10^{-10}	2.4×10^{-10}	16	3×10^{-9}	4×10^{-9}	7×10^{-9}
Toxaphene	0.85	9.7×10^{-9}	1.2×10^{-8}	1.1	1×10^{-8}	1×10^{-8}	2×10^{-8}
Subtotal							**3×10^{-8}**
Total risk (excluding PCB TEQ) across both exposure routes							**1×10^{-6}**
Total risk (excluding total PCBs) across both exposure routes							**1×10^{-6}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] cPAH concentrations are presented as benzo(a)pyrene equivalents.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
na – not available
PCB – polychlorinated biphenyl
TEQ – toxic equivalent

For the tribal clamming RME scenario, the total excess cancer risk for both incidental sediment ingestion and dermal absorption exposure routes was 1×10^{-4} or 2×10^{-4}, depending on the summation approach (i.e., the exclusion of PCB TEQ or total PCBs, respectively) (Table B.5-44). Dioxin/furan TEQ was the greatest contributor to the total excess cancer risk for the tribal clamming RME scenario. Some of the risk attributed to dioxin/furan TEQ is likely related to sources outside the LDW. Similarly, some of the arsenic risk may also be from background sources. Potential contributions to LDW risks from background concentrations of dioxins/furans are discussed in Section B.5.5.

Table B.5-44. Excess cancer risk estimates for the tribal clamming RME scenario (120 days per year) based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Tribal/subsistence clam collectors
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	27	9.9×10^{-6}	3.6×10^{-6}	1.5	1×10^{-5}	5×10^{-6}	2×10^{-5}
cPAHs[b]	0.77	2.8×10^{-7}	4.4×10^{-7}	7.3	2×10^{-6}	3×10^{-6}	5×10^{-6}
Dioxin/furan TEQ	1.42×10^{-3}	5.2×10^{-10}	1.9×10^{-10}	150,000	8×10^{-5}	3×10^{-5}	1×10^{-4}
PCB TEQ	1.84×10^{-4}	6.8×10^{-11}	1.1×10^{-10}	150,000	1×10^{-5}	2×10^{-5}	3×10^{-5}
Total PCBs	4.0[c]	1.5×10^{-6}	$2.5 \times 10\text{-}^{6}$	2	3×10^{-6}	5×10^{-6}	8×10^{-6}
						Subtotal excluding PCB TEQ	**1×10^{-4}**
						Subtotal excluding Total PCBs	**2×10^{-4}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.35	1.3×10^{-7}	4.7×10^{-8}	0.34	4×10^{-8}	2×10^{-8}	6×10^{-8}
Dieldrin	0.013	4.8×10^{-9}	5.8×10^{-9}	16	8×10^{-8}	9×10^{-8}	2×10^{-7}
Toxaphene	6.3	2.3×10^{-6}	2.8×10^{-6}	1.1	3×10^{-6}	3×10^{-6}	6×10^{-6}
						Subtotal	**6×10^{-6}**
Total risk (excluding PCB TEQ) across both exposure routes							**1×10^{-4}**
Total risk (excluding total PCBs) across both exposure routes							**2×10^{-4}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[c] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
SWAC – spatially weighted average concentration
TEQ – toxic equivalent

For the tribal clamming 183-day-per-year scenario, the total excess cancer risk for incidental ingestion and dermal absorption pathways was 2×10^{-4} or 3×10^{-4}, depending on the summation approach used (the exclusion of PCB TEQ or total PCBs, respectively) (Table B.5-45). The individual chemical with highest contribution to the excess cancer risk was dioxin/furan TEQ, with an excess cancer risk of 2×10^{-4}. As noted in Tables B.5-44 and B-5-45, PCB TEQ risks were appreciably greater than total PCB risks. None of the JN-qualified chemicals were significant risk contributors.

Table B.5-45. Excess cancer risk estimates for the tribal clamming 183-day-per-year scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Tribal/subsistence clam collectors
Receptor age: Adult

Chemical	EPC[a] (mg/kg dw)	Cancer CDI (mg/kg-day)		Cancer Slope Factor (mg/kg-day)$^{-1}$	Excess Cancer Risk		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Arsenic	27	1.7×10^{-5}	6.0×10^{-6}	1.5	2×10^{-5}	9×10^{-6}	3×10^{-5}
cPAHs[b]	0.77	4.7×10^{-7}	7.4×10^{-7}	7.3	3×10^{-6}	5×10^{-6}	8×10^{-6}
Dioxin/Furan TEQ	1.42×10^{-3}	8.7×10^{-10}	3.2×10^{-10}	150,000	1×10^{-4}	5×10^{-5}	2×10^{-4}
PCB TEQ	1.84×10^{-4}	1.1×10^{-10}	1.9×10^{-10}	150,000	2×10^{-5}	3×10^{-5}	5×10^{-5}
Total PCBs	4.0[c]	2.5×10^{-6}	4.1×10^{-6}	2	5×10^{-6}	8×10^{-6}	1×10^{-5}
Subtotal excluding PCB TEQ							**2×10^{-4}**
Subtotal excluding total PCBs							**3×10^{-4}**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.35	2.1×10^{-7}	7.8×10^{-8}	0.34	7×10^{-8}	3×10^{-8}	1×10^{-7}
Dieldrin	0.013	8.0×10^{-9}	9.6×10^{-9}	16	1×10^{-7}	2×10^{-7}	3×10^{-7}
Toxaphene	6.3	3.9×10^{-6}	4.7×10^{-6}	1.1	4×10^{-6}	5×10^{-6}	9×10^{-6}
Subtotal							**9×10^{-6}**
Total risk across all exposure routes / pathways excluding PCB TEQ							**2×10^{-4}**
Total risk across all exposure routes / pathways excluding total PCBs							**3×10^{-4}**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] cPAH concentrations are presented as benzo(a)pyrene equivalents.

[c] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
dw – dry weight
EPC – exposure point concentration
PCB – polychlorinated biphenyl
SWAC – spatially weighted average concentration
TEQ – toxic equivalent

For non-cancer hazard estimates for the clamming scenarios, HQs for all chemicals for all three clamming scenarios were less than 1 (Tables B.5-46, B.5-47, and B.5-48). Effect-specific His were not calculated because the total HI across all effects did not exceed 1.

Table B.5-46. Non-cancer hazard estimates for the 7-day-per-year clamming scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Residents/occasional clam collectors
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	15,000	4.0×10^{-4}	na[b]	1	0.0004	na[b]	0.0004
Antimony	1.3	3.5×10^{-8}	na[b]	0.0004	0.00009	na[b]	0.00009
Arsenic	9.5	2.5×10^{-7}	9.2×10^{-8}	0.0003	0.0008	0.0003	0.001
Barium	52	1.4×10^{-6}	na[b]	0.2	0.000007	na[b]	0.000007
Cadmium[c]	0.42	1.1×10^{-8}	1.4×10^{-10}	0.001	0.00001	0.000005	0.00002
Chromium	28	7.5×10^{-7}	na[b]	0.003	0.0002	na[b]	0.0002
Copper	49	1.3×10^{-6}	na[b]	0.04	0.00003	na[b]	0.00003
Iron	24,000	6.4×10^{-4}	na[b]	0.3	0.002	na[b]	0.002
Manganese	310	8.3×10^{-6}	na[b]	0.14	0.00006	na[b]	0.00006
Mercury	0.20	5.3×10^{-9}	na[b]	0.0001	0.00005	na[b]	0.00005
Molybdenum	2.1	5.6×10^{-8}	na[b]	0.005	0.00001	na[b]	0.00001
Total PCBs	1.5	4.0×10^{-8}	6.8×10^{-8}	0.00002	0.002	0.003	0.005
Silver	0.57	1.5×10^{-8}	na[b]	0.005	0.000003	na[b]	0.000003
Thallium	0.071	1.9×10^{-9}	na[b]	0.00007	0.00003	na[b]	0.00003
Vanadium	53	1.4×10^{-6}	na[b]	0.001	0.001	na[b]	0.001
Zinc	140	3.7×10^{-6}	na[b]	0.3	0.00001	na[b]	0.00001
						Subtotal	**0.01**
Tentatively identified chemicals (JN-qualified)							
Total DDT	0.022	5.9×10^{-10}	2.1×10^{-10}	0.0005	0.000001	0.0000004	0.000001
Dieldrin	0.017	4.5×10^{-10}	5.5×10^{-10}	0.00005	0.000009	0.00001	0.00002
						Subtotal	**0.00002**
Total hazard index across both exposure routes[d]							**0.01**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake
dw – dry weight
EPC – exposure point concentration
na – not available
PCB – polychlorinated biphenyl
RfD – reference dose

Table B.5-47. Non-cancer hazard estimates for the tribal clamming RME scenario (120 days per year) based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Tribal/subsistence clam collectors
Receptor age: Adult

Chemical	EPC (mg/kg dw)[a]	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	20,000	8.0×10^{-3}	na[b]	1	0.008	na[b]	0.008
Antimony	8.2	3.3×10^{-6}	na[b]	0.0004	0.008	na[b]	0.008
Arsenic	27	1.1×10^{-5}	3.9×10^{-6}	0.0003	0.04	0.01	0.05
Barium	310	1.2×10^{-4}	na[b]	0.2	0.0006	na[b]	0.0006
Cadmium[c]	4.8	1.9×10^{-6}	2.3×10^{-8}	0.001	0.002	0.0009	0.003
Chromium	81	3.3×10^{-5}	na[b]	0.003	0.01	na[b]	0.01
Copper	450	1.8×10^{-4}	na[b]	0.04	0.005	na[b]	0.005
Iron	33,000	1.3×10^{-2}	na[b]	0.3	0.04	na[b]	0.04
Manganese	650	2.6×10^{-4}	na[b]	0.14	0.002	na[b]	0.002
Mercury	0.23	9.2×10^{-8}	na[b]	0.0001	0.0009	na[b]	0.0009
Molybdenum	3.8	1.5×10^{-6}	na[b]	0.005	0.0003	na[b]	0.0003
Total PCBs	4.0[d]	1.6×10^{-6}	2.7×10^{-6}	0.00002	0.08	0.1	0.2
Silver	6.7	2.7×10^{-6}	na[b]	0.005	0.0005	na[b]	0.0005
Thallium	3.6	1.4×10^{-6}	na[b]	0.00007	0.02	na[b]	0.02
Vanadium	56	2.3×10^{-5}	na[b]	0.001	0.02	na[b]	0.02
Zinc	480	1.9×10^{-4}	na[b]	0.3	0.0006	na[b]	0.0006
						Subtotal	**0.4**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.35	1.4×10^{-7}	5.1×10^{-8}	0.0005	0.0003	0.0001	0.0004
Dieldrin	0.013	5.2×10^{-9}	6.3×10^{-9}	0.00005	0.0001	0.0001	0.0002
						Subtotal	**0.0006**
Total hazard index across both exposure routes[e]							**0.4**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to determine the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

[e] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, His were not calculated for individual endpoints.

CDI – chronic daily intake

dw – dry weight

EPC – exposure point concentration

na – not available

PCB – polychlorinated biphenyl

RfD – reference dose

RME – reasonable maximum exposure

SWAC – spatially weighted average concentration

TEQ – toxic equivalent

Table B.5-48. Non-cancer hazard estimates for the tribal clamming 183-day-per-year scenario based on exposure by incidental sediment ingestion and dermal absorption

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Sediment
Receptor population: Tribal/subsistence clam collectors
Receptor age: Adult

Chemical	EPC[a] (mg/kg dw)	Non-Cancer CDI (mg/kg-day)		Reference Dose (mg/kg-day)	Hazard Quotient		
		Incidental Ingestion	Dermal Absorption		Incidental Ingestion	Dermal Absorption	Total
Aluminum	20,000	1.2×10^{-2}	na[b]	1	0.01	na[b]	0.01
Antimony	8.2	5.0×10^{-6}	na[b]	0.0004	0.01	na[b]	0.01
Arsenic	27	1.7×10^{-5}	6.0×10^{-6}	0.0003	0.06	0.02	0.08
Barium	310	1.9×10^{-4}	na[b]	0.2	0.001	na[b]	0.001
Cadmium[c]	4.8	2.9×10^{-6}	13.6×10^{-8}	0.001	0.003	0.001	0.004
Chromium	81	5.0×10^{-5}	na[b]	0.003	0.02	na[b]	0.02
Copper	450	2.8×10^{-4}	na[b]	0.04	0.007	na[b]	0.007
Iron	33,000	2.0×10^{-2}	na[b]	0.3	0.07	na[b]	0.07
Manganese	650	4.0×10^{-4}	na[b]	0.14	0.003	na[b]	0.003
Mercury	0.23	1.4×10^{-7}	na[b]	0.0001	0.001	na[b]	0.001
Molybdenum	3.8	2.3×10^{-6}	na[b]	0.005	0.0005	na[b]	0.0005
Total PCBs	4.0[d]	2.5×10^{-6}	4.1×10^{-6}	0.00002	0.1	0.2	0.3
Silver	6.7	4.1×10^{-6}	na[b]	0.005	0.0008	na[b]	0.0008
Thallium	3.6	2.2×10^{-6}	na[b]	0.00007	0.03	na[b]	0.03
Vanadium	56	3.4×10^{-5}	na[b]	0.001	0.03	na[b]	0.03
Zinc	480	2.9×10^{-4}	na[b]	0.3	0.001	na[b]	0.001
						Subtotal	**0.6**
Tentatively identified chemicals (JN-qualified)							
Total DDTs	0.35	2.1×10^{-7}	7.8×10^{-8}	0.0005	0.0004	0.0002	0.0006
Dieldrin	0.013	8.0×10^{-9}	9.6×10^{-9}	0.00005	0.0002	0.0002	0.0004
						Subtotal	**0.001**
Total hazard index across all exposure routes/pathways excluding total PCBs[e]							**0.6**

[a] All EPCs are medium-specific, rather than route-specific (i.e., the same EPC was used for the dermal absorption and incidental sediment ingestion exposure routes).

[b] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[c] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose while dermal exposure utilizes an

absorbed dose. The adjustment allows for expression of exposure and toxicity in terms of absorbed dose (EPA 2004d).

[d] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

[e] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs were not calculated for individual endpoints.

CDI – chronic daily intake
dw – dry weight
EPC – exposure point concentration
na – not available
PCB – polychlorinated biphenyl
RfD – reference dose
SWAC – spatially weighted average concentration
TEQ – toxic equivalent

B.5.3.5 Total risks for clamming scenarios (clam collection and clam consumption)

Risks associated with clams were evaluated both in the seafood consumption scenarios (Section B.5.3.1) and in the direct sediment exposure scenarios (Section B.5.3.4). In this section, total risks are calculated as the sum of clam consumption (seafood exposure) and clam collection (sediment exposure). Risk totals were calculated for scenarios with less frequent (Table B.5-49) and more frequent exposures (Table B.5-50 and B.5-51). Almost all (i.e., over 95%) of the total risk for each chemical is attributed to the consumption of clams. Tables B.5-49, B.5-50, and B.5-51 include the chemicals that together represent the majority of the cancer risk for consumption and/or collection (see Section B.7). For risk estimates assuming more frequent clamming (Table B.5-50 and B.5-51), the adult tribal RME clam consumption rate based on Tulalip data was assumed. If the adult tribal clam consumption rate based on Suquamish data had been assumed instead, the relative contribution from consumption (compared to collection) would have been even larger. Total cancer risks and hazards across chemicals are included, but it should be noted that some chemicals were not analyzed in both sediment and tissue. No dioxin/furan data exist for seafood tissue; therefore, any risks resulting from ingesting seafood tissue that contains dioxin/furan residues were not quantified in this risk assessment. However, if dioxin/furan TEQ values were known for seafood, they would likely account for a significant portion of the risk from consumption of resident LDW seafood. The risk associated with dermal contact with sediment containing dioxin/furans is shown here only for comparison.

Table B.5-49. Risk estimates for example chemicals for the 7-day-per-year clamming scenario and the adult one-meal-per-month clam consumption scenario

Chemical	Excess Cancer Risk			Non-Cancer Hazard Quotient		
	Consumption[a]	Collection[b]	Total	Consumption[a]	Collection[b]	Total
Arsenic	1×10^{-4}	3×10^{-7}	1×10^{-4}	0.7	0.001	0.7
cPAHs[c]	7×10^{-6}	1×10^{-7}	7×10^{-6}	na	na	na
Dioxin/Furan TEQ [d]	na	8×10^{-7}	na	na	na	na
PCB TEQ	2×10^{-5}	2×10^{-7}	2×10^{-5}	na	na	na
Total PCBs	5×10^{-5}	9×10^{-8}	5×10^{-5}	3	0.005	3
Total excess cancer risk across all exposure routes / pathways[e]						
excluding PCB TEQ	**2×10^{-4}**	**1×10^{-6}**	**2×10^{-4}**	**na**		
excluding total PCBs	**1×10^{-4}**	**1×10^{-6}**	**1×10^{-4}**			
Non-cancer hazard indices by effect[e]						
HI for cardiovascular endpoint[f]				**0.9**	**0.002**	**0.9**
HI for developmental endpoint[g]				**3**	**0.005**	**3**
HI for hematologic endpoint[h]				**0.03**	**0.0001**	**0.03**
HI for immunological endpoint[i]				**3**	**0.005**	**3**
HI for kidney endpoint[j]				**0.03**	**0.00007**	**0.03**
HI for liver endpoint[k]				**0.1**	**0.00005**	**0.1**
HI for neurological endpoint[l]				**3**	**0.005**	**3**
HI for dermal endpoint[m]				**0.7**	**0.001**	**0.7**
Total HI across all exposure routes/ pathways[n]				**4**	**0.01**	**4**

[a] Risk from the consumption of clams in the adult one-meal-per-month – clam seafood exposure scenario.

[b] Risk from dermal absorption and incidental sediment ingestion during the collection of clams in the 7-day-per-year clamming scenario.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data used to calculate consumption risks are from only 2004 because of high reporting limits in historical data.

[d] Dioxin/furan TEQ data were available only for direct sediment exposure. However, dioxins/furans could represent a significant portion of the true excess cancer risk from seafood consumption, and thus excess cancer risks from clam consumption would be higher if dioxins/furans were included, although it is not known if the increase would change risk estimates that are given with only one significant figure.

[e] Total excess cancer risks and non-cancer HIs represent all COPCs for the collection or consumption scenario, not just example chemicals shown in this table. Because the COPC list for these scenarios was different, the totals for these scenarios represent different lists of chemicals.

[f] Cardiovascular endpoint is for arsenic and vanadium.

[g] Developmental endpoint is for PCBs and mercury.

[h] Hematologic endpoint is for antimony and zinc for the consumption scenario and for antimony, thallium, and zinc for the collection scenario.

[i] Immunological endpoint is for PCBs and TBT for the consumption scenario and for PCBs for the collection scenario.

[j] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol for the consumption scenario, and for barium, cadmium, copper, and molybdenum for the collection scenario.

[k] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol for the consumption scenario. For the collection scenario, the chemicals include copper, total DDTs, and dieldrin.

[l] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs for the consumption scenario and is manganese, mercury, molybdenum and total PCBs for the collection scenario.

[m] Dermal endpoint is for 4-methylphenol and arsenic for the consumption scenario, and is for arsenic and silver for the collection scenario.

[n] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

cPAH – carcinogenic polycyclic aromatic hydrocarbon

dw – dry weight

EPC – exposure point concentration

HI – hazard index

na – not available

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

Table B.5-50. Risk estimates for example chemicals for the tribal clam collection RME scenario and the consumption of clams in the adult tribal RME seafood consumption scenario based on Tulalip data

Chemical	Excess Cancer Risk			Non-Cancer Hazard Quotient		
	Consumption[a]	Collection[b]	Total	Consumption[a]	Collection[b]	Total
Arsenic	1×10^{-3}	2×10^{-5}	1×10^{-3}	3	0.05	3
cPAHs[c]	7×10^{-5}	5×10^{-6}	8×10^{-5}	na	na	na
Dioxin/furan TEQ[d]	na	1×10^{-4}	na	na	na	na
PCB TEQ	2×10^{-4}	3×10^{-5}	2×10^{-4}	na	na	na
Total PCBs[e]	6×10^{-4}	8×10^{-6}	6×10^{-4}	14	0.2	14
Total excess cancer risk across all exposure routes / pathways[f]						
excluding PCB TEQ	**2×10^{-3}**	**1×10^{-4}**	**2×10^{-3}**	**na**		
excluding total PCBs	**1×10^{-3}**	**2×10^{-4}**	**1×10^{-3}**			
Non-cancer hazard indices by effect[f]						
HI for cardiovascular endpoint[g]				**4**	**0.07**	**4**
HI for developmental endpoint[h]				**10**	**0.2**	**10**
HI for hematologic endpoint[i]				**0.1**	**0.03**	**0.1**
HI for immunological endpoint[j]				**11**	**0.2**	**11**
HI for kidney endpoint[k]				**0.1**	**0.009**	**0.1**
HI for liver endpoint[l]				**0.3**	**0.006**	**0.3**
HI for neurological endpoint[m]				**10**	**0.2**	**10**
HI for dermal endpoint[n]				**3**	**0.05**	**3**
Total HI across all exposure routes/ pathways[n]				**15**	**0.4**	**15**

[a] Risk from the consumption of clams in the adult tribal RME seafood exposure scenario based on Tulalip data.

[b] Risk from dermal absorption and incidental sediment ingestion during the collection of clams in the tribal clamming RME scenario (120 days per year).

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data used to calculate consumption risks are from only 2004 because of high reporting limits in historical data.

[d] Dioxin/furan TEQ data were available only for direct sediment exposure. However, dioxins/furans could represent a significant portion of the true excess cancer risk from seafood consumption, and thus excess cancer risks from clam consumption would be higher if dioxins/furans were included, although it is not known if the increase would change risk estimates that are given with only one significant figure.

[e] EPC value for the collection scenario was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

[f] Total excess cancer risks and non-cancer HIs represent all COPCs for the collection or consumption scenario, not just example chemicals shown in this table. Because the COPC list for these scenarios was different, the totals for these scenarios represent different lists of chemicals.

[g] Cardiovascular endpoint is for arsenic and vanadium.

[h] Developmental endpoint is for PCBs and mercury.

[i] Hematologic endpoint is for antimony and zinc for the consumption scenario and for antimony, thallium, and zinc for the collection scenario.

[j] Immunological endpoint is for PCBs and TBT for the consumption scenario and for PCBs for the collection scenario.

[k] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol for the consumption scenario, and for barium, cadmium, copper, and molybdenum for the collection scenario.

[l] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol for the consumption scenario. For the collection scenario, the chemicals include copper, total DDTs, and dieldrin.

[m] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs for the consumption scenario and is manganese, mercury, molybdenum and total PCBs for the collection scenario.

[n] Dermal endpoint is for 4-methylphenol and arsenic for the consumption scenario, and is for arsenic and silver for the collection scenario.

[o] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

cPAH – carcinogenic polycyclic aromatic hydrocarbon

dw – dry weight

EPC – exposure point concentration

HI – hazard index

na – not available

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

SWAC – spatially weighted average concentration

TEQ – toxic equivalent

Table B.5-51. Risk estimates for example chemicals for the tribal clamming 183-day-per-year scenario and the consumption of clams in the adult tribal RME seafood consumption scenario based on Tulalip data

Chemical	Excess Cancer Risk			Non-Cancer Hazard Quotient		
	Consumption[a]	Collection[b]	Total	Consumption[a]	Collection[b]	Total
Arsenic	1×10^{-3}	3×10^{-5}	1×10^{-3}	3	0.08	3
cPAHs[c]	7×10^{-5}	8×10^{-6}	8×10^{-5}	na	na	na
Dioxin/furan TEQ[d]	na	2×10^{-4}	na	na	na	na
PCB TEQ	2×10^{-4}	5×10^{-5}	3×10^{-4}	na	na	na
Total PCBs[e]	6×10^{-4}	1×10^{-5}	6×10^{-4}	14	0.3	14
Total excess cancer risk across all exposure routes / pathways[f]						
excluding PCB TEQ	**3×10^{-3}**	**2×10^{-4}**	**3×10^{-3}**	**na**		
excluding total PCBs	**2×10^{-3}**	**3×10^{-4}**	**2×10^{-3}**			
Non-cancer hazard indices by effect[f]						
HI for cardiovascular endpoint[g]				**4**	**0.1**	**4**
HI for developmental endpoint[h]				**10**	**0.3**	**10**
HI for hematologic endpoint[i]				**0.1**	**0.04**	**0.1**
HI for immunological endpoint[j]				**11**	**0.3**	**11**
HI for kidney endpoint[k]				**0.1**	**0.01**	**0.1**
HI for liver endpoint[l]				**0.3**	**0.008**	**0.3**
HI for neurological endpoint[m]				**10**	**0.3**	**10**
HI for dermal endpoint[n]				**3**	**0.08**	**3**
Total HI across all exposure routes/pathways[n]				**15**	**0.6**	**16**

[a] Risk from the consumption of clams in the adult tribal RME seafood exposure scenario based on Tulalip data.

[b] Risk from dermal absorption and incidental sediment ingestion during the collection of clams in the tribal clamming 183-day-per-year scenario.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data used to calculate consumption risks are from only 2004 because of high reporting limits in historical data.

[d] Dioxin/furan TEQ data were available only for direct sediment exposure. However, dioxins/furans could represent a significant portion of the true excess cancer risk from seafood consumption, and thus excess cancer risks from clam consumption would be higher if dioxins/furans were included, although it is not known if the increase would change risk estimates that are given with only one significant figure.

[e] EPC value for the collection scenario was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

[f] Total excess cancer risks and non-cancer HIs represent all COPCs for the collection or consumption scenario, not just example chemicals shown in this table. Because the COPC list for these scenarios was different, the totals for these scenarios represent different lists of chemicals.

[g] Cardiovascular endpoint is for arsenic and vanadium.

[h] Developmental endpoint is for PCBs and mercury.

[i] Hematologic endpoint is for antimony and zinc for the consumption scenario and for antimony, thallium, and zinc for the collection scenario.

[j] Immunological endpoint is for PCBs and TBT for the consumption scenario and for PCBs for the collection scenario.

[k] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol for the consumption scenario, and for barium, cadmium, copper, and molybdenum for the collection scenario.

[l] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol for the consumption scenario. For the collection scenario, the chemicals include copper, total DDTs, and dieldrin.

[m] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs for the consumption scenario and is manganese, mercury, molybdenum and total PCBs for the collection scenario.

[n] Dermal endpoint is for 4-methylphenol and arsenic for the consumption scenario, and is for arsenic and silver for the collection scenario.

[o] This total is not directly interpretable for risk assessment because it includes hazard quotients across multiple endpoints. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

cPAH – carcinogenic polycyclic aromatic hydrocarbon

dw – dry weight

EPC – exposure point concentration

HI – hazard index

na – not available

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

SWAC – spatially weighted average concentration

TEQ – toxic equivalent

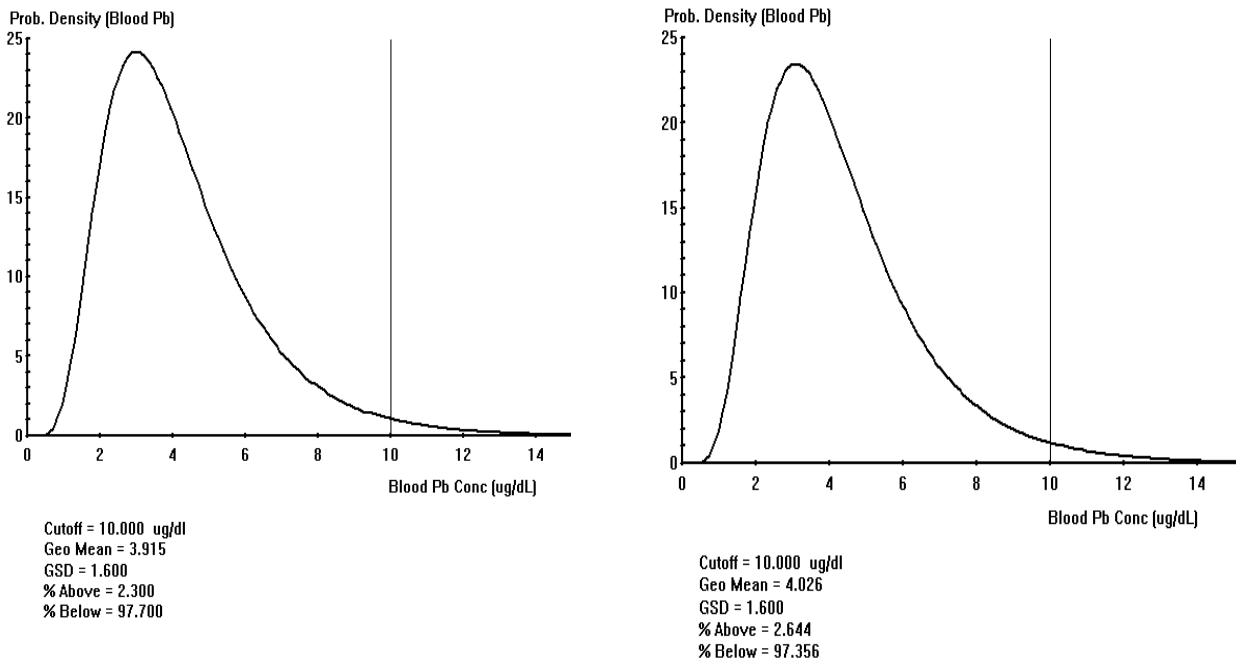
B.5.4 LEAD

As described in Section B.3.4.4, risks from exposure to lead are not quantified following the exposure model used for other COPCs. Because the toxicokinetics (absorption, distribution, metabolism, and excretion) of lead are well understood, health risks from lead exposure are evaluated based on blood lead concentration, which can be modeled. The results of blood lead modeling for children (IEUBK) and adults (ALM) are presented in the subsections below.

B.5.4.1 Children

The IEUBK lead model was run using default parameters, except for the inclusion of site-specific sediment and tissue concentration data, as described earlier in Tables B.3-43 through B.3-46. Model output is provided in the form of a probability density curve that describes the probability of blood lead concentrations occurring in a hypothetical population of children. The Centers for Disease Control and Prevention (CDC) has established 10 µg/dL as a level of concern threshold for children blood level above which appropriate medical follow-up is warranted. The probability density curves designate the percent of children that are predicted to have blood lead levels that may exceed the threshold.

Probability density curves were generated for the LDW using time-weighted average EPCs from the beach play RME scenarios (Figure B.5-1). See Section B.3.4.4.1 for further explanation of the time-weighted average approach to calculating EPC values. The IEUBK model was run using both the highest and lowest of the time-weighted EPCs from the beach play RME scenarios, along with a weighted EPC for seafood consumption (see Section B.3.4.4). The time-weighted average EPCs ranged from 167 mg/kg dw for the Area 7 scenario to 191 mg/kg dw for the Area 2 scenario. Both of these EPCs were evaluated in the model. Note that these concentrations are less than the pre-set soil concentration of 200 mg/kg dw in the model. Based on this range of exposures, fewer than 3% of the modeled child population would have blood lead levels that exceed the CDC level of concern (shown in both graphs in Figure B.5-1 as the area under the curve to the right of the vertical line, which represents 10 µg/dL). EPA's risk reduction goal for contaminated sites is that no more than 5% of the population of children exposed to lead will have blood lead levels greater than 10 µg/dl. Based on the results of the IEUBK model for the LDW, lead is not considered to be a COC in the LDW.



a) Area 7 **b) Area 2**

Figure B.5-1. Probability density curves for predicted blood lead concentrations using input data from children's beach play RME and seafood consumption

B.5.4.2 Adults

The ALM was run to estimate risks from lead exposure to the most sensitive population, which is a developing fetus. Lead risks were assessed by estimating the probability of exceeding the threshold blood lead level of 10 µg/dL in the fetus through evaluation of exposure of a pregnant mother. Results for the beach play RME scenario with the highest EPC (Area 2), as well as the tribal clamming RME and netfishing scenarios, are presented in Table B.5-52. As described in Section B.3.4.4.2, the model was run in two modes (with and without seafood consumption) so that the incremental effects of seafood consumption could be evaluated. The risks from lead exposure in the 7-day-per-year clamming scenario as well as the other beach play areas are not presented because the lead concentrations in sediment for these scenarios were lower than for the above scenarios used in the modeling.

Table B.5-52. Risk estimates for predicted fetal and adult lead levels using the adult lead model

Results	Units	Scenario		
		Beach Play RME Area 2	Tribal Clamming RME	Netfishing RME
Estimates for soil and sediment incidental ingestion only				
Predicted adult blood lead levels, CTE[a]	µg/dL	1.8	2.0	1.8
Predicted fetal blood lead levels, 95th percentile[b]	µg/dL	6.3	7.0	6.3
Probability of fetal blood lead level exceeding 10 µg/dL (lognormal)[c]	%	1.4	1.9	1.4
Estimates Including Adult Tulalip RME seafood consumption				
Predicted adult blood lead levels, CTE[d]	µg/dL	2.3	2.6	2.4
Predicted fetal blood lead levels, 95th percentile[b]	µg/dL	8.1	9.1	8.4
Probability of fetal blood lead level exceeding 10 µg/dL (lognormal)[c]	%	2.9	4.0	3.2

[a] Central tendency estimate of adult blood lead concentration for sediment intake only.

[b] Estimate of 95th percentile fetal blood lead concentration.

[c] Probability of exceeding EPA's threshold for fetal exposure, a blood lead level of 10 µg/dL (EPA 2003c).

[d] Central tendency estimate of adult blood lead concentration for sediment ingestion and seafood consumption.

CTE – central tendency estimate

RME – reasonable maximum exposure

The 95th percentiles of predicted blood lead concentrations for the developing fetus ranged from 6.3 to 7.0 µg/dL for the scenarios that assumed no seafood consumption from the LDW (Table B.5-52). The probability of exceeding the 10 µg/dL blood lead threshold was equal to or less than 2% for each of these soil and sediment exposure scenarios. The 95th percentiles of blood lead concentrations and probabilities of exceeding the 10 µg/dL threshold were slightly higher for the scenarios that included seafood consumption. However, the probability for exceeding the 10 µg/dL blood lead threshold remained less than or equal to 4%. These results are consistent with the results from the IEUBK model and indicate that lead is not considered to be a COC for human health in the LDW.

B.5.5 Background Concentrations For Determining Incremental Risk

Both CERCLA and MTCA include provisions for evaluating chemical concentrations in background areas. Both programs distinguish between natural background and anthropogenic background (called "area" background in MTCA). Natural background is defined in MTCA as "the concentration of hazardous substance consistently present in the environment that has not been influenced by localized human activities." Anthropogenic background is defined as the combination of both natural and anthropogenic substances present in the environment as a result of human activities not specifically related to the CERCLA or MTCA release in question. The chemicals for

which an evaluation of background is appropriate are determined on a site-specific basis based on the most significant contributors to the total risk estimates at the site and a preliminary evaluation of the magnitude of differences between site and background concentrations.

Although CERCLA and MTCA both allow for consideration of background concentrations, they use somewhat different approaches. Under the MTCA cleanup regulation (WAC 173-340), cleanup levels may be based on background concentrations in certain cases. This HHRA does not provide evaluations of background data, including the selection of appropriate datasets and their statistical analysis, for the purpose of selecting cleanup levels under CERCLA or MTCA. Where evaluations of background data are presented in this HHRA, they are intended only to provide additional information relevant to exposure and risk estimates. Further background data evaluations supporting CERCLA and MTCA determinations of cleanup levels will be provided in the RI and/or FS reports.

There is no provision in the MTCA rule to use background concentrations in an incremental risk evaluation. However, consistent with CERCLA guidance, this section provides an incremental risk evaluation for arsenic and dioxins/furans. According to EPA (2002d) guidance, the risks from chemicals with suspected background sources are characterized initially without consideration of those background sources, as has been done in Section B.5.3 in the risk characterization.[26] Next, a separate risk estimate may be made for assumed exposures to these chemicals from background sources. Finally, background risks are compared to site-related risks. The difference between these two estimates, if any, is called the incremental risk.

The three chemicals with the highest contribution to total excess cancer risk estimates for multiple exposure pathways and scenarios are PCBs, arsenic, and dioxins/furans. These chemicals are known to be present in sediments from background areas of Puget Sound (see Section 7 of the main document). Sediment PCB concentrations over broad areas of the LDW are well above concentrations found in background areas, so no incremental risk estimates are presented in this section for PCBs. The sediment concentrations of arsenic and dioxins/furans, on the other hand, are much closer to urban background over most of the LDW, with only a few areas exhibiting substantially higher concentrations. Consequently, an analysis was conducted of arsenic and dioxin/furan concentrations in background areas, so that the incremental risks associated with conditions within the LDW could be estimated.

Focused sampling was conducted in 2004 and 2005 for arsenic and dioxins/furans from background areas as part of the incremental risk approach described in this section and in the Phase 2 RI work plan (Windward 2004d). Tissue and sediment samples were analyzed for arsenic and sediment samples were collected from urban

[26] Risks from exposure to dioxins/furans in LDW seafood were not described in the risk characterization section because no dioxin/furan tissue data were collected.

background areas and analyzed for dioxins/furans. These studies were designed in consultation with EPA and Ecology (Windward 2004c, 2005g, h). The study designs for these background sampling events vary by chemical and medium. An explanation of the rationale for each study design is provided in Section B.5.5.1 for arsenic and in Section B.5.5.2 for dioxins/furans.

B.5.5.1 Arsenic

Excess cancer risks for arsenic exceeded 1×10^{-6} for most of the direct sediment exposure scenarios and exceeded 1×10^{-4} for most of the seafood consumption scenarios. These risk estimates do not consider the percent of the total arsenic risk in the LDW that may be attributable to background sources, such as arsenic that occurs naturally in the Puget Sound basin, or to arsenic from anthropogenic (i.e., man-made) sources outside the LDW. All discussions of background levels for arsenic presented here should be considered preliminary and may be re-evaluated in the RI and FS.

Arsenic occurs naturally in all sediments and soils worldwide and is found in sediments of central and northern Puget Sound as part of native rock and resulting soils and sediments, and as a result of natural geological features, such as volcanoes (NOAA and Ecology 1999, 2000, 2002). In addition, historical, anthropogenic sources within the region have contributed additional arsenic in some areas of the LDW. For example, a recently completed soil survey for arsenic and lead in south King County suggested that the former Asarco smelter located in Ruston, Washington, is likely one of the sources responsible for elevated arsenic and lead concentrations in soil throughout the LDW watershed (Pacific Groundwater Group and TeraStat 2005).

B.5.5.1.1 Sediment

The Duwamish River upstream of the LDW was selected for the collection of sediment samples for the incremental risk approach because it is affected by similar natural (i.e., soils of volcanic origin) and anthropogenic sources outside the LDW (i.e., the former Asarco smelter plume). Therefore, 12 additional sediment samples were collected in 2005 from the Duwamish River upstream of the LDW to supplement existing data from eight previously collected upstream sediment samples. Data from all stations are displayed in Table B.5-53, and station locations are shown on Map B.5-1 (Windward 2005h).

Table B.5-53. Duwamish River surface sediment arsenic data collected upstream of the LDW

River Mile	Location ID	Sample Date	Arsenic Concentration (mg/kg dw)
5.26	LDW-153	3/15/2005	6.3
5.28	DR297	9/16/1998	4.0
5.34	DR298	9/16/1998	5.1
5.38	DR301	9/16/1998	4.4
5.45	DR299	9/16/1998	4.2
5.48	DR300	9/16/1998	4.1
5.57	LDW-154	3/15/2005	7.3
5.65	LDW-155	3/15/2005	5.4
5.80	LDW-156	3/15/2005	3.2
6.12	REF-1	10/18/1997	4.8
6.13	REF-2	10/18/1997	7.2
6.13	REF-3	10/18/1997	4.5
6.18	DR-SS7	2/1/2005	4.6
6.25	DR-SS6	2/1/2005	8.2[a]
6.29	DR-SS5	2/1/2005	8.9
6.60	DR-SS10	2/9/2005	7.3
6.64	DR-SS11	2/9/2005	7.4
6.80	DR-SS13	2/9/2005	7.4
7.01	DR-SS14	2/9/2005	5.4
7.03	DR-SS15	2/9/2005	10.9
Summary statistics:			
Minimum			3.2
Maximum			10.9
Mean			6.0
UCL (calculated using ProUCL)			6.8

[a] Concentration is the average of two field duplicate samples collected at this location.

dw – dry weight

ID – identification

UCL – upper confidence limit

The data presented in Table B.5-53 are normally distributed, as determined by the ProUCL software. The resulting upstream UCL (6.8 mg/kg dw) is compared in Table B.5-54 with arsenic EPCs for the direct sediment exposure scenarios evaluated in this HHRA (taken from Table B.3-6). The LDW EPCs ranged from 8.9 to 27 mg/kg dw. Exposure scenarios analogous to those developed for the LDW have not been developed for upstream reaches, so the EPC for each LDW scenario was compared to the single upstream UCL in Table B.5-54. As noted previously, this background value

should be considered preliminary. Additional data will be evaluated in the RI and FS to determine the appropriate background concentration for the LDW.

Table B.5-54. Sediment exposure point concentrations for arsenic in upstream and LDW exposure scenarios

Exposure Scenario	Upstream Concentration (UCL) (mg/kg dw)	LDW EPC (mg/kg dw)	Difference Between the EPC and the Upstream Concentration (mg/kg dw)
Netfishing RME	6.8	21	14.2
Netfishing CT	6.8	21	14.2
Tribal clamming – 183 days per year	6.8	27	20.2
Tribal clamming RME(120 days per year)	6.8	27	20.2
Clamming – 7 days per year	6.8	9.5	2.7
Beach play RME, Area 1	6.8	15	8.2
Beach play RME, Area 2	6.8	21	14.2
Beach play RME, Area 3	6.8	13	6.2
Beach play RME, Area 4	6.8	11	4.2
Beach play RME, Area 5	6.8	8.9	2.1
Beach play RME, Area 6	6.8	9.8	3.0
Beach play RME, Area 7	6.8	11	4.2
Beach play RME, Area 8	6.8	10	3.2

dw – dry weight

EPC – exposure point concentration

LDW – Lower Duwamish Waterway

Excess cancer risks associated with upstream concentrations of arsenic were calculated for each LDW direct sediment exposure scenario by replacing each LDW EPC with the upstream UCL. Incremental cancer risks were then estimated for each scenario by subtracting the upstream-related risks from the scenario-related risks. The incremental risks for direct sediment exposure scenarios are presented in Table B.5-55. The upstream risks ranged from 2×10^{-7} for the 7-day-per-year clamming scenario to 8×10^{-6} for the tribal clamming 183-day-per-year scenario. Incremental risks for the clamming scenarios in the LDW ranged from 1×10^{-7} for the 7-day-per-year clamming scenario to 2×10^{-5} for the tribal clamming 183-day-per-year scenario. Based on the incremental risk estimates for the netfishing RME scenario, tribal clamming RME, and most beach play RME scenarios areas, arsenic is considered a COC in sediment.

Table B.5-55. Incremental risks from exposure to arsenic in sediment

Exposure Scenario	Upstream Excess Cancer Risk Estimate	LDW Excess Cancer Risk Estimate	Incremental Cancer Risk Estimate
Netfishing RME	2×10^{-6}	6×10^{-6}	4×10^{-6}
Netfishing CT	5×10^{-7}	1×10^{-6}	5×10^{-7}
Tribal clamming – 183 days per year	8×10^{-6}	3×10^{-5}	2×10^{-5}
Tribal clamming RME (120 days per year)	5×10^{-6}	2×10^{-5}	1×10^{-5}
Clamming – 7 days per year	2×10^{-7}	3×10^{-7}	1×10^{-7}
Beach play RME, Area 1	2×10^{-6}	5×10^{-6}	3×10^{-6}
Beach play RME, Area 2	2×10^{-6}	7×10^{-6}	5×10^{-6}
Beach play RME, Area3	2×10^{-6}	4×10^{-6}	2×10^{-6}
Beach play RME, Area 4	2×10^{-6}	4×10^{-6}	2×10^{-6}
Beach play RME, Area 5	2×10^{-6}	3×10^{-6}	1×10^{-6}
Beach play RME, Area 6	2×10^{-6}	3×10^{-6}	1×10^{-6}
Beach play RME, Area 7	2×10^{-6}	4×10^{-6}	2×10^{-6}
Beach play RME, Area 8	2×10^{-6}	3×10^{-6}	1×10^{-6}

CT – central tendency

LDW – Lower Duwamish Waterway

RME – reasonable maximum exposure

B.5.5.1.2 Tissue

The study design to determine background concentrations of inorganic arsenic in seafood tissue focused on Puget Sound areas outside the LDW. Species selected for background sampling consisted of marine fish and shellfish species that were also sampled in the LDW. The estuarine species found in the LDW are not found in the fresh water environment upstream of the LDW. Consequently, it was not possible to use upstream reaches of the LDW to obtain samples to characterize background levels of inorganic arsenic in tissue.

Two background location types were sampled for fish, crabs, and clams: one representing areas with naturally occurring arsenic, and another representing areas that were affected by the Asarco smelter emissions. Two location types were chosen for evaluation because arsenic concentrations may differ between the two environments, and EPA (2002e) policy acknowledges that both natural and anthropogenic sources may be relevant as background for risk characterization. The Asarco smelter is the presumed source of much of the anthropogenic background arsenic in the LDW basin. The areas of anthropogenic (i.e., Asarco smelter) influence and areas of naturally occurring arsenic are shown on Map B.5-2. The areas of influence are defined by soil samples collected by Ecology as part of a smelter plume study (Ecology 2001b). Sediment sampling in the area shown in Map B.5-2 is far more

limited and does not show clear patterns that would make it possible to draw a map of Asarco-influenced sediments.

Data on background arsenic in seafood comes from three field studies. The sampling areas for each of these studies are shown on Map B.5-2. In 2004, 12 composite clam tissue samples were collected from Seahurst Park (Asarco-influenced background) and Bainbridge Island (outside the Asarco plume) and analyzed for both total and inorganic arsenic (Windward 2005b). Because the clam species collected in 2004 from background locations were not the same as the clam species for which arsenic data were available from the LDW, a second background clam sampling event was conducted in 2005. In 2005, 12 composite tissue samples of soft-shell clams (*Mya arenaria,* the same species collected in the LDW) were collected from Vashon Island (Asarco-influenced background) and the Dungeness National Wildlife Refuge (NWR) (representative of an area with naturally occurring arsenic) and analyzed for both total and inorganic arsenic (Windward 2006a). As part of a separate Phase 2 investigation, 53 fish and crab composite tissue samples were collected in 2004 from the East Passage (Asarco-influenced background) and Blake Island (outside the Asarco plume but representative of Puget Sound Basin areas with naturally occurring arsenic) and analyzed for both total and inorganic arsenic (Windward 2005c). Arsenic concentrations for composited samples of different seafood species (i.e., fish, crabs, and clams) are shown in Table B.5-56. As with the LDW samples, only the inorganic arsenic tissue data were used in this HHRA because EPA developed a cancer SF to estimate carcinogenic risks only for inorganic arsenic. Although mussels collected from the LDW were included in the seafood consumption exposure scenario, no inorganic arsenic data were collected for mussels from background areas because of the small fraction of the seafood consumption diet represented by mussels and the relatively low total arsenic concentration in mussels.[27]

The inorganic arsenic EPCs for background and LDW tissue samples calculated using ProUCL are presented in Table B.5-56. EPCs were much higher for clams compared to fish and crabs for both LDW and background areas. Perch inorganic arsenic concentrations from the LDW were also slightly elevated relative to background.

[27] Mussels represent a relatively small fraction of the total seafood consumption rate used for the seafood consumption scenarios. The maximum total arsenic concentration in LDW mussels was 1.07 mg/kg ww, which is much lower than the arsenic concentrations in the other seafood species evaluated in this HHRA.

Table B.5-56. Inorganic arsenic EPCs for tissue samples collected from the LDW and background areas

Area	Tissue Type	Number of Samples	Inorganic Arsenic EPC (mg/kg ww)
Clams			
LDW	*Mya arenaria*	8	1.96
Vashon (Asarco-influenced background)	*Mya arenaria*	6	0.183
Seahurst Park (Asarco-influenced background)	other species[a]	6	0.800
Dungeness NWR (background)	*Mya arenaria*	6	0.087
Bainbridge Island (background)	other species[a]	6	0.444
Perch (shiner surfperch, pile perch, striped perch)			
LDW	whole body + fillet	10	0.086
East Passage (Asarco-influenced background)	whole body + fillet	3	0.01 (max)
Blake Island (background)	whole body + fillet	6	0.03
Crabs (Dungeness crab, slender crab)			
LDW	edible meat	6	0.042
	edible meat + hepatopancreas[b]	6	0.11
East Passage (Asarco-influenced background)	edible meat	6	0.03
	edible meat + hepatopancreas[b]	6	0.05
Blake Island (background)	edible meat	6	0.03
	edible meat + hepatopancreas[b]	6	0.1
Benthic fish (English sole and starry flounder)			
LDW	fillet	8	0.0062
	whole body	8	0.073
East Passage (Asarco-influenced background)	fillet	6	0.004
	whole body	6	0.01
Blake Island (background)	fillet	6	0.004
	whole body	6	0.02

[a] Composite samples were formed from multiple species, including *Saxidomus giganteus, Clinocardium nuttallii, Macoma nasuta, Macoma secta, Tresus capax*, and *Protothaca staminea*.

[b] Data from hepatopancreas composite samples were mathematically combined with data from composite samples of edible meat to form composite samples of edible meat plus hepatopancreas. Whole-body (i.e., edible meat plus hepatopancreas) crab concentrations were calculated assuming 69% (by weight) edible meat and 31% hepatopancreas, based on the relative weight of these tissues in a 16.6-cm Dungeness crab dissected by Windward in 2004 (unpublished data).

EPC – exposure point concentration

LDW – Lower Duwamish Waterway

ww – wet weight

Incremental cancer risk estimates associated with arsenic for the seafood consumption exposure scenarios were calculated by subtracting the background risk estimates from the LDW risk estimates (Table B.5-57). Incremental risks for most of the seafood consumption scenarios are almost identical to the LDW risk estimates, regardless of the type of background data used for the comparison (i.e., Asarco-influenced or non-

Asarco-influenced). This is because the inorganic arsenic concentrations in LDW clams, which are two orders of magnitude higher than the concentrations in fish and crabs (from all locations) are substantially higher than the concentrations in clams from background locations. Inorganic arsenic concentrations in fish and crabs were relatively low for both LDW and background locations, although perch from the LDW also exhibited slightly elevated inorganic arsenic concentrations relative to background samples. These results can be seen most clearly for the adult one-meal-per-month risk estimates shown in Table B.5-57, which are based on single species ingestion, rather than the combination of all seafood categories that was used for the other exposure scenarios. The incremental cancer risk estimates for adult one-meal-per-month consumption of clams and pelagic fish (only in Asarco-influenced background areas) are the most similar to the LDW cancer risk estimates, while the incremental estimates for the other species are consistently lower (i.e., at least two-fold lower for crabs and benthic fish). Table B.5-58 also highlights that clams are the dominant seafood category contributing to incremental arsenic risk for the adult tribal RME scenario based on Tulalip data. This table presents the percentages of total incremental risk from each seafood category for the adult tribal RME scenario based on Tulalip data (similar to Table B.5-17). The percentage of the incremental risk associated with clam consumption is 98 or 96%, depending on background source.

Table B.5-57. Incremental cancer risk estimates associated with inorganic arsenic for the seafood consumption exposure scenarios

Exposure Scenario	LDW Cancer Risk Estimate	Background Type	Background Cancer Risk Estimate[a]	Incremental Cancer Risk Estimate[a]
Adult tribal RME (Tulalip data)	1×10^{-3}	Asarco-influenced	2×10^{-4}	8×10^{-4}
		non-Asarco	1×10^{-4}	9×10^{-4}
Adult tribal CT (Tulalip data)	6×10^{-5}	Asarco-influenced	8×10^{-6}	5×10^{-5}
		non-Asarco	6×10^{-6}	5×10^{-5}
Child tribal RME (Tulalip data)	3×10^{-4}	Asarco-influenced	4×10^{-5}	3×10^{-4}
		non-Asarco	3×10^{-5}	3×10^{-4}
Child tribal CT (Tulalip data)	3×10^{-5}	Asarco-influenced	4×10^{-6}	3×10^{-5}
		non-Asarco	2×10^{-6}	3×10^{-5}
Adult tribal (Suquamish data)	2×10^{-2}	Asarco-influenced	2×10^{-3}	2×10^{-2}
		non-Asarco	8×10^{-4}	2×10^{-2}
Adult API RME	7×10^{-4}	Asarco-influenced	7×10^{-5}	6×10^{-4}
		non-Asarco	4×10^{-5}	7×10^{-4}
Adult API CT	1×10^{-5}	Asarco-influenced	2×10^{-6}	8×10^{-6}
		non-Asarco	1×10^{-6}	9×10^{-6}

Exposure Scenario	LDW Cancer Risk Estimate	Background Type	Background Cancer Risk Estimate[a]	Incremental Cancer Risk Estimate[a]
Adult one meal per month[b]	1×10^{-4} (clam) 6×10^{-6} (pelagic) 3×10^{-6} (crab) 4×10^{-7} (benthic)	Asarco-influenced	1×10^{-5} (clam)	9×10^{-5} (clam)
			7×10^{-7} (pelagic)	5×10^{-6} (pelagic)
			2×10^{-6} (crab)	1×10^{-6} (crab)
			3×10^{-7} (benthic)	1×10^{-7} (benthic)
		non-Asarco	6×10^{-6}(clam)	9×10^{-5} (clam)
			2×10^{-6} (pelagic)	4×10^{-6} (pelagic)
			2×10^{-6} (crab)	1×10^{-6} (crab)
			3×10^{-7} (benthic)	1×10^{-7} (benthic)

[a] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Cancer risk estimates for this exposure scenario are species-specific, in contrast to the other scenarios, which are combined estimates for the consumption of all species.

API – Asian and Pacific Islander

Table B.5-58. Incremental cancer risk estimates associated with inorganic arsenic for the seafood consumption categories in the adult tribal RME seafood exposure scenario based on Tulalip data

Seafood Category	LDW-Wide Cancer Risk Estimate[a]	Background Cancer Risk Estimate[a]	Incremental Cancer Risk Estimate[a]	Percent of Total Risk[b]
Asarco-influenced background				
Benthic fish	9×10^{-7}	6×10^{-7}	3×10^{-7}	0.032%
Clam	1×10^{-3}	2×10^{-4}	8×10^{-4}	97%
Crab – edible meat	3×10^{-5}	2×10^{-5}	1×10^{-5}	1.1%
Crab – whole body	2×10^{-5}	1×10^{-5}	1×10^{-5}	1.1%
Pelagic fish	1×10^{-5}	2×10^{-6}	8×10^{-6}	0.86%
Total risk[c]	**1×10^{-3}**	**2×10^{-4}**	**8×10^{-4}**	**100%**
Non-Asarco background				
Benthic fish	9×10^{-7}	5×10^{-7}	2×10^{-7}	0.041%
Clam	1×10^{-3}	5×10^{-5}	1×10^{-3}	98.3%
Crab – edible meat	3×10^{-5}	2×10^{-5}	1×10^{-5}	1.0%
Crab – whole body	2×10^{-5}	2×10^{-5}	0	0%
Pelagic fish	1×10^{-5}	4×10^{-6}	6×10^{-6}	0.62%
Total risk[c]	**1×10^{-3}**	**1×10^{-4}**	**9×10^{-4}**	**100%**

[a] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] The percent total risk is based on the percent of the total incremental cancer risk estimate.

[c] The total risk represents the LDW-wide risk estimate for the adult Tulalip RME seafood exposure scenario based on all consumption categories included (i.e., benthic fish, benthic fish whole body, clams, crab edible meat, crab whole body, and pelagic fish). However, because of significant figures, this value is not necessarily equal to the sum of the five consumption categories shown in this table.

The clam background data from Seahurst Park and Bainbridge Island were not used to make incremental risk estimates because the background clams were not the same species as those found in the LDW (*M. arenaria*) and different clam species may bioaccumulate arsenic to different extents. However, the incremental risk estimates would not be greatly different if these alternate background clam data were used. Based on the incremental risks, which are attributable largely to arsenic in clams, inorganic arsenic would still be considered a COC in tissue, although it would no longer exceed 1×10^{-6} excess cancer risk for adult one-meal-per-month crab consumption.

B.5.5.2 Dioxins/furans

Excess cancer risks for dioxins/furans were higher than 1×10^{-6} for all of the direct sediment exposure scenarios except for beach play RME at areas 5 and 7, where the risk was below 1×10^{-6}. No cancer risk estimates were possible for beach play RME areas 1, 2, 3, 6, and 8 because there are no dioxin/furan sediment chemistry data from those areas. The sediment risk estimates provided previously do not consider what percent of the total dioxin/furan risk in the LDW could be attributable to natural sources in the Puget Sound Basin or to anthropogenic sources representative of the LDW urban background. No risk estimates were made for human exposure to dioxins/furans in LDW seafood. LDWG, EPA, and Ecology participated in a meeting on the dioxin/furan issue and agreed that elevated dioxin and furan concentrations in sediment would result in unacceptable seafood consumption risks. LDWG, EPA, and Ecology agreed that that dioxins and furans in sediments would be remediated on the basis of direct contact risks and sediment background concentrations rather than on the basis of seafood consumption. Consequently, dioxins and furans were not analyzed in tissue. All discussions of background levels for dioxins and furans presented here should be considered preliminary and may be re-evaluated in the RI and FS. Background data evaluations in this risk assessment are for the purpose of incremental risk calculations only and not for the determination of potential cleanup levels based on background concentrations.

Dioxins/furans enter the aquatic environment through non-point source runoff via storm drains, through direct atmospheric deposition to surface water, or through point source discharges associated with certain industrial practices (e.g., pulp mills or pentachlorophenol [PCP] production) (Ecology 1998). Urban sources of dioxins/furans include fuel combustion (particularly diesel fuel), backyard burning, and wood stoves. Incineration of waste materials that contain chlorine, such as medical waste and municipal solid waste, and certain types of chemical manufacturing can also result in air releases of dioxins/furans. Air emission is one of the most common mechanisms for the release of dioxins/furans into the environment (Ecology 1998). Thus, dioxins/furans are commonly dispersed in urban areas because air emission sources are commonly found in urban areas. Stormwater runoff from

surfaces such as parking lots, streets, and rooftops may contribute significant mass of dioxins/furans.

B.5.5.2.1 Sediment

The objective of the study design for dioxins and furans was to identify background concentrations that would represent non-point urban sources, as reflected in sediments of receiving waters from urban storm drains. Such a design is consistent with EPA (EPA 2002e) guidance for characterizing anthropogenic, or urban, background. The 2005 study design included the collection of 13 sediment samples in the vicinity of storm drains that serve drainage basins in the greater Seattle area (Windward 2005e). Because over 200 storm drains and 10 CSOs discharge into the LDW study area, the background sampling locations, shown in Map B.5-3, were selected to meet the following criteria for characterizing "urban background" in the LDW:

- Receive drainage from areas with land use as similar as could be found to that of areas draining to the LDW
- Not near known industrial point sources of dioxins/furans
- Represent a range of receiving water environments
- Discharge flow characteristics for the nearby storm drains (i.e., a range of discharge frequencies, velocities, and types) similar to those in the LDW

The LDW urban background dataset for dioxins/furans in sediment is summarized in Table B.5-59. The concentrations are represented as dioxin/furan TEQs. TEQs ranged from 2.23 to 187 ng/kg dw. Samples with the highest TEQs were collected in the ship canal and Union Bay near Laurelhurst. Samples with the lowest TEQs were collected upstream of the LDW (see Map B.5-3). The LDW urban background data summarized in Table B.5-59 were compared to national background TEQs from EPA (2000b)[28] as a means of identifying appropriate background concentrations for use in the incremental risk assessment.

[28] The range of dioxin/furan TEQs in sediments of 11 lakes and reservoirs throughout the US, which were selected to represent background conditions in areas removed from known sources, was 0.12 to 16.3 ng/kg dw (EPA 2000c), with an arithmetic mean of 5.3 ng/kg dw.

Table B.5-59. Calculated dioxin/furan TEQs in surface sediment samples collected from the greater Seattle area

LOCATION	LOCATION ID	DIOXIN/FURAN TEQ (ng/kg dw)[a]
Elliott Bay (Terminal 91)	EB-SS2a	13.7 J
	EB-SS2b	18.9 J
Lake Union (I-5 bridge)	LU-SS9a	5.46 J
	LU-SS9b	26.1 J
Lake Washington (Bothell)	LW-SS3	13.2 J[b]
Lake Washington (Bellevue)	LW-SS4	14.7 J
Lake Washington (Renton)	LW-SS5a	14.1 J
	LW-SS5b	14.5 J
Springbrook Creek (Tukwila)	SB-SS6	2.23 J
Duwamish River (Tukwila)	DRD-SS7	2.59 J
Ship canal (Salmon Bay)	SC-SS1a	187 J
	SC-SS1b	63.1 J
Union Bay (Laurelhurst)	UB-SS8	53.4 J

[a] TEQ calculated using concentrations equal to half the reporting limit for congeners that were not detected for a given sample and using mammalian toxic equivalency factors reported in Table B.2-5.

[b] Reported concentration is the average of two field duplicate samples (12.8 and 13.6 ng/kg dw).

dw – dry weight
J – estimated concentration
ID – identification
TEQ – toxic equivalent

Data from samples collected from the ship canal and Union Bay have been omitted from the LDW urban background dataset to be used in the incremental risk analysis because while no specific dioxin/furan sources have been identified at these locations, these TEQs are well above the range of those identified as national background TEQs in either soils or sediments (EPA 2000b; Windward 2005h). The data from upstream of the LDW (i.e., Springbrook Creek and Duwamish River) have also been omitted from the incremental risk analysis because these locations have much lower commercial and industrial land use patterns compared to the LDW, as noted by a land use/land cover map produced for the Green/Duwamish estuary subwatershed (Kerwin and Nelson 2000).

Two samples were collected approximately 30 ft apart at each of the Elliott Bay, Lake Union, and Lake Washington (Renton) locations. While not intended to be field replicates,[29] it is appropriate to average the results from the two samples at each location to better represent area exposures and not overemphasize individual samples. Following elimination of select stations as inappropriate for LDW urban background and consolidation of station data in small areas, five location results are available for characterizing LDW urban background conditions. Data on dioxin/furan TEQs for

[29] The samples were collected to provide a better representation of the general vicinity of the storm drains.

these locations are presented in Table B.5-60. The mean and UCL (calculated using ProUCL) on the mean concentrations for these five locations are 14.9 and 16.0 ng/kg dw, respectively. The UCL is only slightly greater than the mean, reflecting the relatively narrow range of TEQs and their normal distribution as determined by ProUCL. The inclusion of the two upstream locations in the incremental risk calculations would have resulted in a lower mean (11.2 ng/kg dw) but a higher UCL on the mean (21.1 ng/kg dw) because of the skewness of the resulting dataset. The UCL on the mean for the five LDW urban background locations (i.e., without the locations upstream of the LDW) was used as the EPC in the incremental risk assessment.

Table B.5-60. Summary of dioxin/furan TEQs used for the characterization of LDW urban background concentrations of dioxins/furans in surface sediment for the greater Seattle area

LOCATION	DIOXIN/FURAN TEQ (ng/kg dw)[a]
Elliott Bay (Terminal 91)	16.3 J
Lake Union (I-5 bridge)	15.8 J
Lake Washington (Bothell)	13.2 J[b]
Lake Washington (Bellevue)	14.7 J
Lake Washington (Renton)	14.3 J
Mean concentration	14.9
UCL on mean concentration (calculated by ProUCL)	16.0

[a] TEQ calculated using concentrations equal to half the reporting limit for dioxin/furan congeners that were not detected for a given sample and using mammalian toxic equivalency factors reported in Table B.2-5.

[b] Reported concentration is the average of two field duplicate samples (12.8 and 13.6 ng/kg dw).

dw – dry weight

J – estimated concentration

TEQ – toxic equivalent

UCL – upper confidence limit

The dataset for dioxins/furans in LDW sediment is summarized in Table B.5-61 and shown on Map B.3-4. The dataset is a product of reconnaissance-level sampling conducted by EPA in 1998 and focused sampling conducted by LDWG in 2005 to eliminate data gaps and identify hot spots. It is possible that some small hot spots remain uncharacterized. The mean concentration of the data shown in Table B.5-61 is 100 ng/kg dw, but the UCL on that mean is much higher (610 ng/kg dw) because several high concentrations create a highly skewed dataset. Removing the highest concentrations (i.e., seven samples with TEQs higher than 100 ng/kg dw) yields a arithmetic mean TEQ of 9.9 ng/kg dw for the remainder of the dataset and a UCL on the mean TEQ of 12.7 ng/kg dw. Comparison of these values with the results in Table B.5-60 indicates that dioxin/furan TEQ concentrations in most of the LDW sediment are similar to dioxin/furan TEQ sediment concentrations in the greater Seattle urban environment and in national background areas away from known sources (EPA

2000c). Although no remedial decisions about dioxins/furans in LDW sediment have been made, this analysis indicates that cleaning up small areas with the highest dioxin/furan TEQs is likely to significantly lower the average dioxin/furan TEQ in the LDW.

Table B.5-61. Calculated dioxin/furan TEQs in LDW surface sediment samples

Location ID	Sampling Event (year)	Dioxin/Furan TEQ (ng/kg dw)[a]
DR002	EPA SI (1998)	7.20 J
DR008	EPA SI (1998)	180 J
DR021	EPA SI (1998)	13.0 J
DR033	EPA SI (1998)	5.90 J
DR042	EPA SI (1998)	16.0 J
DR046	EPA SI (1998)	7.80 J
DR047	EPA SI (1998)	8.90 J
DR051	EPA SI (1998)	13.0 J
DR092	EPA SI (1998)	8.10 J
DR101	EPA SI (1998)	2.20
DR111	EPA SI (1998)	11.0 J
DR115	EPA SI (1998)	6.90 J
DR154	EPA SI (1998)	12.0 J
DR168	EPA SI (1998)	7.80 J
DR183	EPA SI (1998)	4.40 J
DR203	EPA SI (1998)	2.20 J
DR206	EPA SI (1998)	2.60 J
DR221	EPA SI (1998)	3.90 J
DR224	EPA SI (1998)	2.00
DR246	EPA SI (1998)	8.40 J
DR264	EPA SI (1998)	1.70
DR284	EPA SI (1998)	2.90
DR291	EPA SI (1998)	4.70 J
DR298	EPA SI (1998)	1.10 J
DR301	EPA SI (1998)	1.20 J
LDW-SS14	LDWG (2005)	1.59 J
LDW-SS28	LDWG (2005)	11.7 J
LDW-SS36	LDWG (2005)	27.1 J
LDW-SS37	LDWG (2005)	124 J
LDW-SS43	LDWG (2005)	18.2 J
LDW-SS56	LDWG (2005)	2,100 J

Location ID	Sampling Event (year)	Dioxin/Furan TEQ (ng/kg dw)[a]
LDW-SS57	LDWG (2005)	463 J
LDW-SS58	LDWG (2005)	565 J
LDW-SS59	LDWG (2005)	49.0 J
LDW-SS64	LDWG (2005)	10.1 J
LDW-SS71	LDWG (2005)	13.0 J
LDW-SS83	LDWG (2005)	33.3 J
LDW-SS84	LDWG (2005)	412 J
LDW-SS109	LDWG (2005)	101 J
LDW-SS123	LDWG (2005)	5.08 [b] J
LDW-SS127	LDWG (2005)	13.5 J
LDW-SS131	LDWG (2005)	15.5 J [c]
LDW-SS143	LDWG (2005)	4.69 J
LDW-wide	mean	100 J
LDW-wide	UCL on mean	610 J

[a] TEQ calculated using concentrations equal to half the reporting limit for dioxin/furan congeners that were not detected for a given sample and using mammalian toxic equivalency factors reported in Table B.2-5.

[b] Reported concentration is the average of two field duplicate samples (4.93 and 5.22 ng/kg dw).

[c] Reported concentration is the average of two field duplicate samples (8.29 and 22.7 ng/kg dw).

dw – dry weight

J – estimated concentration

EPA SI – EPA site inspection

TEQ – toxic equivalent

The UCL on the mean (610 ng/kg dw) for the full LDW sediment dataset was used as the EPC for dioxins/furans in the incremental risk assessment for the netfishing scenario because this scenario considers the entire LDW to be the exposure area. Although the UCL is derived from 43 samples using ProUCL, it is strongly influenced by a few high concentrations that are located in areas where netfishing may not be possible. Alternate UCL calculations and associated risk calculations that take into account locations that may not be suitable for netfishing are presented in the uncertainty analysis (see Section B.6.1.7). Other sediment EPCs for dioxins/furans, as shown in Table B.5-62, were used for the beach play and clamming direct sediment exposures because the exposure areas were smaller than the entire LDW and thus included only a subset of the data shown in Table B.5-62. The calculation of UCLs for these EPCs followed the rules provided previously for EPC estimation (Section B.3.4.3). The EPCs for the two clamming scenarios are both strongly influenced by a few samples with much higher dioxin/furan concentrations. In the case of the 7-day-per-year clamming scenario (based on only six samples), the EPC would have been much lower if a single sample with a much higher dioxin/furan concentration had been excluded. In the case of the tribal clamming scenarios (based on only 11 samples),

the EPC was strongly influenced by three samples with high dioxin/furan concentrations collected in a small area on the western side of the LDW between RM 1.4 and RM 1.5. The EPCs for the three beach play RME scenarios were based on only one sample each, in which case the EPC is equivalent to the single TEQ value for that exposure area. In cases where the underlying distribution of chemical concentrations is positively skewed for an exposure area, the use of a single sample to represent the EPC for these beach play areas likely contributes to an underestimation of exposure (see Section B.6.1.1.11), which might affect the incremental risk comparisons presented here. Background exposure scenarios analogous to that developed above for the full LDW dataset with the netfishing scenario have not been developed for each data subset for the other scenarios, so the LDW EPCs for those scenarios were compared to the single urban background UCL from Table B.5-60.

Table B.5-62. Sediment exposure point concentrations for dioxins/furans (TEQ) in the LDW urban background and LDW exposure scenarios

Area	Exposure Scenario	Exposure Point Concentration (ng/kg dw)	Sample Size
LDW urban background	All scenarios	16.0 J	5 locations (Table B.5-60)
LDW	Netfishing	610 J	43 samples (Table B.5-61)
	Tribal clamming[a]	1,420 J	11 samples
	Clamming – 7 days per year	365 J	6 samples
	Beach play RME, Area 4	412 J	1 sample
	Beach play RME, Area 5	2.20 J	1 sample
	Beach play RME, Area 7	1.70 J	1 sample

[a] Includes both the tribal clamming RME scenario and the tribal clamming 183-day-per-year clamming scenario.

dw – dry weight

J – estimated concentration

RME – reasonable maximum exposure

Incremental risks calculated for exposures to dioxins/furans TEQs in LDW sediment for all sediment scenarios are shown in Table B.5-63. Because elevated dioxin/furan TEQs were identified as the dominant contributor for the netfishing and tribal clamming RME scenarios, the cancer risk estimates for those scenarios in general are much higher than for beach play RME scenarios. The incremental risk estimates for the beach play RME area 5 and 7 scenarios are zero because the LDW urban background EPC is as high as or higher than the EPCs for these scenarios (see Table B.5-62).

Table B.5-63. Incremental risk estimates from exposure to dioxins/furans (TEQ) in sediment

Exposure Scenario	LDW Cancer Risk Estimate	LDW Urban Background Cancer Risk Estimate	Incremental Cancer Risk Estimate
Netfishing RME	2×10^{-5}	4×10^{-7}	2×10^{-5}
Netfishing CT	4×10^{-6}	1×10^{-7}	4×10^{-6}
Tribal clamming – 183 days per year	2×10^{-4}	2×10^{-6}	2×10^{-4}
Tribal clamming RME(120 days per year)	1×10^{-4}	1×10^{-6}	1×10^{-4}
Clamming – 7 days per year	8×10^{-7}	4×10^{-8}	8×10^{-7}
Beach play RME, Area 4	1×10^{-5}	6×10^{-7}	9×10^{-6}
Beach play RME, Area 5	8×10^{-8}	6×10^{-7}	0
Beach play RME, Area 7	6×10^{-8}	6×10^{-7}	0

B.5.5.2.2 Tissue

LDWG, EPA, and Ecology agreed that elevated dioxin and furans in sediment would result in unacceptable seafood consumption risks and that dioxins/furans are ubiquitous in urban environments. LDWG, EPA, and Ecology agreed that that dioxins and furans in sediments would be remediated on the basis of direct contact risks and sediment background concentration rather than on the basis of seafood consumption. Consequently, dioxins and furans were not analyzed in tissue. The parties discussed that for the purpose of risk communication, qualitative risks associated with consuming seafood containing dioxins/furans should be discussed in the HHRA.

Dioxins/furans are hydrophobic chemicals that preferentially accumulate in organic-rich sediments and tissues. Dioxins/furans are ubiquitous in the environment and in the food that people consume. As part of the US Food and Drug Administration's ongoing Total Diet Study (http://www.cfsan.fda.gov/~comm/tds-toc.html), hundreds of food samples, representing the major components of the diet of the US population, have been analyzed for dioxins/furans. These chemicals are detected in almost every type of food that people consume. Because dioxins/furans tend to bioaccumulate in the food chain, animal meats have higher TEQs than fruit, vegetables, or grains.

Dioxins/furans have been detected consistently in fish and shellfish samples worldwide as well as in other foods (Institute of Medicine 2003). Fish and shellfish samples collected worldwide, including those collected throughout the Pacific Northwest, from Elliott Bay (EPA 1999b), Olympia (Era-Miller 2005, 2006), Port Orchard (ATSDR 1997), Port Angeles (ATSDR 2005a), the Columbia River basin (EPA 2002b), and the Willamette River (SEA et al. 2003) had dioxin/furan TEQs for almost all of these samples that exceeded the National Toxics Rule human health criterion of

0.07 ng/kg ww (40 CFR 131.36).[30] Most of these data (all but the Columbia River basin data) were collected as part of contaminated site investigations. Many of these dioxin/furan TEQs were also above 0.5 ng/kg ww. In addition, Schecter and Li (1997) and Schecter et al. (1997) analyzed dioxins and furans in food from American supermarkets and fast food restaurants and obtained dioxin/furan TEQ concentrations on the order of 0.2 ng/kg ww. Assuming a consumption rate of 97.5 g/day (the rate used for the tribal adult RME seafood consumption scenario based on Tulalip data), the excess cancer risk for dioxin/furan TEQs of 0.2 to 0.5 ng/kg ww would range from 4×10^{-5} to 9×10^{-5}. Based on these analyses, this HHRA assumes that dioxin/furan risks resulting from consumption of resident LDW seafood would exceed EPA and Ecology's acceptable risk ranges.

The dioxin/furan concentrations in most LDW sediments are within the range of dioxin/furan concentrations in LDW urban background sediments in areas receiving discharges from multiple urban storm drains and CSOs. However, concentrations of dioxins/furans in sediments at a few locations within the LDW are more than 100 times higher than in sediments at LDW urban background locations. Risks associated with exposure to dioxins/furans in the LDW through direct sediment contact were estimated, and these estimates were found to be above the 1×10^{-6} threshold (see Section B.5.1.1). Because of the assumptions that seafood consumption risks would be unacceptable and that sediments with dioxin/furan concentrations above LDW urban background levels would likely require remediation, tissue data are not needed to make remedial decisions.

B.5.6 RISK CHARACTERIZATION SUMMARY

The excess cancer risk and non-cancer hazard estimates for seafood consumption scenarios are summarized in Tables B.5-64 and B.5-65, respectively; the excess cancer risk estimates for direct sediment exposure scenarios are summarized in Table B.5-63. For the purpose of brevity, chemical-specific risk and HQ estimates are provided only for chemicals exceeding a cancer risk estimate of 1×10^{-6} or an HQ of 1 for any scenario.[31]

Risks have been evaluated for a number of different types of exposure scenarios to describe different intensities of site use or seafood consumption. RME scenarios represent the highest exposures that are reasonably expected to occur at a site, and are generally used by EPA to evaluate remedial actions at a site (EPA 1989). RME by definition likely overestimates exposure for many individuals. CT risk estimates are intended to reflect average exposures. CT exposures and risks are not favored in

[30] This TEQ is part of the calculation used to derive the human health water quality criterion for 2,3,7,8-TCDD, based on the consumption of organisms.

[31] Note that a chemical must be associated with greater than 1×10^{-6} and/or HQs greater than 1 for one or more RME scenarios to be designated as a COC. Complete lists of seafood consumption and direct contact COCs are provided in Section B.7.

decision-making because they will underestimate exposure and risk for a substantial number of individuals (EPA 1989). CT exposures and risks are useful in characterizing the exposure/risk range (National Research Council 1994). Another method of examining risk and exposure is to look at risks associated with some unit of exposure that a member of the public can use to assess risks associated with their individual behavior. This last method was used to characterize seafood consumption risk on an individual basis, with the unit of exposure being one meal per month.

Table B.5-64. Summary of estimated excess cancer risks for the seafood consumption scenarios

Chemical	Adult Tribal RME (Tulalip Data)	Adult Tribal CT (Tulalip Data)	Child Tribal RME (Tulalip Data)	Child Tribal CT (Tulalip Data)	Adult Tribal (Suquamish Data)	Adult API RME	Adult API CT	Adult One Meal per Month			
								Benthic Fish	Clam	Crab	Pelagic Fish
Arsenic (inorganic)[a]	1×10^{-3}	6×10^{-5}	3×10^{-4}	3×10^{-5}	2×10^{-2} [b]	7×10^{-4}	1×10^{-5}	4×10^{-7}	1×10^{-4}	3×10^{-6}	6×10^{-6}
Bis(2-ethylhexyl) phthalate	6×10^{-6}	2×10^{-7}	1×10^{-6}	7×10^{-8}	4×10^{-5}	2×10^{-6}	3×10^{-8}	8×10^{-7}	8×10^{-8}	8×10^{-8} [c]	1×10^{-6}
cPAHs[d]	7×10^{-5}	4×10^{-6}	7×10^{-5}	8×10^{-6}	8×10^{-4}	3×10^{-5}	8×10^{-7}	2×10^{-7}	7×10^{-6}	2×10^{-7}	3×10^{-7}
Dioxin/furan TEQ[e]	na	na	na	na	na	na	na	na	na	na	na
PCB TEQ	1×10^{-3}	6×10^{-5}	2×10^{-4}	2×10^{-5}	7×10^{-3}	4×10^{-4}	7×10^{-6}	8×10^{-5}	2×10^{-5}	2×10^{-5}	2×10^{-4}
Total PCBs	2×10^{-3}	6×10^{-5}	3×10^{-4}	3×10^{-5}	1×10^{-2} [b]	5×10^{-4}	8×10^{-6}	1×10^{-4}	5×10^{-5}	2×10^{-5}	2×10^{-4}
Pentachlorophenol[a]	9×10^{-5} [f]	2×10^{-6} [f]	2×10^{-5} [f]	7×10^{-7} [f]	5×10^{-4} [f]	2×10^{-5}	3×10^{-7}	2×10^{-5} [c]	1×10^{-6} [c]	2×10^{-6} [c]	1×10^{-5}
Subtotal (excluding PCB TEQ)[g]	**3×10^{-3}**	**1×10^{-4}**	**7×10^{-4}**	**7×10^{-5}**	**3×10^{-2}**	**1×10^{-3}**	**2×10^{-5}**	**1×10^{-4}**	**2×10^{-4}**	**3×10^{-5}**	**2×10^{-4}**
Subtotal (excluding total PCBs)[g]	**2×10^{-3}**	**1×10^{-4}**	**6×10^{-4}**	**6×10^{-5}**	**3×10^{-2}**	**1×10^{-3}**	**2×10^{-5}**	**1×10^{-4}**	**1×10^{-4}**	**3×10^{-5}**	**2×10^{-4}**
Tentatively identified chemicals (JN-qualified)											
Aldrin	5×10^{-5} [f]	1×10^{-6} [f]	9×10^{-6} [f]	6×10^{-7} [f]	2×10^{-4}	1×10^{-5}	2×10^{-7}	3×10^{-6} [c]	8×10^{-7} [c]	3×10^{-6} [c]	3×10^{-6}
alpha-BHC	2×10^{-5} [f]	5×10^{-7} [f]	3×10^{-6} [f]	2×10^{-7} [f]	6×10^{-5}	3×10^{-6}	6×10^{-8}	1×10^{-6}	1×10^{-7}	1×10^{-6} [c]	1×10^{-6}
beta-BHC	6×10^{-6} [f]	3×10^{-7} [f]	1×10^{-6} [f]	1×10^{-7} [f]	3×10^{-5}	1×10^{-6}	3×10^{-8}	3×10^{-7}	1×10^{-7}	3×10^{-7} [c]	6×10^{-7}
Carbazole	5×10^{-5}	1×10^{-6}	8×10^{-6}	4×10^{-7}	2×10^{-4}	1×10^{-5}	8×10^{-8}	1×10^{-6} [c]	9×10^{-8} [c]	1×10^{-6} [c]	1×10^{-5}
Total chlordane	6×10^{-6}	2×10^{-7}	1×10^{-6}	9×10^{-8}	3×10^{-5}	2×10^{-6}	3×10^{-8}	3×10^{-7}	7×10^{-8}	7×10^{-8}	1×10^{-6}
Total DDTs	2×10^{-5}	1×10^{-6}	4×10^{-6}	4×10^{-7}	1×10^{-4}	6×10^{-6}	1×10^{-7}	1×10^{-6}	2×10^{-7}	4×10^{-7}	4×10^{-6}
Dieldrin	1×10^{-4}	3×10^{-6}	2×10^{-5}	1×10^{-6}	1×10^{-3}	5×10^{-5}	4×10^{-7}	3×10^{-6} [c]	9×10^{-6}	3×10^{-6}	3×10^{-6} [c]
gamma-BHC	6×10^{-6}	1×10^{-7}	1×10^{-6}	5×10^{-8}	3×10^{-5}	1×10^{-6}	1×10^{-8}	2×10^{-7} [c]	1×10^{-7}	2×10^{-7}	1×10^{-7}
Heptachlor	1×10^{-5} [f]	4×10^{-7} [f]	3×10^{-6} [f]	2×10^{-7} [f]	6×10^{-5}	3×10^{-6}	4×10^{-8}	7×10^{-7} [c]	1×10^{-7} [c]	7×10^{-7} [c]	2×10^{-6}
Heptachlor epoxide	3×10^{-5}	1×10^{-6}	6×10^{-6}	5×10^{-7}	2×10^{-4}	9×10^{-6}	1×10^{-7}	1×10^{-6} [c]	6×10^{-7}	9×10^{-7}	4×10^{-6}
Hexachlorobenzene	1×10^{-5}	2×10^{-7}	2×10^{-6}	1×10^{-7}	4×10^{-5}	2×10^{-6}	3×10^{-8}	6×10^{-7}	6×10^{-8}	6×10^{-7}	9×10^{-7}
Subtotal[g]	**3×10^{-4}**	**9×10^{-6}**	**6×10^{-5}**	**4×10^{-6}**	**2×10^{-3}**	**1×10^{-4}**	**1×10^{-6}**	**1×10^{-5}**	**1×10^{-5}**	**1×10^{-5}**	**3×10^{-5}**
Total excess cancer risk (excluding PCB TEQ)[g]	**3×10^{-3}**	**1×10^{-4}**	**8×10^{-4}**	**7×10^{-5}**	**3×10^{-2}**	**1×10^{-3}**	**2×10^{-5}**	**1×10^{-4}**	**2×10^{-4}**	**4×10^{-5}**	**2×10^{-4}**
Total excess cancer risk (excluding total PCBs)[g]	**2×10^{-3}**	**1×10^{-4}**	**7×10^{-4}**	**6×10^{-5}**	**3×10^{-2}**	**1×10^{-3}**	**2×10^{-5}**	**1×10^{-4}**	**1×10^{-4}**	**4×10^{-5}**	**2×10^{-4}**

[a] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Because the excess cancer risk is greater than or equal to 0.01, risk was calculated using the exponential equation in EPA (1989).

[c] No detected values in this seafood category. CDI and risk estimate are based on one-half the maximum reporting limit.

[d] cPAH concentrations are based on benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6). Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for children for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[e] Tissue data for dioxins/furans were not collected. Thus, the calculated risk total does not include the unknown contribution to exposures and risks from dioxins/furans.

[f] Greater than 50% of the risk (based on dose) associated with this chemical is derived from seafood categories with no detected values.

[g] Total and subtotal risk values include the risks associated with all COPCs. Because cancer risks for all COPCs were greater than 1×10^{-6} for at least one scenario, all COPCs are listed in this table.

API – Asian and Pacific Islanders

BHC – benzene hexachloride

CDI – chronic daily intake

cPAH – carcinogenic polycyclic aromatic hydrocarbon

CT – central tendency

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

TEQ – toxic equivalent quotient

Table B.5-65. Summary of estimated non-cancer hazards for the seafood consumption scenarios

Chemical	Adult Tulalip RME (Tulalip Data)	Adult Tribal CT (Tulalip Data)	Child Tribal RME (Tulalip Data)	Child Tribal CT (Tulalip Data)	Adult Tribal (Suquamish Data)	Adult API RME	Adult API CT	Adult One Meal per Month			
								Benthic Fish	Clam	Crab	Pelagic Fish
Arsenic[a, b]	3	0.3	7	0.7	38	3	0.2	0.002	0.7	0.01	0.03
Chromium	0.2	0.02	0.3	0.04	2	0.1	0.01	0.002	0.03	0.006	0.007
Mercury	0.5	0.07	1	0.1	2	0.3	0.02	0.06	0.02	0.07	0.04
Total PCBs	40	4	86	8	274	29	2	6	3	1	10
TBT (as ion)	1	0.2	3	0.3	15	1	0.1	0.002	0.3	0.02	0.06
Vanadium	0.8	0.1	2	0.2	9	0.8	0.07	0.01	0.2	0.01	0.06
Subtotal[c]	**46**	**5**	**101**	**10**	**344**	**35**	**2**	**6**	**4**	**1**	**10**
Hazard indices by effect:											
HI for cardiovascular endpoint[d]	**4**	**0.4**	**9**	**0.9**	**47**	**4**	**0.3**	**0.01**	**0.9**	**0.02**	**0.09**
HI for developmental endpoint[e]	**41**	**4**	**87**	**8**	**276**	**29**	**2**	**6**	**3**	**1**	**10**
HI for hematologic endpoint[f]	**0.2**	**0.03**	**0.5**	**0.05**	**2**	**0.2**	**0.01**	**0.006**	**0.03**	**0.01**	**0.009**
HI for immunological endpoint[g]	**41**	**4**	**89**	**8**	**289**	**30**	**2**	**6**	**3**	**1**	**10**
HI for kidney endpoint[h]	**0.4**	**0.05**	**1.0**	**0.1**	**2**	**0.3**	**0.02**	**0.03**	**0.03**	**0.04**	**0.04**
HI for liver endpoint[i]	**1**	**0.1**	**3**	**0.3**	**7**	**0.8**	**0.05**	**0.1**	**0.1**	**0.09**	**0.3**
HI for neurological endpoint[j]	**41**	**4**	**87**	**8**	**276**	**29**	**2**	**6**	**3**	**1**	**10**
HI for dermal endpoint[k]	**3**	**0.3**	**7**	**0.7**	**38**	**3**	**0.2**	**0.01**	**0.7**	**0.02**	**0.06**
Total HI across all exposure routes/ pathways[c]	**47**	**5**	**103**	**10**	**348**	**35**	**2**	**6**	**4**	**1**	**10**

a No mussel data were available for this chemical. When calculating the risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

b Arsenic risk estimates are based on inorganic arsenic.

c Total and subtotal risk values include the risks associated with all COPCs. However, only those COPCs with a hazard quotient greater than or equal to 1 for at least one scenario are listed in this table.

d Cardiovascular endpoint is for arsenic and vanadium.

e Developmental endpoint is for PCBs and mercury.

f Hematologic endpoint is for antimony and zinc.

[g] Immunological endpoint is for PCBs and TBT.

[h] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[i] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[j] Neurological endpoint is for 4-methylphenol, mercury and total PCBs.

[k] Dermal endpoint is for 4-methylphenol and arsenic.

API – Asian and Pacific Islanders
BHC – benzene hexachloride
COPC – chemical of potential concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
HI – hazard index
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TBT – tributyl tin

Table B.5-66. Summary of estimated excess cancer risks for direct sediment exposure scenarios

Chemical	Netfishing		Beach Play RME								Clamming		
	RME	CT	Area 1	Area 2	Area 3	Area 4	Area 5	Area 6	Area 7	Area 8	Tribal – 183 Days per Year	Tribal RME	7 Days per Year
Arsenic	6×10^{-6}	1×10^{-6}	5×10^{-6}	7×10^{-6}	4×10^{-6}	4×10^{-6}	3×10^{-6}	3×10^{-6}	4×10^{-6}	3×10^{-6}	3×10^{-5}	2×10^{-5}	3×10^{-7}
cPAHs[a]	1×10^{-6}	2×10^{-7}	1×10^{-5}	4×10^{-5}	3×10^{-5}	8×10^{-6}	5×10^{-6}	5×10^{-6}	1×10^{-6}	4×10^{-6}	8×10^{-6}	5×10^{-6}	1×10^{-7}
Dioxin/furan TEQ	2×10^{-5}	4×10^{-6}	na	na	na	1×10^{-5}	8×10^{-8}	na	6×10^{-8}	na	2×10^{-4}	1×10^{-4}	8×10^{-7}
PCB TEQ	4×10^{-6}	6×10^{-7}	4×10^{-9}	3×10^{-7}	na	9×10^{-6}	1×10^{-7}	3×10^{-7}	3×10^{-8}	8×10^{-8}	5×10^{-5}	3×10^{-5}	2×10^{-7}
Total PCBs	2×10^{-6}[b]	3×10^{-7}[b]	7×10^{-8}	1×10^{-7}	1×10^{-7}	6×10^{-6}	1×10^{-7}	5×10^{-7}	1×10^{-7}	1×10^{-7}	1×10^{-5}[b]	8×10^{-6}[b]	9×10^{-8}
Subtotal (excluding PCB TEQ)	**3×10^{-5}**	**5×10^{-6}**	**2×10^{-5}**	**5×10^{-5}**	**3×10^{-5}**	**3×10^{-5}**	**8×10^{-6}**	**9×10^{-6}**	**5×10^{-6}**	**7×10^{-6}**	**2×10^{-4}**	**1×10^{-4}**	**1×10^{-6}**
Subtotal (excluding total PCBs)	**3×10^{-5}**	**6×10^{-6}**	**2×10^{-5}**	**5×10^{-5}**	**3×10^{-5}**	**3×10^{-5}**	**8×10^{-6}**	**8×10^{-6}**	**5×10^{-6}**	**7×10^{-6}**	**3×10^{-4}**	**2×10^{-4}**	**1×10^{-6}**
Tentatively identified chemicals (JN-qualified)													
Toxaphene	2×10^{-6}	3×10^{-7}	1×10^{-8}	7×10^{-9}	2×10^{-8}	2×10^{-7}	1×10^{-7}	1×10^{-8}	1×10^{-8}	1×10^{-8}	9×10^{-6}	6×10^{-6}	2×10^{-8}
Total risk (excluding PCB TEQ) across both exposure routes)[c]	**3×10^{-5}**	**5×10^{-6}**	**2×10^{-5}**	**5×10^{-5}**	**3×10^{-5}**	**3×10^{-5}**	**8×10^{-6}**	**9×10^{-6}**	**5×10^{-6}**	**7×10^{-6}**	**2×10^{-4}**	**1×10^{-4}**	**1×10^{-6}**
Total risk (excluding total PCBs) across both exposure routes)[c]	**3×10^{-5}**	**6×10^{-6}**	**2×10^{-5}**	**5×10^{-5}**	**3×10^{-5}**	**3×10^{-5}**	**8×10^{-6}**	**8×10^{-6}**	**5×10^{-6}**	**7×10^{-6}**	**3×10^{-4}**	**2×10^{-4}**	**1×10^{-6}**

[a] cPAH concentrations are based on benzo(a)pyrene equivalents. Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for beach play RME for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[b] EPC value was derived from an arithmetic UCL for the entire LDW, and therefore likely overestimates the true exposure. An alternate EPC value based on a spatially weighted calculation is presented in Section B.6.1.1.12.

[c] Total risk values include the risks associated with all COPCs. However, only those COPCs with an excess cancer risk greater than or equal to 1×10^{-6} for at least one scenario are listed in this table.

cPAH – carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
na – not available
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
SWAC – spatially weighted average concentration

All seafood consumption scenarios evaluated were associated with upper bound excess cancer risk estimates that exceeded 1×10^{-6}. The highest excess cancer risk and non-cancer hazard estimates were calculated for the seafood consumption scenarios, which were dominated by PCBs (either total or TEQ) in all seafood sources, and arsenic in clams. For the adult tribal RME scenario based on Tulalip data, overall total PCB risks were 2×10^{-3} while PCB TEQ risks were 1×10^{-3}. Risk estimates for arsenic presented in this summary do not include consideration of the contribution from background sources, which is discussed in Section B.5.5.1.2. Site-related arsenic seafood consumption risks were slightly less than the risk values presented in Table B.5-64 for most scenarios. Excess cancer risk estimates for all chemicals combined range from a low of 2×10^{-5} for the adult API CT scenario to a high of 3×10^{-2} for the adult tribal scenario based on Suquamish data (Table B.5-64).

All RME and CT exposure scenarios for seafood consumption are associated with HIs greater than 1. HQ estimates were highest for PCBs and arsenic, although HQs were greater than 1 for two other chemicals (vanadium and TBT) for the child tribal RME scenario based on Tulalip data and the adult tribal scenario based on Suquamish data. For the adult tribal scenario based only on Suquamish data, HQs were greater than 1 for two additional chemicals (mercury and chromium (Table B.5-65). Although these metals were not identified as COCs for seafood consumption (see Section B.7), they were identified as COCs for benthic invertebrates and spotted sandpiper in the ERA (see Appendix A of the RI). Thus, although mercury and chromium are not designated as COCs for the HHRA, they will be considered COCs for the overall FS, including considerations noted in Section B.7. HQs for PCBs ranged from 1 for the one-meal-per-month crab consumption scenario to 274 for the adult tribal scenario based on Suquamish data. Four of the eleven arsenic HQs presented in Table B.5-65 were greater than 1 (adult tribal RME scenario based on Tulalip data, child tribal RME based on Tulalip data, adult API RME, and adult tribal based on Suquamish data), ranging from 3 for the adult tribal RME scenario based on Tulalip data and the adult API RME scenario to 38 for the adult tribal scenario based on Suquamish data.

Excess cancer risks for the direct sediment exposure pathways were much lower than the cancer risks for seafood consumption scenarios, although all but one (7-day-per-year clamming scenario) were associated with upper-bound excess cancer risk estimates that exceeded 1×10^{-6}. Total excess cancer risks were 5×10^{-6} or 6×10^{-6} for the netfishing CT scenario (excluding PCB TEQ and total PCBs, respectively) and 3×10^{-5} for the netfishing RME scenario. Total excess cancer risks ranged from 5×10^{-6} to 5×10^{-5} for the eight beach play RME areas. Total excess cancer risks were 1×10^{-6} for the 7-day-per-year clamming scenario, 1×10^{-4} or 2×10^{-4} for the tribal clamming RME scenario (excluding PCB TEQ and total PCBs, respectively), and 2×10^{-4} or 3×10^{-4} for the tribal clamming 183-day-per-year scenario (excluding PCB TEQ and total PCBs, respectively) (Table B.5-66). No direct sediment exposure scenarios had

HQs for individual chemicals greater than 1 or generated effect-specific HIs in excess of 1, so those scenarios are not included in this summary.

These risk summaries indicate that risks associated with the seafood consumption scenarios are higher than the risks associated with the direct sediment exposure scenarios. Table B.5-67 shows the relative contribution of different chemicals to excess cancer risk estimates for all seafood consumption scenarios. This type of assessment was not done for non-cancer hazards because HQs are not directly additive across endpoints, and therefore the contribution of different chemicals cannot be characterized as fractions of overall hazard. The overwhelming majority of the non-cancer hazards associated with seafood consumption were contributed by total PCBs (> 80% of the total developmental, neurological, and immunological hazard indices). The total PCB HQ for all seafood consumption scenarios, except the adult one-meal-per-month crab scenario, exceeded 1.

The relative excess cancer risks are illustrated in Figure B.5-2 using pie charts based on the average percent contribution of PCBs, arsenic, cPAHs, and other chemicals for both adult (all adult scenarios that included a market basket of seafood categories) and child seafood consumption scenarios (child tribal CT and RME scenarios based on Tulalip data). These three chemicals were selected for further examination because they had excess cancer risks greater than 1×10^{-6} and made up more than 5% of the total excess cancer risk for at least one scenario.

Overall, PCBs (as total PCBs or PCB TEQ) and arsenic were the greatest contributors to excess cancer risk estimates. Carcinogenic PAHs contributed to a much lesser degree to the overall cancer risk estimate, between 2 and 3% for adult scenarios and between 10 and 12% for child scenarios. The greater percentage of overall risk contributed by cPAHs in children relative to adults is a result of EPA risk assessment procedures that treat children as being more sensitive to cPAHs than adults (see Section B.5.1.1). Chemicals in the "other chemicals" group were mainly JN-qualified pesticides. Because tissue data for dioxins/furans were not collected, their contribution to excess cancer risk is not reflected in the percentage risk by chemical descriptions.

Table B.5-67. Risks by chemical for adult and child seafood consumption scenarios

Chemical	Adult Tribal RME (Tulalip Data)	Adult Tribal CT (Tulalip Data)	Child Tribal RME (Tulalip Data)	Child Tribal CT (Tulalip Data)	Adult Tribal (Suquamish data)	Adult API RME	Adult API CT
Percentage contribution to overall excess cancer risk (excluding PCB TEQ)							
Arsenic	41%	44%	38%	39%	54%	50%	55%
cPAHs	2%	3%	10%	12%	3%	3%	3%
Dioxin/furan TEQ[b]	na	na	na	na	na	na	na
Total PCBs	45%	45%	41%	42%	35%	38%	36%
Other chemicals[c]	12%	8%	11%	7%	8%	9%	6%
Percentage contribution to overall excess cancer risk (excluding total PCBs)							
Arsenic	46%	47%	42%	41%	61%	55%	57%
cPAHs	2%	3%	11%	13%	3%	3%	4%
Dioxin/furans TEQ[b]	na	na	na	na	na	na	na
PCB TEQ	38%	42%	35%	38%	27%	32%	33%
Other chemicals[c]	13%	8%	12%	8%	10%	10%	6%

[a] Includes all other detected tissue COPCs.

[b] Tissue data for dioxins/furans was were not collected, and thus their contribution to excess cancer risk is not reflected here.

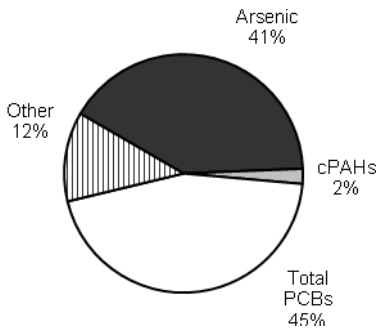
API – Asian and Pacific Islander

CT – central tendency

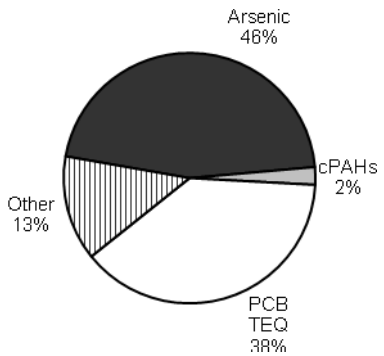
cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

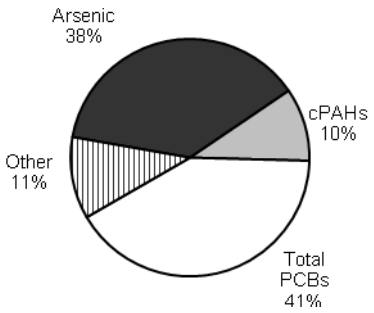
RME – reasonable maximum exposure



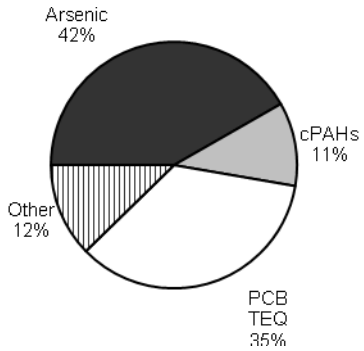
a) Total cancer risk (excluding PCB TEQ) for adult tribal RME seafood consumption scenario based on Tulalip data



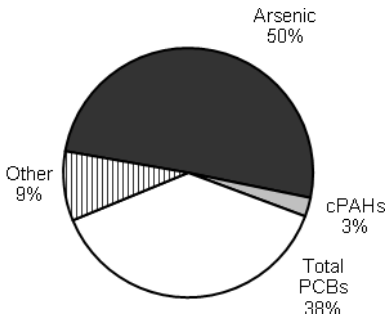
b) Total cancer risk (excluding total PCBs) for adult tribal RME seafood consumption scenario based on Tulalip data



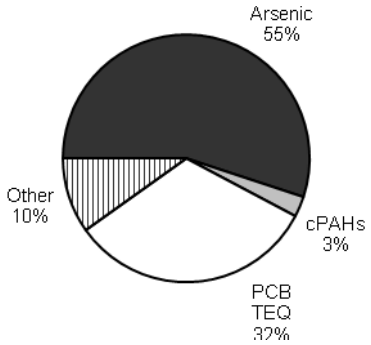
c) Total cancer risk (excluding PCB TEQ) for child tribal RME seafood consumption scenario based on Tulalip data



d) Total cancer risk (excluding total PCBs) for child tribal RME seafood consumption scenario based on Tulalip data



e) Total cancer risk (excluding PCB TEQ) for adult API RME seafood consumption scenario



f) Total cancer risk (excluding total PCBs) for adult API RME seafood consumption scenario

Figure B.5-2. RME seafood consumption scenario risks by chemical

A similar evaluation of risk contribution was made for the direct sediment exposure scenarios (Table B.5-68 and Figure B.5-3). Figure B.5-3 shows both the risk magnitude and the percent risk by chemical for each exposure scenario. Arsenic, cPAHs, dioxin/furans, and PCBs were evaluated because these chemicals had excess cancer risks greater than 1×10^{-6} and made up greater than 5% of the total excess cancer risk for at least one scenario. In addition, these chemicals were detected in greater than 10% of LDW sediment samples.

The results were quite different between the adult (i.e., netfishing and clamming) and child (i.e., beach play RME) scenarios. The percent contribution by chemical was fairly consistent across the five adult sediment exposure scenarios. Dioxin/furan risks contributed the majority (61 to 79%) of the risks associated with the adult sediment scenarios, followed by arsenic (12% to 23%) (Figure B.5-3). For the beach play RME scenarios, the percent contribution by chemical was highly variable. Dioxin/furans were much less important to the overall risk estimate , primarily because there were far fewer data in those areas and the highest dioxin/furan concentrations from the LDW were not in beach play areas (Figure B.5-3). The major contributors to beach play RME risk estimates were cPAHs (19% to 88%) and arsenic (12% to 77%). Total PCBs generally contributed 10% or less of the total cancer risk for a given adult or child direct sediment exposure scenario.

Table B.5-68. Risks by chemical for adult and child direct sediment exposure scenarios

Chemical	Netfishing		Clamming			Beach Play RME							
	RME	CT	Tribal 183 Days per Year	Tribal RME	7 Days per Year	Area 1	Area 2	Area 3	Area 4	Area 5	Area 6	Area 7	Area 8
Arsenic	19%	17%	12%	14%	23%	33%	15%	12%	14%	36%	35%	77%	42%
cPAHs	3%	4%	3%	4%	8%	66%	85%	88%	28%	60%	59%	19%	56%
Dioxins/furans	65%	69%	78%	72%	61%	na	na	na	35%	1%	na	1%	na
Total PCBs	6%	5%	4%	6%	7%	0%	0%	0%	21%	1%	6%	2%	1%
Other chemicals[a]	7%	5%	3%	4%	2%	0%	0%	0%	1%	1%	0%	0%	0%

[a] Includes total DDTs, dieldrin, and toxaphene.

CT – central tendency

na – not available

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

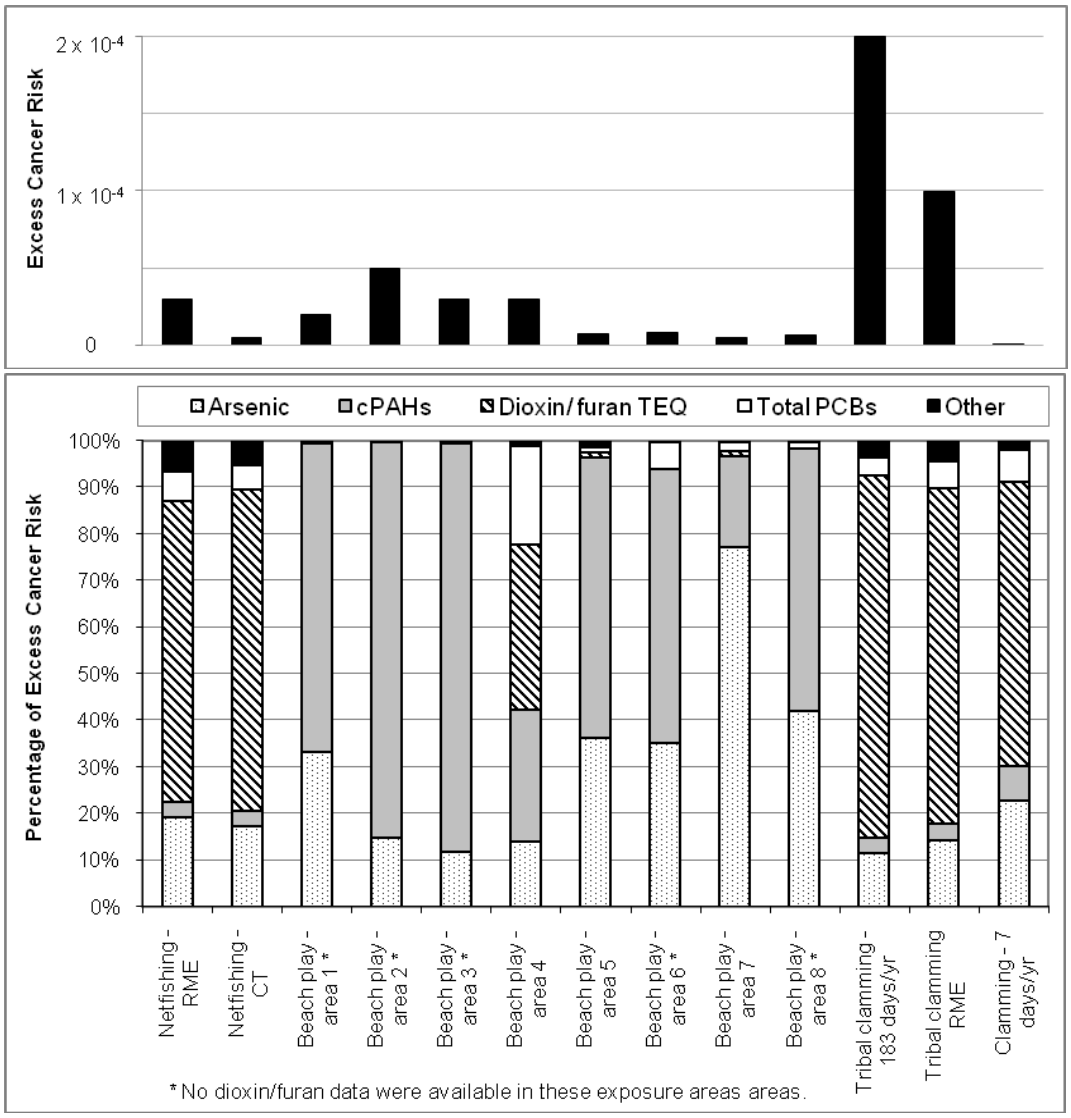


Figure B.5-3. Cancer risks by chemical for direct sediment exposure scenarios

As discussed in Section B.5.2, risks for multiple scenarios are summed to represent possible exposure of the same individuals to LDW chemicals from different activities. Summed risks are presented in Table B.5-69 for the combination of adult tribal RME netfishing, seafood consumption based on Tulalip data, and swimming;[32] child tribal RME seafood consumption based on Tulalip data, beach play RME, and swimming; adult recreational clamming, clam consumption, and swimming; and tribal RME clamming, swimming, and adult seafood consumption. Note that although some individuals might engage in both netfishing and clamming, risks for these two scenarios were not summed. This is because given the high frequency assumed for both activities, engaging in both with the assumed frequency (over 100 days per year) is unlikely. The sum of excess cancer risk estimates for each of these three sets of scenarios in Table B.5-69 is the same as the estimates for their seafood consumption components alone after rounding to one significant figure, as recommended by EPA (1989). This analysis demonstrates that the contributions of netfishing, clamming, beach play RME, and swimming are relatively small in comparison to seafood consumption risk estimates, and highlights the significance of the seafood consumption exposure pathways for all users of the LDW. Summing child beach play RME and swimming increased the risk estimate only slightly over that of beach play RME alone. Overall, swimming was the least important exposure pathway.

Table B.5-69. Excess cancer risk estimates across related scenarios

Activity	Excess Cancer Risk[a]
Adult Tulalip scenarios	
Netfishing RME[b]	3×10^{-5}
Swimming[c]	$< 1 \times 10^{-6}$
Adult tribal RME seafood consumption based on Tulalip data	3×10^{-3}
Total	**3×10^{-3}**
Child scenarios[d]	
Beach play RME – Area 2[e]	5×10^{-5}
Swimming[c]	$< 1 \times 10^{-6}$
Subtotal for beach play RME and swimming	**5×10^{-5}**
Child tribal RME seafood consumption based on Tulalip data	8×10^{-4}
Total	**9×10^{-4}**
Adult low-end clamming scenarios	
Clamming – 7 days per year[b]	1×10^{-6}
Swimming[c]	$< 1 \times 10^{-6}$
Clam consumption – one meal per month	2×10^{-4}
Total	**2×10^{-4}**

[32] Risks associated with exposure to surface water while swimming were estimated previously by King County (1999d) and are incorporated in this HHRA by reference.

ACTIVITY	EXCESS CANCER RISK[a]
Adult RME clamming scenarios	
Tribal clamming RME (120 days per year)	1×10^{-4}
Swimming[c]	$< 1 \times 10^{-6}$
Adult tribal RME seafood consumption based on Tulalip data	3×10^{-3}
Total	$\mathbf{3 \times 10^{-3}}$

[a] All non-swimming risk estimates are from this document. Total excess cancer risk estimates excluding PCB TEQ were used because these were equal to or higher than total excess cancer risk estimates excluding total PCBs, except for the tribal clamming RME scenario, where total risk excluding total PCBs was higher. Thus for the tribal clamming RME scenario, the total risk excluding total PCBs is shown here.

[b] The RME netfishing scenario, rather than the CT netfishing scenario, was used to account for the fact that tribal members may engage in RME seafood consumption and RME netfishing practices simultaneously.

[c] Adult and child swimming risk estimates as reported by King County for Elliott Bay and the Duwamish River for medium exposure assumptions (12 events per year for adults or children aged 1 to 6) (King County 1999b). Exposure pathways consist of dermal contact and incidental sediment ingestion of water during swimming. Risks were estimated based on total PCB concentrations of 14.4 ng/L in the LDW originally modeled by King County (King County 1999b). PCB congener data from samples collected from the LDW by King County in 2005 indicate the previous modeled estimate is an overestimate of the highest empirical total PCB concentrations, which were no greater than 3.14 ng/L during low-flow sampling conducted in August 2005 (Mickelson and Williston 2006). These results indicate that the risk estimates for the swimming scenario presented by King County in the water quality assessment (King County 1999b) are also overestimated.

[d] The child scenario total includes the child tribal seafood consumption scenario based on Tulalip data (calculated as 40% of total adult consumption), which is considered protective of non-tribal children.

[e] Beach Play RME Area 2 is included because it had the highest risk estimate of the beach play RME scenarios.

RME – reasonable maximum exposure

All the risk estimates associated with the seafood consumption scenarios are highly sensitive to consumption rate assumptions. The consumption rates used for RME scenarios were intended to reflect the 95th percentile of consumption. However, although the consumption rates used for these risk estimates are based on recent consumption studies and direction from EPA (2007b), there is uncertainty related to the application of these rates to groups using the LDW. For example, EPA (2007b) states, "The use of consumption rates of Puget Sound-harvested fish and shellfish derived using Tulalip and Suquamish Tribal data as a surrogate for another Tribe in Puget Sound or the Strait of Georgia could lead to either an overestimate or an underestimate of the actual fish and shellfish consumption rate potentially associated with site releases." Risk estimates would change if consumption rate assumptions were substantially different. To illustrate the relationship between the risk estimates and consumption rates, figures were created to show the excess cancer risk estimates for PCB TEQ and total PCBs across a continuum of consumption rates for the different seafood consumption scenarios. Figure B.5-4 shows the PCB TEQ risks for the RME and CT scenarios, and Figure B.5-5 shows the total PCB risks for the RME and CT scenarios. These figures demonstrate the direct correlation between changes in assumptions of overall consumption rate (assuming the same proportional consumption of different species) and risk estimates for all seafood consumption scenarios.

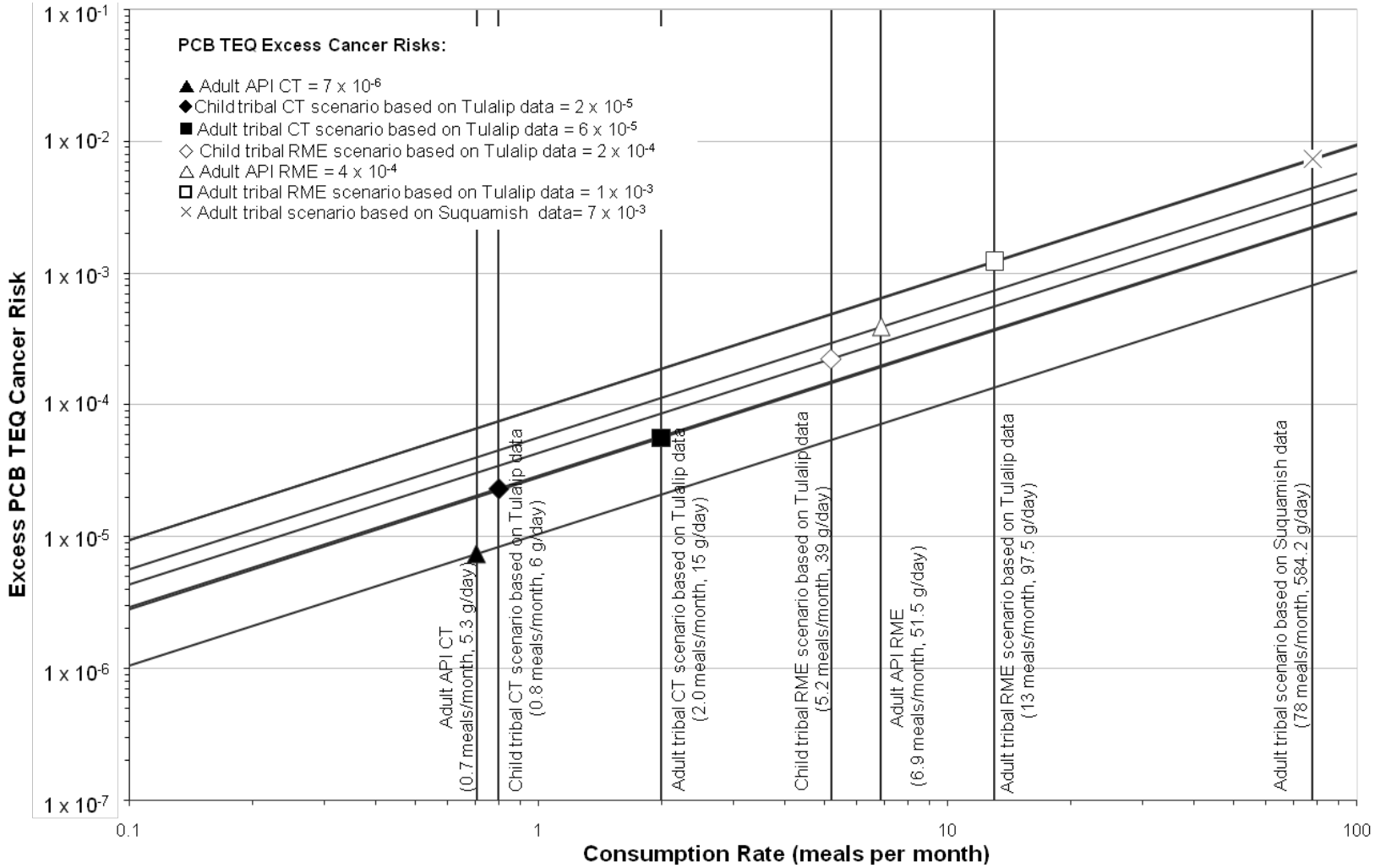


Figure B.5-4. Excess cancer risks from PCB TEQ for seafood consumption scenarios and rates

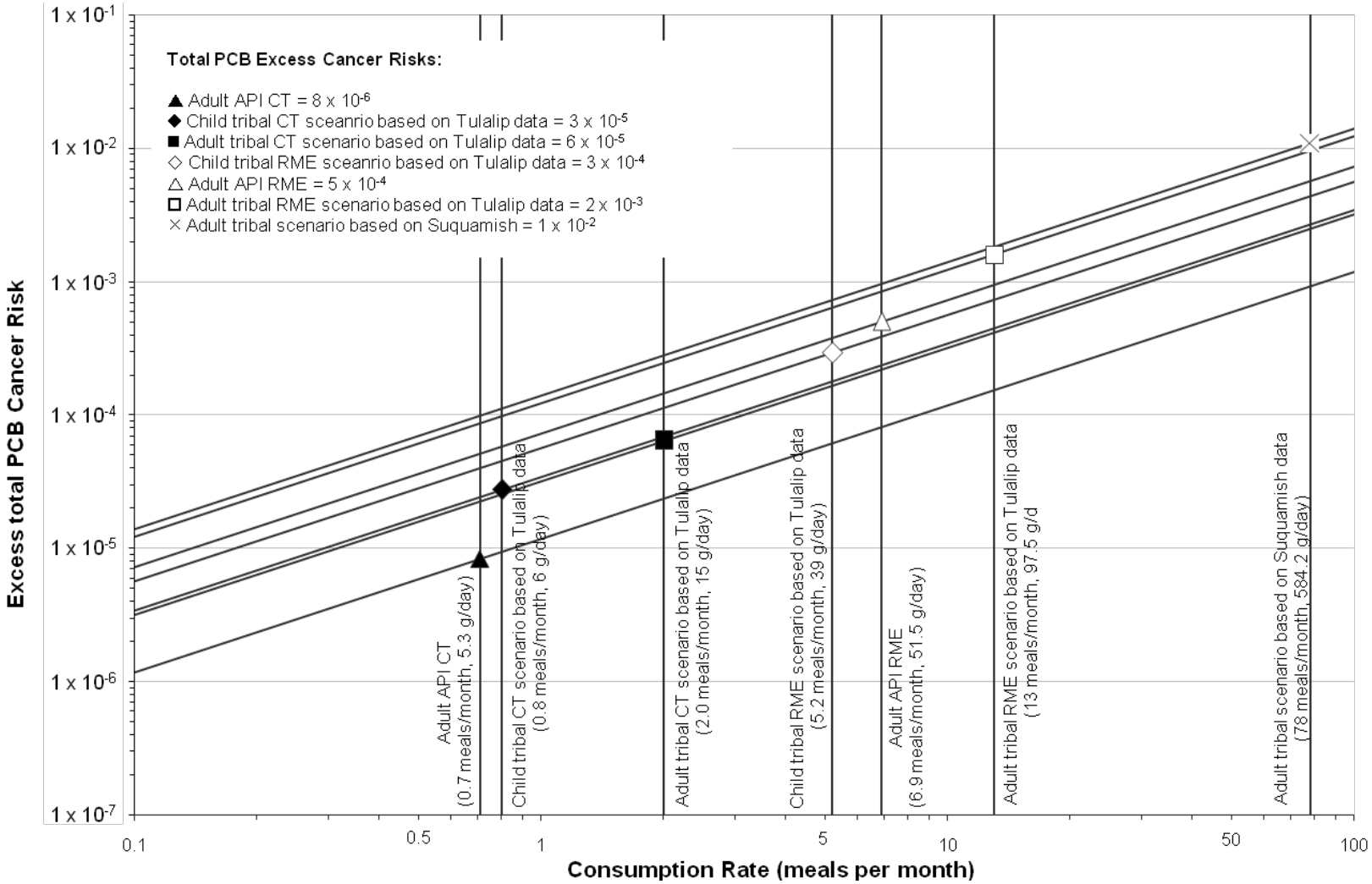


Figure B.5-5. Excess cancer risks from total PCBs for seafood consumption scenarios and rates

B.6 Uncertainty Analysis

There is a degree of uncertainty in any quantitative risk assessment. The exposure and toxicity assumptions used for this risk assessment, which were based on EPA guidance (incorporating policy decisions), current scientific literature, and best scientific judgment, are inherently uncertain. Therefore, the resulting risk estimates carry a degree of uncertainty. This section discusses some of the key uncertainties in this risk assessment and presents alternative risk estimates for many of the exposure scenarios based on different hypothetical exposure or toxicity assumptions.

Table B.6-1 lists some of the key uncertainties in this baseline HHRA. Each uncertainty is characterized qualitatively as low, medium, or high (see table footnotes). Table B.6-1 also characterizes each uncertainty by the impact of additional information or an alternative analysis on the characterization of risk, and whether risk estimates included in the risk characterization section are likely to be underestimates or overestimates.

The uncertainties discussed in this section are grouped by exposure assessment (Section B.6.1), toxicity assessment (Section B.6.2), and risk characterization (Section B.6.3).

Table B.6-1. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Exposure Assessment (B.6.1)					
Exposure point concentrations (B.6.1.1)					
Inclusion of undetected chemicals (B.6.1.1.1)	low	May have no effect or may overestimate risk if chemicals are not present.	Achieve lower reporting limits.	low for total risk estimates	Risk estimates for chemicals never detected are presented in B.6.3.2.
Statistical approach to evaluating datasets with undetected concentrations (B.6.1.1.2)	low	Risk estimates for chemicals with EPCs based on a single detection and multiple undetected concentrations are likely to be overestimated.	More detailed statistical assessment of data for all COPCs with high frequency of undetected concentrations.	low for total risk estimates	Chemicals affected are minor contributors to total risks. Preliminary explorations of alternative approaches for undetected concentrations indicates initial estimates in risk characterization are reasonable.
Infrequently detected organochlorine pesticides in tissue, tentatively identified and JN-qualified (B.6.1.1.3)	high	High RLs for undetected concentrations may lead to overestimation of risk if these compounds are not present. Reanalysis of pesticide results to account for PCB interferences suggests that pesticide risks are lower.	Conduct separate analyses using undetected and detected concentrations. Conduct chemical analyses that are not impacted by interference of PCBs and can achieve lower detection limits.	low because these risks are much lower than other risk estimates	Although not risk drivers, most organochlorine pesticides have excess cancer risk estimates, based on tentative analytical results, that are greater than 1×10^{-6} for all seafood consumption scenarios.
Bis(2-ethylhexyl) phthalate and pentachlorophenol tissue data (B.6.1.1.4)	medium	High RLs for undetected concentrations may lead to overestimation of risk if these compounds are not present.	Collect more tissue data with lower RLs. Based on lower RLs, pentachlorophenol risks are potentially 100-fold lower that the original risks calculated. Bis(2-ethylhexyl) phthalate risks are very likely to be lower, but further analysis would be required to substantiate this.	medium	Some samples were reanalyzed with more sensitive methods resulting in undetected concentrations with lower RLs.
Carcinogenic PAHs in tissue (B.6.1.1.5)	medium	High RLs for undetected concentrations in historical dataset may lead to overestimation of risk. Some other PAHs that may contribute to carcinogenicity were not analyzed. This could contribute to underestimation of risk.	Conduct separate analyses with prior high RL data excluded and included. Analyze tissue for additional PAHs.	medium	Risk estimates using only 2004 LDWG tissue data with lower RLs are an order of magnitude lower than estimates using both 2004 data and non LDWG data but still exceed 1×10^{-6} excess cancer risk for all multi-species scenarios. Impact of unanalyzed PAHs is unknown.

Table B.6-1, cont. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Lack of tissue chemistry data for dioxins and furans and lack of sediment chemistry data for some beach play areas (B.6.1.1.6)	medium	Cannot be accurately estimated without site-specific data.	Collect tissue chemistry data for dioxins and furans. Collect sediment chemistry for beach play areas where data are lacking.	medium	LDWG, EPA, and Ecology agreed that tissue samples would not be analyzed for dioxins and furans because remedial decisions could be made based only on sediment chemistry data.
PCB sediment analytical methods (B.6.1.1.7)	medium	Inclusion of NOAA HPLC/PDA data for PCBs slightly alters risk estimates because the PCB concentrations differ from those based on the standard GC/ECD method.	Exclude NOAA data.	low	Existing PCB data from sources other than NOAA suggest risks from direct exposure to sediment-associated PCBs are below EPA's excess cancer risk threshold of 10^{-4}.
Calculation methods for total PCBs (B.6.1.1.8)	low	Alternative calculation methods could slightly increase the total PCB concentrations.	Compare results from multiple calculation methods.	low	Calculation method used in baseline HHRA is specified in Washington Sediment Management Standards.
PCB EPCs for benthic fish fillets (B.6.1.1.9)	low	Alternative methods to calculate EPCs integrating unequal numbers of benthic fish fillet samples from different areas of the LDW could lead to different estimates of the EPC and risks associated with PCBs in benthic fish fillets.	Compare results from multiple calculation methods.	low	EPCs calculated using alternative methods differed only slightly and did not alter excess cancer risk estimates for total PCBs.
EPCs for infrequently detected chemicals (B.6.1.1.10)	medium	Alternative methods to estimate EPCs for datasets with fewer than six detected concentrations might lead to different EPCs and risk estimates.	Compare results from multiple calculation methods.	low	EPCs calculated with alternative methods significantly affected risk estimates for some chemicals, but most of the chemicals affected are minor contributors to overall risk estimates.
EPCs for small datasets (B.6.1.1.11)	medium	Unlike datasets that were statistically analyzed (i.e., those with six or more samples that were evaluated using ProUCL 4), UCLs on estimates of the mean for datasets of five or fewer samples were not calculated. Therefore, uncertainty related to potential EPC underestimation is greater for EPCs derived for small datasets with fewer than six samples, for which ProUCL 4 was not used. Chemical concentration data are often positively skewed. For small, positively skewed datasets, the true mean may exceed the highest individual sample result. Hence, the selected EPC, based on the maximum concentration, may underestimate the mean.	Collect additional data for datasets with small sample sizes (n = 5 or fewer).	low	The vast majority of tissue, clamming, and netfishing EPCs were developed from datasets with six or more samples. However, small sample sizes affectedEPC development for several of the beach play areas (which included the smallest geographic areas evaluated for direct contact exposure).

Table B.6-1, cont. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Spatial bias in EPC estimates for PCBs (B.6.1.1.12)	high	The arithmetic EPCs calculated for total PCBs in the netfishing and tribal RME scenarios are known to be overestimated because of the spatial bias of the underlying data.	A spatially weighted approach for calculating a UCL would account for the spatial bias and would better represent the potential exposure. Agreed upon methods to verify that SWAC UCLs provide true 95% UCLs are lacking.	high	The SWAC UCL evaluated in this section was approximately five-fold lower than the arithmetic UCLs for total PCBs.
Ingestion rates (B.6.1.2)					
Incidental sediment ingestion rates (B.6.1.2.1)	high	The applicability of incidental soil ingestion rates from EPA guidance to sediment exposure scenarios is unknown.	Compare sediment exposure behaviors to behaviors assumed for EPA default sediment ingestion rates.	unknown	Ingestion rates would be very difficult to measure, so assumed rate is based largely on best professional judgment.
Adult seafood Consumption rates (B.6.1.2.2)	high	Site use is greatly overestimated for tribal populations for current conditions. The degree of overestimation for tribal populations under future conditions is uncertain but likely lower than the degree of overestimation under current conditions. API community members harvest fish from the LDW, but it is uncertain to what degree consumption rates from EPA's 1999 API study overestimate LDW-specific API consumption rates.	Collect additional data that reflects LDW resource use by different populations in urban watersheds that have similar habitat to the LDW but do not have substantial chemical contamination (assuming any such watersheds could be found), which could then be used to draw conclusions about resource use within the LDW.	high	Although site use may increase in the future, the degree of future use assumed in this assessment may overestimate risks for most users.
Exclusion of salmon from overall seafood consumption rate (B.6.1.2.2)	low	Overall risk estimate based on resident fish and shellfish is only slightly underestimated by the exclusion of salmon.	Include salmon in consumption rate and risk estimate; applicability to LDW sediment-related exposures is uncertain.	low	EPA has excluded salmon from the seafood consumption rate to be used for tribal and API seafood consumption risk assessments for bioaccumulative chemicals because LDW sediment-related exposures are likely insignificant compared to exposures that are not site-related (Kissinger 2005; EPA 2007b).

Table B.6-1, cont. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Tribal child seafood consumption rates (B.6.1.2.3)	high	Children's tribal fish consumption rates are generally less well characterized than adult rates. Despite this fact, use of the child-to-adult ratio approach based on the Tulalip data yields a consumption rate that is consistent with other upper percentile children's consumption rates from available studies.	Conduct better tribal children's seafood consumption surveys to support consumption rates.	high	Uncertainties for the adult consumption rates also apply to the child rates with additional uncertainty related to the fact that data are more limited for children. The sample size for the Tulalip Tribes' study is small, and multiple children were sampled from the same households in the Suquamish study.
Fraction of dose obtained from LDW (B.6.1.3)	high	For most individuals, the fraction of Puget Sound- or King County-harvested fish and shellfish intake obtained from the LDW is likely to be moderately to greatly overestimated. This is particularly true for the RME, CT, and adult tribal consumption based on Suquamish data seafood consumption scenarios, because all consumption is assumed to occur from the LDW. There may be only a very small population that currently practices subsistence seafood harvest from the LDW. The representativeness for future-use scenario is unknown. For the beach play and clamming scenarios, the frequency of exposure and therefore intake from the site (as opposed to other locations that have been surveyed) is unknown.	Collect additional data that reflects site-specific usage and habitat suitability in urban watersheds that have habitat similar to that of the LDW but do not have substantial chemical contamination (assuming any such watersheds could be found), which could then be used to draw conclusions about resource use within the LDW.	high	Default assumption of 1 applied as required by EPA because of a lack of site-specific data. Alternative assumptions of site use would still indicate excess cancer risk estimates greater than 1×10^{-6} for most seafood consumption scenarios.
Exposure duration for API seafood consumption scenario (B.6.1.4)	medium	Exposure duration (ED) for API utilizing the LDW as their primary or exclusive fishing location is unknown. In the risk characterization, ED was assumed to be 30 years based on EPA's assessment of the 90th percentile for residence time in the United States.	Perform survey of exposure duration for API who utilize the LDW as their primary or exclusive fishing location. Estimate risks using alternative assumptions of exposure duration.	low	No site-specific survey data were available. An alternative ED assumption based on EPA's assessment of national data was used to estimate risk.
Dermal exposure (B.6.1.5)					
Chemicals lacking guidance on absorption factors (B.6.1.5.1)	medium	Underestimation of dermal risks from metals lacking absorption factors expected to have small effect on overall risk estimates.	Evaluate risk estimates using a range of absorption assumptions	low	Dermal absorption dependent on speciation of metals, but is typically low. Calculations in this uncertainty assessment where absorption was assumed to be 0.001 to 0.03 (the range of available for metals), indicate this pathway likely does not contribute significantly to underestimation.

Table B.6-1, cont. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Dermal adherence factors used for beach play and clamming scenarios (B.6.1.5.2)	medium	May lead to low over- or underestimate for netfishing and clamming scenario, moderate to significant increases with higher adherence factors for beach play scenarios.	Evaluate risk estimates using a range of adherence assumptions	low (netfishing and clamming); medium (beach play RME).	Adherence factor assumptions for beach play are most uncertain and have greatest impact on risk estimates.
Representativeness of fish and shellfish COPC data for all potentially exposed populations (B.6.1.6)	medium	Unknown	Collect additional data for different tissue types and analyze for additional chemicals; consider preparation practices in risk calculations.	low	Changes in chemical concentration resulting from preparation and cooking were not considered. Food preparation and cooking practices may reduce or increase risks. Given the wide range of cooking practices, it is health-protective not to adjust tissue concentrations for cooking and preparation. Although not all tissue samples were analyzed for all chemicals, particularly dioxins/furans (which were not analyzed in any tissue samples) and certain cPAHs, the relatively large tissue database used in the HHRA should reasonably approximate the range of chemical concentrations to which seafood consumers might be exposed.
Spatial coverage of sediment chemistry data (B.6.1.7)	low to medium	Low for most chemicals; unknown for dioxin/furan TEQ. Data for some assumed beach play areas are limited to a few samples.	Research past industrial activities to determine if likely chemical sources have been adequately characterized.	unknown	Available information does not suggest there are large sources that have not been characterized, but some minor gaps in spatial coverage may exist for specific exposure areas.
Spatially weighted EPC estimate (B.6.1.7)	low to high	Low for chemicals that have not been the focus of spatially biased sampling (i.e., sampling was not targeted in areas of suspected contamination); high for chemicals that have been, such as PCBs.	Calculate SWACs for every COPC.	low to high	The level of effort for calculating SWACs for many COPCs is not warranted given the relatively low magnitude of the risk estimates for those COPCs.
Temporal variability in chemistry data (B.6.1.8)	low to high, depending on exposure area	Concentrations from more recent sediment samples may be lower or higher than concentrations in samples taken previously from the same general location. Most of the tissue data are from 2004-2005, so temporal variability in tissue data has only a minor impact on risk estimate.	Temporal variability in sediment chemistry data will be evaluated more thoroughly in the RI.	unknown (sediment) to low (tissue)	Decision to include only valid data collected since 1990 as a single dataset was made early in the project. Temporal relationships with earlier datasets are not evaluated.

Table B.6-1, cont. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Health-protectiveness of sediment exposure scenarios (B.6.1.9)	low	Risk estimates for sediment exposure scenarios may be overestimated for current conditions, especially for beach play and clamming scenarios.	Separating current from future scenarios could provide more realistic exposure conditions for current conditions.	A current exposure scenario would likely yield lower risk estimates.	The current frequency of sediment contact activities has not been well quantified. However, reasonable future exposure must be considered in CERCLA risk assessments. Quantifying future exposure frequencies is very difficult and highly uncertain.
Toxicity Assessment (B.6.2)					
Toxicity assessment for chemicals lacking toxicity values (B.6.2.1)	low	For chemicals detected in tissue with provisional toxicity values but lacking RBCs, RBCs were developed and tissue chemistry data were screened against these values. For chemicals lacking any toxicity benchmarks risks could be underestimated to unknown degree.	unknown	medium	Evaluation using RBCs developed for chemicals with provisional toxicity values indicated that risk contribution from those chemicals would be minimal. Risks were not estimated for the chemicals that lack toxicity values (i.e., RfDs or SFs).
Total PCBs (B.6.2.2)	medium	Moderately overestimated based on selection of PCB SF. However, in other settings, bioaccumulation and environmental weathering have been demonstrated to alter the components of PCB mixtures, which could contribute to greater toxicity of the mixture compared to commercial PCB mixtures.	unknown	low	Environmental mixtures of PCBs differ from Aroclor formulations. Most-health-protective SF derived based on Aroclors 1254 and 1260 probably not representative of the toxicity of all PCB Aroclors and may overestimate carcinogenicity of lower chlorinated Aroclors. However, because Aroclors detected are predominantly the more highly chlorinated Aroclors, uncertainty is low.
PCB TEQ (B.6.2.3)	medium	Unknown	unknown	high	PCB TEFs used to calculate PCB TEQ based primarily on structure activity relationships rather than direct toxicity data. PCB TEQ excess cancer risk evaluation requires use of dioxin SF that is highly uncertain and undergoing review
TEQ approach for sediment (B.6.2.3)	high	Approach likely overestimates the bioavailability of PCB, dioxin, and furan congeners that tend to bind tightly to sediment particles.	Obtain congener-specific bioavailability estimates to adjust the TEFs.	high	The TEQ approach is most appropriately applied to tissue matrices; bioavailability estimates for sediment TEQ would be uncertain.
Chromium speciation (B.6.2.4)	medium	Moderately overestimated because the RfD for hexavalent chromium (the most toxic species) is used for total chromium.	Collect additional data on chromium species present in sediment and tissue.	low	Chromium risks do not exceed acceptable risk levels even with this conservative risk assumption

Table B.6-1, cont. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Mercury speciation (B.6.2.5)	medium	Moderately overestimated because RfD for methyl mercury (the most toxic form) is used for total mercury. Assumption may be more reasonable for fish tissue than sediment	Collect additional data on mercury species present in sediment and tissue.	low	Mercury risks do not exceed acceptable risk levels for any RME seafood consumption scenarios (the adult tribal scenario based on Suquamish data has an HQ of 2) or any sediment scenarios, even with this conservative risk assumption
Risk Characterization (B.6.3)					
Inclusion of PCBs in estimates of total excess cancer risk (B.6.3.1)	medium	Reporting totals with only total PCBs or only PCB TEQ may underestimate total excess cancer risk.	Develop an adjustment process to avoid double-counting when summing total PCB and PCB TEQ risks.	high	The approach in this HHRA has been used at other Superfund sites, although other approaches have been provided in EPA guidance and used in other risk assessments. Risk characterization of environmental PCB mixtures using toxicity estimates derived for commercial PCB mixtures is problematic.
Risk calculations for undetected chemicals (B.6.3.2)	medium	Greatly overestimated if undetected COPCs are not present; uncertain if these COPCs are present at concentrations below the RLs.	Collect additional data with lower RLs, if analytically possible. Conduct risk calculations using RL data to bound potential risks.	low	Many of the chemicals that were never detected have no known LDW source, so lower RLs may not be helpful. Calculations conducted assuming undetected chemicals were present at the RL resulted in relatively low estimates for all but a few undetected chemicals that had high RLs
Risk calculations for discrete areas within the LDW (B.6.3.3)					
Applicability of LDW seafood consumption risk calculations for smaller exposure areas within the LDW (B.6.3.3.1)	medium	May overestimate risk if site use for selected area is overestimated or if harvesting in smaller area is unsustainable.	Evaluate risks for smaller exposure areas within the LDW.	low	Seafood consumption risk estimates for three of four areas in the LDW were generally lower than those for the LDW-wide area.
Smaller spatial scale assessment of sediment beach play exposure scenario (B.6.3.3.2)	low	Risk estimates for the beach play areas may be applicable to various spatial scales.	Additional risk estimates for smaller spatial scales.	low	COPC concentrations in most beach play areas appear to be relatively low compared to other areas evaluated.
Evaluation of smaller spatial scales using screening method for habitat biologist scenario (B.6.3.3.3)	low	Point risk estimates are greatly overestimated compared to more realistic exposure areas.	Develop exposure estimates based on planned or hypothetical restoration areas.	medium	Development of hypothetical restoration areas would require considerable professional judgment.

Table B.6-1, cont. Summary of uncertainties identified in the baseline HHRA

Parameter	Level of Uncertainty[a]	Effect of Uncertainty on Risk Estimate	Potential Means to Reduce Uncertainty	Potential Impact on Risk Estimates[b]	Comment
Evaluation of smaller spatial scales using screening method for netfishing scenario (B.6.3.3.3)	low	Point risk estimates are greatly overestimated compared to more realistic exposure areas.	Develop exposure estimates based on actual spatial extent of netfishing by specific individuals.	medium	Data are not available from the Muckleshoot Tribe that would allow derivation of smaller netfishing exposure areas.
Arsenic risks attributed to clam consumption (B.6.3.4)	high	Current risk estimates are based on only eight composite clam tissue samples. The inorganic arsenic concentrations from LDW samples are higher than those obtained elsewhere.	Collect additional inorganic arsenic data from LDW clams.	medium	Although there are various hypotheses to explain the inorganic arsenic concentrations found in LDW clams, much of the data needed to evaluate these hypotheses do not exist.

[a] Level of uncertainty: low = large and relevant dataset; medium = small dataset or limited information; high = very limited data or no site-specific information.

[b] Potential impact: low = additional data or analysis unlikely to result in a change in determination of whether a chemical exceeds acceptable risk levels or (i.e., HQ greater than 1 or cumulative excess cancer risk greater than 1 × 10-6) or identification of a pathway of concern; medium = additional data or analysis could result in a change in determination of whether a chemical exceeds acceptable risk levels or identification of a pathway of concern; high = additional data or analysis likely to result in a change in determination of whether a chemical exceeds acceptable risk levels or identification of a pathway of concern.

CERCLA – Comprehensive Environmental Response, Compensation, and Liability Act
COPC – chemical of potential concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
ECD – electron capture detection
Ecology – Washington State Department of Ecology
EPA – US Environmental Protection Agency
EPC – exposure point concentration
GC – gas chromatography
HHRA – human health risk assessment
HPLC – high-performance liquid chromatography
J – estimated concentration
LDW – Lower Duwamish Waterway
LDWG – Lower Duwamish Waterway Group
N – tentative identification
NOAA – National Oceanic and Atmospheric Administration
PCB – polychlorinated biphenyl
PDA – photodiode array detection
RBC – risk-based concentration
RfD – reference dose
RL – reporting limit
RME – reasonable maximum exposure
SF – slope factor
SWAC – spatially weighted average concentration
TEQ – toxic equivalent
TEF – toxic equivalency factor
UCL – upper confidence limit

B.6.1 EXPOSURE ASSESSMENT

For most HHRAs, including this one, assumptions made during the exposure assessment contribute a high amount of uncertainty and variability to the risk estimates. Alternative exposure values are possible for all the parameters described in Section B.3.4, most of which would have a linear effect on the resulting risk estimate.[33] For exposure frequency and exposure duration parameters for all exposure scenarios, the values selected were based on EPA guidance and professional judgment. These values have been the subject of considerable debate and analysis during preparation of the Phase 1 HHRA and the present baseline HHRA, and will not be discussed further in this uncertainty assessment. There are several other parameters in the exposure assessment for which possible alternative values warrant discussion, including exposure point concentrations and the consideration of undetected chemicals, seafood consumption rates, incidental sediment ingestion rates, fraction of dose obtained from the LDW, representativeness of existing fish and shellfish data for all potentially exposed populations, and exposure area used for the beach play and clamming scenarios. Each of these topics is discussed in the following subsections.

B.6.1.1 Exposure point concentrations

B.6.1.1.1 Consideration of undetected chemicals

The ProUCL 4 software used to calculate EPCs has the capability of assigning a hypothetical interpolated result for non-detects based on the distribution of detected concentrations, as explained further in Section B.6.1.1.2. Given the statistical treatment on non-detects for EPC calculation in this software, the resulting EPC calculations are not necessarily biased either high or low. The uncertainty associated with these calculations is relatively low.

For chemicals that were detected five or fewer times within a dataset used for EPC calculation, the EPC chosen was the larger of either the maximum detected concentration or half the maximum RL. The use of only a single maximum sample result to represent the EPC is associated with some uncertainty. This is because the use of a detected concentration does not take into account any of the other data that may indicate lower concentrations or the absence of the chemical above the RL. The resulting EPC is likely to be an overestimate of the "true" UCL on the mean (the typical statistic used for the EPC), but there is no statistically reliable means to estimate the UCL. To highlight this uncertainty in the risk estimates for the seafood consumption scenarios, chemicals were footnoted in the risk characterization tables if greater than 50% of the estimated excess cancer risk or non-cancer hazard (based on total dose) was attributable to seafood categories (e.g., crab whole body, clams) with

[33] Changes to consumption rates for individual seafood categories for scenarios other than the one-meal-per-month scenario would not have a directly linear effect on risk estimates because the CDI is the sum of exposures from consumption of multiple seafood categories.

no detected concentrations. This issue does not apply to chemicals that were never detected because they were evaluated only in the uncertainty assessment (see Section B.6.3.2), rather than in the risk characterization.

B.6.1.1.2 Statistical approach to evaluation of infrequently detected COPCs

ProUCL software was used to develop UCLs on mean concentrations of COPCs. ProUCL first evaluates the distribution of the data, then recommends a statistical approach and provides an estimated UCL (EPA 2006e). ProUCL 4 statistical software was created by EPA and its affiliates as an upgrade to the ProUCL 3 software (EPA 2004c). This updated software provides defensible statistical methods and does not rely on simple substitutions for non-detected data points. ProUCL 4 software allows for parametric and non-parametric analysis of both uncensored datasets (i.e., all detected concentrations) and those that contain non-detects to determine a distribution from which a UCL may be calculated. Some of the methods (e.g., Kaplan-Meier method) are able to handle datasets that have multiple detection levels (EPA 2006e). The more accurate methods for identifying distributions make it possible to better define the appropriate UCL value for use in risk assessment.

The ProUCL software generally determined that non-parametric statistics were most appropriate for UCL calculations with more than 50% undetected values and frequently recommended very conservative statistical approaches, such as the 99th percentile Chebyshev for the UCL (Tables B.3-34 through B.3-36, and B.3-39 through B.3-41). The recommended UCLs were generally close to the maximum for these COPCs and sometimes exceeded the maximum. In all cases, the recommended UCL from ProUCL was used in the risk equations.

The UCL calculation methods were intended to provide reasonably health-protective estimates of EPCs for the large number of infrequently detected chemicals present in seafood and sediment. For the seafood consumption scenarios, many chemicals identified as exceeding acceptable risk levels (i.e., upper-bound excess cancer risk estimate > 1×10^{-6} or non-cancer HQ >1) were less frequently detected, or had only non-detected values, in at least one seafood category. Many of these chemicals had excess cancer risk estimates that were an order of magnitude or more below estimates for the most significant risk contributors (i.e., arsenic, PCB TEQ, total PCBs), which were detected in all seafood categories in which they were analyzed (Table B.5-61). In addition, many of the less frequently detected chemicals were qualified JN because identifications and concentration quantifications were tentative. Thus, the uncertainty surrounding the presence and concentrations of chemicals in seafood tissue with lower risk estimates is greater than the uncertainty associated with the chemicals with the highest risk estimates. For the direct sediment exposure scenarios, this was less of an issue because no chemicals exceeding acceptable risk levels had detection frequencies of 50% or less or were only tentatively identified.

B.6.1.1.3 Infrequently detected organochlorine pesticides in tissue

Ten organochlorine pesticides were identified as exceeding acceptable risk levels for one or more seafood consumption scenarios (Table B.5-61). Some of these pesticides, such as aldrin, alpha-BHC, dieldrin, and heptachlor, were detected in less than 5% of the tissue samples used for the risk calculations. Consequently, there is high uncertainty associated with the "true" exposure concentrations of this subset of organochlorine pesticides in tissue.

Another substantial source of uncertainty with regard to the tissue pesticide data is the likely analytical interference in the detection of organochlorine pesticides caused by the presence of PCB congeners in the same tissue samples. This issue was identified by both the analytical laboratory and the data validators. The organochlorine pesticides were analyzed in tissue using EPA Method 8081 (GC/ECD), which is the standard method. The detected results for organochlorine pesticides in the fish and crab tissue samples analyzed in 2004 were JN-qualified by the validator (Windward 2005c), which indicates the presence of an analyte that has been "tentatively identified" and the associated numerical value represents its "approximate concentration" (EPA 1999e). These data were qualified based on the probable interference in the analysis from PCB congeners. The clam tissue data submitted for full validation were also JN-qualified for the same reason (Windward 2005b); however, data undergoing summary validation were not qualified in this manner. To be consistent with the qualification of the fish and crab tissue dataset, all detected organochlorine pesticide results for the benthic invertebrate tissue dataset, which includes the clam tissue data, were qualified JN in consultation with EPA. Analytical results for organochlorine pesticides used in this risk assessment from events prior to 2004 were likely to have been complicated by interference from PCBs; however, the detected results were not all JN-qualified.

The JN-qualified results for organochlorine pesticides are highly uncertain and biased high. For example, a high bias for DDT concentrations in the original dataset was confirmed by gas chromatography/mass spectrometry (GC/MS) reanalysis of 14 sediment, fish, and crab samples that originally had been reported as having high PCB and DDT concentrations based on the less accurate GC/ECD method (Windward 2005f). The GC/MS method was not selected for the original analyses because the achievable RLs were much higher than the target RLs specified in the QAPP (Windward 2004c). The only DDT isomers that were detected in the GC/MS confirmation analyses were 4,4′-DDE and 4,4′-DDD, whereas the GC/ECD analyses detected the 4,4′-DDT, 2,4′DDT, 4,4′-DDE, and 4,4′-DDD isomers, with the highest concentrations reported for the two DDT isomers. The confirmation analysis results verified the JN-qualification of the original sample results, and all the results from the confirmation analyses were much lower than the original results.[34] Thus, the original

[34] The confirmed total DDT concentrations in six sediment samples co-located with benthic invertebrate and clam sample locations ranged from 4 to 60% of the original results. Total DDT was not detected in the confirmation analysis in samples with the two highest of the original total DDT concentrations

reported concentrations of DDT compounds appear to reflect the presence of both PCB congeners and DDT isomers in the sample, and were elevated because of analytical interference.

Despite the findings that the DDT compounds were artificially elevated in the original samples, the results of the reanalysis were not used in this HHRA. The DDT confirmation analyses were run using the original sample extracts, but were conducted 6 months after extraction, which greatly exceeded the maximum 40-day extract holding time. The impact of storage on DDT concentrations is unknown without experimental verification. However, DDT has a long half life in the environment. DDT has remained persistent in the environment despite the US ban on this chemical instituted in 1972, which suggests that the longer holding time may have had minimal effect on the confirmation analyses. The results of these analyses were treated as qualitative and useful as an estimate of the DDT isomer concentrations, but were not incorporated into the project database, or used in the risk calculations presented in this HHRA. No additional confirmation analyses could be conducted in 2004 because the estimated concentrations of other organochlorine pesticides were too low to detect using the low resolution GC/MS method. Since that time, a high-resolution GC/MS method using isotope dilution has become available, but the archived tissue samples collected in 2004 had been discarded because the holding times expired.

Based on the DDT confirmation analyses that were conducted and the known interferences between PCBs and organochlorine pesticides, it is likely that all of the risk estimates for all organochlorine pesticides in the seafood consumption scenarios are overestimated. It is not possible to quantify the magnitude of overestimation at this time.

B.6.1.1.4 Bis(2-ethylhexyl) phthalate and pentachlorophenol tissue data

Bis(2-ethylhexyl) phthalate and pentachlorophenol were both identified as exceeding acceptable risk levels for one or more seafood consumption scenarios (Table B.6-61). Both chemicals were infrequently detected in tissue samples used for this HHRA. Bis(2-ethylhexyl) phthalate was detected in approximately 15% of the tissue samples, and pentachlorophenol was detected in approximately 6% of the tissue samples. Many of the RLs obtained from the original analyses of the tissue samples collected in 2004 as part of Phase 2 were highly elevated because of matrix interferences (Windward 2005c). Consequently, archived subsamples of 49 tissue samples with undetected

---

with RLs that were 3 and 8% of the original detected results. Therefore, the original ECD results overestimated the total DDT concentrations by a factor of 3-20 times. Eight fish and crab tissue samples (three crab hepatopancreas, four shiner surfperch whole-body, and one English sole whole-body) were reanalyzed, and the reanalysis confirmed the presence of DDTs in six of the samples, with confirmed total DDT concentrations that were 5 to 34% of the original results.

concentrations (of 91 total archived fish and crab tissue samples) were submitted for additional analyses of bis(2-ethylhexyl) phthalate and pentachlorophenol to achieve lower RLs than those achieved in the original analyses (Table B.6-2). Additional cleanup steps and an alternative analytical method (for pentachlorophenol) were employed to minimize matrix interferences, resulting in more sensitive instrument response and lower RLs (Windward 2006c). The reanalysis results were used in this HHRA rather than the original results because of the greater sensitivity of the reanalysis methods for these analytes.

Table B.6-2. Number and tissue types of samples selected for bis(2-ethylhexyl) phthalate and pentachlorophenol reanalysis

Species	Number of Samples by Type		
	Fillet	Whole Body	Other Tissue Types
English sole	5	11	0
Pile perch	1	0	0
Striped perch	1	0	0
Shiner surfperch	0	4	0
Starry flounder	1	3	0
Crab – hepatopancreas	0	0	7
Crab – edible meat	0	0	16
Total	**8**	**18**	**23**

The samples selected for reanalysis were those with undetected concentrations for bis(2-ethylhexyl) phthalate and pentachlorophenol, and with the highest RLs of the fish and crab samples analyzed in 2004: 7,200 µg/kg ww for bis(2-ethylhexyl) phthalate and 5,700 or 5,800 µg/kg ww for pentachlorophenol (Windward 2005c). Forty-nine samples, as shown in Table B.6-2, were originally reported at these elevated RLs. The Analytical Resources, Inc. (ARI), reanalysis resulted in a single detection of bis(2-ethylhexyl) phthalate, at an estimated concentration of 100 µg/kg ww, in a slender crab hepatopancreas sample. Bis(2-ethylhexyl) phthalate was not detected by ARI in the remaining 48 samples, with RLs ranging from 66 to 230 µg/kg ww (Windward 2006c).

The initial analyses conducted by Columbia Analytical Services, Inc. (CAS), resulted in 47 undetected values for pentachlorophenol, with RLs ranging from 5,700 to 5,800 µg/kg ww. Pentachlorophenol was detected by CAS in two shiner surfperch whole-body samples, both at estimated concentrations of 2,200 µg/kg ww. Neither of these detected results was confirmed by ARI's GC/ECD analyses; both samples were undetected, one with an RL of 4.5 µg/kg ww and the other with an RL of 4.6 µg/kg ww. However, pentachlorophenol was detected by ARI at low concentrations in 6 of the 47 samples originally reported as undetected by CAS at the higher RLs. Detected results included one shiner surfperch whole-body sample at an estimated concentration of 2.8 µg/kg ww, one starry flounder whole-body sample at an

estimated concentration of 1.3 µg/kg ww, and four English sole whole-body samples at estimated concentrations ranging from 1.1 to 2.3 µg/kg ww. These detected concentrations were more than three orders of magnitude lower than the original RLs reported by CAS. ARI also reported 43 undetected results, with RLs ranging from 3.3 to 11 µg/kg ww.

Some of the reanalyses were conducted slightly outside the 1-year maximum holding time specified in the QAPP (Windward 2004c); 10 samples were extracted 9 to 13 days past the maximum holding time for bis(2-ethylhexyl) phthalate, and three samples were extracted 9 to 13 days past the maximum holding time for pentachlorophenol (Windward 2006c). All analyses of the extracts were conducted within the maximum allowable 40-day extract holding time. The chemicals of concern were not detected in any of the samples that exceeded the 1-year maximum holding time for frozen samples, and all results for these samples were UJ-qualified, indicating some uncertainty around the RL.

The reanalysis results suggested that bis(2-ethylhexyl) phthalate and pentachlorophenol are likely to be at far lower concentrations in LDW fish or shellfish samples than the original elevated RLs obtained in 2004 (Windward 2005c). Because only the samples with the highest RLs were reanalyzed, however, the resulting fish EPCs are based on a mix of reanalyzed and original data, and are still likely to be overestimates of the "true" EPCs for fish and shellfish. Therefore, the risks associated with exposures to these two chemicals in fish and shellfish are likely overestimated as well.

EPCs and risk estimates for bis(2-ethylhexyl) phthalate and pentachlorophenol were recalculated for hypothetical datasets that included replacement of the remaining elevated RLs (for those samples that were not reanalyzed) with the highest RLs obtained from the 2005 reanalysis of fish and shellfish tissue. For bis(2-ethylhexyl) phthalate, all RLs greater than 230 µg/kg ww (the highest RL from the 2005 reanalysis) were replaced with an assumed RL of 230 µg/kg ww. For pentachlorophenol, all RLs and detected concentrations greater than 11 µg/kg ww (the highest RL from the 2005 reanalysis) were replaced by 11 µg/kg ww. Detected pentachlorophenol concentrations were replaced in addition to the RLs, based on the results of the reanalysis of the previously detected results, which were not confirmed in the reanalysis. Following the replacements, EPCs for bis(2-ethylhexyl) phthalate and pentachlorophenol were recalculated (Table B.6-3). Hypothetical risk estimates for two of the seafood consumption scenarios are presented in Table B.6-3 to show the effect of using lower EPCs in the exposure and risk calculations.

Table B.6-3. Comparison of original seafood consumption EPCs and risk estimates for bis(2-ethylhexyl) phthalate and pentachlorophenol based on hypothetical tissue reanalyses

Consumption Category	Detection Frequency	Original Results			Hypothetical Results		
			Excess Cancer Risk Estimate[a]			Excess Cancer Risk Estimate[a]	
		EPC (µg/kg ww)	Adult Tribal RME (Tulalip Data)	Adult API RME	EPC (µg/kg ww)	Adult Tribal RME (Tulalip Data)	Adult API RME
Bis(2-ethylhexyl) phthalate							
Benthic fish, fillet	2/14	1,300[b]	6×10^{-6}	2×10^{-6}	1,300[b]	6×10^{-6}	2×10^{-6}
Benthic fish, whole body	0/24	1,800[c]			120[c]		
Pelagic fish	5/29	2,100[b]			2,100[b]		
Pentachlorophenol							
Benthic fish, fillet	0/14	2,900[c]	9×10^{-5}	2×10^{-5}	5.5[c]	9×10^{-7}	3×10^{-7}
Benthic fish, whole body	6/24	780[d]			2.8[d]		
Clams	0/14	200[c]			5.5[c]		
Crab, edible meat	0/21	290[c]			5.5[c]		
Crab, whole body	0/21	100[c]			5.5[c]		
Mussel	0/22	14[c]			5.5[c]		
Pelagic fish	2/29	2,400[b]			11[b]		

[a] Excess cancer risk estimates represent risks from all seafood consumption categories. Bis(2-ethylhexyl) phthalate EPCs for clams, crab edible meat, crab whole body, and mussels were not changed from their original values provided in Table B.3-36 and are not shown here.

[b] EPC is equal to the maximum detect.

[c] EPC is equal to one-half of the maximum RL.

[d] EPC was calculated using ProUCL 4.0.

API – Asian and Pacific Islander

EPC – exposure point concentration

RL – reporting limit

RME – reasonable maximum exposure

ww – wet weight

Risk estimates for the adult tribal RME scenario based on Tulalip data and the adult API RME seafood consumption scenario calculated using the hypothetical EPCs were unchanged for bis(2-ethylhexyl) phthalate because two fish EPCs were based on the maximum detected concentrations, which were not replaced in the hypothetical dataset. EPCs based on very small numbers of detected concentrations may be overestimated (see Section B.6.1.1.10). The hypothetical risk estimates for

pentachlorophenol were approximately 100 times lower than the original estimates and would no longer exceed the risk threshold of 1×10^{-6}.

B.6.1.1.5 Carcinogenic PAHs in tissue

In the historical dataset used in the Phase 1 HHRA (not collected by LDWG, as described in Table B.2-4), cPAHs were detected in only one of the tissue types (mussels). In addition, RLs for cPAHs in other tissue types were relatively high. To refine estimates of exposure and risk for these chemicals, analytical methods with lower RLs were used in the 2004 data collection (Windward 2005b, c). In risk estimates presented in Section B.5 for cPAHs, only data from 2004 were included in the risk calculations. This approach was used to prevent the possibility that high RLs for historical sample concentrations that are not thought to be representative of actual concentrations might significantly influence risk estimates.

To evaluate this uncertainty, risks from exposure to cPAHs in seafood were also evaluated using both the 2004 and historical tissue data. The EPCs based on this combined dataset are presented in Table B.6-4. The risk estimates using the combined data are higher than risk estimates for the 2004 dataset alone (Table B.6-5). Because there were no 2004 mussel data, mussel consumption was reallocated in risk calculations in the risk characterization section to other seafood classes proportional to consumption of those classes for scenarios including multiple seafood categories. For example, mussel consumption for the adult tribal RME scenario based on Tulalip data (0.082 g/day) was reassigned to other seafood categories based on the relative consumption of the other seafood categories included in the risk calculations. Thus, the majority was reassigned to clams and crabs because those categories were the most consumed categories.

Overall, these calculations indicate that detection issues with the historical data may have moderately affected risk calculations for cPAHs. However, excess cancer risks calculated using more recent data alone or with inclusion of the historical data are both in excess of 1×10^{-6} for all but one multi-species scenario (the adult API CT scenario) and for the one-meal-per-month clam consumption scenario. Thus, cPAHs would be identified as exceeding acceptable risk levels for these scenarios using either dataset. However, for one-meal-per-month benthic fish consumption, use of only the 2004 data results in an excess cancer risk estimate below 1×10^{-6}, while use of the larger dataset with the elevated RLs results in a risk estimate in excess of this threshold. Risk estimates for one-meal-per-month pelagic fish and crab consumption and the adult API CT scenario are not in excess of 1×10^{-6} when calculated using either dataset.

Table B.6-4. Exposure point concentrations and summary statistics for cPAHs using all available tissue data

Consumption Category	No. Detected/ Total No. Samples	Mean Value (µg/kg ww)	Maximum Detection (µg/kg ww)	Maximum RL (µg/kg ww)	Statistic Used	EPC Value (µg/kg ww)
Benthic fish, fillet	5/14	4.0	0. 64 J	29	95% Chebyshev UCL, pooled RL	15[a]
Benthic fish, whole body	21/24	1.4	2.8 J	0. 45	95% Chebyshev UCL	2.3
Clams	14/14	15	44	na	approximate gamma UCL	20
Crab, edible meat[b]	8/21	1.8	0.84 J	29	99% Chebyshev UCL	0.64 [a]
Crab, whole body	19/21	1.5	2.4 JM	17	95% Chebyshev UCL	1.2
Mussels	11/22	23	33	29	Student's-t UCL	31 [a]
Pelagic fish	26/29	2.9	2.2	43	99% Chebyshev UCL	1.2

[a] Detection frequency for this consumption category was less than 50%.

cPAH – carcinogenic polycyclic aromatic hydrocarbon

EPC – exposure point concentration

J – estimated value

M – calculated value as described in Table B.2-4

na – not applicable

RL – reporting limit

t (t-distribution) – statistical method used to calculate the mean for a normally distributed set of samples

UCL – upper confidence limit

ww – wet weight

Table B.6-5. Comparison of excess cancer risks associated with cPAHs based on 2004 and combined datasets

Scenario	2004 cPAH Data[a]		All cPAH Data[b]	
	Cancer CDI	Excess Cancer Risk	Cancer CDI	Excess Cancer Risk
Adult tribal RME (Tulalip data)	9.8×10^{-6}	7×10^{-5}	1.1×10^{-5}	8×10^{-5}
Adult tribal CT (Tulalip data)	5.2×10^{-7}	4×10^{-6}	5.7×10^{-7}	4×10^{-6}
Child tribal RME (Tulalip data)	1.8×10^{-6}	7×10^{-5}	2.1×10^{-6}	8×10^{-5}
Child tribal CT (Tulalip data)	2.1×10^{-7}	8×10^{-6}	2.5×10^{-7}	1×10^{-6}
Adult tribal (Suquamish data)	1.1×10^{-4}	8×10^{-4}	1.2×10^{-4}	9×10^{-4}
Adult Asian and Pacific Islander – RME	4.4×10^{-6}	3×10^{-5}	5.2×10^{-6}	4×10^{-5}
Adult Asian and Pacific Islander – CT	1.0×10^{-7}	8×10^{-7}	1.2×10^{-7}	9×10^{-7}
Adult one meal per month – benthic fish	2.9×10^{-8}	2×10^{-7}	6.7×10^{-7}	5×10^{-6}
Adult one meal per month – clam[c]	9.0×10^{-7}	7×10^{-6}	9.0×10^{-7}	7×10^{-6}
Adult one meal per month – crab	2.9×10^{-8}	2×10^{-7}	2.9×10^{-8}	2×10^{-7}
Adult one meal per month – pelagic fish	4.3×10^{-8}	3×10^{-7}	5.4×10^{-8}	4×10^{-7}

[a] Consists of data from the 2004 investigation (Windward 2005b, c).

[b] Consists of historical and 2004 data (as described in Table B.2-4).

[c] Clam data available from only the 2004 sampling event.

CDI – chronic daily intake

cPAH – carcinogenic polycyclic aromatic hydrocarbon

CT – central tendency

RME – reasonable maximum exposure

An additional uncertainty related to cPAH risk assessment is the fact than not all PAHs were analyzed in tissue. California EPA has generated potency equivalence factors (PEFs, similar to TEFs and used to estimate total cPAHs) for some PAHs that were not analyzed in LDW samples (California EPA 1994). If some of these other PAHs with PEFs were present in the LDW, the uncertainty from the lack of data could result in underestimation of the EPCs for cPAHs in tissue and sediment.

B.6.1.1.6 Lack of dioxin and furan data in tissue and some beach play areas

As noted in Section B.5.5.2.2, LDWG, EPA, and Ecology agreed that no tissue samples would be analyzed for dioxins and furans. In the absence of these data, the overall excess cancer risk estimates made for the seafood consumption scenarios are likely underestimated. The degree to which they may or may not be underestimated cannot be determined precisely. In addition, dioxin and furan sediment chemistry data were lacking for some beach play areas (Areas 1, 2, 3, 6, and 8). Hence, dioxin and furan risk estimates for those areas could not be estimated.

B.6.1.1.7 Sediment PCB analytical methods

As indicated in Section B.2.3.5.1, the sediment PCB data used in this HHRA are from two different analytical methods: GC/ECD, which was used at almost 1,000 locations, and HPLC/PDA, which was used by NOAA at approximately 300 locations. Some uncertainty exists regarding the suitability of the PCB data derived from the HPLC/PDA method. As discussed in Section B.2.3.4.3, this uncertainty occurs primarily because the method does not involve direct quantification of total PCBs. In the Phase 1 HHRA, the implications of these different methods were explored quantitatively (Windward 2003b). At that time, samples from approximately 600 locations had been analyzed for total PCBs (Aroclor sum) using GC/ECD, and samples from 300 locations had been analyzed using HPLA/PDA (i.e., the same samples included in the current baseline HHRA). The Phase 1 evaluation found that risks associated with the netfishing RME scenario were lowered by approximately 20% when the HPLA/PDA data were excluded (Windward 2003b). This is likely a result of the fact that, in general, the NOAA method yields higher concentrations than does the GC/ECD method for high-PCB-concentration samples (e.g., those that exceed 10 mg/kg dw) (Krahn et al. 1998). Because a greater percentage of the overall dataset is based on the GC/ECD results in the current baseline HHRA, the influence of the HPLA/PDA data is expected to be less than in Phase 1. Therefore, this uncertainty was not re-evaluated quantitatively for the baseline HHRA.

B.6.1.1.8 Calculation methods for total PCBs

The concentration of total PCBs in a sample may be calculated as the sum of Aroclors or the sum of PCB congeners. Aroclor data were available for many more tissue samples (n = 221) than were congener data (n = 49).[35] Because of the larger dataset available, total PCBs in tissue were assessed as the sum of Aroclors in the risk characterization. The sample size affects the calculation of UCLs (for EPCs) in that the statistical software used for UCL computation (ProUCL 4) attempts to compensate for the uncertainty of having fewer measurements by selecting calculation approaches that lead to higher UCL estimates. This section explores different approaches to summing Aroclors and then compares risk estimates for total PCBs based on the sum of Aroclors to risk estimates based on the sum of PCB congeners.

Total PCBs in sediment and tissue were calculated as described in Section B.2.2.4 in accordance with the Washington State Sediment Management Standards (SMS; WAC 173-204). That method sums only detected Aroclor concentrations or assigns a value equal to the highest Aroclor RL if all Aroclors are undetected. At other Superfund sites, different methods for calculating total PCBs have been used. For example, EPA is developing guidance for assessing human health risks from total PCBs that includes summing detected Aroclor concentrations and half the RL for particular Aroclors if they were detected in a significant number of samples found elsewhere at the site. This is the approach being used at the Portland Harbor Superfund site.

Table B.6-6 presents a comparison of total PCB concentrations in LDW tissue samples calculated by the primary method used elsewhere in this document and the alternative method described above. The differences between the two methods are small. For fish consumption categories, which have the highest total PCB concentrations, the percentage difference is 2% or less. The differences are of greater magnitude for the shellfish consumption categories, but the concentrations are much lower in shellfish than finfish. Both total PCB calculation methods would yield very similar risk estimates for seafood consumption. Given the small difference between the two methods for summing PCBs, and because of the complexity associated with combining information from two datasets, it was felt that the approach outlined in WAC 173-204 should be applied in this risk assessment. The same approach was used in the LDW ERA.

[35] Of the 221 samples with Aroclor data, 49 were also analyzed for PCB congeners. PCB congener data were available for 3 additional English sole samples that that were included in this dataset (or for PCB TEQ assessments) because the samples were only analyzed for 23 of the 209 PCB congeners (Battelle 1996).

Table B.6-6. Comparison of two different methods for calculating PCB totals in LDW tissue samples

Consumption Category	Average Total PCB Concentration (µg/kg ww)		Absolute Difference Between Two Methods (µg/kg ww)	Percentage Difference Between Two Methods
	Primary Method[a]	Alternative Method[b]		
Benthic fish fillet	700	714	14	2.0
Benthic fish whole body	2,200	2,240	40	1.8
Clams	140	151	11	7.9
Crab edible meat	170	177	7	4.1
Crab whole body	890	890	0	0
Mussel	34	47	13	38
Pelagic fish	1,700	1,700	0	0

[a] Primary method described in Section B.2.2.4 – sum only detected Aroclor concentrations or assign a value equal to highest Aroclor RL if all Aroclors undetected.

[b] Alternative method – sum detected Aroclor concentrations and one-half the RL for undetected Aroclors that were detected elsewhere at the site. The alternative method does not include nine samples from 1998 that had detected concentrations of Aroclor 1016/Aroclor 1242. The analyst could not distinguish between these two Aroclor patterns. The maximum concentration was 16 µg/kg ww. Neither Aroclor was detected in any other sample in any sampling event. For LDW tissue samples, Aroclors 1248, 1254, and 1260 were detected frequently, so the total PCB sums presented above include detected concentrations and half RLs for those three Aroclors.

LDW – Lower Duwamish Waterway

PCB – polychlorinated biphenyl

ww – wet weight

As discussed in Section B.2.3.4.3, 49 tissue samples (approximately one-third of all tissues) were analyzed for both PCBs as Aroclors and PCBs as congeners (all 209). For all species except clam, the Aroclor summation approach (i.e., the method used in risk characterization and primary method shown in Table B.6-6) leads to a total PCB estimate that was 1.5 times or more greater than the concentration estimate based on the sum of the PCB congeners. For clams, the Aroclor summation method yielded estimates that were about 70% of the PCB congener total. Therefore, the total PCB EPCs used in the risk assessment on the whole are likely to contribute to overestimation of risk.

As discussed previously in this section, PCBs were analyzed as individual congeners in a subset of the tissue samples analyzed for Aroclors. EPCs for total PCBs calculated in the following three ways are shown in Table B.6-7: based on the congener data (n = 49), based on the Aroclor data for those samples also analyzed for congeners (n = 49), and based on Aroclor data for all samples (i.e., the same EPCs presented in Section B.3.4.3.1, n = 221). Aroclors were summed according to the primary method described above and in Section B.2.2.4. Total PCBs based on PCB congeners were calculated as the sum of detected congeners in each sample. The samples analyzed for PCB congeners were analyzed for all 209 congeners, with an average detection frequency of 91% (i.e., in each of the 49 samples, an average of 91% of the 209

congeners tested were detected) indicating that the majority of PCBs present in the samples were quantified. It is important to note that a much larger number of samples were analyzed for Aroclors than for PCB congeners; the samples analyzed for PCB congeners were a subset of those analyzed for Aroclors. Overall, the EPCs based on the subset of data (analyzed for both congeners and Aroclors) were higher than the EPCs based on the Aroclor data for the full dataset. This may reflect some bias in the selection of samples to be analyzed for both congeners and Aroclors and greater uncertainty in the estimation of means for the smaller datasets (i.e., the smaller datasets that were analyzed for both congeners and Aroclors may be more variable than larger datasets, leading to higher UCLs on the mean and therefore higher EPCs). For all consumption categories except clams, the EPCs based on congeners were lower than those based on Aroclors for the subset of samples analyzed for both congeners and Aroclors.

Table B.6-7. EPCs for total PCBs based on PCB congeners and Aroclor sums

Consumption Category	Total PCB (Sum of Congeners)		Total PCBs (Sum of Aroclors for Samples with Congener Data)		Total PCBs (Sum of Aroclors for all Available Aroclor Data as Presented in Risk Characterization)	
	Sample Count	EPC (mg/kg ww)	Sample Count	EPC (mg/kg ww)	Sample Count	EPC (mg/kg ww)
Benthic fish, fillet	8	1.11	8	1.69	33	1.16
Benthic fish, whole body	8	2.33	8	3.92	45	2.6
Clams	8	0.554	8	0.452	14	0.6
Crab, edible meat	8	0.170	8	0.282	29	0.204
Crab, whole body	6	1.43	6	1.52	25	1.1
Pelagic fish	11	6.60	11	8.77	53	3.2

EPC – exposure point concentration

PCB – polychlorinated biphenyl

ww – wet weight

Excess cancer risks and non-cancer hazards for the adult tribal RME seafood consumption scenario based on Tulalip data were also estimated using EPCs from the three datasets in Table B.6-7 and are presented in Table B.6-8. For the subset of data (n = 49), the excess cancer risk estimate based on the sum of congeners was the same as the excess cancer risk estimate based on the sum of Aroclors. However, the non-cancer hazards based on the sum of congeners were slightly lower those based on the sum of Aroclors. The risk estimates for the subset of data (n = 49, as the sum of congeners or as the sum of Aroclors) were greater than the risk estimates based on Aroclors for all data (n = 221). The larger Aroclor dataset was used in the risk characterizations because it is expected to more accurately represent LDW PCB risks than the subset of data with PCB congener data available, which, as described above, was composed of samples that generally had higher concentrations.

Table B.6-8. Excess cancer risks and non-cancer hazards for the adult tribal RME scenario based on Tulalip data: risk estimates for total PCBs as the sum of congeners and as the sum of Aroclors

Chemical	Cancer CDI	Non-Cancer CDI	Excess Cancer Risk	Hazard Quotient
Total PCBs (sum of congeners) [a]	1.3×10^{-3}	1.3×10^{-3}	3×10^{-3}	64
Total PCBs (sum of Aroclors for samples with congener data) [a]	1.6×10^{-3}	1.6×10^{-3}	3×10^{-3}	78
Total PCBs (sum of Aroclors, as reported in risk characterization section for all data) [b]	8.0×10^{-4}	8.0×10^{-4}	2×10^{-3}	40

[a] CDIs and risk estimates are based on EPCs for 49 samples (see Table B.6-7).

[b] CDIs and risk estimates are based on EPCs for 221 samples (see Table B.6-7).

CDI – chronic daily intake

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

B.6.1.1.9 PCB EPCs for benthic fish fillets

As noted in Table B.3-37, the PCB EPC calculated for benthic fish fillets (1.2 mg/kg ww) that was used in the risk characterization was based on a weighted approach in consideration of the greater abundance of benthic fish fillet data from the lower part of the LDW (RM 0 to RM 1.5) compared to the rest of the LDW. Two alternative methods for EPC calculation are presented in this section and compared to the results described in Table B.3-35.

The method used in Section B.3 for calculating tissue EPCs for chemicals other than PCBs with more than five detected values was simply to take all results from the composite samples and calculate an EPC in ProUCL. Applying this method to the total PCB data for benthic fish fillets results in an EPC of 0.91 mg/kg ww. This EPC is slightly lower than the EPC used in the risk characterization because it includes unweighted PCB data from the late 1990s, which had lower PCB concentrations than data collected in 2004 and 2005. A second EPC calculation was performed using only data from 2004 and 2005, which were collected using a grid sampling design that avoided the spatial bias associated with including the older data. The PCB EPC for the 2004/2005 data is 1.3 mg/kg ww. The slight differences in the EPCs calculated by the three methods (1.2, 0.91, and 1.3 mg/kg ww, respectively) would not result in any significant change in the risk estimates for any of the seafood consumption scenarios. For example, the total PCB excess cancer risk estimate for the adult tribal RME scenario would be 2×10^{-3} regardless of which EPC calculation method was used.

B.6.1.1.10 EPCs for infrequently detected chemicals

Many data sets in the HHRA would have had an adequate number of samples for EPC development (five or more), but were not suited for development of statistically defensible EPCs because of the high fraction of non-detects. Developing realistic EPCs

for datasets with very small numbers of detected concentrations cannot be done statistically and requires a policy decision. As summarized in Figure B.3-3, a simplifying assumption used for this HHRA was to set the EPC at the higher of one-half the maximum RL or the maximum detected value for datasets with fewer than six detected values. In these cases, the "true" EPC may be overestimated because the complete dataset, including many values that are lower than the maximum, is not considered in the calculation. The EPC may also be underestimated because the true mean may be higher than the maximum value of a small dataset, since the distribution for most chemical contamination data may be positively skewed. This uncertainty is unavoidable when only a few samples are available to characterize exposure (see also Section B.6.1.1.11).

B.6.1.1.11 EPC estimates for small datasets

The approach for EPC estimation based on dataset size and number of detected concentrations is presented in Section B.3.4.3. ProUCL 4 was used to develop EPCs for datasets with six or more samples. ProUCL 4 takes into account sample size and distribution in its recommendation of a UCL. In some cases, the recommended UCL exceeds the maximum sample value. For sample sets smaller than six, a policy decision was made to use the maximum detected concentration or half the maximum RL, whichever was higher. Therefore, uncertainty related to potential EPC underestimation is greater for EPCs derived for small datasets with fewer than six samples, for which ProUCL 4 was not used. This section focuses on the uncertainty related to datasets with fewer than six samples. Uncertainties related to infrequently detected chemicals are discussed in Section B.6.1.1.10.

For some datasets, the available sample size was very small (irrespective of the number of detections). Of the 391 tissue EPCs, four EPCs were developed based on five or fewer samples. EPCs for one chemical (benzidine) for both clamming scenarios were based on just two samples. For the beach play scenarios, 93 of 199 EPC were developed based on sample sizes of five or smaller.[36] The high number of EPCs in beach play areas that were derived from datasets with five or fewer samples was partially a result of the small exposure areas. Nearly all of the EPCs for beaches 1, 2, and 6 had five or fewer samples, accounting for 61 of the small-dataset EPCs. In addition, some chemicals were analyzed in only a subset of samples, including essential elements (e.g., iron or manganese), dioxins/furans, PCB TEQ, and pesticides (e.g., total DDTs or dieldrin).

When only five or fewer samples were available, the EPCs used in the risk calculation were based on a maximum detected concentration (or one-half the maximum RL). In these cases, the uncertainty about whether the EPC is equal to or exceeds the mean is much greater than that for datasets with six or more samples for which ProUCL 4 was

[36] An additional 25 beach play EPCs could not be developed because no samples were analyzed for a given COPC in the beach play exposure area.

applied. In cases where ProUCL 4 was applied, a UCL on the mean was estimated (e.g., 95% Student's t-UCL) and used as the EPC. This was not done for datasets with fewer than six samples, where a policy decision for EPC selection was made, and no UCL on the mean was estimated or incorporated into the EPC. Assuming that the maximum sampled value does not exceed the true mean, this latter approach may underestimate the mean. This may contribute to the underestimation of risk, but the potential magnitude of this uncertainty is unknown.

B.6.1.1.12 Spatial bias in EPC estimates for PCBs

As noted in Section B.2.3.1.1, an EPC based on a simple arithmetic mean, in which all concentrations are given equal weight in the computation of the mean, may overestimate exposure because more highly contaminated areas have been sampled more intensively than less-contaminated areas. Because total PCBs in sediment have been sampled more frequently in several ongoing early action investigations in the LDW than in other areas of the LDW, the arithmetic mean of all sampling results overestimates PCB exposures. Nonetheless, arithmetic EPCs for PCBs were used in the exposure assessment and risk characterization sections of this HHRA. This section discusses the differences between arithmetic EPCs and spatially weighted EPCs for PCBs.

To account for spatial variations in sampling density, many scientists use Thiessen polygons for spatial analysis. The Thiessen polygon associates each point in a plane with the closest sampling location for which a measurement is available (Burmaster and Thompson 1997). The effect of this process is an assumption that the concentration at any point where measurements have not been made is the same as the concentration in the sample closest to that point. Polygon boundaries are defined by the distances to the nearest sampling locations within the baseline surface sediment dataset.

For the netfishing scenario, Thiessen polygons were created around each sediment sampling location in both the intertidal and subtidal depths (Map B.6-1). For the tribal clamming scenarios, only intertidal surface sediment locations within the tribal clamming area (see Maps B.3-2 and B.6-2) were used in the analysis. The UCLs for total PCBs for the other sediment exposure scenarios (i.e., beach play RME and low-end [7 days per year] clamming) were based on the arithmetic mean because they involved much smaller areas that are much less affected by the sampling bias noted above. Consequently, the UCL on the arithmetic mean should reasonably approximate the exposure within these areas for other sediment exposure scenarios.

Although EPA (1989) guidance does not explicitly address spatially weighted EPC calculations, a 95% UCL on the spatially weighted mean was calculated for total PCB for the netfishing and tribal clamming scenarios to remain consistent with the intent of the EPA (1989) guidance. There are several methods for calculating a UCL on a spatially weighted mean, but only a single method is shown here. Other methods are

likely to yield different results, but the results from this method are adequate to provide some indication of the difference between arithmetic and spatial approaches.

A bootstrapping[37] procedure was implemented because the PCB concentrations do not closely match either a normal or log-normal distribution. Commonly used calculation methods other than bootstrapping are based on an assumed distribution, either normal or log-normal. In the bootstrapping procedure, 50,000 bootstrap iterations were created in S-PLUS®.[38] In each iteration, a sample equal to the sample size of the particular dataset (n = 440 for clamming, n = 1,291 for netfishing) was drawn such that each polygon concentration was drawn with a probability proportional to its size. The mean of all the concentrations drawn in each iteration was computed as a simple arithmetic mean (Equation 6-1). The 95th percentile of the 50,000 iteration means was computed as the SWAC UCL (Equation 6-2).

$$\bar{x}_{wtdBootstrap_i} = \sum_{i=1}^{n} x_i \Big/ n \qquad \textbf{Equation 6-1}$$

$$95\%UCL_{Bootstrap} = 95\%\text{ile of } \bar{x}_{wtdBootstrap_{i=1,k}} \qquad \textbf{Equation 6-2}$$

Where:

$\bar{x}_{wtdBootstrap_i}$	=	weighted arithmetic mean concentration of bootstrap iteration
x_i	=	polygon concentration (mg/kg dw)
n	=	sample size of the particular dataset
k	=	number of bootstrap iterations (50,000 in this case)

This particular bootstrap approach is known as the "percentile" bootstrap method and may underestimate 95th percentiles if the bootstrapped distribution of means exhibits skew. Methods do exist to address developing bootstrapped 95% UCLs for skewed distributions of bootstrapped means (e.g., bias corrected bootstrapping). These methods were not explored in this uncertainty analysis. The objective of this analysis was to conduct a preliminary evaluation of the impact of sampling bias in PCB surficial sediment sampling. The SWAC UCLs calculated for total PCBs for the netfishing and tribal clamming (RME and 183 days per year) scenarios were 0.47 and 0.90 mg/kg dw, compared to the arithmetic EPCs of 2.5 and 4.0 mg/kg dw, respectively, shown on Tables B.3-42 and B.3-44. Based on this method of SWAC UCL calculation, the arithmetic UCLs are roughly five-fold higher. The risk implications of these differences are discussed in Section B.6.1.7.

---

[37] Bootstrapping is a technique by which the original dataset is randomly sampled to create pseudoreplicate datasets. The parameter of interest is calculated for each pseudoreplicate dataset, thereby providing information about the variability of that parameter. In this study, the original dataset was not sampled randomly. Sampling was conducted proportionally based on the size of each Thiessen polygon relative to other polygons in the LDW.

[38] S-PLUS® is a statistical software package.

B.6.1.2 Ingestion rates

B.6.1.2.1 Incidental sediment ingestion

Incidental sediment ingestion rates for the netfishing, beach play, and clamming scenarios were evaluated using soil ingestion rates identified in EPA guidance. This approach is commonly used in HHRAs, but it is not clear to what extent incidental soil ingestion rates are applicable to evaluation of incidental sediment ingestion. For example, the amount of sediment transferred to fishermen's hands when handling monofilament gill nets is not known.

B.6.1.2.2 Adult seafood consumption

Site-specific estimates of seafood consumption were not available for the LDW (e.g., Muckleshoot Indian Tribe or recreational users). As described in Section B.3.4.1, the seafood consumption rates assumed for the adult tribal scenario based on Tulalip data, adult tribal scenario based on Suquamish data, and adult API scenario were provided by EPA (EPA 2005a; Kissinger 2005) and based on recent regional seafood consumption studies (EPA 1999a; Suquamish Tribe 2000; Toy et al. 1996). The child tribal consumption rate based on Tulalip data presented in the risk characterization was derived in part from a ratio applied to the adult tribal rate based on Tulalip data as described in Section B.3.4.1. The uncertainties specifically associated with the child tribal consumption rate based on Tulalip data and development of a child tribal consumption rate based on Suquamish data are discussed in Section B.6.1.2.3. The adult seafood consumption rates are based on surveys that appear to fairly represent the populations that were interviewed (EPA 1999a; Suquamish Tribe 2000; Toy et al. 1996). However, because the groups surveyed do not use the LDW as their primary fishing area, the degree to which the rates represent people who presently or may in the future consume fish and shellfish from the LDW is not known. The consumption continuum figures presented in Section B.5.6 illustrate risks associated with different seafood consumption rates. These figures illustrate that assumptions about which consumption rates are appropriate for a given scenario can have significant effects on risk estimates.

EPA's interpretation of the seafood consumption studies to develop consumption rates required numerous assumptions. For example, the total seafood consumption rate was allocated among seven seafood categories based on the reported mean consumption of each seafood category regardless of the source of the seafood; i.e., regardless of whether it was self-caught or store-bought, or from some other source (EPA 2005a; Kissinger 2005). For the tribal populations evaluated in this HHRA, this assumption is reasonable because the majority of the seafood consumed by these populations is self-caught. However, based on the survey of the API population (EPA 1999a), the majority of their consumed seafood is purchased in stores. Less than a quarter of the overall fish consumption reported in the API survey was self-harvested (EPA 1999a). The initial total API seafood consumption rate used in the risk calculations was developed using demographically weighted data for consumers of King County species and is

intended to reflect the 95th percentile of API consumption of seafood from only King County (as described in Section B.3.4.1.3) (Kissinger 2005). The percentages of consumption for the different seafood categories were derived using the same data (demographically weighted) for consumption of only King County seafood. However information about preparation style for the crab (whole body vs. edible meat) and benthic fish (whole body vs. fillet) in the dataset did not distinguish between King County seafood and seafood from other sources (such as store-bought seafood). Thus, the crab and benthic fish apportionments contain uncertainty as to how well they reflect consumption of LDW seafood. In addition, as requested by EPA (2006c), consumption of freshwater fish reported in the survey was reapportioned to other marine categories, and it was assumed that there was no freshwater fish consumption in the scenarios used in the risk assessments (see Section B.6.1.2.3). There is also uncertainty related to how much of the reported King County harvested seafood is harvested in the LDW.

Another uncertainty regarding the interpretation of the API survey relates to the difficulties of characterizing consumption for the many diverse ethnic groups included in the study. As discussed in Section B.3.4.1, the sample sizes for the 10 ethnic groups included in the study were generally not demographically representative of the API population in King County. Survey results were adjusted statistically to account for this uneven representation (Kissinger 2005). Despite this adjustment, several ethnic groups were represented by small sample sizes (n = 10 or less). Defining a consumption rate for a large population that includes several groups of small sample sizes entails substantial uncertainty. For example, many individuals in the API survey reported no consumption of King County seafood during the interviews, while others reported very high percentages (EPA 1999a). The reported estimate for 50th percentile consumption was 5.8 g/day, as compared to the 95th percentile estimate of 57.1 g/day, which was used for risk estimates in this document (Kissinger 2005)[39]. The uncertainties related to a characterization of a single seafood consumption rate to represent the many diverse API ethnic groups included in the survey are reflected in the wide range of the upper and lower confidence bounds for the estimate of total King County 95th percentile consumption (approximately 25 g/day to approximately 80 g/day) (Kissinger 2005) and should be considered in interpreting the API risk estimates.

The seafood consumption rates from the Suquamish (2000) and Tulalip Tribes (Toy et al. 1996) studies are based on seafood consumption surveys of tribal members consuming seafood from outside the LDW. The Suquamish and Tulalip Tribes consume seafood from Puget Sound habitats that differ considerably in terms of quality and quantity from the LDW. Consequently, it is highly uncertain how well these tribal seafood consumption rates apply as surrogates for tribal seafood

[39] As described in Section B.3.4.1.3, the anadromous fish portion of consumption was not included in the adult API RME and CT exposure scenarios.

consumption rates specific to the LDW, particularly for clams. The consumption rates used in calculating the CDI equate to approximately 900 and 11,000 clams per year for the adult tribal RME scenario based on Tulalip data and the adult tribal scenario based on Suquamish data, respectively, for each individual consuming clams, assuming a weight of approximately 15 g ww for each clam. EPA (2007b) acknowledged the importance of habitat quality in selecting seafood consumption rates for application to the LDW: "As a policy decision, for sites in the Puget Sound and Strait of Georgia that lack extensive intertidal habitat, the consumption rate derived by EPA from data from the Tulalip Tribes represents a sustainable consumption rate." Based on these considerations, EPA selected the consumption rates based on the Tulalip Tribes as most appropriate for the LDW (EPA 2005a). Furthermore, as stated in the LDW application of the EPA tribal seafood consumption framework (EPA 2005a), "EPA believes that use of Suquamish exposure parameters will not provide the best estimate of LDW tribal seafood consumption risks due to the degraded habitat in the LDW and questions whether the high Suquamish shellfish consumption rate could be sustained."

The ability of LDW habitats to support the clam populations that would be necessary to sustainably achieve the clam consumption rates that were assumed in this HHRA is unknown. Although an intertidal clam survey was conducted in the LDW in 2004, data were insufficient to make LDW-wide population estimates for adult clams likely to be targeted by harvesters (Windward 2004a). Suitable clam habitat is limited in the LDW by physical qualities (e.g., grain size) of the intertidal sediments and the fluctuating salinity (varying from nearly freshwater at the surface to nearly full-strength seawater at depth), particularly in more upstream areas (Windward 2004a). Clams of harvestable size within the LDW are nearly all *Mya arenaria*, a species known to be more tolerant of finer-grained sediments and low salinities than are the clam species more typically found in Puget Sound. In addition to these natural physical constraints that limit clam habitat in the LDW, habitat has also been reduced as a result of steep banks of riprap, concrete, and other construction materials. There is high interest among stakeholders in improving the habitat quality in the LDW. Such habitat improvement may increase the quantity of harvestable clams, but some physical constraints (especially the fluctuating salinity regime) are likely to remain.

The seafood consumption rates used in this HHRA do not include salmon, as explained in Sections B.3.4.1. Some of the chemicals found in adult salmon returning to the LDW originated in the LDW, during juvenile outmigration. Given the great size difference between juvenile and adult salmon, however, a significant growth dilution effect takes place. In addition, most of the salmon's life cycle takes place outside the LDW, such that the body burden of bioaccumulative chemicals in an adult salmon is largely attributed to contaminant uptake that occurred outside the LDW. An example calculation presented in Section B.2.1.2 suggests that the fraction of the PCB body burden in an adult chinook salmon that can be attributed to direct exposure within the LDW during the juvenile outmigration is less than 1%. Recent studies indicate that

adult salmon have higher PCB body burdens as a result of time spent in Puget Sound (PSAT 2007; Missildine et al. 2005). Transport of contaminants from the LDW to Puget Sound could also result in indirect but site-related uptake of site-related contaminants during residence within Puget Sound. The magnitude of any such site-related uptake is unknown but likely to be small relative to other sources. The exclusion of salmon from seafood consumption scenarios and ingestion rates will underestimate site-related contaminant exposures and overall seafood consumption risks. Because the portion of total body burden resulting from uptake of contaminants from the LDW site is expected to be small, the effect of this underestimate on risk estimates is thought to be negligible.

B.6.1.2.3 Child seafood consumption

There are a number of uncertainties with tribal children's seafood consumption information. In general, regional tribal seafood consumption surveys included smaller numbers of children than adults and had a higher percentage of children reported as non-consumers than adults (Table B.6-9). Therefore, estimates of children's consumption have additional uncertainties beyond many of those described above for the adult seafood consumption scenarios. In the risk characterization, the child seafood consumption scenario based on Tulalip data was derived as a percentage of the adult tribal consumption rate based on Tulalip data. Because there are uncertainties in this approach and because actual child consumption rate data are available, an alternative approach using actual Tulalip child consumption rate data is presented here.

This section also presents a child tribal scenario based on Suquamish data and the associated risk estimates. As has been noted previously in this risk assessment regarding the evaluation of seafood consumption for individual API ethnic groups, drawing conclusions from small numbers of individuals creates uncertainty. In addition, as for any non-observational survey of young children, children's consumption rates were obtained by interviewing adults in the same household. Finally, for the Suquamish survey, multiple children may have been selected from the same household, leading to a lack of independence in the recorded data. This issue is discussed further as part of the Suquamish child seafood consumption scenario.

Table B.6-9. Number of participants in child and adult tribal seafood surveys

Tribe(s) (Source)	No. of Children Surveyed	No. of Children Consuming Seafood	No. of Adults Surveyed	No. of Adults Consuming Seafood
Tulalip Tribes (Toy et al. 1996)[a]	21	15	73	73
Squaxin Island (Toy et al. 1996)[a]	48	36	117	117
Suquamish Tribe (Suquamish Tribe 2000)[b]	31	31	92	92
Nez Perce, Yakama, Warm Springs, Umatilla (CRITFC 1994)	194	153	513	477

[a] Less than 1% of those (adults) contacted were excluded due to non-consumption of fish (Toy et al. 1996).

[b] All (adult) respondents consumed at least one type of fish or shellfish. Thus, no respondents were excluded because of non-consumption.

In comparing upper percentiles of children's seafood consumer-only consumption rates, the Tulalip Tribes' children's rates are below those reported for other tribes. Hence, use of the Tulalip Tribes' children's rate may underestimate children's exposures for other tribes. For this reason, 40% of the adult Tulalip Tribes' 95th percentile consumption rate (194 g/day),[40] or 77.6 g/day, was used to assess tribal children's seafood consumption risks in the risk characterization section. The rate of 77.6 g/day falls within the 95th percentiles of tribal children's seafood consumption rates estimated from other studies (Table B.6-10).

Table B.6-10. Child tribal seafood consumption

Tribe(s) (Source)	90th Percentile Child Seafood Consumption (g/day)	95th Percentile Child Seafood Consumption (g/day)
Tulalip Tribes (Toy et al. 1996)[a]	13.3[a]	20.4[b]
Squaxin Island Tribe (Toy et al. 1996)[b]	42.4[a]	115[a]
Nez Perce, Yakama, Warm Springs, Umatilla (CRITFC 1994)	53.2[c]	71.6[c]
Suquamish Tribe (Suquamish Tribe 2000)	50.6	122.2

[a] Based on re-analysis of original study data (EPA 2006d).

[b] The 95th percentile was computed using a lognormal distribution fit to the Tulalip children's consumption data (EPA 2006d; Kissinger 2007b).

[c] Derived for consumers only from CRITFC (1994).

Child Tribal 95th Percentile Seafood Consumption Scenario Based on Tulalip Data

Several approaches to developing child Tulalip consumption rates have been proposed and discussed in this HHRA. In the risk characterization, a ratio approach recommended by EPA (EPA 2006d) was used to develop the child tribal RME exposure scenario based on Tulalip data (Section B.3.4.1.2). Despite the issues of small sample size noted above, the children's data for the Tulalip Tribes are a measure of existing tribal children's seafood consumption. Thus, the Tulalip Tribes children's seafood consumption data (Toy et al. 1996) were used to provide an alternative estimate of children's seafood consumption risks.

EPA (2006d) calculated a 95th percentile total seafood consumption rate for only child consumers of 20.4 g/day based on seafood consumption reported in the Tulalip Tribes

[40] Forty percent of the adult seafood consumption rate is an option provided in EPA's draft *Framework for Selecting and Using Tribal Fish and Shellfish Consumption Rates for Risk-Based Decision Making at CERCLA and RCRA Cleanup Sites in Puget Sound and the Strait of Georgia* (EPA 2007b).

survey(Kissinger 2007b).[41] This rate reflects total reported seafood consumption from any source. The apportionment of this rate into seafood categories, based on the Tulalip child data (Toy et al. 1996), is presented in Tables B.6-11 through B.6-13. When data from the children's survey were available, apportionment was based on the children's consumption data. When such data were lacking, apportionment was based on Tulalip Tribes' adult consumption data (Toy et al. 1996). The approach for the apportionment is the same as that used for the adult tribal RME scenario based on Tulalip data, as described in Section B.3.4.1.2. The total consumption was first broken down into broad seafood groups and then into edible-meat and whole-body portions, as applicable.

Table B.6-11. Percentages and rates associated with different seafood categories for the child tribal 95th percentile seafood consumption scenario based on Tulalip data

Seafood Category	Percentage of Seafood Consumption[a]	Consumption Rate (g/day)[b]
Anadromous fish [c]	28	5.7[c]
Pelagic fish	18	3.7
Benthic fish	1	0.2
Shellfish	53	10.8

[a] Calculated from reported average consumption rates by seafood category based on the Tulalip child data (Toy et al. 1996).

[b] Calculated from 95th percentile of total seafood consumption (20.4 g/day) (Kissinger 2007b) using data from Toy et al. (1996) for children multiplied by percentage of consumption for Tulalip children of the various seafood categories.

[c] Consumption rate not used in this HHRA.

Table B.6-12. Consumption of crabs, clams, and mussels for the child tribal 95th percentile seafood consumption scenario based on Tulalip data

Shellfish Type	Percentage of Shellfish Consumption[a]	Consumption Rate (g/day)[b]
Crabs	53	5.7
Clams[c]	46	5.0
Mussels	1	0.11

[a] Same consumption percentages as for adult tribal RME scenario based on Tulalip data (Table B.3-26).

[b] Calculated from total child Tulalip shellfish rate (10.8 g/day, Table B.6-12) multiplied by percentage of adult Tulalip shellfish consumption for each category.

[c] Includes Manila/littleneck clams, horse clams, butter clams, cockles, oysters, and scallops.

[41] The Tulalip Tribes survey included 21 children aged 0 to 5 years, although only 15 consumed seafood (Toy et al. 1996).

Table B.6-13. Portions of crab consumed – child tribal 95th percentile scenario based on Tulalip data

Crab Portion	Percentage of Crab Consumption[a]	Consumption Rate (g/day)[b]
Crab edible meat	76	4.3
Crab whole-body	24	1.4

[a] Used same consumption percentages as in adult tribal RME scenario based on Tulalip data (Table B.3-27).

[b] Consumption percentages multiplied by total crab consumption (5.7 g/day from Table B.6-12).

The child tribal 95th percentile consumption rates based on Tulalip data were used with EPCs for the different seafood categories (Section B.3.4.3) to develop upper bound cancer risk and non-cancer hazard estimates (Table B.6-14). In this uncertainty assessment, estimated child tribal 95th percentile consumption rates based on Tulalip data (Table B.6-11 to B.6-13) were used with other exposure parameters (e.g., body weight, exposure duration) from Table B.3-9. As shown in Table B.6-14, the total cancer risk estimates based on the 95th percentile consumption (from any source) exceeded 1×10^{-6}, but were less than half the total cancer risk estimates for the child tribal RME scenario based on Tulalip data (using the 40% adult ratio, Table B.5-3). The most significant contributors to cancer risk were the same for both means of evaluating this scenario: PCBs (total PCBs and PCB TEQ) and arsenic. The risks associated with three tentatively identified (JN-qualified) pesticides that exceeded 1×10^{-6} (Table B.5-3) for the child tribal RME scenario based on Tulalip data (using the 40% adult ratio) did not exceed this threshold for child tribal 95th percentile scenario based on Tulalip data (Table B.6-14). The HQs for the child tribal 95th percentile scenario based on Tulalip data (Table B.6-15) were approximately one-half of the estimates for the child tribal RME scenario based on Tulalip data (using the 40% adult ratio) presented in Table B.5-11. TBT and vanadium had HQs that exceeded 1 for the child tribal RME scenario based on Tulalip data (using the 40% adult ratio, Table B.5-11) , but do not exceed that threshold for the child tribal 95th percentile scenario based on Tulalip data (Table B.6-15).

Table B.6-14. Excess cancer risk estimates for the child tribal 95th percentile seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor (kg/mg-day)$^{-1}$	Excess Cancer Risk
Arsenic (inorganic)[b,c]	Table B.3-34	6.1×10^{-5}	1.5	9×10^{-5}
Bis(2-ethylhexyl) phthalate	Table B.3-36	5.3×10^{-5}	0.014	7×10^{-7}
cPAHs[b,d,e]	Table B.3-36	6.1×10^{-7}	7.3	2×10^{-5}
PCB TEQ[b]	Table B.3-35	9.5×10^{-10}	150,000	1×10^{-4}
Total PCBs	Table B.3-35	7.2×10^{-5}	2	1×10^{-4}
Pentachlorophenol	Table B.3-36	6.7×10^{-5}	0.12	8×10^{-6}
			Subtotal excluding PCB TEQ	**2×10^{-4}**
			Subtotal excluding total PCBs	**2×10^{-4}**
Tentatively identified chemicals (JN-qualified)				
Aldrin[f]	Table B.3-35	2.1×10^{-7}	17	4×10^{-6}
alpha-BHC	Table B.3-35	2.0×10^{-7}	6.3	1×10^{-6}
beta-BHC	Table B.3-35	3.2×10^{-7}	1.8	6×10^{-7}
Carbazole	Table B.3-36	3.4×10^{-4}	0.02	7×10^{-6}
Total chlordane	Table B.3-35	2.2×10^{-6}	0.35	8×10^{-7}
Total DDTs	Table B.3-35	7.0×10^{-6}	0.34	2×10^{-6}
Dieldrin	Table B.3-35	5.4×10^{-7}	16	9×10^{-6}
gamma-BHC	Table B.3-35	2.7×10^{-7}	1.3	4×10^{-7}
Heptachlor	Table B.3-35	3.2×10^{-7}	4.5	1×10^{-6}
Heptachlor epoxide	Table B.3-35	3.4×10^{-7}	9.1	3×10^{-6}
Hexachlorobenzene	Table B.3-36	5.4×10^{-7}	1.6	9×10^{-7}
			Subtotal	**3×10^{-5}**
Total excluding PCB TEQ				**2×10^{-4}**
Total excluding total PCBs				**2×10^{-4}**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-36 through B.3-38).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic

[d] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for carcinogenic PAHs is based on dose adjustments across the 0-6 year age range of children. See Section B.5.1 for more information.

[e] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[f] Greater than 50% of the risk associated with this chemical (based on total dose) is derived from seafood categories with no detected values.

CDI – chronic daily intake
EPC – exposure point concentration
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
TEQ – toxic equivalent
ww – wet weight

Table B.6-15. Non-cancer hazard estimates for the child tribal 95th percentile seafood consumption scenario based on Tulalip data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (kg/mg-day)	Hazard Quotient
4-Methylphenol	Table B.3-36	5.8×10^{-4}	0.005	0.1
Antimony	Table B.3-36	3.4×10^{-5}	0.0004	0.08
Arsenic (inorganic)[b, c]	Table B.3-36	7.1×10^{-4}	0.0003	2
Bis(2-ethylhexyl) phthalate	Table B.3-36	6.2×10^{-4}	0.02	0.03
Butyl benzyl phthalate	Table B.3-36	5.0×10^{-4}	0.2	0.003
Cadmium	Table B.3-34	6.9×10^{-5}	0.001	0.07
Chromium	Table B.3-34	3.7×10^{-4}	0.003	0.1
Copper	Table B.3-34	6.4×10^{-3}	0.04	0.2
Mercury	Table B.3-34	4.0×10^{-5}	0.0001	0.4
Nickel	Table B.3-34	3.7×10^{-4}	0.02	0.02
Total PCBs	Table B.3-35	8.4×10^{-4}	0.00002	42
Pentachlorophenol	Table B.3-36	7.8×10^{-4}	0.03	0.03
TBT (as ion)	Table B.3-34	1.6×10^{-4}	0.00015	1
Vanadium	Table B.3-34	6.9×10^{-4}	0.001	0.7
Zinc	Table B.3-34	2.8×10^{-2}	0.3	0.09
			Subtotal	**47**
Tentatively identified chemicals (JN-qualified)				
Aldrin[d]	Table B.3-35	2.5×10^{-6}	0.00003	0.08
alpha-BHC	Table B.3-35	2.3×10^{-6}	0.0005	0.005
beta-BHC	Table B.3-35	3.7×10^{-6}	0.0002	0.02
Total chlordane	Table B.3-35	2.5×10^{-5}	0.0005	0.05
Total DDTs	Table B.3-35	8.1×10^{-5}	0.0005	0.2
Dieldrin	Table B.3-35	6.3×10^{-6}	0.00005	0.1
Endrin	Table B.3-35	3.1×10^{-6}	0.0003	0.01
Endrin aldehyde	Table B.3-35	2.1×10^{-5}	0.0003	0.07
gamma-BHC	Table B.3-35	3.2×10^{-6}	0.0003	0.01
Heptachlor	Table B.3-35	3.8×10^{-6}	0.0005	0.008
Heptachlor epoxide	Table B.3-35	4.0×10^{-6}	0.000013	0.3
Hexachlorobenzene	Table B.3-36	6.2×10^{-6}	0.0008	0.008
			Subtotal	**0.9**
Hazard indices by effect:				
Hazard Index for Cardiovascular Endpoint[e]				**3**
Hazard Index for Developmental Endpoint[f]				**42**
Hazard Index for Hematologic Endpoint[g]				**0.2**
Hazard Index for Immunological Endpoint[h]				**43**
Hazard index for Kidney Endpoint[i]				**0.4**

CHEMICAL	EPC (mg/kg ww)[a]	NON-CANCER CDI (mg/kg-day)	REFERENCE DOSE (kg/mg-day)	HAZARD QUOTIENT
Hazard index for Liver Endpoint[j]				**1**
Hazard index for Neurological Endpoint[k]				**43**
Hazard index for Dermal Endpoint[l]				**2**
Total Risk across all exposure routes/pathways[m]				**48**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[e] Cardiovascular endpoint is for arsenic and vanadium.

[f] Developmental endpoint is for PCBs and mercury.

[g] Hematologic endpoint is for antimony and zinc.

[h] Immunological endpoint is for PCBs and TBT.

[I] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[j] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[k] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[l] Dermal endpoint is for 4-methylphenol and arsenic.

[m] This total is not directly interpretable for risk assessment. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Child Tribal Scenario Based on Suquamish Data

Estimates of child tribal risks for the scenario based on Suquamish data were not provided in the risk characterization section of this document because of the high uncertainty associated with this scenario and EPA's assessment that the Tulalip Tribes' consumption rates were more appropriate for the LDW than the Suquamish consumption rates (EPA 2005a). The Suquamish Tribe survey (Suquamish Tribe 2000) included 31 children from 0 to 6 years old from 21 different households, and provides a 95th percentile estimate of children's seafood consumption[42] equal to 122.2 g/day.[43] This report also provides data on what kinds of seafood Suquamish children consume.

Child-specific rates appropriate for apportionment of total seafood consumption to different seafood categories were based on information on categories of seafood consumed by Suquamish children presented in the consumption survey by the Suquamish Tribe (2000). Children's seafood consumption is potentially influenced by the consumption patterns of adults living in the same household. Multiple children from the same household were selected for the Suquamish survey. Consequently, consumption data for child participants living in the same household are not independent. The effect of the lack of independence in children's consumption rates on the overall calculated consumption rate is unclear.

Suquamish child total consumption data was apportioned into seafood categories using the same basic approach for apportionment used above for the child tribal 95th percentile scenario based on Tulalip data (described in detail in Section B.3.4.1.2) was used to apportion the child tribal total consumption based on Suquamish data into seafood categories (Tables B.6-16 and B.6-17). Again, the conservative assumption was made that all seafood consumed by children was from the LDW. The survey did not report the portion of children's seafood consumption from Puget Sound versus other sources. For adults, an average of 19% or more consumption from each of the major seafood categories (e.g., anadromous, shellfish) was reported as from sources other than "caught in Puget Sound" (Suquamish Tribe 2000). The apportionment approach involved first dividing the total seafood consumption into broad categories, and then dividing the shellfish portion into the specific shellfish types consumed. No children's benthic fish consumption other than fillet (e.g., organs or whole fish) was reported (Suquamish Tribe 2000), so all benthic fish consumption was assumed to be fillet. Similarly, no children's consumption of crab other than edible meat was reported (Suquamish Tribe 2000), so all crab consumption was assumed to be edible meat. The apportionment of consumption for the scenario based on Suquamish children's

---

[42] The Suquamish seafood consumption study included 31 children 0 to 6 years old, although the survey included responses from only 20 adults reflecting children's consumption in 21 households. The 95th percentile rate was provided for consumers and non consumers (combined), but all children reported consumption (Suquamish Tribe 2000).

[43] Product of consumption in reported 95th percentile consumption of 7.272 g/kg/day and average children's body weight of 16.8 kg (Suquamish Tribe 2000).

consumption rates was very similar to that of the scenario based on Suquamish adult consumption rates (Section B.3.4.1).

Table B.6-16. Percentages and rates associated with different seafood categories for the child tribal seafood consumption scenario based on Suquamish data

Seafood Category	Percentage of Seafood Consumption[a]	Consumption Rate (g/day)[b]
Anadromous fish	21.9	26.8[c]
Pelagic fish	10.9	13.3
Benthic fish	2.4	3.0[d]
Shellfish	64.8	79.1

[a] Calculated from reported average children's consumption rates by seafood category (Suquamish Tribe 2000).

[b] Calculated from 95th percentile of reported child Suquamish total seafood consumption (122.2 g/day) multiplied by percentage of consumption of the various seafood categories.

[c] Consumption rate not used in this HHRA.

[d] No children's consumption of benthic fish other than fillet was reported (Suquamish Tribe 2000), so all benthic fish consumption was assumed to be benthic fish fillet.

Table B.6-17. Consumption of crabs, clams, and mussels for the child tribal seafood consumption scenario based on Suquamish data

Shellfish Type	Percentage of Shellfish Consumption[a]	Consumption Rate (g/day)[b]
Crabs	43.5	34.5[c]
Clams[d]	56.4	44.6
Mussels	0.1	0.11

[a] Calculated from reported average children's consumption rates by seafood category (Suquamish Tribe 2000).

[b] Consumption percentages multiplied by total shellfish consumption (79.1 g/day from Table B.6-16).

[c] Adults were asked about children's consumption of crab parts. No children's consumption of whole crab or crab butter (i.e., heptaopancreas) was reported (Suquamish Tribe 2000), so all crab consumption was assumed to be edible meat.

[d] Includes Manila/littleneck clams, horse clams, butter clams, cockles, oysters, and scallops.

The child tribal consumption rates based on Suquamish data were used with EPCs for the different seafood categories (Section B.3.4.3) to develop cancer and non-cancer risk estimates. Consumption rates from Tables B.6-16 and B.6-17 were used with other exposure parameters (e.g., exposure duration) from Table B.3-9 with one exception. The reported average body weight for Suquamish children (16.8 kg) from the Suquamish survey (Suquamish Tribe 2000) was used for this parameter.

Upper bound cancer risk and non-cancer hazard estimates are presented in Tables B.6-18 and B.6-19. Upper bound cancer risk estimates exceeded 1×10^{-6}, and HQs exceeded 1 for several individual chemicals and most endpoints. Upper bound cancer risk estimates and non-cancer hazards were less than those for the adult tribal

scenario based on Suquamish data (Tables B.5-5 and B.5-13), but they exceeded those for the child tribal 95th percentile scenario based on Tulalip data (Tables B.6-14 and B.6-15) and the child tribal RME scenario based on Tulalip data (using the 40% adult ratio, Tables B.5-3 and B.5-11).

Table B.6-18. Excess cancer risk estimates for the child tribal seafood consumption scenario based on Suquamish data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor (kg/mg-day)$^{-1}$	Excess Cancer Risk
Arsenic (inorganic)[b, c]	Table B.3-34	4.7×10^{-4}	1.5	7×10^{-4}
Bis(2-ethylhexyl) phthalate	Table B.3-36	2.1×10^{-4}	0.014	3×10^{-6}
cPAHs[b, d, e]	Table B.3-36	4.7×10^{-6}	7.3	2×10^{-4}
PCB TEQ[b]	Table B.3-35	3.6×10^{-9}	150,000	5×10^{-4}
Total PCBs	Table B.3-35	3.2×10^{-4}	2	6×10^{-4}
Pentachlorophenol	Table B.3-36	3.0×10^{-4}	0.12	4×10^{-5}
			Subtotal excluding PCB TEQ	**2×10^{-3}**
			Subtotal excluding total PCBs	**1×10^{-3}**
Tentatively identified chemicals (JN-qualified)				
Aldrin[f]	Table B.3-35	1.2×10^{-6}	17	2×10^{-5}
alpha-BHC[f]	Table B.3-35	1.0×10^{-6}	6.3	7×10^{-5}
beta-BHC	Table B.3-35	1.6×10^{-6}	1.8	3×10^{-6}
Carbazole	Table B.3-36	1.3×10^{-3}	0.02	3×10^{-5}
Total chlordane	Table B.3-35	7.9×10^{-6}	0.35	3×10^{-6}
Total DDTs	Table B.3-35	2.5×10^{-5}	0.34	9×10^{-6}
Dieldrin	Table B.3-35	3.7×10^{-6}	16	6×10^{-5}
gamma-BHC	Table B.3-35	1.5×10^{-6}	1.3	2×10^{-6}
Heptachlor[f]	Table B.3-35	1.5×10^{-6}	4.5	7×10^{-6}
Heptachlor epoxide	Table B.3-35	1.4×10^{-6}	9.1	1×10^{-5}
Hexachlorobenzene	Table B.3-36	2.6×10^{-6}	1.6	4×10^{-6}
			Subtotal	**2×10^{-4}**
Total excluding PCB TEQ				**2×10^{-3}**
Total excluding total PCBs				**2×10^{-3} [g]**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 through B.3-36).

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[d] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for cPAHs is based on dose adjustments across the 0-to-6-year age range of children. See Section B.5.1 for more information.

[e] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from 2004 only because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

[f] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[g] The total excess cancer risk value does not equal the sum of the two subtotals. This occurs because the total value is calculated by summing the the risks for each individual chemical, rather than the two subtotals.

BHC – benzene hexachloride

CDI – chronic daily intake

cPAH – carcinogenic polycyclic aromatic hydrocarbon

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

Table B.6-19. Non-cancer hazard estimates for the child tribal seafood consumption scenario based on Suquamish data

Scenario timeframe: Current/future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor population: Tribal fish and shellfish consumers
Receptor age: Child

Chemical	EPC (mg/kg ww)[a]	Non-Cancer CDI (mg/kg-day)	Reference Dose (kg/mg-day)	Hazard Quotient
4-Methylphenol[b]	Table B.3-36	2.6×10^{-3}	0.005	0.5
Antimony	Table B.3-34	2.6×10^{-4}	0.0004	0.7
Arsenic (inorganic)[c, d]	Table B.3-34	5.5×10^{-3}	0.0003	18
Bis(2-ethylhexyl) phthalate	Table B.3-36	2.5×10^{-3}	0.02	0.1
Butyl benzyl phthalate[b]	Table B.3-36	2.1×10^{-3}	0.2	0.01
Cadmium	Table B.3-34	3.6×10^{-4}	0.001	0.4
Chromium	Table B.3-34	2.6×10^{-3}	0.003	0.9
Copper	Table B.3-34	3.5×10^{-2}	0.04	0.9
Mercury	Table B.3-34	2.3×10^{-4}	0.0001	2
Nickel	Table B.3-34	2.3×10^{-3}	0.02	0.1
Total PCBs	Table B.3-35	3.7×10^{-3}	0.00002	186
Pentachlorophenol	Table B.3-36	3.5×10^{-3}	0.03	0.1
TBT (as ion)	Table B.3-34	1.2×10^{-3}	0.00015	8
Vanadium	Table B.3-34	4.7×10^{-3}	0.001	5
Zinc	Table B.3-34	1.6×10^{-1}	0.3	0.5
			Subtotal	**223**
Tentatively identified chemicals (JN-qualified)				
Aldrin[b]	Table B.3-35	1.4×10^{-5}	0.00003	0.5
alpha-BHC[b]	Table B.3-35	1.2×10^{-5}	0.0005	0.02
beta-BHC	Table B.3-35	1.8×10^{-5}	0.0002	0.09
Total chlordane	Table B.3-35	9.2×10^{-5}	0.0005	0.2
Total DDTs	Table B.3-35	2.9×10^{-4}	0.0005	0.6
Dieldrin	Table B.3-35	4.3×10^{-4}	0.00005	0.9
Endrin	Table B.3-35	1.5×10^{-5}	0.0003	0.05
Endrin aldehyde	Table B.3-35	7.6×10^{-5}	0.0003	0.3
gamma-BHC	Table B.3-35	1.7×10^{-5}	0.0003	0.06
Heptachlor[b]	Table B.3-35	1.7×10^{-5}	0.0005	0.03
Heptachlor epoxide	Table B.3-35	1.7×10^{-5}	0.000013	1
Hexachlorobenzene	Table B.3-36	3.0×10^{-5}	0.0008	0.04
			Subtotal	**4**
Hazard indices by effect:				
Hazard Index for Cardiovascular Endpoint[e]				**23**
Hazard Index for Developmental Endpoint[f]				**188**
Hazard Index for Hematologic Endpoint[g]				**1**
Hazard Index for Immunological Endpoint[h]				**194**
Hazard index for Kidney Endpoint[i]				**2**

CHEMICAL	EPC (mg/kg ww)[a]	NON-CANCER CDI (mg/kg-day)	REFERENCE DOSE (kg/mg-day)	HAZARD QUOTIENT
Hazard index for Liver Endpoint[j]				**6**
Hazard index for Neurological Endpoint[k]				**189**
Hazard index for Dermal Endpoint[l]				**19**
Total Risk across all exposure routes/pathways[m]				**227**

[a] An EPC for each seafood category was calculated in the exposure section (see Tables B.3-34 through B.3-36).

[b] Greater than 50% of the risk associated with this chemical is derived from seafood categories with no detected values.

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[d] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[e] Cardiovascular endpoint is for arsenic and vanadium.

[f] Developmental endpoint is for PCBs and mercury.

[g] Hematologic endpoint is for antimony and zinc.

[h] Immunological endpoint is for PCBs and TBT.

[I] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[j] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[k] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[l] Dermal endpoint is for 4-methylphenol and arsenic.

[m] This total is not directly interpretable for risk assessment. The value indicates that the HI may exceed 1 for individual endpoints. Therefore, HIs were calculated for individual endpoints.

BHC – benzene hexachloride

CDI – chronic daily intake

EPC – exposure point concentration

PCB – polychlorinated biphenyl

TBT – tributyltin

ww – wet weight

B.6.1.3 Exposure duration for Asian and Pacific Islander seafood consumption scenario

Uncertainty regarding the ingestion rate for the API RME scenario was discussed above (Section 6.1.2). There is also uncertainty surrounding the value selected for the exposure duration in that scenario. EPA (1997) has calculated a 90th percentile residence time in the same household for the general US population of approximately 30 years. The API population in King County is concentrated in areas near the LDW. However, the residence time of API in the vicinity of the LDW may be different from that of the general population. The mobility of API individuals who may use the LDW as a primary or exclusive fishing resource is unknown. There are two main sources of uncertainty regarding exposure duration of API individuals consuming seafood from the LDW. First, it is possible that API residents remain in areas near the LDW for longer than 30 years, and second, it is possible that even when they move away from the LDW, they may return in order to maintain connection with their communities and to catch seafood in the river.

No modeling studies have yet been completed to analyze residence time of API in neighborhoods bordering the LDW, and there are no known studies of similar settings or populations to use as a surrogate for the API population. A modeling effort was conducted for the Hudson River HHRA (TAMS and Gradient 2000) to examine residence time in the five counties directly adjacent to the Hudson River. The 90th percentile value for residence time in that analysis was found to be 40 years. However, because of differences between the Hudson River and LDW including scale (i.e., the Hudson study included five counties and 40 miles of river; the LDW includes a single county and just 5 miles of river), the 40-year exposure duration identified in the Hudson River HHRA may not be applicable to the LDW. However, this modeling effort indicates that exposure durations may differ from the EPA-recommended default for some populations.

In the absence of site-specific information or appropriate surrogate information, EPA's *Exposure Factors Handbook* (1997) reports in Table 15-167 that the general US population has a 95th percentile residence time of 41 years. In order to investigate the effect of assumptions of longer exposure durations on the risk estimates for the API scenario, risks were calculated using an exposure duration of 41 years. This exposure duration is significantly longer than the current exposure duration of 30 years for the API RME scenario. Table B.6-20 presents the results of the CDI and excess cancer risk calculations derived with this longer exposure duration and well the 30-year duration presented previously in the risk characterization.

Table B.6-20. Comparison of API RME risks with 41-year exposure duration

Chemical	30-Year Exposure Duration		41-Year Exposure duration	
	Cancer CDI (mg/kg-day)	Excess Cancer Risk	Cancer CDI (mg/kg-day)	Excess Cancer Risk
Arsenic	4.4×10^{-4}	7×10^{-4}	6.0×10^{-4}	9×10^{-4}
Bis(2-ethylhexyl) phthalate	1.3×10^{-4}	2×10^{-6}	1.8×10^{-4}	3×10^{-6}
cPAHs	4.4×10^{-6}	3×10^{-5}	6.1×10^{-6}	4×10^{-5}
PCB TEQ	2.6×10^{-9}	4×10^{-4}	3.6×10^{-9}	5×10^{-4}
Total PCBs	2.5×10^{-4}	5×10^{-4}	4.0×10^{-4}	8×10^{-4}
Pentachlorophenol	1.8×10^{-4}	2×10^{-5}	2.4×10^{-4}	3×10^{-5}
Subtotal excluding PCB TEQ		**1×10^{-3}**		**2×10^{-3}**
Subtotal excluding total PCBs		**1×10^{-3}**		**1×10^{-3}**
Tentatively identified chemicals (JN-qualified)				
Aldrin	6.0×10^{-7}	1×10^{-5}	8.2×10^{-7}	1×10^{-5}
alpha-BHC	5.0×10^{-7}	3×10^{-6}	6.8×10^{-7}	4×10^{-6}
beta-BHC	8.0×10^{-7}	1×10^{-6}	1.1×10^{-6}	2×10^{-6}
Carbazole	5.9×10^{-4}	1×10^{-5}	8.1×10^{-4}	2×10^{-5}
Total chlordane	5.0×10^{-6}	2×10^{-6}	6.9×10^{-6}	2×10^{-6}
Total DDTs	1.7×10^{-5}	6×10^{-6}	2.3×10^{-5}	8×10^{-6}
Dieldrin	2.8×10^{-6}	5×10^{-5}	3.9×10^{-6}	6×10^{-5}
gamma-BHC	1.0×10^{-6}	1×10^{-6}	1.4×10^{-6}	2×10^{-6}
Heptachlor	7.2×10^{-7}	3×10^{-6}	1.4×10^{-6}	4×10^{-6}
Heptachlor epoxide	9.8×10^{-7}	9×10^{-6}	1.3×10^{-6}	1×10^{-5}
Hexachlorobenzene	1.5×10^{-6}	2×10^{-6}	2.0×10^{-6}	3×10^{-6}
Subtotal		**1×10^{-4}**		**1×10^{-4}**
Total risk across all exposure routes/ pathways excluding PCB TEQ		**1×10^{-3}**		**2×10^{-3}**
Total risk across all exposure routes/ pathways excluding total PCBs		**1×10^{-3}**		**1×10^{-3}**

API – Asian and Pacific Islander
CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

As shown in Table B.6-20, increasing the exposure duration from 30 to 41 years doubles the current estimate of total excess cancer risk (see Section B.5.3.1.1) from 1×10^{-3} to 2×10^{-3} when the total risk across all pathways excluding PCB TEQ is summed. The doubling of the excess cancer risk estimate when the exposure duration increased by only 37% is a consequence of representing the excess cancer risk

estimates to only one significant digit. However, when the excess cancer risks are summed excluding total PCBs, the total risks associated with the longer exposure duration are unchanged. Although the appropriate exposure duration for API users of the LDW is unknown, these results provide an upper-bound risk estimate for members of the general population who may be less mobile or who move away but choose to continue to use the LDW as their harvesting resource.

B.6.1.4 Fraction of dose obtained from site

For the Phase 1 and baseline HHRAs, the fractional intake of dose obtained from the LDW was set at 1 by default for all exposure pathways. This assumption is appropriate for the netfishing scenario, which occurs primarily within the LDW. For the seafood consumption scenario, however, there is more uncertainty regarding the degree to which seafood consumers would use only the LDW for collection of fish and shellfish, and thus there is uncertainty in the selection of an appropriate FI value. For the beach play RME scenario, it is also possible that beaches outside the LDW are utilized.

B.6.1.4.1 Fraction of dose obtained from site for seafood consumption scenarios

There are a number of factors to consider in selecting an FI for the seafood consumption scenario. EPA's draft framework document for tribal seafood consumption rates (EPA 2007b) used the fraction of seafood caught in Puget Sound by the Tulalip or Suquamish tribal members, and assumed that all catch from Puget Sound could be obtained from the LDW (i.e., FI = 1). Based on discussions with EPA, an FI value of 1 was selected for all seafood consumption scenarios in this HHRA because site-specific data are insufficient to derive specific quantitative estimates of FI values that are applicable to the RME individuals within the tribal or API consumer groups. The applied FI of 1 likely overestimates current exposures associated with the LDW for most individuals, specifically those individuals who consume a portion of their seafood intake from outside the LDW or whose seafood intake is partly made up of species not found in the LDW. However, such an approach was required by EPA as consistent with an RME approach.

Another important factor in selecting an FI value is the consideration of future resource quality. For at least the highest consumption scenario explored in the risk assessment, there is concern about the availability of shellfish, "EPA believes that use of Suquamish exposure parameters will not provide the best estimate of LDW tribal seafood consumption risks due to the degraded habitat in the LDW and questions whether the high Suquamish shellfish consumption rate could be sustained" (EPA 2005a). There is a high interest in improving habitat quality in the LDW; however, current and future restoration efforts may affect the quantity of harvestable seafood. It is possible that resource availability and use could increase in the future following remediation. For the one-meal-per-month scenarios, an FI of 1 for the LDW is

appropriate because the consumption rates for these scenarios are intended to provide information on health risks for individuals on a per-meal basis.

Because the use of an FI of 1 may overestimate risks for many site users, and to offer different perspectives for risk management decisions, order-of-magnitude variations (i.e., 0.1 and 0.01, which correspond to 10% and 1% site use) of the default FI value of 1 were evaluated for the tribal and API seafood consumption scenarios. Even at an FI of 0.01, the combined excess cancer risk estimates for all chemicals were greater than 1×10^{-6} for all but the child tribal CT scenario based on Tulalip data and the adult API CT scenario (Table B.6-21). However, at an FI of 0.01, only the Suquamish estimates are greater than the upper end of EPA's identified range of acceptable risks of 10^{-4}, and at the 0.01 fractional intake, the tribal and adult API risk estimates are less than or similar to one-meal-per-month risk estimates assuming an FI of 1. Some chemicals, such as some of the JN-qualified organochlorine pesticides, would no longer exceed acceptable risk levels if an FI of 0.1 or 0.01 was assumed.

Table B.6-21. Excess cancer risk estimates for seafood consumption exposure scenarios using alternative assumptions for fractional intake of dose obtained from the site

Seafood Consumption Scenario[a]	Excess Cancer Risk		
	Fractional Intake = 1	Fractional Intake = 0.1	Fractional Intake = 0.01
Adult tribal RME (Tulalip data)	3×10^{-3}	3×10^{-4}	3×10^{-5}
Adult tribal CT (Tulalip data)	1×10^{-4}	1×10^{-5}	1×10^{-6}
Child tribal RME (Tulalip data)	7×10^{-4}	7×10^{-5}	7×10^{-6}
Child tribal CT (Tulalip data)	7×10^{-5}	7×10^{-6}	7×10^{-7}
Adult tribal (Suquamish data)	3×10^{-2}	3×10^{-3}	3×10^{-4}
API – RME	1×10^{-3}	1×10^{-4}	1×10^{-5}
API – CT	2×10^{-5}	2×10^{-6}	2×10^{-7}
Adult one meal per month – benthic fish	1×10^{-4}	[b]	[b]
Adult one meal per month – clam	2×10^{-4}	[b]	[b]
Adult one meal per month – crab	3×10^{-5}	[b]	[b]
Adult one meal per month – pelagic fish	2×10^{-4}	[b]	[b]

[a] Excess cancer risk estimates represent totals for all chemicals, excluding PCB TEQ.

[b] Fractional intake of 1 considered most appropriate for one-meal-per-month scenario.

CT – central tendency

RME – reasonable maximum exposure

B.6.1.4.2. Fraction of dose obtained from site for direct sediment exposure scenarios

For the beach play RME scenario, exposure frequency represents how often children may play in LDW sediments. The HHRA uses a health-protective FI value of 1 for all of the beach play RME scenarios and alternative FI assumptions were not quantitatively explored. It seems reasonable that on days when children are playing in LDW intertidal areas, particularly those areas within walking distance of

neighborhoods such as South Park (e.g., area 5), the majority of their incidental (upland) sediment/soil intake would consist of LDW sediments as opposed to terrestrial soil or sediment from other non-LDW beaches. However, for areas not accessible by walking from a neighborhood, people may choose to drive or bus to other nearby beach areas, such as Alki Beach, rather than use LDW beaches exclusively. In such cases, a lower FI may be warranted, leading to proportionally lower risk estimates.

As with the exposure to sediments related to seafood-gathering scenarios, alternatives to the default FI value of 1 (i.e., 0.5 and 0.1, which correspond to 50% and 10% site use) were evaluated for the clamming and netfishing scenarios (Table B.6-22). For the netfishing and clamming scenarios at an FI of 0.5, the highest risk estimate was 2×10^{-5}, and with an FI of 0.1, all excess cancer risk estimates for the clamming scenarios were equal to or below 1×10^{-5} and some were below 1×10^{-6}. The risk estimates for clamming and netfishing, as well as beach play, would be altered if the beaches visited (for clamming and netfishing) or areas used (for netfishing) within the site were different from those assumed for this risk assessment.

Table B.6-22. Excess cancer risk estimates for direct sediment exposure scenarios using alternative assumptions for fractional intake of dose obtained from the site

Direct Sediment Exposure Scenario	Excess Cancer Risk[a]		
	Fractional Intake = 1	Fractional Intake = 0.5	Fractional Intake = 0.1
Netfishing RME	3×10^{-5}	1×10^{-5}	[b]
Netfishing CT	5×10^{-6}	3×10^{-6}	[b]
Clamming – 7 days per year	2×10^{-6}	7×10^{-7}	1×10^{-7}
Tribal Clamming RME (120 days per year)	1×10^{-4}	7×10^{-5}	1×10^{-5}
Tribal Clamming – 183 days per year	2×10^{-4}	1×10^{-4}	2×10^{-5}

[a] Excess cancer risk estimates represent totals for all chemicals, excluding PCB TEQ.

[b] Not evaluated because majority of netfishing thought to occur in the LDW.

CT – central tendency

RME – reasonable maximum exposure

B.6.1.5 Dermal exposure

B.6.1.5.1 Chemicals lacking guidance on absorption factors

Dermal exposure to 13 metals identified as COPCs for the direct sediment exposure scenarios was not evaluated because these chemicals lacked dermal absorption factors. EPA guidance states that "for inorganics, the speciation of the compound is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value" (EPA 2004d). Therefore, only incidental ingestion for these 13 metals was considered in the risk characterization (Sections B.5.3.2 to B.5.3.4). To investigate whether this approach may have resulted in a significant underestimation of risk from

exposure to these metals, risk estimates were calculated for these metals using the MTCA default dermal absorption value of 0.01 for inorganics. No HQ exceedances of 1 occurred for any scenario when this value was applied to these chemicals.

In addition, risks were calculated for one of the metals assuming several different absorption factors to assess the impacts on risk estimates of different assumptions for dermal absorption values. Vanadium was selected because the maximum concentration in sediment exceeded its sediment risk-based criterion by more than 10-fold in the COPC screening process, and it has one of the lowest RfDs of the metals that lack an absorption factor term.[44] Therefore, the inclusion of dermal exposure to vanadium would be expected to have a more significant impact on sediment risk estimates than the inclusion of dermal exposure to other metals. Only non-cancer hazards were considered because vanadium, as with other metals that lack absorption factors, has not been demonstrated to cause cancer.

Table B.6-23 presents hypothetical HQs for the direct sediment exposure scenarios assuming a range of possible dermal absorption factors for vanadium. A value of 0.01 has been presented by California EPA (2005) and is the MTCA default dermal absorption value for inorganics. Exposure via incidental sediment ingestion was also included so that total risks associated with direct sediment exposure could be assessed (see Sections B.3.3 and B.3.4 for details on incidental sediment ingestion risk estimates). Assuming the highest proportion of dermal absorption recommended by EPA (2004d) for any metal to date (0.03 for arsenic), hypothetical HQs were still less than 1 for all direct-contact scenarios. At an assumed dermal absorption factor of 0.01, which was also the dermal absorption factor recommended in the EPA dermal guidance prior to the current guidance, the dermal absorption contribution to the HQ was equal to or greater than the contribution from incidental sediment ingestion for all scenarios except the netfishing CT and beach play RME (Areas 2, 4, 7, and 8) scenarios (Table B.6-23).

Thus, the risk estimates for those chemicals that lack dermal absorption factors are somewhat uncertain because it is not possible to quantitatively identify the risk without a dermal absorption factor. However, the hypothetical dermal absorption factors assumed in this example (Table B.6-23) provide boundaries for the range of possible risk values associated with exposure to vanadium. Specifically, EPA guidance provides dermal absorption factors for only two metals (0.03 for arsenic and 0.001 for cadmium). The hypothetical dermal absorption factors assumed in this table (0.03, 0.01, and 0.001) were selected to represent a range of possible values. Application of

[44] Mercury, antimony, and thallium lack absorption fractions and have lower RfDs than vanadium. In the HQ calculation for dermal vanadium exposure, the vanadium RfD was multiplied by an oral adjustment factor (0.026) because vanadium lacks a dermal RfD (see footnote b for Table B.6-18). This adjustment effectively gives vanadium a lower dermal RfD than antimony and thallium, which do not have oral adjustment factors (EPA 2004d). Mercury was not selected as an example chemical because mercury speciation affects toxicity and is poorly characterized for sediment.

these assumed absorption values indicates that this source of uncertainty (i.e., lack of dermal absorption factors for some metals) is unlikely to affect overall conclusions from the risk characterization about risks associated with dermal exposure to metals in sediment.

Table B.6-23. Hypothetical non-cancer hazard estimates for vanadium using three dermal absorption factors for the direct sediment exposure scenarios

Dermal Absorption Factor[a]	CDI (mg/kg-day)		Hazard Estimate		
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption[b]	Total
Netfishing RME					
0.03	1.2×10^{-5}	5.2×10^{-6}	0.01	0.2	0.2
0.01	1.2×10^{-5}	1.7×10^{-6}	0.01	0.07	0.08
0.001	1.2×10^{-5}	1.7×10^{-7}	0.01	0.007	0.02
Netfishing CT					
0.03	6.3×10^{-6}	2.7×10^{-7}	0.006	0.01	0.02
0.01	6.3×10^{-6}	9.1×10^{-8}	0.006	0.004	0.01
0.001	6.3×10^{-6}	9.1×10^{-9}	0.006	0.0004	0.006
Beach Play RME, Area 1					
0.03	1.2×10^{-4}	7.5×10^{-6}	0.1	0.3	0.4
0.01	1.2×10^{-4}	2.5×10^{-6}	0.1	0.1	0.2
0.001	1.2×10^{-4}	2.5×10^{-7}	0.1	0.01	0.1
Beach Play RME, Area 2					
0.03	1.8×10^{-4}	1.1×10^{-5}	0.2	0.4	0.6
0.01	1.8×10^{-4}	3.5×10^{-6}	0.2	0.1	0.3
0.001	1.8×10^{-4}	3.5×10^{-7}	0.2	0.01	0.2
Beach Play RME, Area 3					
0.03	1.3×10^{-4}	7.9×10^{-6}	0.1	0.3	0.4
0.01	1.3×10^{-4}	2.6×10^{-6}	0.1	0.1	0.2
0.001	1.3×10^{-4}	2.6×10^{-7}	0.1	0.01	0.1
Beach Play RME, Area 4					
0.03	1.5×10^{-4}	9.0×10^{-6}	0.2	0.3	0.5
0.01	1.5×10^{-4}	3.0×10^{-6}	0.2	0.1	0.3
0.001	1.5×10^{-4}	3.0×10^{-7}	0.2	0.01	0.2
Beach Play RME, Area 5					
0.03	1.5×10^{-4}	8.7×10^{-6}	0.1	0.3	0.4
0.01	1.5×10^{-4}	2.9×10^{-6}	0.1	0.1	0.2
0.001	1.5×10^{-4}	2.9×10^{-7}	0.1	0.01	0.1

Dermal Absorption Factor[a]	CDI (mg/kg-day)		Hazard Estimate		
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption[b]	Total
Beach Play RME, Area 6					
0.03	1.3×10^{-4}	7.8×10^{-6}	0.1	0.3	0.4
0.01	1.3×10^{-4}	2.6×10^{-6}	0.1	0.1	0.2
0.001	1.3×10^{-4}	2.6×10^{-7}	0.1	0.01	0.1
Beach Play RME, Area 7					
0.03	1.7×10^{-4}	1.0×10^{-5}	0.2	0.4	0.6
0.01	1.7×10^{-4}	3.4×10^{-6}	0.2	0.1	0.3
0.001	1.7×10^{-4}	3.4×10^{-7}	0.2	0.01	0.2
Beach Play RME, Area 8					
0.03	1.5×10^{-4}	9.0×10^{-6}	0.2	0.3	0.5
0.01	1.5×10^{-4}	3.0×10^{-6}	0.2	0.1	0.3
0.001	1.5×10^{-4}	3.0×10^{-7}	0.2	0.01	0.2
Clamming – 7 days per year					
0.03	1.4×10^{-6}	5.1×10^{-7}	0.001	0.02	0.02
0.01	1.4×10^{-6}	1.7×10^{-7}	0.001	0.007	0.008
0.001	1.4×10^{-6}	1.7×10^{-8}	0.001	0.0007	0.002
Tribal clamming RME					
0.03	2.3×10^{-5}	8.2×10^{-6}	0.02	0.3	0.3
0.01	2.3×10^{-5}	2.7×10^{-6}	0.02	0.1	0.1
0.001	2.3×10^{-5}	2.7×10^{-7}	0.02	0.01	0.03
Tribal Clamming – 183 days per year					
0.03	3.4×10^{-5}	1.2×10^{-5}	0.03	0.5	0.5
0.01	3.4×10^{-5}	4.1×10^{-6}	0.03	0.2	0.2
0.001	3.4×10^{-5}	4.1×10^{-7}	0.03	0.02	0.05

Note: The incidental sediment ingestion estimates were presented in the risk characterization (Section B.5) and are included here for completeness.

[a] EPA guidance provides an dermal absorption factor for only two metals (0.03 for arsenic and 0.001 for cadmium). The hypothetical dermal absorption factors assumed in this table (0.03, 0.01, and 0.001) were selected to represent a range of possible values.

[b] Oral adjustment factor for vanadium = 0.026 (EPA 2004b). Dermal HQ = CDI/(RfD × 0.026) (EPA 2004d).

CDI – chronic daily intake

B.6.1.5.2 Dermal adherence factors used for sediment exposure scenarios

Dermal adherence factors are used to estimate the amount of sediment that adheres to exposed skin in the assessment of risks posed by dermal exposure to sediment. EPA (2004d) recommends a dermal adherence value of 0.2 mg/cm^2 as a default health-protective factor for exposures of children and adults to moist soil. There are three main sources of uncertainty surrounding the use of this parameter in risk assessment

scenarios involving marine sediments such as found at the LDW. The first source of uncertainty is related to the limited data from field studies used as the basis for the EPA recommendation. Nearly all the studies used by EPA in determining dermal adherence factors focused on exposure to terrestrial soil. However, direct sediment exposure data were derived from only two studies: an investigation of sediment adherence for adults gathering reeds in marine sediment and an inland study of children playing in mud along the shoreline of a lake (EPA 2004d). Neither of these studies involved children exposed to intertidal marine sediments similar to those found in the LDW.

The second main source of uncertainty regarding the dermal adherence factor relates to the differences in the particulate make-up of soil and sediment. Marine sediments generally have a higher sand fraction than freshwater sediments and may potentially have a greater percentage of larger particles, which are less prone to dermal adherence than small particles. However, higher moisture content in sediment, the third source of uncertainty, will likely increase the adherence of particles of all sizes. Also important in the discussion of particle size and skin adherence is the concept of mono-layer loading of the skin surface. As sediment loading of the skin surface increases, the fraction of chemical that is available to be absorbed will remain constant until all of the skin is covered by a thin layer of sediment (known as the mono-layer) (Duff and Kissel 1996). The fraction of chemical that can be absorbed across the skin will increase as dermal loading increases until the point when the mono-layer threshold is crossed. At that point, the fraction of chemical that can be absorbed will decrease because not all of the sediment is in constant, direct contact with skin. Both the amount of sediment required to form the mono-layer and the associated adherence capability of the soil depend directly on the size of the sediment particles and the moisture content of the sediment. In general, larger, drier particles will have a lower adherence factor than smaller, more moist particles.

Since publication of the EPA (2004d) guidance for dermal risk assessment, additional studies that focus specifically on dermal adherence of marine sediments during clamming activities (for adults) and beach play (for children) have been conducted (Shoaf et al. 2005a, b). Both of these newer studies included unscripted clamming or beach play activities and identified post-exposure dermal sediment loadings. Surface-area-weighted sediment dermal adherence factors were calculated from the body-part-specific sediment loadings presented in these studies. For adults digging in a clam flat, the sediment dermal adherence factor was 0.3 mg/cm^2, quite similar to EPA's recommended value of 0.2 mg/cm^2. This value was also similar to that presented in the study that investigated individuals gathering reeds in marine sediment in Washington State (Kissel et al. 1996). Table B.6-24 summarizes the effect on total risk estimates when the adult exposure dermal adherence factor of 0.3 mg/cm^2 is used instead of the value of 0.2 mg/cm^2. There are only slight changes in the non-cancer and cancer risk estimates, and therefore it can be concluded that the effect of higher

skin adherence on the overall risk estimates for netfishing and clamming is not significant.

Table B.6-24. Effect of increased dermal adherence factors on risk estimates for adult sediment exposure scenarios

Dermal Adherence Factor	Totaling Approach	Hypothetical Risk by Exposure Scenario				
		Netfishing		Clamming		
		RME	CT[a]	Tribal 183 Days per Year	Tribal RME	7 Days per Year
Total Excess Cancer Risk						
0.2	total risk excluding PCB TEQ	3×10^{-5}	9×10^{-6}	2×10^{-4}	1×10^{-4}	1×10^{-6}
0.3	total risk excluding PCB TEQ	3×10^{-5}	1×10^{-5}	3×10^{-4}	2×10^{-4}	2×10^{-6}
Total Non-Cancer Hazard[b]						
0.2	na	0.1	0.08	0.6	0.4	0.01
0.3	na	0.2	0.09	0.7	0.5	0.01

[a] To characterize risks for the netfishing CT scenario, a dermal adherence factor of 0.02 was used as specified as the default value for a CT industrial worker in EPA (2004d). However, for consistency, the values of 0.2 and 0.3 were used in this table.

[b] Estimates for total non-cancer hazards are provided as HQs. Non-cancer hazards do not include estimates of dermal risk conferred from metals other than arsenic and cadmium because of the lack of dermal absorption factors for all other metals (see Section B.6.1.4.1).

CT – central tendency

na – not applicable

RME – reasonable maximum exposure

TEQ – toxic equivalent

The body-part-weighted sediment dermal adherence factor for children engaged in beach play based on sediment loadings reported in the study conducted by Shoaf et al. (2005b) was 3.9 mg/cm^2. Note that a higher dermal adherence factor of 20.3 mg/cm^2, based on data gathered by Kissel et al. (1996) during a study of children playing on an inland lake shore, was not used by EPA in development of their dermal adherence factor (EPA 2001b) and, therefore, is also not included in this analysis. The Shoaf et al. (2005b) study is the most relevant study to the children's beach play scenarios evaluated in this risk assessment; however, it was not available when EPA's most recent dermal guidance was developed (EPA 2004d) and has not received the same level of review as the studies included in that guidance. Using the Shoaf (2005b) dermal adherence factor of 3.9 as an alternative value led to an increase in total risk of approximately one order of magnitude for each of the beach play RME scenarios (Table B.6-25). The higher value for dermal adherence would affect all beach play areas similarly with respect to cancer risk, but has the largest effect on non-cancer HQs in areas 3 and 4. The effect of increasing the dermal adherence factor on non-cancer hazards is limited by the fact that the dermal portion of the risk is only included in the total risk calculation if a dermal absorption factor is available for that chemical. Use of

the Shoaf (2005b) dermal adherence factor may be inappropriate given the monolayer concept (Duff and Kissel 1996), which limits the dermal adherence to 0.2 mg/cm^2.

Table B.6-25. Effect of increased dermal adherence factor on risk estimates for child sediment exposure scenarios

Dermal Adherence Factor	Hypothetical Risk by Exposure Scenario							
	Area 1	Area 2	Area 3	Area 4	Area 5	Area 6	Area 7	Area 8
Total Excess Cancer Risk[a]								
0.2	2×10^{-5}	5×10^{-5}	3×10^{-5}	3×10^{-5}	8×10^{-6}	9×10^{-6}	5×10^{-6}	7×10^{-6}
3.9	7×10^{-5}	2×10^{-4}	1×10^{-4}	1×10^{-4}	3×10^{-5}	3×10^{-5}	1×10^{-5}	3×10^{-5}
Total Non-Cancer Hazard[b]								
0.2	0.3	0.5	1	2	0.5	0.3	0.8	0.7
3.9	0.6	0.8	2	10	0.8	1	1	0.9

[a] Excess cancer risk total is the sum excluding PCB TEQ.

[b] Estimates for total non-cancer hazards are provided as HQs. Additionally, non-cancer hazards do not include estimates of dermal risk conferred from metals other than arsenic and cadmium because of the lack of dermal absorption factors for all other metals (see Section B.6.1.4.1)

CT – central tendency

na – not applicable

RME – reasonable maximum exposure

TEQ – toxic equivalent

B.6.1.5.3 Cumulative effects of alternative dermal absorption and alternative dermal adherence factors used for sediment exposure scenarios

Risks were estimated using alternative dermal absorption factors and high-end dermal adherence factors in order to assess the potential cumulative effects of these uncertainties. Excess cancer risks and non-cancer HQs were calculated using the default MTCA dermal absorption factor of 0.01 for inorganic chemicals lacking such values. A high-end dermal adherence value of 3.9 was used for the beach play scenarios (increased from 0.2), and a value of 0.3 was used for the clamming and netfishing scenarios (increased from 0.2 for the clamming and netfishing RME scenarios and from 0.02 for the clamming CT scenario). These are the same values that were used in Section B.6.1.5.2.

Overall, changes to risk conclusions provided in Section B.5 were limited. As was determined in Section B.5, no netfishing or clamming scenario HQs exceeded 1. With use of the higher dermal adherence factor, three excess cancer risk estimates slightly exceeded the 10^{-6} threshold. Excess cancer risks for cPAHs in the netfishing RME scenario and for arsenic and PCB TEQ in the netfishing CT scenario were equal to 2×10^{-6}. It should be noted that these changes in excess cancer risks were not caused by the default dermal absorption factor because these chemicals have specific literature values, as indicated in Table B.3-34.

For the beach play scenarios, several HQ changes occurred from results presented in Section B.5. With the alternative dermal absorption and dermal adherence factors, the HQs for vanadium were 2 or 3 for all areas, exceeding the threshold HQ of 1. In addition, the HQ for chromium at Area 4 increased to 1, and the HQ for total PCBs at Area 4 increased from 1 to 8. The new dermal parameters also resulted in several new exceedances of the excess cancer risk threshold of 10^{-6}, including for cPAHs at Area 7 and for total PCBs at Area 6.

B.6.1.6 Representativeness of fish and shellfish COPC data for all potentially exposed populations

In contrast to the Phase 1 HHRA (Windward 2003b), where tissue chemistry data were available for a relatively small number of samples, the tissue chemistry dataset is much larger for this baseline HHRA. Chemistry data from over 120 composite tissue samples of fish, crabs, and clams, representing eight different species (details of individuals included in the composite samples may be found in Table B.2-4), were collected in 2004 and 2005 (Windward 2005c, 2006b). The relatively large tissue database used in this HHRA should approximate the range of chemical concentrations to which seafood consumers might be exposed.

As discussed in the Section B.3.4.1, seven seafood categories were assumed to reasonably characterize the consumption of the several diverse groups of consumers. In the surveys used to develop the consumption rates for this risk assessment, consumption of several dozen different seafood species was reported. These were assigned to a handful of seafood categories in the reports based on the initial consumption studies. Seven consumption categories were then used in this risk assessment, with tissue data assumed to be representative of each category. For example, what was reported in a survey as benthic fish consumption might have included English sole, flounder, and rockfish. The data used to develop an EPC for this category may have included only English sole. This uncertainty may have led to either over or underestimation of risk.

The tissue samples used in this HHRA were uncooked portions of the total organism (e.g., whole body and fillets for benthic fish, hepatopancreas and muscle meat for crab). These portions represent the consumption habits of many, but not all, of the potentially exposed populations. For example, most people cook fish or shellfish before eating them. Data from uncooked or raw tissue samples were used in this HHRA because most chemistry data were collected for this type of sample. There is no standard cooking preparation that is used for environmental investigations. The King County Water Quality Assessment (King County 1999b) included analysis of two composite tissue samples of crabs that had been cooked and two composite tissue samples of crabs that had not been cooked. Mean concentrations of arsenic and PCBs, which are two COCs identified in the risk characterization section, were 9.95 mg/kg and 156 µg/kg, respectively, in the uncooked samples, and 4.84 mg/kg and 89.5 µg/kg, respectively, in the cooked samples. In the same assessment, risk estimates for

PCBs were approximately double for cooked sole compared to uncooked sole and approximately one-half for cooked crab compared to raw crab. For arsenic, risks associated with cooked sole were only slightly higher than for raw sole and were three times lower for cooked crab compared to raw crab (King County 1999b). Thus, risk estimates may be either increased on decreased when cooking is considered. Because there are no standard cooking practices, the assumption that risks would be uniformly reduced by cooking is inappropriate. For example, preparation of soups or stews from seafood would not likely reduce chemical concentrations to the same degree as broiling, where fats drip away. Given the uncertainties in both chemical concentration reduction associated with different cooking practices, as well as the cooking practices employed by different groups, uncooked tissue samples were used for risk assessment purposes.

In addition to uncertainties related to cooking, there are also uncertainties related to other preparation methods. Many individuals depurate clams (i.e., hold clams alive in water to remove the sediment in the clam digestive system) prior to consumption. The clams used for development of clam EPCs were not depurated. This is an uncertainty and may lead to over- or underestimation of risk, depending on whether chemical concentrations in clam gut contents are higher or lower than chemical concentrations in clam tissue and on how the clams are actually prepared prior to consumption (i.e., depurated or not). For example, if arsenic concentrations in the sediment exceeded those in clam tissue, the undepurated clams would likely have higher arsenic concentrations than depurated clams. If the clams are generally depurated before they are consumed, using undepurated tissue samples could contribute to overestimation of risk.

B.6.1.7 Spatial coverage of sediment chemistry data

As described in Section B.2.3, the sediment chemistry database is reasonably representative of both site-related contamination and human use patterns. Although sampling coverage was generally thorough, the number of analyses conducted for each chemical differs (see Attachment 1). Many chemicals were analyzed in hundreds of sediment samples, but some chemicals, such as dioxins/furans, organochlorine pesticides, and benzidine, were analyzed much less frequently. Dioxins/furans were identified as posing high risks in some scenarios in the risk characterization section, and hypothetical benzidine risks are discussed in Section B.6.3.2. In addition, for some exposure scenarios, there were only a few sediment samples for the relevant exposure area.

The limited number of samples in some sediment areas is particularly important for the beach play RME scenarios, where exposure areas are relatively small compared to the exposure areas for the netfishing (which included the entire LDW) and clamming scenarios (see Maps B.3-1 and B.3-2). Thus, exposure estimates for the beach play RME scenarios are based on only a few sediment samples (sometimes only one) for some

chemicals (see Tables B.3-39 through B.3-41). EPCs based on a single sample[45] carry far more uncertainty, as do resulting risk estimates, than EPCs based on larger datasets. Chemical concentration data are often positively skewed. For small, positively skewed datasets (n = 5 or fewer) where the maximum value was selected as the EPC, the maximum value might have underestimated the true mean. Unlike larger datasets (n = 6 or more) where an upper confidence limit on the mean was estimated with Pro UCL 4 (e.g., 95% Student's t-UCL) and selected as the EPC, there is much greater uncertainty in the EPCs for small datasets (n = 5 or fewer) where the maximum value was selected as the EPC and no upper confidence limit on the mean was estimated or incorporated into the EPC.

The representativeness of the limited dioxin/furan sediment data to the full LDW site is also uncertain. The dioxin/furan data used in this HHRA are shown on Map B.3-4 and in Table B.5-58. These data are from two sources: the EPA Site Inspection (Weston 1999), which was focused on reconnaissance of the entire LDW and provided reasonably good coverage of the LDW with 25 dioxin/furan sampling locations; and the 2005 sediment sampling effort, which differed from the EPA Site Inspection in that it was focused, in part, on potential sources, characterization of hot spots identified in EPA's 1998 data collection effort, and areas without existing data (Windward 2005h). Consequently, the spatial coverage of the 18 locations where samples were collected in 2005 for dioxin/furan analyses is greater in areas with potential dioxin/furan sources. This design resulted in an arithmetic mean concentration, based on all 43 locations summarized in Table B.5-58, that is likely biased high for exposure scenarios that include large areas of the LDW, such as netfishing and clamming. It is also possible, however, that because the overall sampling density was low, additional dioxin hot spots were not identified. Identification of additional high dioxin results could increase EPCs.

As an example of the potential high bias of the dioxin/furan TEQ data, three of the highest dioxin/furan TEQs in the baseline surface sediment dataset are located in a relatively small area at RM 1.5 west (Map B.3-4) near a potential dioxin/furan source. Each of these three locations was treated as a separate data point in the EPC calculations. Although there are not enough dioxin/furan data to estimate a reasonably accurate spatially weighted mean concentration, some simple calculations illustrate the degree of spatial bias in the existing dataset. The mean dioxin/furan TEQ for the three highest TEQs located at RM 1.5 west was 1,033 ng/kg dw. Substituting this area sub-average for the three more variable original data points (2,100, 565, and 463 ng/kg dw) results in a revised LDW-wide EPC of 320 ng/kg dw, compared to the EPC used in the netfishing sediment exposure scenario of 610 ng/kg dw. Using this alternative EPC would reduce the excess cancer risk estimate for the dioxin/furan TEQ in the RME netfishing scenario from 2×10^{-5} to 9×10^{-6}.

---

[45] For datasets with fewer than five detected concentrations, EPCs were based on the highest detected concentration or the highest reporting limit divided by two, whichever was greater.

The relatively high dioxin/furan excess cancer risk estimates for netfishing and tribal clamming scenarios are driven by a small number of elevated concentrations, some of which are clustered (see Section B.5.5.2). An example of the clustering of samples with high dioxin/furan concentrations occurs at approximately RM 1.5 west, in an area surrounding an inlet (the same location discussed above). Because of the presence of a large pier near the entrance to the inlet and the very shallow depths in the western side of the inlet, it is unknown if netfishers deploy their nets in this area, or would do so in the future. If tribal netfishers did not use this relatively small and shallow area, direct contact risks would be much lower compared to estimates presented in Section B.5, which are based on the assumption that this area can be used. However, tribal members clamming by boat could theoretically use this area because the constraints on netfishing would not apply. Therefore, the risk estimates for netfishing were re-calculated without the three high dioxin samples clustered in the inlet. Table B.6-26 presents the changes to both the EPC, CDI and risk estimates for the full dataset as well as the adjusted version. Using the full dataset, the risks for netfishing CT and netfishing RME are 6×10^{-6} and 2×10^{-5}, respectively. When the cluster of three high samples is removed, the risks for the netfishing scenarios are reduced by approximately 80%, to 1×10^{-6} (for CT) and 4×10^{-6} (for RME).

Table B.6-26. Comparison of dioxin/furan TEQ risks with and without cluster of high concentrations at RM 1.5

Dioxin/Furan TEQ	N	EPC	CDI (mg/kg-day)		Cancer Slope Factor	Cancer Risk		Total Risk
			Ingestion	Dermal		Ingestion	Dermal	
Full Dataset								
Netfishing CT	43	0.00061	2.7×10^{-11}	1.2×10^{-12}	150,000	4×10^{-6}	2×10^{-7}	4×10^{-6}
Netfishing RME	43	0.00061	7.6×10^{-11}	3.3×10^{-11}	150,000	1×10^{-5}	5×10^{-6}	2×10^{-5}
Without RM 1.5 West Samples								
Netfishing CT	40	0.00014	6.1×10^{-12}	2.6×10^{-13}	150,000	9×10^{-7}	4×10^{-8}	1×10^{-6}
Netfishing RME	40	0.00014	1.8×10^{-11}	7.6×10^{-12}	150,000	3×10^{-6}	1×10^{-6}	4×10^{-6}

CDI – chronic daily intake

CT – central tendency

EPC – exposure point concentration

RM – river mile

RME – reasonable maximum exposure

TEQ – toxic equivalent

Although the spatial sampling density for many COPCs is adequate, many more samples exist in areas of higher concentrations for some COPCs than in areas of lower concentrations. This spatial bias reflects the greater interest in characterizing areas with higher concentrations for making remedial decisions. This bias is particularly evident for PCBs in surface sediment, triggering an alternative EPC calculation method, as noted in Section B.6.1.1.12. The PCB risk estimates presented in this

document for the netfishing and clamming scenarios are based on the arithmetic approach described in Section B.3.4.3.2 and are known to be overestimated because of the spatial bias of the underlying data. These risk estimates are approximately four- to five-fold higher than risk estimates made using the spatially weighted approach described in Section B.6.1.1.12 (Table B.6-27).

Table B.6-27. PCB risk estimates for sediment exposure scenarios using two EPC calculation methods

Scenario	Arithmetic Mean Approach		SWAC Approach	
	EPC[a] (mg/kg dw)	Cancer Risk Estimate	EPC[b] (mg/kg dw)	Cancer Risk Estimate
Netfishing RME	2.5	2×10^{-6}	0.47	4×10^{-7}
Tribal clamming RME	4.0	8×10^{-6}	0.90	2×10^{-6}

[a] EPC was calculated as a 95th UCL on the mean.

[b] EPC was calculated as a SWAC (spatially weighted average concentration).

EPC – exposure point concentration

RME – reasonable maximum exposure

SWAC – spatially weighted average concentration

UCL – upper confidence limit

B.6.1.8 Temporal variability in chemistry data

Most of the chemistry data used in this HHRA were collected over a 10-year period (1996 to 2005), as summarized in Section B.2.1; although some sediment samples were also collected in the early 1990s. There is some uncertainty about whether the conditions characterized by some of the older samples remain in effect today. However, for the purposes of this HHRA, these data are used to represent baseline conditions in the absence of any remedial actions. Technical memoranda have been submitted and approved by EPA documenting the acceptability of historical datasets for all uses in the RI/FS (Windward 2005j, k). Temporal trends in both sediment and tissue chemistry data will be discussed in the RI report.

The large majority of the tissue chemistry data used in the HHRA was collected by LDWG in 2004 and 2005. Historical data collected in the mid- to late-1990s were also used. The largest historical tissue chemistry dataset for a single tissue type exists for English sole fillets. Fifteen samples collected from 1996 to 1998 were analyzed for PCBs, mercury, and TBT. Some samples were also analyzed for additional COPCs. A quantitative example of the uncertainty associated with the use of historical tissue chemistry data is presented using English sole fillet samples analyzed for total PCBs. The total PCB EPC used in this HHRA for the benthic fish fillet seafood category was 1.16 mg/kg ww (n = 33). Using only the benthic fish fillet (one starry flounder fillet sample was included) data collected by LDWG in 2004 and 2005, the EPC would be 1.29 mg/kg ww (n = 18). An EPC based only on the 2005 data would be 1.11 mg/kg ww (n = 10). This example suggests that the risk estimates that incorporate both

historical and more recent tissue chemistry data would be very similar to risk estimates that included only the more recently collected data, at least for total PCBs.

B.6.1.9 Health-protectiveness of sediment exposure scenarios

The sediment exposure scenarios summarized in Section B.3.2 and used for risk characterization in this HHRA were selected because they represent activities that may commonly occur in the LDW or may commonly occur in the future. They were also selected to represent activities that result in a relatively higher amount of exposure than other activities. There are other activities that may occur in the LDW that were not explicitly discussed in the HHRA, such as walking along the shoreline, habitat restoration, and occupational exposure associated with specific industrial or commercial facilities. The risks from these other activities are expected to be lower than the risks for the scenarios that were quantified. Additional quantitative analysis is presented in this section to support that conclusion. These other activities are described briefly below.

Walking adjacent to the LDW may be a common activity, particularly in residential neighborhoods near the LDW, such as South Park. No formal survey of the frequency of walking has been conducted. It is likely that walking occurs both on the top of the bank, above the high water line, and within the intertidal zone. Dog walking may also commonly occur in the South Park neighborhood. People walking their dogs may have some contact with sediment when they throw sticks or balls for their dogs in the intertidal zone. Any dermal contact with sediment that occurs during dog walking would most likely be limited to hands and forearms. Exposure during dog walking would primarily be to hands, for example, during activities such as playing "fetch" with a ball or stick. This assessment assumes that an individual walks a dog 200 days per year and that on each of those days the individual's hands are totally covered with sediment. In this way, it is a health-protective assumption and likely overestimates risks for most site users. Although not specifically addressed in the risk calculations, which included dermal contact on the hands only, activities such as hugging or picking up a muddy dog to carry it along the beach may lead to dermal contact with sediment on the forearms as well as hands. Because of uncertainty regarding the frequency with which exposures to the forearms occur, the exposure area for calculation of risks for a dog walker was limited to hands only. However, if the activity is 200 days per year and the forearms, as well as hands, are exposed, an increase in risk would be predicted. Several habitat restoration projects have been completed in the past 10 years in the LDW, and there will undoubtedly be more projects in the future, as well as possible environmental research projects. People conducting intertidal habitat restoration routinely come in contact with sediment. The duration and frequency of such contact may be much lower than other sediment exposure scenarios quantified in this HHRA because restoration projects occur only episodically. Habitat restoration workers can be paid staff or volunteers, including:

- Biologists conducting on-site supervision or sampling activities

- Washington Conservation Corps (WCC) volunteers
- King County special operations crews
- Citizen volunteers

Although there is generally little public access at industrial facilities, there may be occupational exposure to sediment at specific facilities, but such exposure is probably very low compared to the occupational exposure of netfishing that was quantitatively evaluated in the HHRA. The spatial scale at which facility-specific occupational exposure may occur is different, and perhaps smaller, than the spatial scale of netfishing. A concern is that despite lower contact rates, there may be significant risks in certain areas with higher sediment contaminant concentrations. A method for evaluating occupational exposure and risk at smaller spatial scales is presented in Section B.6.3.3.3. No additional quantitative analysis of occupational exposure is presented here.

For the purpose of this uncertainty assessment, hypothetical exposure parameters were derived for the dog walking and habitat restoration scenarios described above (Table B.6-28).

Table B.6-28. Exposure parameters for dog walking and habitat restoration scenarios

Scenario	Exposure Frequency (days/yr)	Exposure Duration (yrs)	Sediment Ingestion Rate (mg/day)	Adherence Factor (mg/cm^2)	Skin Surface Area (cm^2)
Dog walking	200[a]	30[b]	25[c]	0.2[d]	904[e]
Research and habitat restoration scenarios					
Biologist	15[f]	20[g]	100[h]	0.2[d]	6040[i]
WCC volunteer	90[j]	2[k]	100[h]	0.2[d]	6040[i]
King County special operations	31[l]	20[g]	100[h]	0.2[d]	6040[i]
Citizen volunteer	5[m]	20[m]	100[h]	0.2[d]	6040[i]

[a] Approximately 4 days per week.

[b] EPA default for RME scenario.

[c] One-half of the EPA default for industrial workers; assumes adult dog walking exposure is less than typical industrial exposures because of duration per event and specific behavior differences.

[d] Default health-protective factor for exposures of children and adults to moist soil recommended by EPA (2004d).

[e] Total surface area of hands (EPA 1997).

[f] Biologists are typically on site only periodically during a restoration activity.

[g] Accounts for a reasonably long career in the same position, but assumes that the most senior scientists will spend very little time in the field.

[h] Default for agricultural and residential exposure (EPA 1997).

[l] Skin surface area used for adult clamming scenario in this HHRA.

[j] Assumes that 75% of the work days are in the field (180 days/yr) and that 50% of that time is in the LDW (90 days/yr).

[k] WCC (Washington Conservation Corps) volunteers serve for a defined duration (i.e., 2 years).

[l] King County special operations road crews may spend 50% of their time at habitat restoration projects (125 days/yr). This scenario assumes they might spend 50% of that time in the LDW (62 days/yr) in a single year, but the long-term average is unlikely to exceed 25% of that time (31 days/yr).

[m] Opportunities for citizen volunteers are more limited compared to opportunities for the paid crews.

To provide a comparison of the relative risks associated with the different exposure scenarios, risk estimates were developed using standardized, hypothetical EPCs for example chemicals. The exposure parameters described in Table B.6-28 as well as exposure parameters for beach play, clamming, and netfishing from Section B.3.4 were applied to hypothetical EPCs for arsenic (10 mg/kg dw), cPAHs (1 mg/kg dw), and total PCBs (1 mg/kg dw) to yield hypothetical risk estimates for carcinogenic endpoints (Table B.6-29). The hypothetical arsenic EPC was similar to or slightly lower than EPCs used for the beach play RME (8.9 to 21 mg/kg dw), tribal clamming RME (27 mg/kg dw), and netfishing RME (21 mg/kg dw) scenarios. The hypothetical total PCB and cPAH EPCs were higher than all but one or two of the beach play area EPCs. The risk estimates include both sediment ingestion and dermal contact exposure routes.

Risk estimates were also made for sediment exposure scenarios that were quantified in the HHRA so that the health protectiveness of these scenarios could be evaluated. The hypothetical risks for the dog walking and habitat restoration scenarios were all lower than the risks for the beach play, clamming, and netfishing scenarios shown in Table B.6-29. The risks for sediment contact scenarios evaluated are more fully quantified and discussed in the risk characterization section. However, these results indicate that risk estimates for these primary scenarios (i.e., beach play, clamming, and netfishing) are health-protective of activities that may occur more frequently but with lesser sediment contact, such as dog walking. The risk estimates are also health-protective of habitat restoration activities with a similar degree of sediment contact but with lower frequency and/or exposure duration. Risks for the habitat restoration scenarios might decrease over time if restoration involves altering the existing substrate to a new condition with lower chemical concentrations. If exposure does decrease over time as a result of remedial activities, the risks associated with exposure in the remediated locations would also decrease.

Table B.6-29. Cancer risk estimates using hypothetical EPC values for arsenic, cPAHs, and PCBs

Scenario	Hypothetical Risk for Arsenic[a]	Hypothetical Risk for cPAHs[b]	Hypothetical Risk for PCBs[b]
Dog walking	1×10^{-6}	1×10^{-6}	3×10^{-7}
Research and habitat restoration scenarios			
Biologist	3×10^{-7}	3×10^{-7}	9×10^{-8}
WCC volunteer	2×10^{-7}	2×10^{-7}	5×10^{-8}
King County special operations	7×10^{-7}	6×10^{-7}	2×10^{-7}

Scenario	Hypothetical Risk for Arsenic[a]	Hypothetical Risk for cPAHs[b]	Hypothetical Risk for PCBs[b]
Citizen volunteer	1×10^{-7}	1×10^{-7}	3×10^{-8}
Scenarios already quantified in the HHRA			
Beach play RME (65 days/yr)	4×10^{-6}	3×10^{-6}	6×10^{-7}
Clamming (120 days/yr)	2×10^{-5}	7×10^{-6}	2×10^{-6}
Netfishing (119 days/yr)	3×10^{-6}	3×10^{-6}	8×10^{-7}

[a] Based on a hypothetical EPC of 10 mg/kg dw.

[b] Based on a hypothetical EPC of 1 mg/kg dw.

cPAH – carcinogenic polycyclic aromatic hydrocarbon

EPC – exposure point concentration

HHRA – human health risk assessment

PCB – polychlorinated biphenyl

WWC – Washington Conservation Corps

Because the dog walking scenario results in the highest hypothetical risks of the scenarios not previously evaluated in the risk characterization section, the EPCs for the beaches that showed the highest and lowest risks for beach play, along with the largely residential beach play RME area 5 (see Map B.3-1), were used to calculate risks to a person walking and playing with a dog on those beaches. Beach play RME area 2 represents the high-end dog walker exposure, and beach 7 represents the low end. Tables B.6-30 and B.6-31 show the results of these calculations. Based on the relative risk comparison from Table B.6-29, the child beach play RME risk estimates for these areas (Section B.5.3.3) were higher than for dog walking. The total upper bound cancer risk estimates are above 1×10^{-6} for dog walking for all three areas, but, as with the child beach play at these areas, the HQs are all below 1. Carcinogenic PAHs and arsenic are the greatest contributors to cancer risk estimates. If the LDW beaches are used less than 200 days per year, or if contact rates are less than the assumed rates (e.g., if sediment contact does not occur each of the 200 days or does not cover the entire hand on each day), the risk estimates would be lower.

Table B.6-30. Cancer risk estimates for dog walking at beach play areas 2, 5, and 7

Chemical	Cancer Risk		
	Area 2	Area 5	Area 7
Arsenic	3×10^{-6}	1×10^{-6}	2×10^{-6}
cPAHs[a]	3×10^{-6}	5×10^{-7}	1×10^{-7}
Dioxin/Furan TEQ	nd	3×10^{-8}	3×10^{-8}
PCB TEQ	2×10^{-7}	6×10^{-8}	1×10^{-8}
Total PCBs	6×10^{-8}	6×10^{-8}	8×10^{-8}
Subtotal excluding PCB TEQ	**6×10^{-6}**	**2×10^{-6}**	**2×10^{-6}**
Subtotal excluding total PCBs	**6×10^{-6}**	**2×10^{-6}**	**2×10^{-6}**
Tentatively identified chemicals (JN-qualified)			
Total DDTs	4×10^{-10}	1×10^{-9}	9×10^{-11}
Dieldrin	1×10^{-9}	6×10^{-9}	2×10^{-9}
Toxaphene	4×10^{-9}	5×10^{-8}	8×10^{-9}
Subtotal	**5×10^{-9}**	**6×10^{-8}**	**1×10^{-8}**
Total Risk across all exposure routes/ pathways excluding PCB TEQ	**6×10^{-6}**	**2×10^{-6}**	**2×10^{-6}**
Total Risk across all exposure routes/ pathways excluding total PCBs	**6×10^{-6}**	**2×10^{-6}**	**2×10^{-6}**

[a] cPAH concentrations are presented as benzo(a)pyrene equivalents.

J – estimated concentration

N – tentative identification

nd – not detected in this area but included for completeness and comparison to other areas

cPAH – carcinogenic polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

TEQ – toxic equivalent

Table B.6-31. Non-cancer risk estimates for dog walking at beach play areas 2, 5, and 7

Chemical	Non-Cancer Risk		
	Area 2	Area 5	Area 7
Aluminum[a]	nd	0.005	0.008
Antimony[a]	0.002	0.004	0.004
Arsenic	0.03	0.01	0.02
Barium[a]	nd	0.00007	0.0001
Cadmium[b]	0.0004	0.0001	0.0004
Chromium[a]	0.006	0.003	0.003
Copper[a]	0.001	0.0006	0.0003
Iron[a]	nd	0.03	0.04
Manganese[a]	nd	0.0007	0.001
Mercury[a]	0.002	0.0004	0.0004
Molybdenum[a]	0.0002	0.0001	0.00009
Total PCBs	0.006	0.007	0.008
Silver[a]	0.00003	0.00001	0.00002
Thallium[a]	0.001	0.0003	0.001
Vanadium[a]	0.02	0.02	0.02
Zinc[a]	0.0005	0.0001	0.0001
Subtotal excluding total PCBs	**0.1**	**0.1**	**0.1**
Tentatively identified chemicals (JN-qualified)			
Total DDTs	0.00001	0.00003	0.000002
Dieldrin	0.000006	0.00003	0.00001
Subtotal	**0.00002**	**0.00006**	**0.00001**
Total risk across all exposure routes/ pathways excluding total PCBs[c]	**0.1**	**0.1**	**0.1**

[a] No absorption factor available for this chemical. Dermal exposure for this chemical is discussed in the uncertainty analysis (Section B.6).

[b] Because of the lack of a dermal RfD, an adjustment factor was applied to the oral RfD to estimate the non-cancer hazard from dermal absorption of this chemical. This adjustment was necessary in order to account for the fact that the oral reference dose is based on an administered dose and dermal exposure is based on an absorbed dose. The adjustment allows exposure and toxicity to be expressed in terms of absorbed dose.

[c] This total is not directly interpretable for risk assessment. The value indicates that the HI does not exceed 1 for individual endpoints. Therefore, HIs for individual endpoints were not calculated.

J – estimated concentration

N – tentative identification

nd – chemical not detected in this area.

PCB – polychlorinated biphenyl

B.6.2 TOXICITY ASSESSMENT

Three topics related to uncertainty in the toxicity assessment are discussed here in greater detail: toxicity benchmarks, PCB toxicity assessment, and chromium toxicity assessment.

B.6.2.1 Toxicity benchmarks

The toxicity benchmarks used in this HHRA are based on the most recent guidance provided by EPA. They are health-protective in that they include uncertainty factors or extrapolations to account for sensitive sub-populations or other limitations of the toxicity data on which they are based. The toxicity benchmarks presented in Section B.4 are based on many different studies using both experimental animals and human populations. The RfDs published by EPA included consideration of data available at that time for effects on children (based in some cases on developmental effects in animal studies), particularly the developing fetus. They are designed to be protective of sensitive sub-populations, but the inherent uncertainty may span one or more orders of magnitude. For example, the RfD for methylmercury, which is used as a surrogate for mercury in this HHRA, is based on developmental effects on children following exposure during gestation. EPA's RfD for methylmercury has been extensively peer-reviewed and is thought to be sufficiently health-protective for children (NRC 2000).

Some chemicals may have developmental effects, but other effects were used by EPA to develop the RfDs. For example, several studies have documented developmental effects from exposure of pregnant women to PCBs through fish consumption (Fein et al. 1984; Jacobson and Jacobson 1996, 1997), but the RfD published in IRIS is based on an immunological effect because it was considered to be more health-protective than the developmental effect (i.e., to occur at a lower dose level). Studies published since the publication of the PCB RfD have investigated possible reproductive effects and neurotoxic effects in children. It is unclear whether consideration of these more recent neurotoxicity studies would result in a change to the current PCB RfD, which is based on immunotoxicity. Similarly, arsenic may have some developmental effects at sufficient dose levels (ATSDR 2005b), but the critical study described in IRIS documenting dermal and cardiovascular effects was used to set the RfD because EPA considered these effects to be more health-protective than the developmental effect.

Of all the chemicals that were analyzed in LDW tissue samples, 18 do not have screening levels (e.g., RBCs or PRGs). Table B.6-32 lists these chemicals and indicates whether or not they have been detected in LDW tissue samples. More information on the results of the analysis of these chemicals in LDW tissue is available in Table 3 of Attachment 1.

Table B.6-32. Chemicals in LDW tissue samples without toxicity benchmarks

Detected Chemicals	Undetected Chemicals
4-Nitrophenol[a]	2-Nitroaniline[a]
Benzo(g,h,i)perylene	2-Nitrophenol
bis(2-chloroethoxy)methane[a]	4-Bromophenyl phenyl ether
Cobalt[a]	4-Chloro-3-methylphenol
delta-BHC	4-Chlorophenyl phenyl ether
Dibenzofuran[a]	Coprostanol
Dibutyltin as ion[a]	Di-n-octyl phthalate[a]
Dimethyl phthalate[a]	Tetrabutyltin as ion
Monobutyltin as ion	
Phenanthrene	

[a] Provisional toxicity values are available for these chemicals.

Toxicity information for these 18 chemicals is not provided in the EPA Region 3 table used for fish tissue screening purposes in this risk assessment (EPA 2005b). Toxicity benchmarks could be developed for these chemicals by requesting a review from the National Center for Exposure Assessment (NCEA), as indicated in EPA guidance. However, inasmuch as these chemicals were not identified as COPCs through screening, they were not included in risk estimates. Overall risks may have been underestimated if there are significant toxic effects associated with these chemicals.

As indicated in the table, toxicity benchmarks for some chemicals were available from provisional sources that were not used by EPA Region 3 to develop fish RBCs (EPA 2005b). To evaluate the potential risks associated with these chemicals, fish RBCs were calculated using equations provided by EPA Region 3 (EPA 2005b). Only three chemicals had maximum values that exceeded these calculated RBCs, including 4-nitrophenol (detected in 2 of 145 samples), bis(2-chloroethoxy)methane (detected in 1 of 145 samples) and 2-nitroaniline (never detected). Because of the high uncertainty with the calculated RBCs based on provisional toxicity data and the low detection frequency of these chemicals, they are considered unlikely to contribute significantly to seafood consumption risk for the LDW.

B.6.2.2 Total PCBs

One uncertainty associated with PCB risk assessment is the difference between the PCB mixtures found in the environment and the mixtures used in laboratory toxicity studies. As reviewed by Cogliano (1998), the commercial PCB mixtures released into the environment may be altered by volatilization, vaporization, differential sorption, bacterial degradation, photolysis, and metabolism and elimination. In particular, differential bioaccumulation of more highly chlorinated PCB congeners may alter the toxicity of the bioaccumulated mixtures relative to unweathered Aroclors. PCB mixtures that have been altered by bioaccumulation processes are more toxic to mink

than unweathered Aroclors (EPA 1996b). Individual PCB congeners have a range of toxic effects (e.g., cancer, immune system, neurodevelopmental, cardiovascular, reproductive, endocrine/thyroid, dermal), and it would be expected that environmental mixtures that differ in congener composition from unweathered Aroclors would also exhibit differences in toxicity. This uncertainty was considered in EPA's dose response evaluation and in their recommendation of the PCB RfD and SF (EPA 1996b).

EPA (1996b) has recommended a tiered approach for establishing the most appropriate SF for assessing cancer risk from PCBs. The PCB cancer SF associated with high risk and persistence was used for the seafood consumption scenarios. It is intended that this SF be applied to total PCBs rather than to any specific Aroclor mixture (EPA 1996b). While alternative SFs for PCBs do exist, application of an alternative SF for the seafood consumption scenarios would not be appropriate because of the highly persistent nature of many of the PCB congeners that bioaccumulate in fish (Lake et al. 1995) and sediment (Cogliano 1998). EPA derived a range of upper-end SFs with greater potency observed for the more highly chlorinated Aroclors (EPA 1996b). The SF of 2 $(mg/kg\text{-}day)^{-1}$ was derived based on carcinogenicity data for Aroclor 1260 and 1254. Ultimately, rather than using toxicity data on unweathered Aroclor mixtures to predict the toxicity of environmental mixtures, it might be helpful to have direct toxicity studies on relevant environmental mixtures to reflect the enrichment of persistent congeners, including dioxin-like PCBs. Such studies are outside the scope of the LDW RI/FS. The SF for PCBs is based on a study of carcinogenicity data for Aroclor 1260 and 1254 (EPA 1996b), with the estimated SF for Aroclor 1260 being higher than for Aroclor 1254. However, there are some uncertainties related to the toxicity evaluation of Aroclor 1254 from that study. The Aroclor 1254 mixture evaluated initially differed from the formulation of most Aroclor 1254 produced in that it had a higher proportion of PCB 126 and polychlorinated dibenzofurans (PCDFs). To make the formulation more like the standard Aroclor 1254 formulation, 99% of the PCDFs in the mixture were removed, as well as some portion of the PCB 126, prior to the study (Mayes et al. 1998). However, the amount of PCB 126 in the mixture used was still three to five times greater than in the standard Aroclor 1254 formulation. The use of an atypical and altered formulation might have influenced the toxicity results, compared to a study conducted using the standard Aroclor 1254 formulation. If toxicity of Aroclor 1254 was significantly affected by this experimental issue, the cancer SF could potentially be affected as well.

B.6.2.3 PCB and dioxin/furan TEQs

To address toxicity associated with dioxin-like PCB congeners, excess cancer risk was evaluated based on PCB TEQ exposure and the cancer SF associated with 2,3,7,8-TCDD. The use of toxic equivalency introduces an additional level of uncertainty because the TEFs used to calculate the PCB TEQ are estimates of congener toxicity relative to TCDD, which have been rounded to a value of 1 or 3, regardless of

the order of magnitude(Van den Berg et al. 2006). The PCB TEQ is then multiplied by the 2,3,7,8-TCDD cancer SF to calculate an excess cancer risk estimate. Excess cancer risk estimates based on PCB TEQ were the same or higher for most seafood consumption scenarios compared to excess cancer risk estimates based on total PCBs (see Table B.5-61). The implications of these two methods for the calculation of excess cancer risk associated with exposure to PCBs in the risk characterization step are discussed in Section B.6.3.1.

The TEQ approach is widely used in risk assessments for both dioxin/furan and dioxin-like PCB congeners. In a recent World Health Organization re-evaluation of TEFs, it was noted that the "present TEF scheme (see Table 1)[46] and TEQ methodology are primarily meant for estimating exposure via dietary intake situations because present TEFs are based largely on oral uptake studies often through diet." The application of the TEQ approach based on oral TEFs to environmental matrices such as sediment where exposures are largely dermal may greatly overestimate the potential toxicity of the mixture because the highly hydrophobic polychlorinated dibenzo-*p*-dioxins (PCDDs) and PCDFs bind strongly to particles, thereby significantly reducing their bioavailability to living organisms. The bioavailability of these chemicals is largely dependent upon the organic carbon content and the age of the particles. This problem could be reduced if the degree of absorption of specific PCDDs/PCDFs was considered for direct-contact sediment exposure assessments.

Toxicological studies using abiotic matrices with dioxin-like compounds that would allow the development of sediment-based TEFs are almost nonexistent (Van den Berg et al. 2006). Thus, it is not possible to estimate the degree of overestimation included in the risk estimates for dioxin/furan and PCB congeners via direct sediment pathways.

B.6.2.4 Chromium

The available chromium data for both sediment and tissue are based on total chromium. However, the RfD used for chromium in this HHRA is based on hexavalent chromium, which is orders of magnitude lower than the RfD for chromium III, and which would likely make up only a portion of the total chromium. This health-protective assumption provides an overestimate of the risks from chromium, because chromium VI is unlikely to be present in any substantial quantity in a riverine environment. In addition, this chemical was not identified as exceeding acceptable risk levels (i.e., HQ was not greater than 1) for any scenario except the adult tribal seafood consumption scenario based on Suquamish data, and thus the overall impact to the risk conclusions is thus low.

B.6.2.5 Mercury

Total mercury concentrations were used as a surrogate for methylmercury concentrations, the toxic form of mercury for which the RfD was developed. For the seafood consumption scenarios, concentrations of total mercury and methylmercury

[46] Refers to Table 1 in Van den Berg et al. (2006), which is not included in this document.

are expected to be similar, based on the available site-specific data for English sole (see Section B.2.1.2). Thus, there is relatively low uncertainty associated with the risk estimates for mercury in the seafood consumption scenarios. For all seafood consumption scenarios except for the adult tribal scenario based on Suquamish data, HQs for mercury were less than or equal to one. In addition, the child tribal 95th percentile seafood consumption scenario based on Tulalip data (Section 6.1.2.3) had a mercury HQ less than one.

No site-specific data for methylmercury in sediment have been collected. Data on total mercury and methylmercury from other estuaries suggest that methylmercury makes up a very small fraction of total mercury (Mason and Lawrence 1999). This suggests the risks associated with exposure to mercury through direct sediment contact are greatly overestimated. However, given that the HQs for mercury for all direct sediment contact exposure scenarios are much less than one, the overall impact to the risk conclusions is very low.

B.6.3 RISK CHARACTERIZATION

Because of and in addition to the uncertainties related to exposure and toxicity, the risk characterization step can also have significant uncertainty. The first area of uncertainty discussed in this section relates to the total excess cancer risk estimates for multiple chemicals, particularly PCBs. Uncertainties related to potential health risks of chemicals that were never detected in tissue or sediment samples are also evaluated. Finally, to address the possibility that harvesting organisms for consumption and beach play may occur over areas smaller or different than those assumed in the risk characterization section (i.e., LDW-wide for seafood consumption and eight defined areas for beach play), risk estimates for seafood consumption and beach play for other subareas within the LDW are presented.

B.6.3.1 Inclusion of PCBs in estimates of total excess cancer risk

As discussed in Section B.5.3, PCBs consist of 209 individual congeners. Aroclors are commercial mixtures of PCB congeners that contain a large number of individual congeners. The different Aroclors contain many of the same congeners and vary mostly in terms of the relative abundance of specific congeners. After a commercial mixture is released into the environment, the original congener composition of the commercial PCB mixture changes over time through various processes (e.g., partitioning between environmental media, chemical transformation, and preferential bioaccumulation) (Cogliano 1998). The assessment of cancer risks for environmental PCB mixtures is complicated in that carcinogenicity data are available for commercial but not environmental mixtures. Consequently, the carcinogenicity of commercial mixtures must be used to estimate the toxicity of environmental mixtures that may have a different congener composition than the Aroclors used to develop the carcinogenicity data. Cancer risks for environmental PCB mixtures may be estimated on the basis of either: 1) commercial Aroclor toxicity (hereafter referred to as total PCB

risks), or 2) the toxicity of specific components of Aroclor mixtures (i.e., co-planar PCB congeners that have a mode of toxicity similar to that of dioxin [hereafter referred to as PCB TEQ risks]). Total PCB cancer risks are computed by multiplying the total PCB CDI by the SF for PCBs (as Aroclors). As discussed in Section B.4, after making appropriate adjustments for the TEFs for the individual dioxin-like PCB congeners, PCB TEQ cancer risks are computed by multiplying the PCB TEQ CDI by the dioxin SF.

Challenges exist in using total PCB and PCB TEQ cancer risk estimates to represent the true risks posed by environmental PCB mixtures. As will be subsequently noted in examples from guidance and site-specific risk assessments, descriptions of the cancer risks posed by environmental PCB mixtures are bounded on the low end by use of total PCB or PCB TEQ cancer risk estimates and bounded on the high end by adding total PCB and PCB TEQ cancer risk estimates together. There are issues with both of these approaches. Environmental processes (e.g., bioaccumulation) may increase levels of more highly chlorinated and potentially more toxic congeners (e.g., co-planar PCBs with dioxin-like toxicity) relative to those found in commercial PCB mixtures (EPA 1996b). Hence, using either total PCB or PCB TEQ cancer risk estimates alone to describe overall environmental PCB cancer risks may underestimate the true risk posed by an environmental PCB mixture. However, adding total PCB and PCB TEQ cancer risks may overestimate the true risk posed by an environmental PCB mixture. Co-planar PCBs were present in the commercial mixtures used to derive the Aroclor SF; hence, there is a likely potential for "double counting" the risk posed by the co-planar PCBs when adding total PCB and PCB TEQ cancer risks.

A further uncertainty is the degree to which potential co-planar PCB enrichment in environmental vs. commercial PCB mixtures is the primary cause for enhanced carcinogenicity in environmental PCB mixtures. The EPA Science Advisory Board cited the van der Plas et al. (2000) study of rats exposed to Aroclor 1260, which suggests that most of the tumor promotion potential of PCB mixtures is attributable to the non-dioxin-like fraction (EPA 2001a). Because this fraction is not included in the TEQ calculation, van der Plas et al. (2000) concluded that the tumor promotion potential of PCBs might be underestimated by the TEQ approach alone. This is also supported by estimates of TEQs for the different Aroclors. Although the cancer SF included consideration of several Aroclors, the SFs for 1260, followed by 1254, were the highest in the studies evaluated and were used for the development of the SF for total PCBs (EPA 1996b). The TEQ potency for Aroclor 1260 on a mass basis is lower than the potencies for several other Aroclors (Rushneck et al. 2004; Van den Berg et al. 2006). This also suggests that some of its carcinogenic potency is not attributable to dioxin-like PCB congeners.

However, there is uncertainty related to the carcinogenic potency of non-dioxin-like PCB congeners. Knerr and Schrenk (2006) reviewed the carcinogenicity of non-dioxin-like PCB congeners across numerous studies and concluded that in most cases,

dioxin-like PCB congeners were more potent tumor promoters than non-dioxin-like congeners. However, they also stated that a weak carcinogenic potency of some non-dioxin-like congeners could not be excluded. In the case of the van der Plas (2000) study, Knerr and Schrenk (2006) asserted that the purity data provided in that study was not enough to exclude the potential contribution of some dioxin-like congeners to the observed toxicity.

Several approaches are available to address the fact that commercial Aroclor mixtures contain PCB congeners that have dioxin-like activity, although the benefits and limitations of these approaches are still being evaluated. These approaches include recommendations made in EPA's *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures* (EPA 2000e), an example given in EPA's document *PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures* (EPA 1996b), PCB and PCB TEQ approaches discussed in the risk assessment for the Housatonic Superfund site (Weston Solutions 2005), and PCB and PCB TEQ risks from Columbia Basin fish (EPA 2000e). The implications of different approaches to addressing this issue were explored quantitatively to estimate the PCB cancer risk for the adult tribal RME seafood consumption scenario based on Tulalip data. As recommended by EPA Region 10 (EPA 2006c), several options were explored:

1. Aroclor sum cancer risk alone
2. PCB TEQ cancer risk alone
3. Aroclor sum cancer risk plus PCB TEQ cancer risk
4. PCB TEQ cancer risk plus cancer risk computed using the sum of Aroclor mass minus the mass of dioxin-like PCB congeners, and using the total PCB SF

The first two approaches may lead to underestimates of risk, and the third may overestimate risk because the mass and toxicity of PCB congeners may be double counted. The basis for Option 4 is to prevent the double counting of the mass of the dioxin-like PCB congeners. However, this approach does not address the potential double counting of the toxicity of dioxin-like PCB congeners that were present in the Aroclor test material used to generate the PCB SF.

The sum of Aroclor risks was calculated using the total PCB SF, and PCB TEQ risks were calculated using the dioxin SF. The EPCs, CDIs, and risk estimates for the adult tribal RME seafood consumption scenario based on Tulalip data needed for the first three options and the first part of Option 4 were previously presented in Sections B.3 and B.5. To calculate the sum of Aroclor mass minus the mass of the dioxin-like PCB congeners (for the second part of Option 4 above), the difference of the sum of Aroclors and the mass of dioxin-like PCB congeners was calculated for each sample, and the EPCs for each tissue type were recalculated using ProUCL 4. The resulting EPCs are presented in Table B.6-33.

Table B.6-33. EPCs for sum of Aroclor mass minus the mass of dioxin-like PCB congeners

Seafood Category	No. Detected/ Total No. of Samples[a]	Mean Value (mg/kg ww)	Maximum Detection (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[b]
Benthic fish, fillet	8/8	1.20	1.86	na	Student's-T UCL	1.27
Benthic fish, whole body	8/8	2.76	4.00	na	Student's-T UCL	3.63
Clams	8/8	0.14	0.53	na	95% Chebyshev UCL	0.41
Crab, edible meat	8/8	0.21	0.36	na	Gamma UCL	0.26
Crab, whole body	6/6	0.98	1.70	na	Gamma UCL	1.36
Pelagic fish	11/11	3.20	16.95	na	95% Chebyshev UCL	7.57

[a] The total number of samples with PCB congener data (n = 49) available for this analysis, and for the PCB TEQ EPCs presented in Section B.3, were fewer than the total number of samples with Aroclor data (n = 221) available for the total PCB EPC presented in Section B.3.

[b] No PCB congener data were available for mussels. Therefore, the EPC for total PCBs (Aroclors) was used for mussels in CDI estimations.

EPC – exposure point concentration

na – not applicable

PCB – polychlorinated biphenyl

RL – reporting limit

UCL – upper concentration limit

ww – wet weight

Table B.6-34 presents the cancer risk estimates calculated for each of the four approaches using EPCs presented in Section B.3 (for Options 1 through 3 and the first part of Option 4) and from Table B.6-34 (for the second part of Option 4) and the appropriate SFs. Options 1 and 2, which were presented in Section B.5, have the lowest risk estimates of the four options evaluated (2×10^{-3} and 1×10^{-3}, respectively). The risk estimates for the other two options are 3×10^{-3} and 4×10^{-3} (Table B.6-34).

An issue with the comparison of risks associated with these four options is that different datasets with different numbers of samples were available for different portions of the analysis. The total PCB data as sum of Aroclors were available for a larger dataset (sample size = 221, used for Option 1 and the first part of Option 3). A subset of samples from this larger data was also analyzed for PCB congeners (sample size = 49, used for Options 2, the second part of Option 3, and both parts of Option 4). The larger dataset was used for risk characterization in this HHRA because it was expected to more accurately represent LDW PCB risks than the PCB congener subset. Excess cancer risk estimates for total PCBs (without consideration of PCB TEQ) for the larger dataset and the congener subset are not equivalent, with risk estimates for the subset being 50% higher (see Section B.6.1.1.8). Differences in EPCs for total PCBs

between the larger dataset and the congener subset are due to differences in the specific samples selected (i.e., the total PCB concentrations in the congener subset were higher on average than in the larger dataset) as well as differences in the number of samples (i.e., smaller sample sizes contribute to lower confidence in estimates of the mean, and therefore higher UCLs and EPCs).

Table B.6-34. PCB cancer risk estimates using various risk calculation methods

Risk Calculation Method	Risk Constituent	Cancer CDI (mg/kg-day)	Adult Tulalip RME Cancer Risk
1. Aroclor sum cancer risk alone	Aroclor PCBs	9.2×10^{-4}	2×10^{-3}
2. PCB TEQ cancer risk alone	PCB TEQ	8.1×10^{-9}	1×10^{-3}
3. Aroclor sum cancer risk plus PCB TEQ cancer risk	Aroclor PCB risk and PCB TEQ risk	na	3×10^{-3}
4. PCB TEQ cancer risk plus cancer risk computed using sum of Aroclor mass minus mass of dioxin-like PCB congeners, and using total PCBs SF	Aroclor mass minus mass of dioxin-like PCB congeners, and using total PCBs SF	1.3×10^{-3}	3×10^{-3}
	PCB TEQ	8.1×10^{-9}	1×10^{-3}
Final estimate[a]			**4×10^{-3}**

[a] Final estimate is sum of above two constituents.

CDI – chronic daily intake

na – not applicable

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

SF – slope factor

TEQ – toxic equivalent

B.6.3.2 Risk calculations for undetected chemicals

As indicated in Section B.5.2, risks were characterized only for those chemicals that were detected in the medium specific to that exposure scenario (i.e., sediment or tissue). Several chemicals in each scenario were never detected, but a sufficient number of sample RLs exceeded the applicable RBCs, and these undetected chemicals were thereby identified as COPCs (see Section B.3.3).[47] Hypothetical EPCs were calculated for these undetected chemicals and are presented in Section B.3.4.3. The hypothetical EPCs correspond to half the highest RL for that chemical. Risks calculated using one-half RL values overestimate risks if these COPCs are not present (or are present only at concentrations lower than one-half the highest RL), or

[47] A total of 26 of 58 COPCs in tissue were non-detects in all seafood categories, 2 of 20 COPCs in sediment for the netfishing scenario were non-detects, and 5 of 28 COPCs in sediment for the beach play and clamming scenarios were non-detects. N-nitroso-di-n-propylamine was detected in one tissue sample, which was JN-qualified. The detected concentration was less than half the highest RL. Therefore, this chemical was considered with the non-detected chemicals, and one-half of the highest RL was used for its EPC.

underestimate risks if the COPCs are present at an average concentration greater than one-half the RL. Laboratory RLs and the degree of spatial coverage of the samples are important factors to consider in determining whether the lack of detection truly indicates that a substance is not present. Information on possible chemical sources and environmental conditions (e.g., that affect the transport or speciation of chemicals) is also useful. These issues are discussed after the presentation of the risks and hazards associated with undetected chemicals. If these COPCs are truly present in the samples, then the effect of using one-half the RL in the risk analysis is uncertain because the true concentration could be anywhere between zero and the RL.

Similar to the risk results for detected carcinogenic COPCs in tissue, all undetected carcinogenic COPCs in tissue had hypothetical excess cancer risk estimates higher than 1×10^{-6} for the adult tribal RME seafood consumption scenario based on Tulalip data (Table B.6-35). Nearly all of the risk estimates for undetected chemicals were above this excess cancer risk level for the child tribal RME scenario based on Tulalip data, adult API RME scenario, and one-meal-per-month seafood consumption scenarios. The adult tribal CT scenario based on Tulalip data, child tribal CT scenario based on Tulalip data, and adult API CT scenario had approximately half of the undetected chemicals exceed the 1×10^{-6} risk level. The highest hypothetical excess cancer risk estimates were for benzidine (1 for both the adult tribal scenario based on Suquamish data and the adult tribal RME scenario based on Tulalip data) and n-nitrosodimethylamine[48] (3×10^{-1} for the adult tribal scenario based on Suquamish data and 2×10^{-1} for the adult tribal RME scenario based on Tulalip data). The total hypothetical excess cancer risk estimates for these 16 COPCs were approximately three orders of magnitude higher than the excess cancer risk estimates for detected tissue COPCs. However, the quantifiable presence or absence of undetected chemicals in tissue is unknown. Consequently, it would not be appropriate to sum excess cancer risk estimates from detected COPCs with hypothetical excess cancer risk estimates from undetected COPCs.

[48] The detected concentration was less than half the highest RL. Therefore, this chemical was considered with the non-detected chemicals, and one-half of its highest RL was used for its EPC.

Table B.6-35. Summary of hypothetical excess cancer risk estimates for seafood consumption scenarios from COPCs that were never detected in LDW tissue samples

Undetected Chemicals in Seafood Tissue	Hypothetical Excess Cancer Risks by Seafood Consumption Scenario										
	Adult Tribal RME (Tulalip Data)	Adult Tribal **CT (Tulalip Data)**	Child Tribal RME (Tulalip Data)	Child Tribal CT (Tulalip Data)	Adult Tribal (Suquamish Data)	Adult API RME	Adult API CT	Adult One Meal Per Month			
								Benthic Fish	Clam	Crab	Pelagic Fish
1,2-Diphenylhydrazine[a]	3×10^{-5}	2×10^{-6}	3×10^{-6}	4×10^{-7}	2×10^{-4}	8×10^{-6}	2×10^{-7}	1×10^{-6}	na	1×10^{-6}	1×10^{-6}
1,4-Dichlorobenzene	5×10^{-6}	2×10^{-7}	9×10^{-7}	1×10^{-7}	1×10^{-5}	8×10^{-7}	2×10^{-8}	3×10^{-7}	2×10^{-8}	3×10^{-7}	3×10^{-7}
2,4,6-Trichlorophenol	2×10^{-5}	5×10^{-7}	3×10^{-6}	2×10^{-7}	8×10^{-5}	4×10^{-6}	4×10^{-8}	7×10^{-7}	2×10^{-8}	7×10^{-7}	4×10^{-6}
2,4- and 2,6-Dinitrotoluene[b]	1×10^{-3}	2×10^{-5}	2×10^{-4}	9×10^{-6}	5×10^{-3}	2×10^{-4}	2×10^{-6}	2×10^{-5}	1×10^{-6}	5×10^{-5}	2×10^{-4}
3,3'-Dichlorobenzidine[c]	5×10^{-3}	2×10^{-4}	9×10^{-4}	1×10^{-4}	1×10^{-2} [d]	9×10^{-4}	2×10^{-5}	3×10^{-4}	2×10^{-5}	3×10^{-4}	3×10^{-4}
3-Nitroaniline	7×10^{-5}	2×10^{-6}	1×10^{-5}	1×10^{-6}	3×10^{-4}	2×10^{-5}	2×10^{-7}	3×10^{-6}	2×10^{-7}	3×10^{-6}	1×10^{-5}
4-Nitroaniline	6×10^{-5}	1×10^{-6}	1×10^{-5}	6×10^{-7}	3×10^{-4}	1×10^{-5}	1×10^{-7}	1×10^{-6}	9×10^{-8}	3×10^{-6}	1×10^{-5}
Aniline	2×10^{-5}	1×10^{-6}	4×10^{-6}	5×10^{-7}	7×10^{-5}	4×10^{-6}	9×10^{-8}	2×10^{-6}	1×10^{-7}	2×10^{-6}	2×10^{-6}
Benzidine[c]	$1 \times 10^{+0}$ [d]	3×10^{-1} [d]	6×10^{-1} [d]	1×10^{-1} [d]	$1 \times 10^{+0}$ [d]	6×10^{-1} [d]	2×10^{-2} [d]	2×10^{-1} [d]	3×10^{-2} [d]	3×10^{-1} [d]	3×10^{-1} [d]
bis(2-chloroethyl)ether	4×10^{-4}	1×10^{-5}	7×10^{-5}	6×10^{-6}	9×10^{-4}	6×10^{-5}	1×10^{-6}	1×10^{-5}	1×10^{-6}	3×10^{-5}	1×10^{-5}
bis(2-chloroisopropyl)ether	1×10^{-5}	6×10^{-7}	3×10^{-6}	3×10^{-7}	4×10^{-5}	2×10^{-6}	5×10^{-8}	9×10^{-7}	6×10^{-8}	9×10^{-7}	9×10^{-7}
Hexachlorobutadiene	2×10^{-5}	7×10^{-7}	3×10^{-6}	3×10^{-7}	5×10^{-5}	3×10^{-6}	6×10^{-8}	1×10^{-6}	7×10^{-8}	1×10^{-6}	1×10^{-6}
Hexachloroethane	3×10^{-6}	1×10^{-7}	5×10^{-7}	6×10^{-8}	8×10^{-6}	5×10^{-7}	1×10^{-8}	2×10^{-7}	1×10^{-8}	2×10^{-7}	2×10^{-7}
n-Nitrosodimethylamine	2×10^{-1} [d]	5×10^{-3}	3×10^{-2} [d]	2×10^{-3}	3×10^{-1} [d]	2×10^{-2} [d]	3×10^{-4}	7×10^{-3}	5×10^{-5}	1×10^{-2} [d]	7×10^{-3}
n-Nitroso-di-n-propylamine[e]	1×10^{-3}	6×10^{-5}	3×10^{-4}	3×10^{-5}	4×10^{-3}	2×10^{-4}	5×10^{-6}	9×10^{-5}	6×10^{-6}	9×10^{-5}	9×10^{-5}
n-Nitrosodiphenylamine	2×10^{-6}	5×10^{-8}	3×10^{-7}	2×10^{-8}	7×10^{-6}	4×10^{-7}	4×10^{-9}	6×10^{-8}	4×10^{-9}	6×10^{-8}	3×10^{-7}
Toxaphene	5×10^{-4}	8×10^{-6}	9×10^{-5}	4×10^{-6}	3×10^{-3}	1×10^{-4}	1×10^{-6}	1×10^{-5}	6×10^{-6}	9×10^{-6}	1×10^{-4}
Total excess cancer risk	**$1 \times 10^{+0}$**	**3×10^{-1}**	**6×10^{-1}**	**1×10^{-1}**	**$1 \times 10^{+0}$**	**6×10^{-1}**	**2×10^{-2}**	**2×10^{-1}**	**3×10^{-2}**	**3×10^{-1}**	**3×10^{-1}**

[a] No clam or benthic whole-body fish data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories, as described in Section B.5.3.1.1.

[b] EPA's IRIS database presents a slope factor for the mixture 2,4- and 2,6-dinitrotoluene but does not provide individual slope factors for these chemicals. Therefore, excess cancer risks are presented here only for the mixture.

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories, as described in Section B.5.3.1.1.

[d] Because the excess cancer risk is greater than 0.01, risk was calculated using the exponential equation in EPA (1989).

[e] N-nitroso-di-n-propylamine was detected in one benthic fish (whole-body) sample. Because of uncertainty surrounding the validity of this single detection, which was JN-qualified (see Section B.6.1.1.5), n-nitroso-di-n-propylamine was evaluated in the uncertainty section. In order to be consistent with other undetected chemicals, the EPC values used to generate the risk numbers for this chemical were based on one-half the maximum RL. This approach was conservative because one-half the maximum RL was greater than the single detected concentration.

API – Asian and Pacific Islander

CDI – chronic daily intake

COPC – chemical of potential concern

CT – central tendency

EPC – exposure point concentration

LDW – Lower Duwamish Waterway

RL – reporting limit

RME – reasonable maximum exposure

There were 10 chemicals that were never detected in LDW tissue samples and that had hypothetical non-cancer HQs greater than 1 for the adult tribal RME seafood consumption scenario based on Tulalip data (Table B.6-36). The non-cancer HIs for all scenarios ranged from < 1 for one-meal-per-month consumption of clams to > 1,000 for the adult tribal scenario based on Suquamish data (> 500 for the adult tribal RME scenario based on Tulalip data). N-nitrosodimethylamine contributed two-thirds or more of the non-cancer HI.

Table B.6-36. Summary of hypothetical non-cancer hazard estimates for seafood consumption scenarios from COPCs that were never detected in LDW tissue samples

Undetected Chemicals	Hypothetical Non-Cancer Hazard Estimates by Seafood Consumption Scenario										
	Adult Tribal RME (Tulalip Data)	Adult Tribal **CT (Tulalip Data)**	Child Tribal RME (Tulalip Data)	Child Tribal CT (Tulalip Data)	Adult Tribal (Suquamish Data)	Adult API RME	Adult API CT	Adult One Meal per Month			
								Benthic Fish	Clam	Crab	Pelagic Fish
1,3-Dichlorobenzene	0.07	0.007	0.1	0.02	0.2	0.03	0.002	0.01	0.0007	0.01	0.01
1,4-Dichlorobenzene	0.007	0.0007	0.01	0.002	0.02	0.003	0.0002	0.001	0.00007	0.001	0.001
2,4,6-Trichlorophenol	16	1	34	2	70	9	0.3	2	0.04	2	8
2,4-Dichlorophenol	0.1	0.02	0.3	0.03	0.4	0.06	0.004	0.02	0.001	0.02	0.02
2,4-Dinitrophenol	3	0.2	5	0.5	9	1	0.06	0.3	0.02	0.3	0.8
2,4-Dinitrotoluene	0.8	0.04	2	0.08	3	0.4	0.01	0.04	0.002	0.08	0.4
2,6-Dinitrotoluene	2	0.07	3	0.1	7	0.8	0.02	0.08	0.002	0.2	0.8
2-Chlorophenol	0.1	0.009	0.2	0.02	0.2	0.03	0.002	0.01	0.0008	0.01	0.01
3-Nitroaniline	11	0.8	23	2	49	6	0.2	1	0.07	1	5
4,6-Dinitro-o-cresol	32	2	69	5	146	17	0.6	3	0.2	3	16
4-Chloroaniline[a]	0.3	0.03	0.6	0.07	0.8	0.1	0.008	0.04	0.003	0.04	0.04
4-Nitroaniline	1	0.06	2	0.1	5	0.6	0.01	0.05	0.003	0.1	0.5
Aniline	0.6	0.07	1	0.2	2	0.2	0.02	0.09	0.006	0.09	0.09
Benzidine[a]	8	1	17	2	24	4	0.3	0.9	0.09	1	1
Bis(2-chloroisopropyl) ether	0.005	0.0005	0.01	0.001	0.02	0.002	0.0001	0.0008	0.00005	0.0008	0.0008
Hexachlorobutadiene	1	0.1	2	0.2	3	0.4	0.03	0.2	0.01	0.2	0.2
Hexachlorocyclopentadiene	7	0.5	14	1	29	4	0.1	0.6	0.04	0.6	3
Hexachloroethane	0.2	0.02	0.4	0.05	0.6	0.08	0.006	0.03	0.002	0.03	0.03
Nitrobenzene	0.4	0.04	0.9	0.09	1	0.2	0.01	0.06	0.004	0.06	0.06
n-Nitrosodimethylamine	421	26	905	56	866	141	6	38	0.3	78	38
n-Nitrosodiphenylamine	0.02	0.001	0.03	0.002	0.07	0.009	0.0003	0.002	0.0001	0.002	0.008
Total non-cancer hazard	**506**	**32**	**1,080**	**69**	**1,216**	**184**	**8**	**46**	**0.8**	**87**	**74**

[a] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

API – Asian and Pacific Islander

CDI – chronic daily intake

COPC – chemical of potential concern

CT – central tendency

LDW – Lower Duwamish Waterway

RME – reasonable maximum exposure

Hypothetical excess cancer risk and non-cancer hazard estimates for the sediment COPCs that were never detected in LDW sediments are summarized in Tables B.6-37 and B.6-38, respectively. For all direct sediment exposure scenarios assessed for which benzidine was analyzed, benzidine had excess cancer risk estimates greater than 1×10^{-6} based on the use of one-half the maximum RLs for the EPCs. The hypothetical excess cancer risks from benzidine alone were greater than the excess cancer risks from all other sediment COPCs combined. The hypothetical excess cancer risk estimates for N-nitrosodimethylamine exceeded 1×10^{-6} for both netfishing scenarios and the 120-day-per-year clamming scenario. Total hypothetical excess cancer risk estimates for undetected chemicals were higher or lower than total excess cancer risk estimates for detected chemicals (Table B.5-63), depending on the scenario.

Table B.6-37. Summary of hypothetical excess cancer risks for direct sediment exposure scenarios from COPCs that were never detected in LDW sediment samples

	Hypothetical Excess Cancer Risks by Direct Sediment Exposure Scenario												
	Netfishing		Beach Play **RME**								Clamming		
Undetected Chemicals	RME	CT	Area 1	Area 2	Area 3	Area 4	Area 5	Area 6	Area 7	Area 8	7 Days per Year	Tribal RME	183 Days per Year
Benzidine	6×10^{-5}	1×10^{-5}	na	na	3×10^{-5}	na	na	na	na	na	3×10^{-6}	1×10^{-4}	2×10^{-4}
Bis(2-chloroethyl) ether	nc	nc	3×10^{-9}	8×10^{-9}	4×10^{-9}	5×10^{-8}	2×10^{-8}	9×10^{-9}	9×10^{-9}	9×10^{-9}	4×10^{-9}	1×10^{-7}	2×10^{-7}
n-Nitrosodimethylamine	1×10^{-5}	2×10^{-6}	7×10^{-7}	9×10^{-7}	1×10^{-6}	2×10^{-6}	7×10^{-7}	2×10^{-7}	2×10^{-7}	2×10^{-7}	3×10^{-7}	2×10^{-5}	3×10^{-5}
n-Nitroso-di-n-propylamine	nc	nc	3×10^{-8}	1×10^{-7}	7×10^{-8}	1×10^{-6}	4×10^{-8}	3×10^{-8}	4×10^{-8}	4×10^{-8}	1×10^{-7}	4×10^{-6}	7×10^{-6}
Total excess cancer risk	**7×10^{-5}**	**1×10^{-5}**	**7×10^{-7}**	**1×10^{-6}**	**3×10^{-5}**	**3×10^{-6}**	**7×10^{-7}**	**3×10^{-7}**	**2×10^{-7}**	**2×10^{-7}**	**3×10^{-6}**	**1×10^{-4}**	**2×10^{-4}**

COPC – chemical of potential concern

CT – central tendency

LDW – Lower Duwamish Waterway

RME – reasonable maximum exposure

na – not applicable; no samples were analyzed for benzidine

nc – not a COPC for the netfishing scenarios based on a lack of exceedance of the industrial-based RBC used for the netfishing COPC screening (note that the selection of COPCs for the clamming and beach play RME scenarios used lower residential RBCs resulting in more exceedances)

Table B.6-38. Summary of hypothetical non-cancer hazards for direct sediment exposure scenarios from COPCs that were never detected in LDW sediment samples

	Hypothetical Non-Cancer Hazard by Direct Sediment Exposure Scenario (HQ)												
	Netfishing		Beach Play **RME**								Clamming		
Undetected Chemicals	RME	CT	Area 1	Area 2	Area 3	Area 4	Area 5	Area 6	Area 7	Area 8	7 Days per Year	Tribal RME	183 Days per Year
4,6-Dinitro-o-cresol	nc	nc	0.003	0.008	0.003	0.05	0.02	0.009	0.01	0.01	0.0009	0.01	0.02
Benzidine	0.0001	0.00003	na	na	0.0006	na	na	na	na	na	0.00001	0.0002	0.0003
n-Nitrosodimethylamine	0.05	0.01	0.02	0.03	0.04	0.06	0.02	0.006	0.007	0.007	0.002	0.06	0.09
Total non-cancer hazard (HI)	**0.05**	**0.01**	**0.02**	**0.04**	**0.04**	**0.1**	**0.04**	**0.02**	**0.02**	**0.02**	**0.003**	**0.07**	**0.1**

COPC – chemical of potential concern

CT – central tendency

HI – hazard index, equals the sum of hazard quotients

LDW – Lower Duwamish Waterway

RBC – risk-based concentration

RME – reasonable maximum exposure

na – not applicable; no samples were analyzed for benzidine

nc – not a COPC for the netfishing scenarios based on a lack of exceedance of the industrial-based RBC used for the netfishing COPC screening (note that the selection of COPCs for the clamming and beach play RME scenarios used lower residential RBCs resulting in more exceedances)

Hypothetical non-cancer HQs for the COPCs that were never detected in sediment were low (0.1 or lower) for all assessed direct sediment exposure scenarios (Table B.6-38). Non-cancer HIs for all direct sediment exposure scenarios, based on both hypothetical hazard estimates for undetected chemicals (Table B.6-38) and actual hazard estimates for detected chemicals (Tables B.5-25, B.5-26, B.5-35 to B.5-42, B.5-45, and B.5-46), were all below 1. The hypothetical non-cancer HIs were many times lower than those based on detected chemicals, and indicate that these undetected chemicals would not pose non-cancer health hazards in the LDW at the RLs used in this assessment.

The sample-specific RL is based on the lowest point of the calibration curve associated with each analytical batch of samples. The most common reason for elevated RL values is sample extract dilution. For example, elevated RLs for some chemicals in some areas reflect the greater degree of analytical dilution required for quantification of other analytes, such as PCBs. In addition, there is a group of analytes that are known to be analytically difficult to quantify. These compounds tend to have chemical characteristics that differ from those of other analytes being analyzed using the same method. For example, benzidine, benzoic acid, benzyl alcohol, phenols, and n-nitrosodiphenylamine are all more chemically reactive than the other SVOCs analyzed by EPA (EPA 2003a). More reactive compounds can be difficult to extract and often degrade during analysis. The group of analytically difficult compounds included the following chemicals: chlorobenzenes, phenol, methyl phenols, pentachlorophenol, benzoic acid, benzyl alcohol, hexachlorobutadiene, hexachlorobenzene, and n-nitrosodiphenylamine. These compounds are analytically difficult to quantify at the concentrations required for comparison to risk-based analytical concentration goals and are generally very rarely detected.

Although EPA and Ecology's efforts to identify current and historical sources of contamination to the LDW are not yet complete, it does not appear that the major industries that used these undetected chemicals are or have been present in the LDW area. Benzidine was used as an intermediate in the production of azo dyes, sulfur dyes, fast color salts, naphthols, and other dyeing compounds. Similarly, 3,3′-dichlorobenzidine, which also showed hypothetical unacceptable risks, is used in the manufacture of pigments for printing inks, textile, plastics, enamels, paint, leather, and rubber. N-nitrosodimethylamine can be released from the manufacture of pesticides, rubber tires, alkylamines, and dyes and also may form under natural conditions in air, water, and soil as a result of chemical, photochemical, and biological processes. It is possible that these products were used or handled by industries along the LDW, but current research has not identified any reason to believe that there are undetected high concentrations of these chemicals in the LDW.

B.6.3.3 Risk calculations for discrete areas within the LDW

B.6.3.3.1 Seafood consumption scenarios by tissue sampling area

The seafood consumption scenarios presented in the risk characterization (Section B.5) were based on the assumption that fish and shellfish are consumed from throughout the LDW. However, individual anglers may utilize smaller areas of the LDW, although the spatial scale in the LDW at which these consumption rates could be supported is unknown. To investigate the risk implications of this behavior, seafood consumption scenarios were evaluated for the four tissue sampling areas of the LDW defined for the collection of fish and crabs (Windward 2004c). The four tissue sampling areas of the LDW are presented on Map B.2-2. This evaluation was limited to the four chemicals with the highest risk estimates for the LDW-wide assessment (see Tables B.5-61 and B.5-62) for the adult tribal RME scenario based on Tulalip data, the child tribal RME scenario based on Tulalip data, and the adult API RME scenario. This evaluation focuses specifically on risk estimates for tissue sampling areas 1-4 and comparison of those risk estimates to LDW wide estimates.

In accordance with a request from EPA for this uncertainty assessment, risks associated with clam consumption in these four areas were evaluated despite the lack of any studies capable of determining whether current or future clam populations would be sufficient in most areas to support the clam consumption rates assumed in these scenarios (e.g., 900 clams per year per individual assumed to be consumed for the adult tribal RME scenario based on Tulalip data) (see Map B.3-2). Area-specific EPCs for arsenic (inorganic), cPAHs, PCB TEQ, and total PCBs are presented in Table B.6-39. These EPCs were included in the exposure and risk equations described in Section B.3 to calculate excess cancer risk and non-cancer hazard estimates for the four areas (Tables B.6-40 and B.6-41). The EPCs for cPAHs are based on only the 2004 cPAH data , as was done in the risk characterization section. All other exposure parameters remained the same as listed in Section B.3 (i.e., it was assumed that the fractional intake, ingestion rates, and exposure durations remained the same as for the LDW-wide seafood consumption scenarios). LDW-wide risk estimates for the seafood categories included are provided for comparison.

Table B.6-39. Exposure point concentrations for chemicals on a tissue sampling area-specific basis for the seafood consumption scenarios

Chemical	Consumption Category	No. Detected/ No. of Samples	Mean Value (mg/kg ww)	Maximum Detected Conc. (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Tissue Sampling Area 1							
Arsenic (inorganic)	benthic fish, fillet	2/2	0.004	0.004	na	maximum detect	0.004
	benthic fish, whole body	2/2	0.055	0.090	na	maximum detect	0.090
	clams	3/3	0.555	0.885	na	maximum detect	0.885
	crab, edible meat	2/2	0.020	0.030	na	maximum detect	0.030
	crab, whole body	2/2	0.034	0.046	na	maximum detect	0.046
	pelagic fish	2/2	0.020	0.020	na	maximum detect	0.020
cPAHs	benthic fish, fillet	1/2	3.4×10^{-4}	4.6×10^{-4} J	4.5×10^{-4}	maximum detect	4.6×10^{-4}
	benthic fish, whole body	6/6	2.2×10^{-3}	2.8×10^{-3} J	na	Student's-t UCL	2.8×10^{-3}
	clams	5/5	9.8×10^{-3}	0.012	na	maximum detect	0.012
	crab, edible meat	3/6	5.1×10^{-4}	8.4×10^{-4} J	6.5×10^{-4}	maximum detect	0.00084
	crab, whole body	6/6	6.6×10^{-4}	8.1×10^{-4} JM	na	Modified t-UCL	7.4×10^{-4}
	pelagic fish	6/6	5.9×10^{-4}	7.0×10^{-4} J	na	Student's-t UCL	6.5×10^{-4}
PCB TEQ	benthic fish, fillet	5/5	1.12×10^{-5}	1.29×10^{-5}	na	maximum detect	1.29×10^{-5}
	benthic fish, whole body	2/2	2.15×10^{-5}	2.47×10^{-5}	na	maximum detect	2.47×10^{-5}
	clams	2/2	4.66×10^{-7}	4.91×10^{-7}	na	maximum detect	4.91×10^{-7}
	crab, edible meat	3/3	2.28×10^{-6}	2.93×10^{-6}	na	maximum detect	2.93×10^{-6}
	crab, whole body	2/2	7.18×10^{-6}	7.32×10^{-6}	na	maximum detect	7.32×10^{-6}
	pelagic fish	2/2	1.26×10^{-5}	1.57×10^{-5}	na	maximum detect	1.57×10^{-5}

Table B.6-39, cont. Exposure point concentrations for chemicals on a tissue sampling area-specific basis for the seafood consumption scenarios

Chemical	Consumption Category	No. Detected/ No. of Samples	Mean Value (mg/kg ww)	Maximum Detected Conc. (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Total PCBs	benthic fish, fillet	17/17	0.51	1.6	na	99% Chebyshev UCL	1.8
	benthic fish, whole body	12/12	2.6	4.7	na	Student's-t UCL	3.3
	clams	5/5	0.028	0.033	na	maximum detect	0.033
	crab, edible meat	12/13	0.17	0.39 J	0.020	95% KM (t) UCL	0.223
	crab, whole body	9/9	0.88	1.4	na	Student's-t UCL	1.1
	mussel	15/19	0.031	0.056	0.013	95% KM (Percentile Bootstrap) UCL	0.039
	pelagic fish	17/17	0.87	1.83	na	Student's-t UCL	1.1
Tissue Sampling Area 2							
Arsenic (inorganic)	benthic fish, fillet	1/2	0.003	0.004 J	0.003	maximum detect	0.004
	benthic fish, whole body	2/2	0.055	0.070	na	maximum detect	0.070
	clams	3/3	1.97	3.27	na	maximum detect	3.3
	crab, edible meat	2/2	0.030	0.030	na	maximum detect	0.030
	crab, whole body	2/2	0.111	0.111	na	maximum detect	0.111
	pelagic fish	2/2	0.10	0.16	na	maximum detect	0.16
cPAHs	benthic fish, fillet	1/2	3.3×10^{-4}	4.4×10^{-4} J	2.3×10^{-4}	maximum detect	4.4×10^{-4}
	benthic fish, whole body	6/6	2.1×10^{-3}	2.8×10^{-3}	na	Student's-t UCL	2.6×10^{-3}
	clams	3/3	1.6×10^{-2}	2.3×10^{-2}	na	maximum detect	2.3×10^{-2}
	crab, edible meat	1/6	3.3×10^{-4}	3.3×10^{-4}	3.3×10^{-4}	maximum detect	3.3×10^{-4}
	crab, whole body	6/6	6.3×10^{-4}	6.7×10^{-4} JM	na	Modified t-UCL	7.0×10^{-4}
	pelagic fish	6/6	1.1×10^{-3}	1.9×10^{-3}	na	approximate gamma UCL	1.9×10^{-3}
PCB TEQ	benthic fish, fillet	2/2	1.34×10^{-5}	1.41×10^{-5}	na	maximum detect	1.41×10^{-5}
	benthic fish, whole body	2/2	2.08×10^{-5}	2.35×10^{-5}	na	maximum detect	2.35×10^{-5}
	clams	2/2	4.92×10^{-7}	5.42×10^{-7}	na	maximum detect	5.42×10^{-7}
	crab, edible meat	2/2	2.20×10^{-6}	2.65×10^{-6}	na	maximum detect	2.65×10^{-6}
	crab, whole body	2/2	5.45×10^{-6}	5.76×10^{-6}	na	maximum detect	5.76×10^{-6}
	pelagic fish	2/2	4.56×10^{-5}	7.30×10^{-5}	na	maximum detect	7.30×10^{-5}

Table B.6-39, cont. Exposure point concentrations for chemicals on a tissue sampling area-specific basis for the seafood consumption scenarios

Chemical	Consumption Category	No. Detected/ No. of Samples	Mean Value (mg/kg ww)	Maximum Detected Conc. (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Total PCBs	benthic fish, fillet	6/6	1.2	2.0	na	Student's-t UCL	1.8
	benthic fish, whole body	12/12	2.9	4.2	na	Student's-t UCL	3.4
	clams	3/3	0.036	0.043	na	maximum detect	0.043
	crab, edible meat	7/7	0.18	0.26	na	Student's-t UCL	0.23
	crab, whole body	7/7	0.70	0.84	na	Student's-t UCL	0.85
	pelagic fish	12/12	2.8	18.4	na	95% Chebyshev UCL	9.0
Tissue Sampling Area 3							
Arsenic (inorganic)	benthic fish, fillet	2/2	0.006	0.006	na	maximum detect	0.006
	benthic fish, whole body	2/2	0.040	0.040	na	maximum detect	0.040
	clams	2/2	1.2	2.1	na	maximum detect	2.1
	crab, edible meat	2/2	0.020	0.030	na	maximum detect	0.030
	crab, whole body	2/2	0.079	0.12	na	maximum detect	0.12
	pelagic fish	2/2	0.090	0.10	na	maximum detect	0.10
cPAHs	benthic fish, fillet	2/2	4.5×10^{-4}	5.3×10^{-4}	na	maximum detect	5.3×10^{-4}
	benthic fish, whole body	4/6	9.6×10^{-4}	2.2×10^{-3}	4.5×10^{-4}	maximum detect	2.2×10^{-3}
	clams	6/6	0.019	0.044	na	Student's-t UCL	0.031
	crab, edible meat	3/6	4.6×10^{-4}	6.3×10^{-4} J	6.5×10^{-4}	maximum detect	6.3×10^{-4}
	crab, whole body	6/6	6.5×10^{-4}	6.7×10^{-4} JM	na	Student's-t UCL	6.7×10^{-4}
	pelagic fish	6/6	1.1×10^{-3}	2.2×10^{-3}	na	Student's-t UCL	1.6×10^{-3}
PCB TEQ	benthic fish, fillet	2/2	7.34×10^{-6}	9.68×10^{-6}	na	maximum detect	9.68×10^{-6}
	benthic fish, whole body	2/2	1.34×10^{-5}	1.37×10^{-5}	na	maximum detect	1.37×10^{-5}
	clams	4/4	2.47×10^{-6}	5.65×10^{-6}	na	maximum detect	5.65×10^{-6}
	crab, edible meat	2/2	1.69×10^{-6}	1.73×10^{-6}	na	maximum detect	1.73×10^{-6}
	crab, whole body	1/1	1.16×10^{-5}	1.16×10^{-5}	na	maximum detect	1.16×10^{-5}
	pelagic fish	3/3	2.47×10^{-5}	3.61×10^{-5}	na	maximum detect	3.61×10^{-5}

Table B.6-39, cont. Exposure point concentrations for chemicals on a tissue sampling area-specific basis for the seafood consumption scenarios

Chemical	Consumption Category	No. Detected/ No. of Samples	Mean Value (mg/kg ww)	Maximum Detected Conc. (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Total PCBs	benthic fish, fillet	6/6	0.80	1.6	na	Student's-t UCL	1.2
	benthic fish, whole body	12/12	2.0	4.3	na	Student's-t UCL	2.5
	clams	6/6	0.29	0.58	na	Student's-t UCL	0.43
	crab, edible meat	6/7	0.18	0.3	0.020	95% KM (t) UCL	0.24
	crab, whole body	7/7	1.0	1.6	na	approximate gamma UCL	1.6
	mussel	3/3	0.050	0.060	na	maximum detect	0.060
	pelagic fish	12/12	2.6	8.8	na	approximate gamma UCL	4.1
Tissue Sampling Area 4							
Arsenic (inorganic)	benthic fish, fillet	1/2	0.003	0.005	0.003	maximum detect	0.005
	benthic fish, whole body	2/2	0.075	0.090	na	maximum detect	0.090
	clams	na	na	na	na	na	na
	crab, edible meat	na	na	na	na	na	na
	crab, whole body	na	na	na	na	na	na
	pelagic fish	2/2	0.070	0.090	na	maximum detect	0.090
cPAHs	benthic fish, fillet	1/2	4.3×10^{-4}	6.4×10^{-4}	4.5×10^{-4}	maximum detect	6.4×10^{-4}
	benthic fish, whole body	5/6	5.2×10^{-4}	6.6×10^{-4}	4.5×10^{-4}	maximum detect	6.6×10^{-4}
	clams	na	na	na	na	na	na
	crab, edible meat	1/1	6.3×10^{-4}	6.3×10^{-4}	na	maximum detect	6.3×10^{-4}
	crab, whole body	1/1	2.4×10^{-3}	2.4×10^{-3}	na	maximum detect	2.4×10^{-3}
	pelagic fish	6/6	5.0×10^{-4}	6.5×10^{-4}	na	Student's-t UCL	6.0×10^{-4}
PCB TEQ	benthic fish, fillet	2/2	3.57×10^{-6}	4.23×10^{-6}	na	maximum detect	4.23×10^{-6}
	benthic fish, whole body	2/2	8.12×10^{-6}	1.21×10^{-5}	na	maximum detect	1.21×10^{-5}
	clams	na	na	na	na	na	na
	crab, edible meat	1/1	1.37×10^{-6}	1.37×10^{-6}	na	maximum detect	1.37×10^{-6}
	crab, whole body	1/1	9.35×10^{-6}	9.35×10^{-6}	na	maximum detect	9.35×10^{-6}
	pelagic fish	2/2	9.13×10^{-6}	1.07×10^{-5}	na	maximum detect	1.07×10^{-5}

Table B.6-39, cont. Exposure point concentrations for chemicals on a tissue sampling area-specific basis for the seafood consumption scenarios

Chemical	Consumption Category	No. Detected/ No. of Samples	Mean Value (mg/kg ww)	Maximum Detected Conc. (mg/kg ww)	Maximum RL (mg/kg ww)	Statistic Used	EPC Value (mg/kg ww)[a]
Total PCBs	benthic fish, fillet	4/4	0.56	0.71	na	maximum detect	0.71
	benthic fish, whole body	9/9	1.1	1.8	na	Student's-t UCL	1.4
	clams	na	na	na	na	na	na
	crab, edible meat	1/2	0.13	0.24	0.020	maximum detect	0.24
	crab, whole body	2/2	1.2	1.9	na	maximum detect	1.9
	pelagic fish	10/10	0.71	0.96	na	Student's-t UCL	0.80

Note: LDW-wide EPC values are presented in Section B.3.

[a] EPC was calculated assuming one-half the maximum RL for undetected values.

cPAH – carcinogenic polyaromatic hydrocarbon

EPC – exposure point concentration

J – estimated value

KM – Kaplan Meier method for calculating a UCL

M – calculated value, see Table B.2-4

na – not applicable

nd – not detected

PCB – polychlorinated biphenyl

RL – reporting limit

t (t-distribution) – statistical method used to calculate the mean for a normally distributed set of samples

UCL – upper confidence limit

ww – wet weight

Table B.6-40. Excess cancer risk estimates for select COPCs for seafood consumption scenarios for all tissue sampling areas compared to the LDW-wide area

Chemical	Seafood Category	Area 1			Area 2			Area 3			Area 4[a]			LDW-Wide		
		Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME
Arsenic[b, c]	clam	6×10^{-4}	1×10^{-4}	3×10^{-4}	2×10^{-3}	4×10^{-4}	1×10^{-3}	1×10^{-3}	3×10^{-4}	7×10^{-4}	nd	nd	nd	1×10^{-3}	3×10^{-4}	6×10^{-4}
	crab[d]	3×10^{-5}	5×10^{-6}	4×10^{-6}	4×10^{-5}	7×10^{-6}	8×10^{-6}	4×10^{-5}	8×10^{-6}	9×10^{-6}	nd	nd	nd	5×10^{-5}	9×10^{-6}	9×10^{-6}
	fish[e]	4×10^{-6}	7×10^{-7}	1×10^{-6}	2×10^{-5}	5×10^{-6}	9×10^{-6}	2×10^{-5}	3×10^{-6}	6×10^{-6}	9×10^{-5}[f]	4×10^{-5}[f]	4×10^{-5}[f]	1×10^{-5}	3×10^{-6}	5×10^{-6}
	total	**6×10^{-4}**	**1×10^{-4}**	**3×10^{-4}**	**2×10^{-3}**	**4×10^{-4}**	**1×10^{-3}**	**1×10^{-3}**	**3×10^{-4}**	**7×10^{-4}**	**9×10^{-5}**	**4×10^{-5}**	**4×10^{-5}**	**1×10^{-3}**	**3×10^{-4}**	**6×10^{-4}**
cPAHs[c,g]	clam	4×10^{-5}	7×10^{-6}	2×10^{-5}	8×10^{-5}	1×10^{-5}	4×10^{-5}	1×10^{-4}	2×10^{-5}	5×10^{-5}	nd	nd	nd	7×10^{-5}	1×10^{-5}	3×10^{-5}
	crab	3×10^{-6}	6×10^{-7}	5×10^{-7}	2×10^{-6}	3×10^{-7}	3×10^{-7}	2×10^{-6}	5×10^{-7}	4×10^{-7}	7×10^{-6}	1×10^{-6}	2×10^{-6}	3×10^{-6}	5×10^{-7}	4×10^{-7}
	fish	8×10^{-7}	1×10^{-7}	3×10^{-7}	2×10^{-6}	3×10^{-7}	6×10^{-7}	2×10^{-6}	3×10^{-7}	5×10^{-7}	1×10^{-6}	3×10^{-7}	6×10^{-7}	1×10^{-5}	2×10^{-6}	2×10^{-6}
	total	**4×10^{-5}**	**8×10^{-6}**	**2×10^{-5}**	**8×10^{-5}**	**1×10^{-5}**	**4×10^{-5}**	**1×10^{-4}**	**2×10^{-5}**	**5×10^{-5}**	**8×10^{-6}**	**1×10^{-6}**	**3×10^{-6}**	**8×10^{-5}**	**1×10^{-5}**	**3×10^{-5}**
PCB TEQ[c]	clam	3×10^{-5}	6×10^{-6}	2×10^{-5}	4×10^{-5}	7×10^{-6}	2×10^{-5}	4×10^{-4}	7×10^{-5}	2×10^{-4}	nd	nd	nd	2×10^{-4}	4×10^{-5}	1×10^{-4}
	crab	3×10^{-4}	6×10^{-5}	6×10^{-5}	3×10^{-4}	5×10^{-5}	5×10^{-5}	3×10^{-4}	6×10^{-5}	7×10^{-5}	4×10^{-4}	8×10^{-5}	2×10^{-4}	3×10^{-4}	6×10^{-5}	7×10^{-5}
	fish	4×10^{-4}	8×10^{-5}	1×10^{-4}	1×10^{-3}	2×10^{-4}	4×10^{-4}	7×10^{-4}	1×10^{-4}	2×10^{-4}	4×10^{-4}	7×10^{-5}	2×10^{-4}	7×10^{-4}	1×10^{-4}	2×10^{-4}
	total	**7×10^{-4}**	**1×10^{-4}**	**2×10^{-4}**	**1×10^{-3}**	**3×10^{-4}**	**5×10^{-4}**	**1×10^{-3}**	**2×10^{-4}**	**5×10^{-4}**	**8×10^{-4}**	**1×10^{-4}**	**4×10^{-4}**	**1×10^{-3}**	**2×10^{-4}**	**4×10^{-4}**
Total PCBs	clam[h]	3×10^{-5}	6×10^{-6}	2×10^{-5}	4×10^{-5}	7×10^{-6}	2×10^{-5}	4×10^{-4}	7×10^{-5}	2×10^{-4}	nd	nd	nd	6×10^{-4}	1×10^{-4}	2×10^{-4}
	crab	5×10^{-4}	8×10^{-5}	9×10^{-5}	4×10^{-4}	7×10^{-5}	8×10^{-5}	6×10^{-4}	1×10^{-4}	1×10^{-4}	1×10^{-3}	2×10^{-4}	4×10^{-4}	4×10^{-4}	8×10^{-5}	9×10^{-5}
	fish	5×10^{-4}	1×10^{-4}	1×10^{-4}	2×10^{-3}	4×10^{-4}	7×10^{-4}	1×10^{-3}	2×10^{-4}	3×10^{-4}	5×10^{-4}	9×10^{-5}	2×10^{-4}	6×10^{-4}	1×10^{-4}	2×10^{-4}
	total	**1×10^{-3}**	**2×10^{-4}**	**2×10^{-4}**	**2×10^{-3}**	**5×10^{-4}**	**8×10^{-4}**	**2×10^{-3}**	**4×10^{-4}**	**6×10^{-4}**	**2×10^{-3}**	**3×10^{-4}**	**6×10^{-4}**	**2×10^{-3}**	**3×10^{-4}**	**5×10^{-4}**
Total excess cancer risk (excluding PCB TEQ)[i]		**1×10^{-3}**	**3×10^{-4}**	**5×10^{-4}**	**4×10^{-3}**	**9×10^{-4}**	**2×10^{-3}**	**3×10^{-3}**	**7×10^{-4}**	**1×10^{-3}**	**2×10^{-3}**	**3×10^{-4}**	**6×10^{-4}**	**3×10^{-3}**	**6×10^{-4}**	**1×10^{-3}**
Total excess cancer risk (excluding total PCBs)[i]		**1×10^{-3}**	**2×10^{-4}**	**5×10^{-4}**	**3×10^{-3}**	**7×10^{-4}**	**2×10^{-3}**	**2×10^{-3}**	**5×10^{-4}**	**1×10^{-3}**	**9×10^{-4}**	**1×10^{-4}**	**4×10^{-4}**	**2×10^{-3}**	**5×10^{-4}**	**1×10^{-3}**

[a] No clam data were available in this area. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to clams was divided proportionally among the remaining seafood categories.

Table B.6-40, cont. Excess cancer risk estimates for select COPCs for seafood consumption scenarios for all tissue sampling areas compared to the LDW-wide area

[b] Arsenic concentrations are based on inorganic arsenic

[c] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining seafood categories.

[d] Includes crab whole body and crab edible meat consumption categories.

[e] Includes benthic fish fillet, benthic fish whole body, and pelagic fish consumption categories.

[f] Arsenic data were available only for fish in area 4. The total amount of seafood ingested per day was apportioned between pelagic and benthic fish to calculate this risk.

[g] cPAH concentrations are based on benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All carcinogenic PAH data are analyzed in the uncertainty analysis (Section B.6). Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for children for cPAHs is based on dose adjustments across the 0-6 year age range of children. See Section B.5.1 for more information.

[h] Clam category includes mussel here. This was the only chemical evaluated on a smaller spatial scale for which mussel data were available.

[i] Total excess cancer risks include only chemicals presented in this table, and thus underestimate the actual total.

Note - The LDW-wide risk number is not an average or sum of the other four areas, but rather a separate risk assessment.

API – Asian and Pacific Islander
CDI – chronic daily intake
COPC – chemical of potential concern
LDW – Lower Duwamish Waterway
nd – no data
cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
TEQ – toxic equivalent

Table B.6-41. Non-cancer hazard estimates for select COPCs for seafood consumption scenarios for all tissue sampling areas compared to the LDW-wide area

Chemical[a]	Seafood Category	Area 1			Area 2			Area 3			Area 4			LDW-wide		
		Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip data)	Adult API RME	Adult Tribal RME (Tulalip Data)	Child Tribal RME (Tulalip Data)	Adult API RME
Arsenic[b]	clam	1	3	1	5	11	6	3	7	4	nd	nd	nd	3	7	3
	crab[c]	0.06	0.1	0.02	0.09	0.2	0.04	0.09	0.2	0.04	nd	nd	nd	0.1	0.2	0.05
	fish[d]	0.008	0.02	0.01	0.05	0.1	0.05	0.04	0.08	0.03	0.2[e]	1[e]	0.2[e]	0.03	0.07	0.02
	total	**1**	**3**	**1**	**5**	**11**	**6**	**3**	**7**	**4**	**0.2**	**1**	**0.2**	**3**	**7**	**3**
Total PCBs	clam	0.8	2	0.9	1	2	1	10	21	10	nd	nd	nd	14	30	14
	crab	11	25	5	10	22	5	15	32	7	28	60	24	11	24	5
	fish	14	29	8	53	115	43	26	56	19	12	26	13	15	32	10
	total	**26**	**56**	**14**	**64**	**139**	**49**	**51**	**109**	**36**	**40**	**86**	**38**	**40**	**86**	**29**

[a] Includes only arsenic and total PCBs because no reference dose is available for carcinogenic PAHs or PCB TEQ.

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining seafood categories.

[c] Includes crab whole body and crab edible meat consumption categories.

[d] Includes benthic fish fillet, benthic fish whole body, and pelagic fish consumption categories.

[e] No clam data were available in this area. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to clams was divided proportionally among the remaining seafood categories.

Note – The LDW-wide risk number is not an average or sum of the other four areas, but rather a separate risk assessment.

API – Asian and Pacific Islander
CDI – chronic daily intake
COPC – chemical of potential concern
HQ – hazard quotient
nd – no data
PCB – polychlorinated biphenyl

Because of the limited number of samples for some of the tissue areas, over half of the EPCs used in the area-specific risk calculations were maximum values and some EPCs were based on single values (Table B.6-39). The smaller sample sizes for the tissue areas indicate that there is greater uncertainty in the risk estimates for the tissue areas compared to LDW-wide risk estimates. In general, both excess cancer risk and non-cancer hazard estimates for fish and crab were fairly similar across the four tissue sampling areas (Tables B.6-40 and B.6-41). All area-specific excess cancer risk estimates that exceeded LDW-wide estimates were within an order of magnitude or less of the LDW-wide estimates. Risks associated with clam consumption for all scenarios tended to be lower in area 1 compared to LDW-wide, particularly for total PCBs. More-detailed evaluations of the area-specific risk estimates are presented below by area.

For area 1, excess cancer risks for all scenarios for all seafood categories were lower than LDW-wide estimates for the four chemicals assessed, with the exception of cPAHs and PCB TEQ in crab for all scenarios and PCB TEQ in crab for the child tribal RME scenario based on Tulalip data (Table B.6-40). The excess cPAHs cancer risks associated with crab consumption were slightly higher for area 1 than LDW-wide. Excess cancer risk estimates for PCB TEQ exposure from child tribal crab consumption based on Tulalip data were the same for area 1 and LDW-wide. All non-cancer hazard estimates were lower for area 1 than LDW-wide hazard estimates, except for the HQ for total PCBs associated with crab consumption, which was the same for area 1 and LDW-wide (Table B.6-41). PCB cancer and non-cancer risks associated with clam consumption were much lower for area 1 compared to LDW-wide estimates.

Overall, risk estimates for area 2 differed the most from LDW-wide estimates, with many risk estimates for this area (both excess cancer and non-cancer) being higher than LDW-wide estimates (Table B.6-40). Specifically, area 2 excess cancer risk and hazard estimates were higher than the LDW-wide estimates for all chemicals evaluated for all of the seafood consumption scenarios for the fish category. The area 2 pelagic fish total PCBs EPC (Table B.6-39) was much higher than the LDW-wide pelagic fish EPC (Table B.3-35). The pelagic fish EPC estimate was strongly influenced by a single composite tissue sample that contained the highest PCB concentration of all LDW tissue samples.

Estimates for area 3 and area 4 were both similar to LDW-wide estimates; some excess cancer risk estimates were slightly higher or lower than LDW-wide risk estimates and many were equal (Table B.6-40). HQs for area 3, area 4, and LDW-wide were also fairly similar for arsenic and total PCBs, although crab HQs were higher for area 4 than LDW-wide and fish HQs were higher for area A than LDW-wide for all the seafood consumption scenarios (Table B.6-41).

B.6.3.3.2 Smaller spatial scale assessment of sediment beach play RME exposure scenario

The characterization of risks for children who are exposed to sediment while playing at the beach along the LDW were evaluated by dividing the waterway into eight exposure areas (see Map B.3-1). The assumption was that children were likely to visit particular areas along the waterway, rather than playing at all beaches along the LDW equally. However, it is possible that the assessment of risk using these larger areas could be different from the risks posed by playing at certain smaller beach areas. To investigate this issue, risks were characterized for the public beach at the Duwamish Waterway Park in the South Park neighborhood at RM 3 because this is likely to be more widely visited by children than other areas along the LDW because of its close proximity to the park and neighborhood. Risks were estimated for a selection of beach play COPCs (arsenic, carcinogenic PAHs, total DDTs, PCB TEQs, and total PCBs,) from the five sediment samples in the vicinity of the park using the same exposure assumptions described in Tables B.3-19 and B.3-20. Given the limited number of samples, the risk estimate is uncertain.

Risk estimates for the Duwamish Waterway Park tended to be lower or in some cases similar to the risks for the larger area (Area 5) (Table B.6-42). All HQs are below the risk threshold value of 1; and therefore, no chemicals are identified as exceeding acceptable risk levels for non-cancer hazards. Cancer risks exceed the total excess cancer risk of 1×10^{-6} for arsenic based only on the Duwamish Waterway Park sediment data.

Table B.6-42. Comparison of beach play RME excess cancer risks and non-cancer hazards at the Duwamish Waterway Park in the South Park neighborhood and in beach play RME area 5

Chemical	Non-Cancer Hazard		Cancer Risk	
	Duwamish Waterway Park Beach[a]	Area 5	Duwamish Waterway Park Beach[a]	Area 5
Arsenic	0.09	0.09	3×10^{-6}	3×10^{-6}
cPAHs[b]	na	na	1×10^{-6}	5×10^{-6}
Total DDTs	0.0002	0.0002	3×10^{-9}	3×10^{-9}
PCB TEQ	na	na	1×10^{-8}	1×10^{-7}
Total PCBs	0.01	0.04	6×10^{-8}	1×10^{-7}

[a] Because of the limited number of samples available (five or fewer for each chemical) for the Duwamish Waterway Park beach area, the maximum detected concentration was used as the EPC for all chemicals.

[b] Because of the potential for increased susceptibility of children to carcinogens with mutagenic activity, as described in EPA guidance (EPA 2005e), the risk estimate for children for cPAHs is based on dose adjustments across the 0-6 year age range of children. See Section B.5.1 for more information.

na – not applicable

TEQ – toxic equivalent

PCB – polychlorinated biphenyl

cPAH – carcinogenic polycyclic aromatic hydrocarbon

RME – reasonable maximum exposure

B.6.3.3.3 Evaluating smaller spatial scales using screening method for habitat biologist scenario

The sediment exposure areas quantified in the HHRA vary by scenario. Some are relatively small, such as beach play exposure areas, while others are much larger, such as the netfishing exposure area, which includes all intertidal and subtidal sediment in the LDW. The appropriate spatial scale for exposure areas is difficult to determine because many individuals, and certainly a population of individuals, may use multiple areas in the LDW.

To address the issue of evaluating smaller spatial scales, a screening method was developed for intertidal sediments using the habitat biologist scenario described in Section B.6.1.9. This scenario is considered to be appropriate for this purpose because there could be various habitat restoration projects in the future over a range of spatial scales. In this screening method, the chemical concentration at each sampling location was compared to a sediment threshold concentration that corresponded to a certain risk value (either 1×10^{-6}, 1×10^{-5}, or 1×10^{-4}), and then presented graphically on a map as order-of-magnitude ranges. The habitat biologist sediment risk threshold concentrations were calculated using the specific exposure parameters for the habitat biologist scenario presented in Table B.6-28 and rearranging the risk estimate equations to solve for a sediment concentration (given a specified risk estimate).

The primary benefit of this approach is that various spatial scales can be evaluated visually without a formal risk estimate. For example, if a region of interest includes 10 sediment samples in a particular intertidal section, and the hypothetical risk estimate for each of the 10 samples is below a particular risk threshold, then it is safe to assume that the risk for that area is also below that risk threshold.

This screening method was applied to four chemicals of concern: arsenic, PCBs, cPAHs, and dioxins/furans. The sediment threshold concentrations for the habitat biologist scenario are presented in Table B.6-43.

Table B.6-43. Sediment concentrations (mg/kg dw) corresponding to specific risk thresholds for the habitat biologist scenario for arsenic, total PCBs, cPAHs, and dioxins/furans

Chemical	10^{-6} Excess Cancer Risk	10^{-5} Excess Cancer Risk	10^{-4} Excess Cancer Risk
PCBs (total Aroclors)	14	140	1,400
Arsenic	47	470	4,700
Carcinogenic PAHs	4.0	40	400
Dioxins/furans	0.000299	0.00299	0.0299

Maps B.6-3, B.6-4, B.6-5, and B.6-6 present the point-by-point comparison of sediment concentrations of total PCBs, arsenic, cPAHs, and dioxins/furans, respectively, to the threshold concentrations presented in Table B.6-43. Each point on the map is portrayed relative to risk ranges calculated from the concentration divided by a

specific threshold concentration. Although the data are presented as risk ranges on these maps (e.g., >10^{-6} and < 10^{-5}), this presentation should not be equated with a formal risk estimate at a specific point. The exposure parameters developed for this scenario are appropriate for the assumed spatial scale for the scenario, which would be the size of a hypothetical restoration area. Such a restoration area would be larger than the area associated with a specific sediment sampling point. For smaller spatial scales, different exposure parameters would be more appropriate. In addition, the heterogeneity of chemical concentrations in sediment make single point risk estimates inappropriate.

Map B.6-7 presents a cumulative comparison of all four chemicals simultaneously, using an additive approach. For example, if the ratios of the concentrations of PCBs, arsenic, and cPAHs to the chemical-specific 10^{-6} risk threshold concentrations at a particular point were each 5, the sum of the ratios would be 15 and the point would be portrayed as > 10^{-5} on Map B.6-7.[49] Most points fall below the 1 x 10^{-6} threshold for excess cancer risk for the habitat biologist scenario; those that are above the 1 x 10^{-6} threshold are generally located in early action areas.

B.6.3.3.4 Evaluating smaller spatial scales using screening method for netfishing scenario

Because individual netfishers may use smaller areas than the entire LDW (i.e., the assumed exposure area used in the rest of the HHRA), an uncertainty analysis based on different combined spatial scales was conducted. Such an analysis may be useful to tribal members who conduct netfishing in smaller portions of the LDW for netfishing activities. Sufficient data do not exist to identify smaller areas within the LDW that may be preferentially used for netfishing, nor are there data to suggest an appropriate size of a smaller area.

Because netfishing may occur over an area that is larger than individual habitat restoration areas, a slightly different approach from the habitat biologist approach was used. A weighted concentration was created for each point and compared to sediment threshold concentrations. The threshold concentrations were calculated using the specific exposure parameters for the RME netfishing scenario presented in Tables B.3-15 and B.3-16 and rearranging the risk estimate equations to solve for a sediment concentration (given a specified risk estimate). The weighting factor was 25% toward the concentration at that location and 75% toward the LDW-wide EPC. For total PCBs, the SWAC UCL calculated as described in Section B.6.1.1.12 was used to represent the hypothetical LDW-wide exposure by compensating for spatial bias in the distribution of surface sediment samples analyzed for total PCBs (i.e., because a disproportionate number of samples were collected from areas known to be

[49] A ratio of 5 times the 10^{-6} threshold concentration is equivalent to 5 x 10^{-6} for a single chemical. Because cancer risks are additive for multiple chemicals, the threshold concentration ratios are also additive. A combined ratio of 15 is equivalent to 15 x 10^{-6} or 1.5 x 10^{-5}.

contaminated with PCBs, the UCL on the arithmetic mean may overestimate the EPC). As noted in B.6.1.1.12, further analysis is needed with regards to how to appropriately account for spatial sampling bias. Mathematically, this is equivalent to assuming that netfishing occurs at a single location[50] for 11 years and throughout the LDW for the other 33 years of the 44-year exposure duration assumed for this scenario.

Maps B.6-8, B.6-9, B.6-10, and B.6-11 present the point-by-point comparison of sediment concentrations of total PCBs, arsenic, cPAHs, and dioxins/furans, respectively, to the calculated sediment thresholds. As with the habitat biologist scenario, each point on the map is portrayed as falling within one of four risk ranges, depending on the magnitude of the weighted concentration relative to a specific threshold concentration. Although the data are presented as risk ranges on these maps (e.g., > 10^{-6} and < 10^{-5}), this presentation should not be equated with a formal risk estimate at a specific point. The exposure parameters developed for the netfishing RME scenario are appropriate for the assumed spatial scale for netfishing as presented in Section B.3. For smaller spatial scales, different exposure parameters would be needed.

A cumulative and simultaneous comparison of all four chemicals using an additive approach is presented on Map B.6-12. Cumulative risk estimates greater than 10^{-5} are seen in the vicinity of early action areas, such as Duwamish/Diagonal (RM 0.4 to RM 0.6 on the east side), Slip 4, Terminal 117 (RM 3.5 to RM 3.6 on the west side), and RM 3.3 to RM 3.8 on the east side. Other locations with cumulative risk estimates greater than 10^{-5} include RM 0.1 on the east side, RM 0.5 to RM 0.6 on the west side, Slip 1, RM 1.3 to RM 1.5 on the east side, and Slip 6 (Map B.6-10).

B.6.3.4 Arsenic risks attributed to clam consumption

The excess cancer risks for inorganic arsenic in the seafood consumption scenarios were high and similar to the risks for PCBs. As indicated in the risk characterization (Section B.5.3.1.3) and incremental risk discussions (Section B.5.5.1.2), the excess cancer risks for inorganic arsenic are attributable almost entirely to clams, with the concentrations of inorganic arsenic in remaining seafood categories being similar to concentrations at reference locations. The inorganic arsenic concentrations in clams were many times higher than those in other seafood categories. Consequently, the uncertainty associated with the arsenic risk estimates should be evaluated in the context of the associated sediment chemistry data.

Co-located arsenic sediment chemistry data were collected with the clams that were analyzed in 2004 for inorganic arsenic, as presented in Table B.6-44 (Windward 2005b). The sediment samples were analyzed for total (unspeciated) arsenic, while the clams were analyzed for inorganic arsenic, the form of greatest health concern. This matched dataset has been examined to evaluate a potential relationship between total arsenic

[50] It should be noted that although netfishing does not occur at a single point, because spatial-scale information is not available, this approach was used and assumed to be health-protective.

concentrations in sediment and inorganic arsenic concentrations in clams, although it is not known whether co-located sediment data are the most appropriate data for determining whether such a relationship exists. This issue is discussed at the end of this section.

Table B.6-44. Inorganic arsenic concentrations in clam samples and total arsenic concentrations in co-located sediment samples from the LDW

Location ID	Total Arsenic Concentration in Sediment (mg/kg dw)	Inorganic Arsenic Concentration in Clams (mg/kg ww)
C1	3.53	0.132
C2-1	5.79	0.648
C3-1	4.63	0.885
C4	49.0	3.27
C5	4.72	0.795
C6	5.52	1.85
C7-2	6.80	2.11
C9	3.94	0.233
All LDW	1.2-1,100[a]	0.132-3.27[b]

[a] All LDW intertidal sediment (n = 330, including 302 detected concentrations). For entire LDW, including subtidal, concentration range is the same.

[b] All LDW clam samples, n = 8 analyzed for inorganic arsenic (all detected concentrations)

dw – dry weight

ID – identification

LDW – Lower Duwamish Waterway

ww – wet weight

The eight inorganic arsenic concentrations in composite clam tissue samples from the LDW that were used for the excess cancer risk estimates range from 0.132 to 3.27 mg/kg ww. The highest inorganic arsenic concentrations in clams (3.27 mg/kg ww) and the highest total arsenic concentration in the co-located sediment samples (49.0 mg/kg dw) were both detected at location C4. The next highest inorganic arsenic concentrations in clams (1.85 and 2.11 mg/kg ww) were detected at locations with much lower total arsenic concentrations in sediment (5.52 and 6.80 mg/kg dw, respectively) than the maximum detected at C4. The relationship between arsenic concentrations in sediment and clams is discussed below and in more detail in the RI main document, Section 8.3.2.

There is a positive correlation between the inorganic arsenic concentrations in clams and the total arsenic concentrations in sediment ($R^2 = 0.76$, based on a linear regression using log-transformed arsenic concentrations in sediment; Figure B.6-1 represents this relationship using untransformed data). The sediment arsenic concentration at C4 is more than seven times higher than the next highest sediment total arsenic concentration at C7-2. Without more synoptic clam/sediment samples taken from

locations with sediment arsenic concentrations intermediate between the cluster of arsenic sediment concentrations of 4 to 8 mg/kg dw and the high arsenic concentration of 49 mg/kg dw, it is difficult to determine how appropriate it is to use data from location C4 to characterize the arsenic sediment/tissue relationship. It would be interesting to examine the relationship between inorganic arsenic in *Mya arenaria* tissue and co-located sediment total arsenic for areas outside the LDW to determine if the patterns found in these areas are similar to that found for the LDW and to determine if sediment total arsenic is predictive of tissue inorganic arsenic elsewhere. Unfortunately, the background datasets do not cover a broad enough range of sediment arsenic concentrations to permit such comparisons.

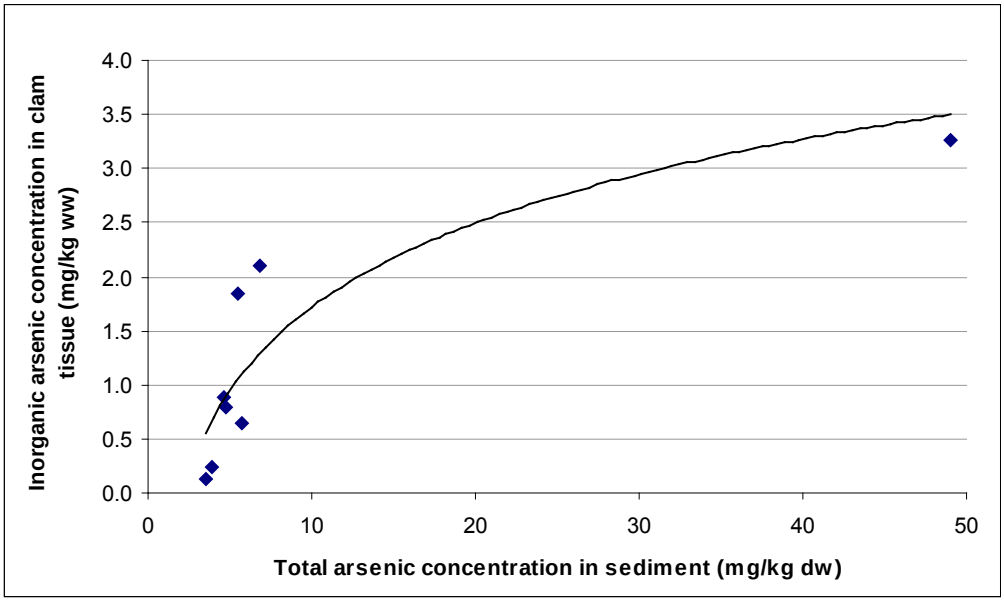


Figure B.6-1. Inorganic arsenic in LDW clams compared to total arsenic in co-located sediment

Although there is uncertainty associated with the arsenic data from location C4, this result was nonetheless included in the baseline HHRA. However, for comparison purposes, an alternative seafood consumption risk estimate was also generated that excluded the maximum inorganic arsenic concentration in clams from location C4. The inorganic arsenic EPC for clams that is used in the seafood consumption scenarios in the baseline HHRA is 1.96 mg/kg ww (n = 8). Excluding the maximum concentration at location C4, the EPC would be almost as high (1.50 mg/kg ww, n = 7). The excess cancer risk estimate that would result using this alternative clam EPC rather than the original clam EPC (with EPCs for all other seafood classes remaining unchanged) would be 1×10^{-3} for the adult tribal seafood consumption scenario based on Tulalip data, compared to the original risk estimate of 2×10^{-3}.

The alternative risk estimate presented above highlights a source of great uncertainty in the excess cancer risk estimate for inorganic arsenic in the seafood consumption

scenarios. Except for the maximum sediment total arsenic concentration detected at C4, the sediment total arsenic concentrations associated with the clam sampling locations are relatively low compared to the rest of the LDW and to the upstream area. For example, the UCL on the mean sediment total arsenic concentration for seven locations shown in Table B.6-44 (all but C4) is 5.81 mg/kg dw, which is lower than UCLs for the entire LDW intertidal area (27 mg/kg dw) and for the upstream area (6.80 mg/kg dw). An unacceptable risk estimate of 1×10^{-3} can be generated from LDW clam data collected from areas with synoptic sediment total arsenic concentrations lower than sediment total arsenic concentrations upstream of the LDW (i.e., in unaffected areas). This finding highlights the uncertainty associated with the possible source of the inorganic arsenic in LDW clams.

Because the clams evaluated in this HHRA are filter feeders, the areal extent of the sediment source is likely to be greater than the single co-located composite sediment sample that was evaluated. Alternative sediment exposure areas were evaluated in this uncertainty analysis. SWACs based on both the entire extent of the clam beach and an additional area that includes subtidal habitat, as shown on Map B.6-13, were calculated and regressed against the clam chemistry data shown in Table B.6-44. The regression statistics (not shown) were similar for both these approaches to the statistics calculated for the co-located samples. Similar regression analyses were conducted using sediment total arsenic concentrations normalized to percent fines, based on the premise that fine-grained particles would be more likely to be suspended in the water column and potentially consumed by clams. These regression statistics (not shown) were also similar to those for the co-located samples. Overall, these data explorations illustrate the uncertainty in the relationship between arsenic concentrations in clams compared to arsenic concentrations in various sediment exposure areas. At this time, there is no clear explanation of why these clams have such high inorganic arsenic concentrations.

B.7 Identification of Risk Drivers

This section presents the rationale for the identification of chemicals as "risk drivers" (EPA 1999c) based on estimated human health risks. As applied herein, the term "risk drivers" is synonymous with the term "indicator hazardous substances" under MTCA (WAC 173-340-703). The risk drivers from both this HHRA and the ERA will be the focus of remedial analyses in the FS.

Chemicals considered to be risk drivers are a subset of the COCs[51] (summarized in Table B.7-1 and B.7-3). The relative percentage of the total human health risk and the absolute magnitude of the risk posed by these COCs was considered in designating a

[51] Note that a chemical must be associated with greater than 1×10^{-6} and/or HQs greater than 1 for one or more RME scenarios to be designated as a COC. See Tables B.5-64 through B.5-66 for summary of risk estimates for all scenarios.

risk driver. Other factors that were also considered include the criteria identified in WAC 173-340-703 for designating an indicator hazardous substance: a) toxicological characteristics that influence its ability to adversely affect human health or the environment relative to the concentration of the hazardous substance at the site, b) tendency to persist in the environment, c) tendency to move into and through environmental media, d) natural background concentrations, e) thoroughness of testing, f) detection frequency, and g) chemicals that readily break down into less toxic byproducts.

COCs not selected as risk drivers in the HHRA will be further evaluated in subsequent steps in the cleanup process, in consultation with EPA and Ecology. This evaluation may include:

- Assessment of reductions in sediment concentrations or residual risks from these chemicals following the selection of the preferred alternative in the FS
- Review of any new toxicological effects data, as part of the 5-year review that is conducted once a CERCLA cleanup is completed
- Inclusion of these chemicals as part of the post-cleanup monitoring program

B.7.1 SEAFOOD CONSUMPTION SCENARIOS

Table B.7-1 summarizes the selection of risk drivers for the seafood consumption scenarios based primarily on risk magnitude, percent contribution of total risk, and detection frequency in LDW seafood tissue. More detailed discussion is provided below for each chemical or group of chemicals. Based on the analysis presented in this section, three chemicals (i.e., total PCBs, arsenic, and cPAHs) were identified as risk drivers based on the health-protective scenario that uses the adult and child tribal seafood consumption rates based on Tulalip data and a fractional intake of 1. In addition, dioxins/furans were assumed to be a risk driver, as described in Section B.7.1.1.

Table B.7-1. COCs and identification of risk drivers for seafood consumption scenarios

COC	RISK DRIVER?	RISK MAGNITUDE[a]	% CONTRIBUTION TO TOTAL EXCESS CANCER RISK[b]	DETECTION FREQUENCY IN LDW SEAFOOD TISSUE (%)
PCBs	yes	2×10^{-3}	58	97
Arsenic	yes	1×10^{-3}	29	100
cPAHs	yes	7×10^{-5}	2	72
Dioxins/furans	yes	nd[b]	nd[c]	nd[c]
SVOCs				
bis(2-ethylhexyl) phthalate	no	6×10^{-6}	<1	15
Pentachlorophenol	no	9×10^{-5}	3	6

COC	Risk Driver?	Risk Magnitude[a]	% Contribution to Total Excess Cancer Risk[b]	Detection Frequency in LDW Seafood Tissue (%)
Metals				
Tributyltin	no	3 (HQ) [d]	na	78
Vanadium	no	2 (HQ) [d]	na	68
Organochlorine pesticides				
Aldrin	no	5×10^{-5}	1	4
alpha-BHC	no	2×10^{-5}	1	5
beta-BHC	no	6×10^{-6}	<1	28
Carbazole	no	5×10^{-5}	1	1
Total chlordane	no	6×10^{-6}	<1	87
Total DDTs	no	2×10^{-5}	1	90
Dieldrin	no	1×10^{-4}	3	5
gamma-BHC	no	6×10^{-6}	<1	11
Heptachlor	no	1×10^{-5}	<1	2
Heptachlor epoxide	no	3×10^{-5}	1	41
Hexachlorobenzene	no	1×10^{-5}	<1	14

[a] Except where noted, the risk estimates shown are excess cancer risk estimates for the adult tribal RME seafood consumption scenario based on Tulalip data, which had the highest cancer risk estimates among the RME seafood consumption scenarios. See Tables B.5-64 and B.5-65 for complete risk estimates for all scenarios.

[b] Total excess cancer risk excluding PCB TEQ (total PCBs included in sum).

[c] It was assumed that dioxin/furan tissue concentrations were above acceptable risk-based concentrations. Consequently, no tissue samples were collected for dioxin/furan analysis.

[d] HQs are for the child tribal RME seafood consumption scenario based on Tulalip data because HQs were below 1 for the adult tribal RME scenario based on Tulalip data. BHC – benzene hexachloride

COC – chemical of concern

cPAH – carcinogenic polycyclic aromatic hydrocarbon

LDW – Lower Duwamish Waterway

nd – no data

na – not applicable

PCB – polychlorinated biphenyl

SVOC – semivolatile organic compound

B.7.1.1 PCBs, arsenic, cPAHs, and dioxins/furans

Risk estimates for both PCBs and arsenic were 1×10^{-3} or greater for the adult tribal seafood consumption scenario based on Tulalip data, which are above the acceptable cancer risk thresholds for CERCLA. Consequently, these chemicals were selected as risk drivers. Although the risk estimates for carcinogenic PAHs were considerably lower than the estimates for PCBs and arsenic, they were still 7×10^{-5} for the adult tribal RME scenario based on Tulalip data. This risk estimate is close to the upper end of EPA's acceptable risk range. Consequently, cPAHs were also selected as a risk driver.

There were no LDW tissue dioxin/furan chemistry data to estimate risks from seafood consumption. However, as discussed earlier, if such data did exist, risk estimates associated with the seafood consumption rates applied herein would likely be high enough to justify the selection of dioxins/furans as a risk driver. Therefore, at the direction of EPA and Ecology, dioxins/furans were also identified as a risk driver for the seafood consumption pathway.

B.7.1.2 Bis(2-ethylhexyl) phthalate

Bis(2-ethylhexyl) phthalate is an insignificant contributor (< 1%) to the total risk estimate (Table B.7-1) and it was rarely detected in LDWG tissue samples. RLs were elevated in the initial analysis because of sample dilution requirements. A subset of samples was reanalyzed, and lower RLs were achieved (see Section B.6.1.1.4 for additional details). The results of the reanalysis suggest that the results of the initial analysis were biased high and that use of the original elevated RLs for the non-detected chemicals greatly overestimated their concentrations in tissue samples.

Bis(2-ethylhexyl) phthalate was identified as a risk driver in the ERA because surface sediment concentrations exceeded the sediment quality standards (SQS) of the SMS in one or more locations. Approximately 80% of the locations with bis(2-ethylhexyl) phthalate concentrations above the SQS also had PCB concentrations above the SQS. Thus, remediation of PCBs in areas with these SQS exceedances will reduce bis(2-ethylhexyl) phthalate concentrations in sediment. Because of expected reductions in sediment concentrations following remediation of PCBs, and the fact that bis(2-ethylhexyl) phthalate comprises a very small percentage of the total risk, bis(2-ethylhexyl) phthalate was not selected as a risk driver in the HHRA.

B.7.1.3 Pentachlorophenol

Pentachlorophenol's contribution to the total risk estimate is minor (< 1%) (Table B.7-1) and it was rarely detected in LDW tissue samples. RLs were elevated in the initial analysis because of sample dilution requirements. A subset of samples was reanalyzed, and much lower RLs were achieved (see Section B.6.1.1.4 for additional details). In addition, although there were two detected results in the original analysis, the detected results were not confirmed in the reanalysis. The results of the reanalysis suggested that the results of the initial analysis were biased high and that the use of elevated RLs associated with pentachlorophenol would greatly overestimate its concentrations in tissue samples. The risk for pentachlorophenol using the original analysis was 9×10^{-5} for the adult tribal RME scenario based on Tulalip data, which is close to EPA's excess cancer risk threshold of 1×10^{-4}. However, if the pentachlorophenol concentrations from the re-analysis are applied, the pentachlorophenol risks decrease to 9×10^{-7} for the adult tribal RME scenario based on Tulalip data (see Table B.6-3). Based on considerations of fraction of risk contributed and the overall magnitude of risk, designation of pentachlorophenol as a risk driver is not warranted.

B.7.1.4 Other metals

TBT and vanadium slightly exceeded an HQ of 1 for only one RME seafood consumption scenario, the child tribal RME scenario based on Tulalip data. The HQs for these chemicals were 3 for TBT and 2 for vanadium (Table B.7-1). The child tribal RME scenario based on Tulalip data has considerable uncertainty as discussed in Section B.6.1.2.3. Because TBT and vanadium HQs for this scenario were only slightly higher than 1, TBT and vanadium were not designated as a risk drivers.

B.7.1.5 Organochlorine pesticides

Eleven organochlorine pesticides (i.e., DDTs, aldrin, alpha-BHC, beta-BHC, carbazole, total chlordane, dieldrin, gamma-BHC, heptachlor, heptachlor epoxide, and hexachlorobenzene) had risk estimates that exceeded risk thresholds. Analytical interference with the quantification of organochlorine pesticides from the presence of PCB congeners was first identified as an issue many years ago (Reynolds 1969). The similarities in the physical and chemical properties between organochlorine pesticides and PCB congeners result in similar chromatographic peaks that can interfere with the accurate quantification of the pesticides. The interference is most significant in cases where PCB concentrations are high relative to the pesticide concentrations. The most commonly used method for the analysis of organochlorine pesticides in tissue and sediment is EPA Method 8081A (gas chromatography with electron capture detection [GC/ECD]). Potential interference in the analysis of organochlorine pesticides because of the presence of PCBs is identified as an issue in the method documentation (EPA Method 8081A, 1996).

A detailed discussion of PCB interference with quantitation of organochlorine pesticides is given in Section B.6.1.1.3 and summarized here. Analytical interference occurred during the organochlorine pesticide analysis of benthic invertebrate tissue and co-located sediment samples (Windward 2005b) and of fish and crab tissue samples (Windward 2005c). This issue was identified by both the analytical laboratory and the data validators. Organochlorine pesticides were analyzed using EPA Method 8081A. The detected results for all organochlorine pesticides for benthic invertebrates, clams, and co-located sediment samples (Windward 2005b) and fish and crab tissue samples collected in 2004 (Windward 2005c) were qualified JN, which indicates "the presence of an analyte that has been 'tentatively identified,' and the associated numerical value represents its approximate concentration" (EPA 1999e). These data were qualified based on the probable interference in the analysis from PCB congeners.

The JN-qualified organochlorine pesticide results are highly uncertain and likely biased high. The high bias for DDTs was confirmed by reanalyzing eight fish and crab tissue samples that had high PCB and DDT concentrations using a GC/MS method that is not susceptible to analytical interference by PCBs for organochlorine pesticides. The GC/MS method is less sensitive than EPA Method 8081A, and, therefore, could not be used for the original analyses and could only be used for confirmation in the

high-concentration samples. The results of the reanalysis confirmed that the pesticide quantitations in the original analysis were influenced by the presence of PCB congener peaks. Specifically, all the organochlorine pesticide concentrations in the reanalyses were substantially lower than the original results.

In addition, most organochlorine pesticides had low detection frequencies in sediment and tissue from the LDW (Table B.7-2). Similarly low detection frequencies were observed throughout Puget Sound, as documented in multiple PSAMP monitoring events (Table B.7-2), suggesting most of these compounds are not generally of concern in this region. Detected organochlorine pesticide concentrations in sediment from the LDW, particularly those above 10 µg/kg dw, were often associated with elevated PCB concentrations. For example, approximately 70% of the locations with total DDT concentrations above 10 µg/kg dw also had PCB concentrations above the SQS. The analytical interference discussed above for tissue also occurs in sediment because of the co-occurrence of PCBs. Therefore, there are uncertainties in the pesticide results for sediments as well. Overall, organochlorine pesticides were not identified as risk drivers because of uncertainties with the data that likely inflate detected concentration estimates, low detection frequencies in sediment, and the low contribution to the overall risk (Table B.7-1).

Table B.7-2. Summary of LDW and Puget Sound organochlorine pesticide data

Chemical	Detection Frequency – LDW Sediment	Detection Frequency – LDW Tissue	Detection Frequency – Puget Sound Sediment[a]	Detection Frequency – Puget Sound Tissue[b]
Aldrin	2%	4%	0%	0%
alpha-BHC	2%	5%	0%	2%
beta-BHC	2%	28%	0%	0%
Carbazole	52%	1%	na	0%
Total chlordane	18%	87%	2%	7%
Total DDTs	40%	90%	24%	41%
Dieldrin	5%	5%	1%	1%
gamma-BHC	6%	11%	1%	0%
Heptachlor	4%	2%	0%	0%
Heptachlor epoxide	3%	41%	0%	0%
Hexachlorobenzene	6%	14%	14%	0%

[a] Based on 300 sediment samples collected during a 3-year reconnaissance survey of Puget Sound (NOAA and Ecology 1999, 2000, 2002). RLs for these samples were similar to those achieved for LDW sediment samples.

[b] Based on approximately 400 English sole and rockfish muscle samples from the PSAMP database (West et al. 2001). RLs for these samples were similar to those achieved for LDW tissue samples.

na – not analyzed

BHC – benzene hexachloride

LDW – Lower Duwamish Waterway

B.7.2 DIRECT SEDIMENT CONTACT SCENARIOS

Table B.7-3 provides a summary of chemicals with risk estimates above thresholds for the direct sediment exposure scenarios. Factors evaluated for the designation of risk drivers for these scenarios included the magnitude of the excess cancer risk estimates, percent contribution to total risk, and frequency of detection in LDW surface sediment samples. Based on this analysis, four chemicals (i.e., arsenic, cPAHs, PCBs, and dioxins/furans) were identified as risk drivers. The risk estimates for PCBs were lower than those for the other risk drivers. Only one of the eight beach play areas and neither the netfishing area nor any of the clamming areas had a risk estimate for PCBs greater than 1×10^{-6}. However, given the importance of PCBs in the seafood consumption scenario and the likely focus of remedial activities on this chemical, PCBs were selected as a risk driver.

Toxaphene contributed insignificantly to the overall risk estimate for the tribal clamming RME scenario and was detected in only two intertidal sediment samples used for the clamming risk estimate. Consequently, the designation of toxaphene as a risk driver was not warranted. Both of the locations with detected toxaphene concentrations also had concentrations of one or more other chemicals greater than the cleanup screening level of the SMS, thus reductions of toxaphene concentrations in sediment are expected based on the remediation of other chemicals.

Table B.7-3. COCs and identification of risk drivers for direct sediment exposure scenarios

COC	RISK DRIVER?	RISK MAGNITUDE[a]	CONTRIBUTION TO TOTAL RISK (%)	DETECTION FREQUENCY IN BASELINE SURFACE SEDIMENT SAMPLES (%)[b]
Arsenic	yes	2×10^{-5} (clamming) 6×10^{-6} (netfishing) 7×10^{-6} (beach play)	14 19 15	92
cPAHs	yes	5×10^{-6} (clamming) 1×10^{-6} (netfishing) 4×10^{-5} (beach play)	4 3 85	94
PCBs	yes	8×10^{-6} (clamming) 2×10^{-6} (netfishing) 6×10^{-6} (beach play)	6 6 21	93
Dioxins/furans (as TEQ)	yes	1×10^{-4} (clamming) 2×10^{-5} (netfishing) 1×10^{-5} (beach play)	72 65 35	100
Toxaphene	no	6×10^{-6} (clamming) 2×10^{-6} (netfishing) 2×10^{-7} (beach play)	4 6 1	1

[a] RME scenarios were used to designate COCs. Clamming is tribal clamming RME scenario (120 days/yr); netfishing is the RME scenario (119 days/yr); risks shown for beach play were the highest for any of the eight assumed beach play RME exposure areas. See Table B.5-65 for risk estimates for all scenarios.

[b] Detection frequency is for all LDW baseline surface sediment data, not just the data used for a particular scenario.

COC – chemical of concern

cPAH – carcinogenic polycyclic aromatic hydrocarbon
PCB – polychlorinated biphenyl
TEQ – toxic equivalent

B.8 Conclusions

This baseline HHRA characterizes risks to the public from site-related exposures in support of risk management decisions and evaluation of remedial options. In addition, the HHRA serves to inform the public of health risks resulting from exposures to site-related chemicals. Both seafood consumption and direct sediment exposure scenarios were evaluated. In addition, water exposures were evaluated previously by King County (1999d). Many different exposure scenarios were evaluated to provide a range of risk estimates. People can evaluate their own risks by comparing their behavior with the assumptions included in each of the exposure scenarios. A summary of the risk estimates and uncertainties associated with these estimates is provided in Section B.5.6 and Table B.6-1, respectively.

The large majority of the estimated risks for all media and all exposure scenarios are attributable to four chemicals called risk drivers (PCBs, arsenic, cPAHs, and dioxins/furans), as summarized in Section B.7. These risk driver chemicals together account for 88% of the excess cancer risk for the adult tribal RME seafood consumption scenario and 96% of the excess cancer risk for the tribal clamming scenario. Human health risks attributable to the many other chemicals found in the LDW are considerably lower, either because the concentrations of those chemicals are relatively low or the chemicals are not particularly toxic or both. For all chemicals other than the risk drivers under all the RME or CT exposure scenarios, risk estimates do not exceed EPA's cancer risk threshold of 10^{-4}. The total risks (all chemicals combined) from exposure to sediment and water (the highest RME estimate was 1×10^{-4} for the tribal clamming RME scenario) are much lower than the total risks associated with seafood consumption (the highest RME estimate was 3×10^{-3} for the adult tribal RME scenario based on Tulalip data, which assumed a consumption rate of 97.5 g/day or approximately 13 meals per month). Total risks from exposure to all chemicals in sediment and water are within or less than EPA's acceptable risk range of 10^{-4} and 10^{-6}, whereas risks from seafood consumption are higher. The risk estimates for each of the risk drivers are summarized briefly below.

8.1 PCBs

Excess cancer risks and non-cancer hazards for PCBs from the consumption of seafood from the LDW exceeded acceptable risk thresholds (excess cancer risks were greater than 1×10^{-6} and HQs were greater than 1 for non-cancer hazards) for all RME seafood consumption scenarios. For example, the highest excess cancer risk associated with total PCBs or PCB TEQ for an RME seafood consumption scenario of 3×10^{-3} was estimated for the adult tribal RME scenario based on Tulalip data. PCBs were

identified as a risk driver (referred to as an indicator hazardous substance under MTCA) for the seafood consumption pathway, based on the high risk estimates presented in this HHRA. The risks are highest for consumption of the pelagic seafood category (i.e., perch) and clams (depending on the seafood consumption scenario evaluated). There is considerable uncertainty about the applicability of some of the seafood consumption rates used in this HHRA, particularly for clams, given the quality and quantity of shellfish habitat in the LDW.

Although risks from sediment contact were much lower than risks associated with seafood consumption, PCBs were also identified as a risk driver for sediment because risks exceeded the excess cancer risk threshold of 1×10^{-6} for at least one direct sediment exposure scenario and because of the importance of PCBs in seafood risk estimates. For example, the highest excess cancer risk associated with PCBs for an RME scenario was 3×10^{-5} for PCB TEQ for the tribal clamming RME.

8.2 ARSENIC

Excess cancer risks and non-cancer hazards for inorganic arsenic from the consumption of seafood from the LDW exceeded acceptable risk thresholds for all RME seafood consumption scenarios (e.g., the highest excess cancer risk associated with inorganic arsenic for an RME seafood consumption scenario of 1×10^{-3} was estimated for the adult tribal RME scenario based on Tulalip data). This chemical was identified as a risk driver for the seafood consumption pathway based on the high risk estimates presented in this HHRA. The source of the elevated inorganic arsenic concentrations found in LDW clams is uncertain. All but one of the co-located sediment samples collected with the clam samples in the LDW had arsenic concentrations that were clustered in a narrow range similar to or lower than both the average total arsenic concentration in LDW sediments and natural background arsenic, thereby making it difficult to discern the relationship between sediment and tissue arsenic.

Arsenic was also identified as a risk driver for the direct sediment exposure scenarios. Although much lower than risks associated with inorganic arsenic for seafood consumption (the highest excess cancer risk associated with inorganic arsenic for an RME scenario was 2×10^{-5} for PCB TEQ for the tribal clamming RME), excess cancer risk estimates were higher than 1×10^{-6} for most of the direct sediment exposure scenarios. Some of the arsenic in the LDW is likely attributable to background sources that are unrelated to the site.

8.3 CARCINOGENIC PAHS

Excess cancer risk estimates for cPAHs in the seafood consumption scenarios are much lower than estimated risks for PCBs and arsenic, but still exceed the risk threshold of 1×10^{-6}. The highest excess cancer risk estimate (7×10^{-5}) was for the adult tribal RME scenario based on Tulalip data. Most of the risk can be attributed to the consumption

of clams. Based on the risk magnitude, cPAHs were selected as a risk driver for seafood consumption.

Carcinogenic PAHs were identified as a risk driver for sediment based on the fact that excess cancer risk estimates were higher than 1×10^{-6} for most of the direct sediment contact exposure scenarios and cPAHs contributed a large portion of the total excess cancer risk for these scenarios. Risks for cPAHs for the RME direct contact pathways ranged from 1×10^{-6} to 4×10^{-5}.

8.4 DIOXINS/FURANS

The dioxin/furan concentrations in most LDW sediments that have been sampled are within the range of dioxin/furan concentrations in sediments from urban background areas. However, concentrations of dioxins/furans in surface sediments at a few locations within the LDW are more than 100 times higher than in sampled background sediment locations in the greater Seattle area. Excess cancer risk estimates were higher than 1×10^{-6} for most of the direct sediment contact exposure scenarios (the highest excess cancer risk associated with dioxin/furan TEQ for an RME scenario was 1×10^{-4} for direct contact with sediments in the tribal clamming RME). Because dioxin/furans made up a large portion of the cancer risk from direct sediment contact, they were identified as a risk driver.

Dioxin/furans were also identified as a risk driver for the seafood consumption pathway even though LDW tissue samples were not analyzed for dioxins/furans. It was assumed that unacceptable risks would be found based on the high toxicity of dioxin/furans, high dioxin/furan sediment concentration in limited areas of the LDW, bioaccumulation of dioxins/furans into seafood from sediments, and exposure to these chemicals via seafood consumption.

B.9 References

American Cancer Society. 2006. Cancer facts and figures 2006. American Cancer Society, Atlanta, GA.

Anderson ME. 1994. Systematics and osteology of the Zoarcidae (Teleostei: Perciformes). Ichthyol Bull JLB Smith Inst Ichthyol 60:1-120.

Armstrong RH. 1996. Alaska's fish. A guide to selected species. Alaska Northwest Books, Portland, OR.

ATSDR. 1995. Toxicological profile for vanadium and compounds. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1997. Public health assessment, Old Navy Dump/Manchester Laboratory (USEPA/NOAA), Manchester, Kitsap County, Washington. CERCLIS No. WA8680030931. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2000. Toxicological profile for polychlorinated biphenyls (PCBs). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2004. Toxicological profile for copper. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2005a. Rayonier Mill site exposure investigation. Evaluation of dioxins in crab and geoduck tissue from a Lower Elwha Klallam tribe fishing area near Port Angeles, Washington. Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services, Atlanta, GA.

ATSDR. 2005b. Toxicological profile for arsenic (update). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2005c. Toxicological profile for hexachlorocyclohexane (lindane). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2006. Draft toxicological profile for cresols. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

Battelle. 1996. Final report for the PCB Aroclor and congener analyses on fish tissue samples from the Elliott Bay/Duwamish River project. Pacific Northwest Division, Battelle Marine Research Laboratory, Sequim, WA.

Burmaster DE, Thompson KM. 1997. Estimating exposure point concentrations for surface soils for use in deterministic and probabilistic risk assessments. Hum Ecolog Risk Assess 3(3):363-384.

California EPA. 1994. Health effects of benzo(a)pyrene. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Berkeley, CA.

California EPA. 2005. Human-exposure-based screening numbers developed to aid estimation of cleanup costs for contaminated soil. November 2004; January 2005 revision. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Berkeley, CA.

Clemens WA, Wilby GV. 1961. Fishes of the Pacific coast of Canada. 2nd edition. Publication no. 68. Fisheries Resources Board of Canada.

Cogliano VJ. 1998. Assessing the cancer risk from environmental PCBs. Environ Health Perspect 106:317-323.

Cooper JA, Chapleau F. 1998. Monophyly and intrarelationships of the family Pleuronectidae (Pleuronectiformes), with a revised classification. Fish Bull 96(4):686-726.

Cordell JR, Tear LM, Jensen K. 2001. Biological monitoring at Duwamish River coastal America restoration and reference sites: A seven-year retrospective. SAFS-UW-0108. Wetlands Ecosystem Team, School of Aquatic and Fisheries Sciences, University of Washington, Seattle, WA.

Cox G, Francis M. 1997. Sharks and rays of New Zealand. Canterbury University Press, Christchurch, New Zealand.

CRITFC. 1994. A fish consumption survey of the Umatilla, Nez Perce, Yakama, and Warm Springs tribes of the Columbia River basin. Technical report 94-3. Columbia River Inter-Tribal Fish Commission, Portland, OR.

Dawson CE. 1985. Indo-Pacific pipefishes (Red Sea to the Americas). The Gulf Coast Research Laboratory, Ocean Springs, MS.

Demetropoulos CL, Braithwaite LF, Maurer BA, Whiting D. 1990. Foraging and dietary strategies of two sublittoral cottids, *Jordania zonope* and *Artedius harringtoni*. J Fish Biol 37(1):19-32.

Duff RM, Kissel JC. 1996. Effect of soil loading on dermal absorption efficiency from contaminated soils. J Toxicol Env Health 48(11):93-106.

Ecology. 1998. Washington State dioxin source assessment. No. 98-320. Washington State Department of Ecology, Olympia, WA.

Ecology. 2001a. Model Toxics Control Act Cleanup Regulation, Chapter 173-340 WAC. Publication No. 94-06. Toxics Cleanup Program, Washington State Department of Ecology, Olympia, WA.

Ecology. 2001b. Tacoma smelter plume. South King County mainland soil study [online]. Washington Department of Ecology. [Cited May 27, 2004]. Available from: www.ecy.wa.gov/programs/tcp/sites/tacoma_smelter/soil_study.htm.

Ecology. 2003. Norfolk combined sewer overflow (Duwamish River) sediment cap recontamination. Phase I investigation. Publ. no. 03-03-004. Environmental Assessment Program, Washington Department of Ecology, Olympia, WA.

Ecology. 2007. Letter from B. Helland, Toxics Cleanup Program, dated July 10, 2007 to M. Johns, Windward Environmental, regarding Ecology review commments on Lower Duwamish Waterway MTCA Site draft baseline human health risk assessment. Washington Department of Ecology, Northwest Regional Office, Bellevue, WA.

EPA. 1988. Guidance for conducting remedial investigations and feasibility studies under CERCLA. Interim final. EPA/540/G-89/004, OSWER Directive 9355.3. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1989. Risk assessment guidance for Superfund, volume 1: Human health evaluation manual, Part A. EPA/540/1-89/002. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1991a. Risk assessment guidance for Superfund, human health evaluation manual, supplemental guidance: standard default exposure factors. Directive No. 9285.6-03. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1991b. Role of the baseline risk assessment in Superfund remedy selection decisions. OSWER Directive 9355.0-30. April 21, 1991. Office of Solid Waste and Emergency Response, US Environmental Protection Agency, Washington, DC.

EPA. 1992a. Dermal exposure assessment: principles and applications. Interim report. EPA-600/8-91/011-B. Office of Health and Environmental Assessment, Exposure Assessment Group, US Environmental Protection Agency, Washington, DC.

EPA. 1992b. Guidance for data useability in risk assessments (Part A). PB92 - 963356. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1993. Provisional guidance for quantitative risk assessment of polycyclic aromatic hydrocarbons. EPA-600/R-93/089. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, US Environmental Protection Agency, Cincinnati, OH.

EPA. 1994. Guidance manual for the Integrated Exposure Uptake Biokinetic Model for lead in children. EPA 540-R-93-081. Technical Review Workgroup for Lead, US Environmental Protection Agency, Washington, DC.

EPA. 1996a. EPA Region 10 supplemental risk assessment guidance for Superfund. Draft. Office of Environmental Assessment, Risk Evaluation Unit, US Environmental Protection Agency, Region 10, Seattle, WA.

EPA. 1996b. PCBs: cancer dose-response assessment and application to environmental mixtures. EPA/600/P-96/001F. Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC.

EPA. 1997. Exposure factors handbook. EPA/600/P-95/002Fa. Office of Research and Development, National Center for Environmental Assessment, US Environmental Protection Agency, Washington, DC.

EPA. 1998. Risk assessment guidance for Superfund. Volume 1. Human health evaluation manual. Part D, Standardized planning, reporting, and review of Superfund risk assessments. Interim Publication No. 9825.7-01D. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1999a. Asian and Pacific Islander seafood consumption study in King County, Washington. Exposure information obtained through a community-centered approach. Study results and education outreach. EPA 910/R-99-003. Office of Environmental Assessment, Risk Evaluation Unit, US Environmental Protection Agency Region 10, Seattle, WA.

EPA. 1999b. EPA Superfund record of decision: Pacific Sound Resources (PSR) site upland and marine sediments OUs, Seattle, WA. PB99-96465. EPA541-R99-083. US Environmental Protection Agency, Washington, DC.

EPA. 1999c. A guide to preparing Superfund proposed plans, records of decision, and other remedy selection decision documents. EPA 540-R-98-031; OSWER 9200.1-23P; PB98-963241. Office of Solid Waste and Emergency Response, US Environmental Protection Agency, Washington, DC.

EPA. 1999d. Risk assessment guidance for Superfund. Volume 1. Human health evaluation manual. Supplemental guidance dermal risk assessment, interim guidance. EPA 540/R-99/005. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1999e. USEPA contract laboratory program national functional guidelines for organic data review. EPA-540/R-99/008. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 2000a. Bioaccumulation testing and interpretation for the purpose of sediment quality assessment: status and needs. EPA-823-R-00-001. Bioaccumulation Analysis Workgroup, US Environmental Protection Agency, Washington, DC.

EPA. 2000b. Exposure and human health reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 1: Estimating exposure to dioxin-like compounds. EPA/600/P-00/001Bc. Draft final report. National Center for Environmental Assessment, US Environmental Protection Agency, Washington, DC.

EPA. 2000c. Exposure and human health reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 1: estimating exposure to dioxin-like compounds. Vol 3: Properties, environmental levels, and background exposures. EPA/600/P-00/001Bc. Draft final report. National Center for Environmental Assessment, US Environmental Protection Agency, Washington, DC.

EPA. 2000d. Guidance for assessing chemical contaminant data for use in fish advisories. Volume 2: Risk assessment and fish consumption limits. Third ed. EPA 823-B-00-008. US Environmental Protection Agency, Washington, DC.

EPA. 2000e. Supplementary guidance for conducting health risk assessment of chemical mixtures. EPA/630/R-00/002. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.

EPA. 2001a. Dioxin reassessment -- an SAB review of the Office of Research and Development's reassessment of dioxin. Review of the revised sections (dose response modeling, integrated summary, risk characterization, and toxicity equivalency factors) of the EPA's reassessment of dioxin by the dioxin reassessment review subcommittee of the EPA Science Advisory Board (SAB). EPA-SAB-EC-01-006. US Environmental Protection Agency, Washington, DC.

EPA. 2001b. Risk assessment guidance for Superfund (RAGS), volume 1: Human health evaluation manual (Part E, supplemental guidance for dermal risk assessment) interim. Review draft for public comment, September 2001. EPA/540/R/99/005. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 2002a. Blood lead concentrations of US adult females: Summary statistics from Phases 1 and 2 of the National Health and Nutrition Evaluation Survey (NHANES III). Technical Review Workgroup for Lead, US Environmental Protection Agency, Washington, DC.

EPA. 2002b. Columbia River basin fish contaminant survey 1996-1998. US Environmental Protection Agency Region 10, Seattle, WA.

EPA. 2002c. Estimated per capita fish consumption in the United States. EPA-821-C-02-003. US Environmental Protection Agency, Washington, DC.

EPA. 2002d. Guidance for comparing background and chemical concentrations in soil for CERCLA sites. EPA 540-R-01-003. OSWER 9285.7-41. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 2002e. Role of background in the CERCLA cleanup program. OSWER 9285.6-07P. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, DC.

EPA. 2002f. Users guide for the integrated exposure uptake biokinetic model for lead in children (IEUBK). EPA-540-K-01-005. OSWER #9285.7-42. Office of Solid Waste and Emergency Response, US Environmental Protection Agency, Washington, DC.

EPA. 2003a. Analyses of laboratory and field studies of reproductive toxicity in birds exposed to dioxin-like compounds for use in ecological risk assessment. EPA/600/R-03/114F. National Center for Environmental Assessment, US Environmental Protection Agency, Cincinnati, OH.

EPA. 2003b. Human health toxicity values in Superfund risk assessments. OSWER Directive 9285.7-53. Office of Solid Waste and Emergency Response, US Environmental Protection Agency, Washington, DC.

EPA. 2003c. Recommendations of the technical review workgroup for lead for an approach to assessing risks associated with adult exposures to lead in soil. EPA-540-R-03-001. Technical Review Workgroup for Lead, US Environmental Protection Agency, Washington, DC.

EPA. 2004a. EPA Region IX preliminary remediation goals: 2004 PRG table [online]. US Environmental Protection Agency Region IX, San Francisco, CA. Updated October 2004. Available from: http://www.epa.gov/region09/waste/sfund/prg/index.html.

EPA. 2004b. ProUCL Version 3.0 user guide. Technical Support Center for Monitoring and Site Characterization, US Environmental Protection Agency.

EPA. 2004c. ProUCL Version 3.0. Statistical software to compute Upper Confidence Limits of the unknown population mean [online]. Technical Support Center for Monitoring and Site Characterization, US Environmental Protection Agency. Updated 5/6/2004. Available from: http://www.epa.gov/nerlesd1/tsc/download.htm.

EPA. 2004d. Risk assessment guidance for Superfund, Volume 1: Human health evaluation manual (Part E, supplemental guidance for dermal risk assessment). Final, July 2004. EPA/540/R/99/005. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 2005a. Application of the draft framework for selecting and using tribal fish and shellfish consumption rates for risk-based decision making at CERCLA and RCRA cleanup sites in Puget Sound and the Strait of Georgia to the Lower Duwamish Waterway sediment cleanup action - June 16, 2005. US Environmental Protection Agency, Region 10, Seattle, WA.

EPA. 2005b. EPA Region III risk-based concentration table [online]. US Environmental Protection Agency Region III, Philadelphia, PA. Updated October 2005. Available from: http://www.epa.gov/reg3hwmd/risk/human/index.htm.

EPA. 2005c. Guidelines for carcinogen risk assessment. EPA/630/P-03/001F. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.

EPA. 2005d. Rhône-Poulenc (Rhodia) sediment & porewater investigation Aug/Sept 2004. Data report: volatile organic compounds, metals, and pesticides/PCBs detected in sediment and porewater. Draft. US Environmental Protection Agency Region 10, Seattle, WA.

EPA. 2005e. Supplemental guidance for assessing susceptibility from early-life exposure to carcinogens. EPA/630/R/03/003F. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.

EPA. 2006a. Child-specific exposure factors handbook (external review draft). EPA/600/R/06/096A. US Environmental Protection Agency, Washington, DC.

EPA. 2006b. Draft framework for selecting and using tribal fish and shellfish consumption rates for risk-based decision making at CERCLA and RCRA cleanup sites in Puget Sound and the Strait of Georgia. Revised draft, May 23, 2006. U.S. Environmental Protection Agency, Region 10, Seattle, WA.

EPA. 2006c. EPA comments on Lower Duwamish Waterway baseline human health risk assessment, dated August 4, 2006 (draft), and accompanying cover letter to D.M. Johns, Windward Environmental, from A. Hiltner dated September 27, 2006. US Environmental Protection Agency Region 10, Seattle, WA.

EPA. 2006d. EPA responses to LDWG's HHRA outstanding issues - 11/22/06. US Environmental Protection Agency Region 10, Seattle, WA.

EPA. 2006e. ProUCL Version 4.0 user guide. National Exposure Research Laboratory, US Environmental Protection Agency, Las Vegas, NV.

EPA, Ecology. 2007. EPA and Ecology comments on revised draft Lower Duwamish Waterway baseline human health risk assessment, Lower Duwamish Waterway Superfund Site, Seattle, Washington, dated April 13, 2007, and accompanying cover letter to D.M. Johns, Windward Environmental, from A. Hiltner dated June 15, 2007 US Environmental Protection Agency Region 10, Seattle, WA.

EPA. 2007a. EPA response to December 15, 2006 memoranda regarding resolution of outstanding issues related to Lower Duwamish Waterway HHRA exposure

scenarios. Memorandum to Tad Deshler, Windward Environmental, from Allison Hiltner and Lon Kissinger, dated 1/3/07. US Environmental Protection Agency Region 10, Seattle, WA.

EPA. 2007b. Framework for selecting and using tribal fish and shellfish consumption rates for risk-based decision making at CERCLA and RCRA cleanup sites in Puget Sound and the Strait of Georgia. U.S. Environmental Protection Agency, Region 10, Seattle, WA.

EPA. 2007c. Frequent questions from risk assessors on the Adult Lead Methodology (ALM) [online]. US Environmental Protection Agency, Washington, DC. Updated 8/2/07. Available from: http://www.epa.gov/superfund/lead/almfaq.htm#fish%20ingestion.

EPA, Ecology. 2000. Administrative order on consent for remedial investigation/feasibility study, Lower Duwamish Waterway; Port of Seattle, King County, City of Seattle, The Boeing Company, respondents. US EPA docket no. CERCLA-10-2001-0055; Ecology docket no. 00TCPNR-1895. US Environmental Protection Agency, Region 10, Seattle, WA; State of Washington Department of Ecology, Olympia, WA.

Era-Miller B. 2005. Quality assurance project plan. South Puget Sound fish and shellfish tissue verification of 303(d) listings. Publication number 05-03-116. Environmental Assessment Program, Washington Department of Ecology, Olympia, WA.

Era-Miller B. 2006. Personal communication (unpublished dioxin/furan mussel data e-mailed to Tad Deshler, Windward Environmental, LLC, Seattle, WA, from Cascade Pole sediment site, Olympia, WA). Project Manager, Environmental Assessment Program, Washington Department of Ecology, Olympia, WA. April 25, 2006.

Eschmeyer WN, Herald ES, Hammann H. 1983. Pacific coast fishes. Peterson Field Guide Series. Houghton Mifflin, Boston, MA.

ESG. 1999. Waterway sediment operable unit, Harbor Island Superfund site. Assessing human health risks from the consumption of seafood: human health risk assessment report. Prepared for Port of Seattle, Todd Shipyards, and Lockheed-Martin for submittal to US Environmental Protection Agency, Region 10, Seattle, WA. Environmental Solutions Group, Inc., Seattle, WA.

Exponent. 1998. Duwamish Waterway phase I site characterization report. Prepared for The Boeing Company. Exponent, Bellevue, WA.

Fein GG, Jacobson JL, Jacobson SW. 1984. Prenatal exposure to polychlorinated biphenyls: effects on birth size and gestational age. J Pediatr 105:315-320.

Fitch JE, Lavenberg RJ. 1975. Tidepool and nearshore fishes of California. California Natural History Guides no. 38. University of California Press, Berkeley and Los Angeles, CA.

Florida Museum of Natural History. 2005. Biological profiles: longnose skate. Ichthyology at the Florida Museum of Natural History: Education-Biological Profiles [online]. Florida Museum of Natural History, University of Florida, Gainesville, FL. [Cited 26 August 2005]. Available from: www.flmnh.ufl.edu/fish/Gallery/Descript/LongnoseSkate/LongnoseSkate.html.

Floyd Snider McCarthy. 2004. Boeing Plant 2, Seattle/Tukwila, Washington: Phase 1 transformer PCB investigation report. Floyd Snider McCarthy, Inc., Seattle, WA.

Frame M. 2001. Personal communication (telephone conversation with Marcia Henning, Washington Department of Health, regarding general observations about behavior of human anglers in Lower Duwamish Waterway). Washington Department of Fish and Wildlife, Enforcement Officer, Seattle, WA. October 3.

Frontier Geosciences. 1996. Mercury results in 18 fish samples for the Elliott Bay/Duwamish River project. Frontier Geosciences, Seattle, WA.

Gall GAE, Crandell PA. 1992. The rainbow trout. Aquaculture 100:1-10.

Gilbert CR, Williams JD. 2002. National Audobon Society field guide to fishes. Alfred A. Knopf, Inc., New York, NY.

Groot C, Margolis L, eds. 1998. Pacific salmon life histories. UBC Press, Vancouver, BC.

Grossman GD. 1979. Demographic characteristics of an intertidal bay goby (*Lepidogobius lepidus*). Environ Biol Fish 4(3):207-218.

Hart Crowser. 1993. Results of sampling and analysis, sediment monitoring plan, Duwamish Shipyard, Inc., Seattle, Washington. Hart Crowser, Inc., Seattle, WA.

Hart JL. 1973. Pacific fishes of Canada. Fisheries Research Board of Canada, Ottawa, ON.

Herrera. 1997. Seaboard Lumber Site. Phase 2 site investigation. Draft. Prepared for Seattle Department of Parks and Recreation. Herrera Environmental Consultants, Inc., Seattle, WA.

Hiltner A. 2007. Personal communication (e-mail to Tad Deshler and Nancy Judd, Windward Environmental, regarding Beach Plan and CT scenarios). US Environmental Protection Agency Region 10, Seattle, WA. January 18, 2007.

Hobson ES. 2000. Interannual variation in predation on first-year *Sebastes* spp. by three northern California predators. Fish Bull 99(2):292-302.

Institute of Medicine. 2003. Dioxins and dioxin-like compounds in the food supply: strategies to decrease exposure. Institute of Medicine of the National Academies, The National Academies Press, Washington, DC.

Integral. 2004. Lower Duwamish Waterway slip 4 early action area: cruise and data report. Prepared for City of Seattle and King County. Integral Consulting, Inc., Mercer Island, WA.

Jacobson JL, Jacobson SW. 1996. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N Engl J Med 335(11):783-789.

Jacobson JL, Jacobson SW. 1997. Evidence for PCBs as neurodevelopmental toxicants in humans. Neurotoxicol 8(2):415-424.

Kerwin J, Nelson TS, eds. 2000. Habitat limiting factors and reconnaissance assessment report, Green/Duwamish and Central Puget Sound watersheds (WRIA 9 and Vashon Island). Washington Conservation Commission, Lacey, WA, and King County Department of Natural Resources, Seattle, WA.

King County. 1996. Norfolk CSO sediment cleanup study. Prepared for Elliott Bay/Duwamish Restoration Program. King County Department of Natural Resources, Seattle, WA.

King County. 1999a. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Vol 1, Appendix B1: Hydrodynamic and fate and transport numerical model. King County Department of Natural Resources, Seattle, WA.

King County. 1999b. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Vol 1, Appendix B2, B3, & B4: human health, wildlife, and aquatic life risk assessments. King County Department of Natural Resources, Seattle, WA.

King County. 1999c. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Vol 1, Appendix C: Issue papers. King County Department of Natural Resources, Seattle, WA.

King County. 1999d. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Vol 1: Overview and interpretation, plus appendices. King County Department of Natural Resources, Seattle, WA.

King County. 1999e. Norfolk CSO sediment remediation project five-year monitoring program: April 1999 monitoring report. Prepared for Elliott Bay/Duwamish Restoration Program. King County Department of Natural Resources, Seattle, WA.

King County. 2000a. Duwamish/Diagonal CSO/SD site assessment report. Draft. Prepared for Elliott Bay/Duwamish Restoration Program. King County Department of Natural Resources, Seattle, WA.

King County. 2000b. King County environmental laboratory quality assurance review for estuarine sediment analytical data. Norfolk CSO sediment remediation project five-year monitoring program. February 2000 sampling event. Environmental Laboratory, King County Department of Natural Resources, Seattle, WA.

King County. 2000c. Norfolk CSO sediment remediation project five-year monitoring program. Annual monitoring report-year one, April 2000. Prepared for Elliott Bay/Duwamish Restoration Program. King County Department of Natural Resources, Seattle, WA.

King County. 2000d. Norfolk CSO sediment remediation project five-year monitoring program. Six-month post-construction monitoring report, October 1999. Prepared for Elliott Bay/Duwamish Restoration Program. King County Department of Natural Resources, Seattle, WA.

King County. 2001a. Duwamish/Diagonal CSO/SD cleanup study report. Draft. Prepared for Elliott Bay/Duwamish Restoration Program panel. King County Department of Natural Resources, Seattle, WA.

King County. 2001b. Norfolk CSO sediment remediation project five-year monitoring program. Annual monitoring report-year two, April 2001. Prepared for Elliott Bay/Duwamish Restoration Program. King County Department of Natural Resources, Seattle, WA.

King County. 2002. Norfolk CSO sediment remediation project five-year monitoring program: Annual monitoring report - year 3, April 2002. Prepared for the Elliott Bay/Duwamish Restoration Panel. King County Department of Natural Resources and Parks, Seattle, WA.

King County. 2005. Norfolk CSO sediment remediation project five-year monitoring program: Final monitoring report - year 5, April 2004. Prepared for the Elliott Bay/Duwamish Restoration Panel. King County Department of Natural Resources and Parks, Seattle, WA.

King County, Anchor, EcoChem. 2005. Duwamish/Diagonal CSO/SD sediment remediation project closure report. Prepared for the Elliott Bay Duwamish Restoration Program Panel. King County Department of Natural Resources, Anchor Environmental LLC, and EcoChem, Inc., Seattle, WA.

Kissel JC, Richter KY, Fenske RA. 1996. Field measurement of dermal soil loading attributable to various activities: implications for exposure assessment. Risk Anal 16:115-125.

Kissinger L. 2002. Personal communication (email to Tad Deshler, Windward Environmental, Seattle, WA, regarding parameterization of Adult Lead Model). US Environmental Protection Agency, Region 10, Seattle, WA. October 17, 2002.

Kissinger L. 2005. Application of data from an Asian and Pacific Islander (API) seafood consumption study to derive fish and shellfish consumption rates for risk assessment. Office of Environmental Assessment, US Environmental Protection Agency Region 10, Seattle, WA.

Kissinger L. 2006a. Personal communication (e-mail to Nancy Judd, Windward Environmental, regarding development of Asian/Pacific Islander seafood consumption rates). Toxicologist, Office of Environmental Assessment, US Environmental Protection Agency Region 10, Seattle, WA. March 7, 2006.

Kissinger L. 2006b. Personal communication (e-mail to Nancy Judd, Windward Environmental, regarding mean harvested crab, mussel, and bivalve consumption rates). Toxicologist, Office of Environmental Assessment, US Environmental Protection Agency Region 10, Seattle, WA. July 7, 2006.

Kissinger L. 2007a. Personal communication (e-mail and attached Excel file to Nancy Judd, Windward Environmental, regarding API fraction of seafood parts consumed). Toxicologist, Office of Environmental Assessment, US Environmental Protection Agency Region 10, Seattle, WA. July 20, 2007.

Kissinger L. 2007b. Personal communication (e-mail and attached Word file to Nancy Judd, Windward Environmental, regarding draft memo, 10/31/06: Derivation of a 95th percentile consumer only consumption rate for Tulalip Tribes' children). Toxicologist, Office of Environmental Assessment, US Environmental Protection Agency Region 10, Seattle, WA. August 31, 2007.

Kissinger L. 2007c. Personal communication (e-mail and attached Word file to Nancy Judd, Windward Environmental, regarding memo, 8-31-07: LDW HHRA clamming exposure duration and frequency). Toxicologist, Office of Environmental Assessment, US Environmental Protection Agency Region 10, Seattle, WA. September 4, 2007.

Knerr S, Schrenk D. 2006. Carcinogenicity of "non-dioxinlike" polychlorinated biphenyls. Crit Rev Toxicol 36(9):663-694.

Krahn MM, Buzitis J, Ylitalo GM, Sloan CA, Brown D, Boyd D, Bolton J, Stein JE. 1998. Comparison of PCB concentrations in split samples of sediments from the Duwamish River, Seattle, WA. National Marine Fisheries Service and National Oceanic and Atmospheric Administration, Seattle, WA.

Lake JL, McKinney R, Lake CA, Osterman FA, Heltshe J. 1995. Comparisons of patterns of polychlorinated biphenyl congeners in water, sediment, and indigenous organisms from New Bedford Harbor, Massachusetts. Arch Environ Contam Toxicol 29:207-220.

Lamb A, Edgell P. 1986. Coastal fishes of the Pacific Northwest. Harbour Publishing Co. Ltd., Madeira Park, BC.

Landolt ML, Hafer FR, Nevissi A, Van Belle G, Van Ness K, Rockwell C. 1985. Potential toxicant exposure among consumers of recreationally caught fish from urban embayments of Puget Sound. NOAA technical memorandum NOS OMA 23. National Oceanic and Atmospheric Administration, Rockville, MD.

Landolt ML, Hafer FR, Nevissi A, Van Belle G, Van Ness K, Rockwell C. 1987. Potential toxicant exposure among consumers of recreationally caught fish from urban embayments of Puget Sound: final report. NOAA technical memorandum NOS OMA 33. National Oceanic and Atmospheric Administration, Rockville, MD.

Longnecker MP, Wolff MS, Gladen BC, Brock JWG, P, Jacobson JL, Korrick SA, Rogan WJ, Weisglas-Kuperus N, Hertz-Picciotto I, Ayotte P, Stewart P, Winneke G, Charles MJ, Jacobson SW, Dewailly E, Boersma ER, Altshul LM, Heinzow B, Pagano JJ, Jensen AA. 2003. Comparison of polychlorinated biphenyl levels across studies of human neurodevelopment. Environ Health Persp 111(1):65-70.

Malins DC, McCain BB, Brown DW, Sparks AK, Hodgins HO. 1980. Chemical contaminants and biological abnormalities in central and southern Puget Sound. NOAA Technical Memorandum OMPA-2. Environmental Conservation Division, National Marine Fisheries Service, Seattle, WA.

Mason RP, Lawrence AL. 1999. Concentration, distribution, and bioavailability of mercury and methylmercury in sediments of Baltimore Harbor and Chesapeake Bay, Maryland, USA. Environ Toxicol Chem 18(11):2438-2447.

Matsuda RI, Isaac GW, Dalseg RD. 1968. Fishes of the Green-Duwamish River. Water Quality Series No. 4. Municipality of Metropolitan Seattle, Seattle, WA.

Mayes BA, McConnell EE, Heal BH, Brunner MJ, Hamilton SB, Sullivan TM, Peters AC, Ryan MJ, Toft JD, Singer AW, Brown JF, Jr, Menton RG, Moore JA. 1998. Comparative carcinogenicity in Sprague-Dawley rats of the polychlorinated biphenyl mixtures Aroclors 1016, 1242, 1254, and 1260. Tox Sci 41:62-76.

McCallum M. 1985. Seafood catch and consumption in urban bays of Puget Sound. Division of Health, Washington Department of Social and Health Services, Olympia, WA.

McEachran JD, Dunn CA. 1998. Phylogenetic analysis of skates, a morphologically conservative clade of elasmobranchs (Chondrichthyes: Rajidae). Copeia 1998(2):271-290.

Mickelson S, Williston D. 2006. Technical memorandum: Duwamish River/Elliott Bay/Green River water column PCB congener survey: transmittal of data and quality assurance documentation. King County Department of Natural Resources, Seattle, WA.

Miller BS, Wingert RC, Borton SF. 1975. Ecological survey of demersal fishes in the Duwamish River and at West Point 1974. Prepared for Municipality of Metropolitan Seattle. Report no. FRI-UW-7509. Fisheries Research Institute, University of Washington, Seattle, WA.

Miller BS, McCain BB, Wingert RC, Borton SF, Pierce KV, Griggs DT. 1977a. Ecological and disease studies of demersal fishes in Puget Sound near METRO-operated sewage treatment plants and in the Duwamish River. FRI-UW-7721. Fisheries Research Institute, University of Washington, Seattle, WA.

Miller BS, Simenstad CA, Moulton LL, Fresh KL, Funk FC, Karp WA, Borton SF. 1977b. Puget Sound baseline program nearshore fish survey. Final report, July 1974-June 1977. Prepared for Washington Department of Ecology. Fisheries Research Institute, University of Washington, Seattle, WA.

Missildine BR, Peters RJ, Chin-Leo G, Houck DR. 2005. Polychlorinated biphenyl concentrations in adult chinook salmon (*Oncorhynchus tshawytscha*) returning to coastal and puget sound hatcheries of Washington State. Environ Sci Tech 39:6944-6951.

Morgan JN, Berry MR, Graves RL. 1997. Effects of commonly used cooking practices on total mercury concentration in fish and their impact on exposure assessments. J Exp Anal Environ Epidem 7:119-34.

Morrow JE. 1980. The freshwater fishes of Alaska. Animal Resources Ecology Library, University of British Columbia, Vancouver, BC.

National Research Council. 1994. Science and judgment in risk assessment. National Academy Press, Washington, DC.

NCI. 2006. National Cancer Institute Surveillance Epidemiology and End Results (SEER) Cancer Stat Fact Sheets [online]. National Cancer Institute, Bethesda, MD. [Cited June 28, 2006]. Available from: http://seer.cancer.gov/statfacts/html/all.html?statfacts_page=all.html&x=12&y=17.

NMFS. 2002. Unpublished data on PCB concentrations in juvenile chinook salmon captured in the Lower Duwamish Waterway during 1993 and 2000. Environmental Conservation Division, National Marine Fisheries Service, Seattle, WA.

NOAA. 1997. Duwamish River sediment study: sampling and analysis plan, quality assurance plan. Environmental Conservation Division, Northwest Fisheries Science Center, National Oceanic and Atmospheric Administration, Seattle, WA.

NOAA. 1998. Duwamish Waterway sediment characterization study report. Damage Assessment Center, National Oceanic and Atmospheric Administration, Seattle, WA.

NOAA. 2006. National Ocean Service water level tidal predictions, 2004-2006 [online]. National Oceanic and Atmospheric Administration, Washington, DC. [Cited March 2006]. Available from: http://tidesandcurrents.noaa.gov/tide_pred.html.

NOAA, Ecology. 1999. Sediment quality in Puget Sound. Year 1- northern Puget Sound. Ecology publication no. 99-347. National Oceanic and Atmospheric Administration, Silver Spring, MD and Washington Department of Ecology, Olympia, WA.

NOAA, Ecology. 2000. Sediment quality in Puget Sound. Year 2 - central Puget Sound. Ecology publication no. 00-03-055. National Oceanic and Atmospheric Administration, Silver Spring, MD and Washington Department of Ecology, Olympia, WA.

NOAA, Ecology. 2002. Sediment quality in Puget Sound. Year 3- southern Puget Sound. Ecology publication no. 02-03-03. National Oceanic and Atmospheric Administration, Silver Spring, MD and Washington Department of Ecology, Olympia, WA.

NRC. 2000. Toxicological effects of methylmercury. National Research Council, National Academy Press, Washington, DC.

O'Neill SM, West JE, Hoeman JC. 1998. Spatial trends in the concentration of polychlorinated biphenyls (PCBs) in chinook (*Oncorhynchus tshawytscha*) and coho salmon (*O. kisutch*) in Puget Sound and factors affecting PCB accumulation: Results from the Puget Sound Ambient Monitoring Program. Puget Sound Research '98:312-328.

Pacific Groundwater Group, TeraStat. 2005. Tacoma Smelter plume project: extended footprint study. Prepared for Washington State Department of Ecology. Pacific Groundwater Group and TeraStat Inc, Seattle, WA.

Page LM, Burr BM. 1991. A field guide to freshwater fishes of North America north of Mexico. Houghton Mifflin Company, Boston, MA.

Parametrix. 2003. Results of a human use survey for shoreline areas of Lake Union, Lake Washington, and Lake Sammamish. Sammamish-Washington Analysis and Modeling Program (SWAMP). Prepared for King County Department of Natural Resources. Parametrix, Kirkland, WA.

Pearcy WG, Hancock D. 1978. Feeding habits of Dover sole, *Microstomus pacificus*; rex sole, *Glyptocephalus zachirus*; slender sole, *Lyopsetta exilis*; and Pacific sanddab,

Citharichthys sordidus, in a region of diverse sediments and bathymetry off Oregon. Fish Bull 76(3):641-651.

PSAT. 2007. 2007 Puget Sound Update: ninth report of the Puget Sound Assessment and Monitoring Program, February 2007. Puget Sound Action Team, Office of the Governor, Olympia, WA.

PSEP. 1988. Health risk assessment of chemical contamination in Puget Sound seafood. Prepared for the Puget Sound Estuary Program, U.S. Environmental Protection Agency, Region 10, Office of Puget Sound, Seattle, WA. TC-3338-28. Tetra Tech, Inc., Bellevue, WA.

Reynolds LM. 1969. Polychlorobiphenyls (PCB's) and their interference with pesticide residue analysis. Bull Environ Contam Toxicol 4:128-143.

Rhône-Poulenc. 1995. RCRA facility investigation (RFI) report for the Marginal Way facility. Vol 1: RFI results and conclusions. Prepared for US Environmental Protection Agency, Region 10. Rhône-Poulenc, Tukwila, WA.

Rhône-Poulenc. 1996. RCRA facility investigation (RFI) report for the Marginal Way facility. Round 3 data and sewer sediment technical memorandum. Prepared for US Environmental Protection Agency, Region 10. Rhône-Poulenc, Tukwila, WA.

Rieman BE, McIntyre JD. 1993. Demographic and habitat requirements for conservation of bull trout. USFS Publication IN-GTR-302. US Fish and Wildlife Service, Washington, DC.

Rushneck DR, Beliveau A, Fowler B, Hamilton C, Hoover D, Kaye K, Berg M, Smith T, Telliard WA, Roman H, Ruder E, Ryan L. 2004. Concentrations of dioxin-like PCB congeners in unweathered Aroclors by HRGC/HRMS using EPA method 1668A. Chemosphere 54:79-87.

Schecter A, Li L. 1997. Dioxins, dibenzofurans, dioxin-like PCBs, and DDE in U.S. fast food 1995. Chemosphere 34:1449-57.

Schecter A, Cramer P, Boggess K, Stanley J, Olson JR. 1997. Levels of dioxins, dibenzofurans, PCB and DDE congeners in pooled food samples collected in 1995 at supermarkets across the United States. Chemosphere 34:1437-1447.

Scott WB, Crossman EJ. 1973. Freshwater fishes of Canada. Bulletin 184. Fisheries Research Board of Canada, Ottawa, ON, Canada.

SEA, Fishman, Ellis, Windward, Anchor, Kennedy/Jenks. 2003. Portland Harbor remedial investigation/feasibility study round 1 field sampling report. Prepared for Lower Willamette Group. Striplin Environmental Associates, Inc., Olympia, WA; Fishman Environmental Services, Portland, OR; Ellis Environmental Services, Estacada, OR; Windward Environmental LLC, Seattle,

WA; Anchor Environmental, Seattle, WA; Kennedy/Jenks Consultants, Portland, OR.

Shannon J. 2006. Personal communication (e-mails to Matt Luxon, Windward Environmental, regarding Duwamish-Elliott beach seine data collected by Taylor Associates in 1998, 2000, and 2002-2003. Biologist, Taylor Associates, Seattle, WA. March 30, 2006.

Shoaf MB, Shirai JH, Kedan G, Schaum J, Kissel JC. 2005a. Adult dermal sediment loads following clam digging in tide flats. Soil Sed Contam 14:463-470.

Shoaf MB, Shirai JH, Kedan G, Schaum J, Kissel JC. 2005b. Child dermal sediment loads following play in a tide flat. J Expos Anal Environ Epidemiol 15:407-412.

Skea J, Simonin H, Harris E, Jackling S, Spagnoli J, Symula J, Colquhoun J. 1979. Reducing levels of mirex, Aroclor 1254, and DDE by trimming and cooking Lake Ontario brown trout (*Salmo trutta* Linnaeus) and smallmouth bass (*Micropterus dolomieui* lacepede). Internat Assoc Great Lakes Res 5:153-159.

Suquamish Tribe. 2000. Fish consumption survey of the Suquamish Indian Tribe of the Port Madison Indian Reservation, Puget Sound region. The Suquamish Tribe, Suquamish, WA.

TAMS, Gradient. 2000. Phase 2 report: further site characterization and analysis. Volume 2F - revised human health risk assessment, Hudson River PCBs reassessment RI/FS. Prepared for US Environmental Protection Agency Region 2 and U.S. Army Corps of Engineers, Kansas City District. TAMS Consultants, Inc., New York, NY, and Gradient Corporation, Cambridge, MA.

Tanner CD. 1991. Potential intertidal habitat restoration sites in the Duwamish River Estuary. EPA 910/9-91-050. US Environmental Protection Agency, Region 10, Seattle, WA.

Taylor WJ, Shreffler DK, Cordell JR. 1999. Duwamish East Waterway channel deepening project: alternative dredge disposal sites juvenile salmonid and epibenthic prey assessment. Technical report. Preliminary draft. Prepared for Port of Seattle. Taylor Associates, Seattle, WA.

Toy KA, Polissar NL, Liao S, Mittelstaedt GD. 1996. A fish consumption survey of the Tulalip and Squaxin Island tribes of Puget Sound region. Department of Environment, Tulalip Tribes, Marysville, WA.

USFWS. 2000. LDW intertidal metadata (Intertidal-hab.shp), electronic file dated April 18, 2000. Follows FGDC content standards for digital geospatial metadata standard. US Fish and Wildlife Service, Lacey, WA.

Van den Berg M, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, Fiedler H, Hakansson H, Hanberg A, Haws L, Rose M, Safe S, Schrenk D, Tohyama C, Tritscher A, Tuomisto J, Tysklind M, Walker N, Peterson RE. 2006. The 2005

World Health Organization reevaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Tox Sci 93(2):223-241.

van der Plas SA, Sundberg H, van den Bert H, Scheu G, Wester P, Jensen S, Berbman A, de Boer J, Koeman JH, Brouwer A. 2000. Contribution of planar (0-1 *ortho*) and nonplanar (2-4 *ortho*) fractions of Aroclor 1260 to the induction of altered hepatic foci in female Sprague-Dawley rats. Toxicol Appl Pharmacol 169:255-268.

Varanasi U, Casillas E, Arkoosh MR, Hom T, Misitano D, Brown DW, Chan S-L, Collier TK, McCain BB, Stein JE. 1993. Contaminant exposure and associated biochemical effects in outmigrant juvenile chinook salmon (*Onchorynchus tschawytscha*) from urban and nonurban estuaries of Puget Sound, Washington. NOAA technical memorandum NMFS-FWFSC-8. Northwest Fisheries Science Center, National Marine Fisheries Service, Seattle, WA.

Warner EJ, Fritz RL. 1995. The distribution and growth of Green River chinook salmon (*Oncorhynchus tshawytscha*) and chum salmon (*Oncorhynchus keta*) outmigrants in the Duwamish estuary as a function of water quality and substrate. Water Resources Division, Muckleshoot Indian Tribe, Auburn, WA.

Weitkamp DE, Campbell RF. 1980. Port of Seattle Terminal 107 fisheries study. Parametrix, Inc., Bellevue, WA.

West JE, O'Neill SM, Lippert G, Quinnell S. 2001. Toxic contaminants in marine and anadromous fishes from Puget Sound, Washington. Results of the Puget Sound ambient monitoring program fish component 1989-1999. Washington Department of Fish and Wildlife, Olympia, WA.

Weston. 1993. Harbor Island remedial investigation report (part 2-sediment). Vol 1-report. Prepared for US Environmental Protection Agency, Region 10. Roy F. Weston, Inc., Seattle, WA.

Weston. 1998a. Comprehensive RCRA facility investigation report, Boeing Plant 2, Seattle/Tukwila, Washington. Roy F. Weston, Inc., Seattle, WA.

Weston. 1998b. Human health and environmental evaluation, RCRA facility investigation, Duwamish Waterway sediment, Boeing Plant 2, Seattle/Tukwila, Washington. Roy F. Weston, Inc., Seattle, WA.

Weston. 1999. Site inspection report, Lower Duwamish River (RK 2.5-11.5), Seattle, Washington. Vol 1-Report and appendices. Prepared for US Environmental Protection Agency, Region 10. Roy F. Weston, Inc., Seattle, WA.

Weston. 2000. Outfall and nearshore sediment sampling report, Duwamish facility. Prepared for James Hardie Gypsum, Seattle, Washington. Roy F. Weston, Inc., Seattle, WA.

Weston Solutions. 2005. Human health risk assessment, GE/Housatonic River Site - Rest of river. Volume IV, Appendix C, Consumption of fish and waterfowl risk assessment. Submitted to US Army Corps of Engineers, New England District, and US Environmental Protection Agency, New England Region, Boston, MA. Weston Solutions, Inc., Seattle, WA.

Whitehead PJP. 1985. Clupeoid fishes of the world (suborder Clupeioidei). An annotated and illustrated catalogue of the herrings, sardines, pilchards, sprats, shads, anchovies and wolf-herrings. Part 1 - Chirocentridae, Clupeidae and Pristigasteridae. FAO species catalogue. FAO Fish Synopses 125, vol 7. Food and Agriculture Organization of the United Nations, Rome, Italy.

Windward. 2001. Lower Duwamish Waterway remedial investigation. Task 2: Site characterization - Technical memoranda: 1. Criteria for evaluating and accepting data sets, 2. List of reports for historical site characterization, 3. Conceptual design for database. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2003a. Lower Duwamish Waterway remedial investigation. Phase 1 remedial investigation report. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2003b. Lower Duwamish Waterway remedial investigation. Phase 1 remedial investigation report. Appendix B: Human health risk assessment. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2004a. Lower Duwamish Waterway remedial investigation. Intertidal clam survey data report. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2004b. Lower Duwamish Waterway remedial investigation. Juvenile chinook salmon data report. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2004c. Lower Duwamish Waterway remedial investigation. Quality assurance project plan: Fish and crab tissue collection and chemical analyses. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2004d. Lower Duwamish Waterway remedial investigation. Task 8: Phase 2 RI work plan. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005a. Lower Duwamish Waterway remedial investigation. Benthic data report addendum: PCB congener data and SIM analyses. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005b. Lower Duwamish Waterway remedial investigation. Data report: Chemical analyses of benthic invertebrate and clam tissue samples and co-located sediment samples. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005c. Lower Duwamish Waterway remedial investigation. Data report: Fish and crab tissue collection and chemical analyses. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005d. Lower Duwamish Waterway remedial investigation. Data report: Round 1 surface sediment sampling for chemical analyses and toxicity testing. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005e. Lower Duwamish Waterway remedial investigation. Data report: Round 2 surface sediment sampling for chemical analyses and toxicity testing. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005f. Lower Duwamish Waterway remedial investigation. Fish and crab data report addendum: PCB congener data, MS/MSD analyses, and DDT confirmation. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005g. Lower Duwamish Waterway remedial investigation. Quality assurance project plan: Benthic sampling of Lower Duwamish Waterway. Addendum: Additional clam sampling in background area. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005h. Lower Duwamish Waterway remedial investigation. Quality assurance project plan: surface sediment sampling and toxicity testing of the Lower Duwamish Waterway. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005i. Lower Duwamish Waterway remedial investigation. Technical memorandum: Results from survey of potential human access locations on the Lower Duwamish Waterway. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005j. Technical Memorandum: Summary of sediment and tissue chemistry datasets to be used in the Phase 2 RI/FS. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2005k. Technical Memorandum: Summary of sediment and tissue chemistry datasets to be used in the Phase 2 RI/FS: Addendum 1. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2006a. Lower Duwamish Waterway remedial investigation. Data report: Arsenic concentrations in clam tissue samples and co-located sediment samples collected from background areas in 2005. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2006b. Lower Duwamish Waterway remedial investigation. Data report: chemical analyses of fish and crab tissue samples collected in 2005. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2006c. Lower Duwamish Waterway remedial investigation. Fish and crab tissue data report addendum: bis(2-ethylhexyl)phthalate and pentachlorophenol re-analyses. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2006d. Lower Duwamish Waterway remedial investigation. Technical memorandum: Criteria for defining the baseline surface sediment dataset for use in the Lower Duwamish Waterway Phase 2 RI/FS. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2007a. Lower Duwamish Waterway remedial investigation. 2005 fish and crab data report addendum: PCB congener data. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward. 2007b. Lower Duwamish Waterway remedial investigation. Technical memorandum: Summary of chemistry datasets to be used in the Phase 2 RI/FS—Addendum 2. Draft final. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.

Windward, DOF, Onsite. 2004a. Lower Duwamish Waterway Superfund site, Terminal 117 early action area. Sediment field sampling, cruise and data report. Prepared for the Port of Seattle. Windward Environmental LLC, Dalton, Olmsted & Fuglevand, Inc., and Onsite Enterprises, Inc., Seattle, WA.

Windward, DOF, Onsite. 2004b. Lower Duwamish Waterway Superfund site, Terminal 117 early action area. T-117 sediment and soil supplemental data report. Prepared for the Port of Seattle. Windward Environmental LLC, Dalton, Olmsted & Fuglevand, Inc., and Onsite Enterprises, Inc., Seattle, WA.

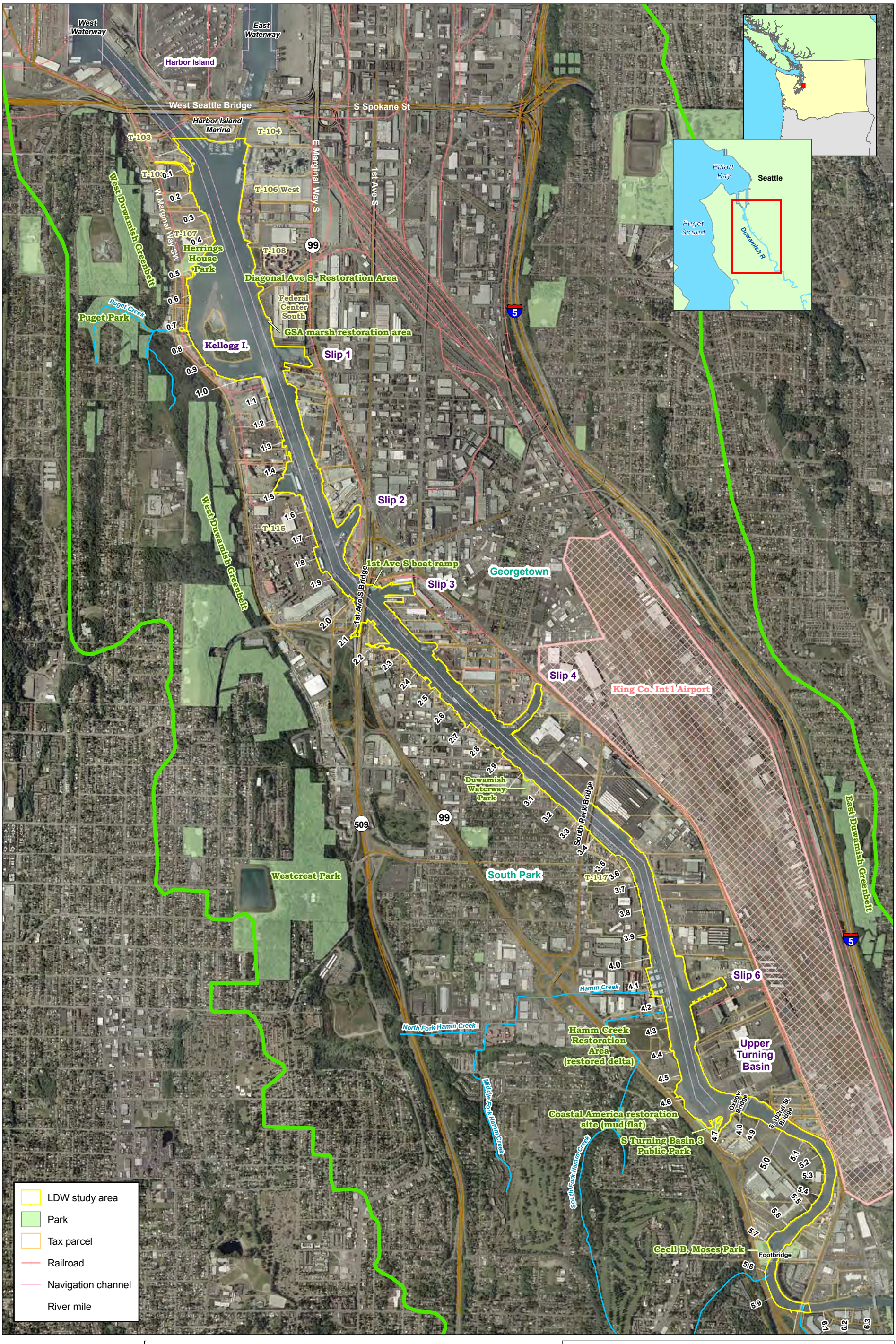
WR Consulting. 2004. Final sediment characterization report, Boyer Towing, Inc. dock replacement, Seattle, Washington. WR Consulting, Inc., Seattle, WA.

WSDOH. 2005. Health consultation - Lower Duwamish Waterway site: updated fish consumption advisory and evaluation of marine tissue collected from the Lower Duwamish Waterway in August and September 2004, Seattle, Washington. DOH Pub. #333-103. Office of Environmental Health Assessments, Washington State Department of Health, Olympia, WA.

Zabik ME, Hoojjat P, Weaver CM. 1979. Polychlorinated biphenyls, dieldrin, and DDT in lake trout cooked by broiling, roasting, or microwave. Bull Environ Contam Toxicol 21:136-143.

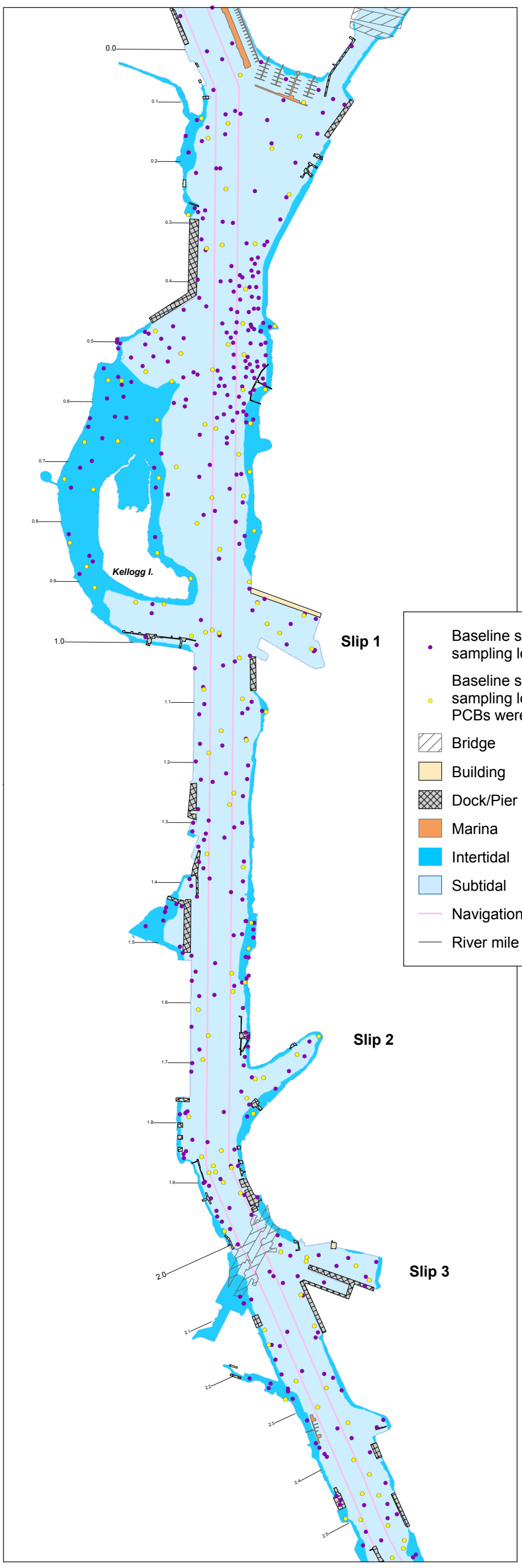
Zabik ME, Merrill C, Zabik M. 1982. PCBs and other xenobiotics in raw and cooked carp. Bull Environ Contam Toxicol 28:710-715.

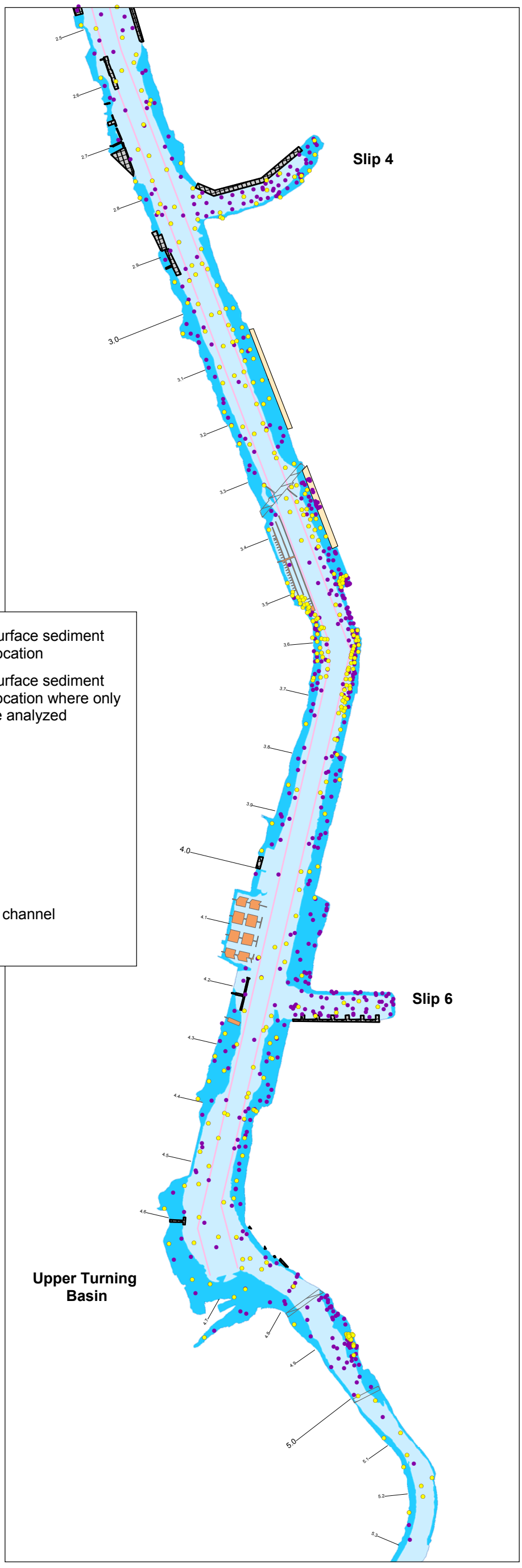
GIS MAPS



Map B.1-1. Lower Duwamish Waterway study area

Prepared by CEH 11/12/07; Map 2242; \data\gis\HHRA





- Baseline surface sediment sampling location
- Baseline surface sediment sampling location where only PCBs were analyzed
- Bridge
- Building
- Dock/Pier
- Marina
- Intertidal
- Subtidal
- Navigation channel
- River mile

Wind Ward environmental LLC

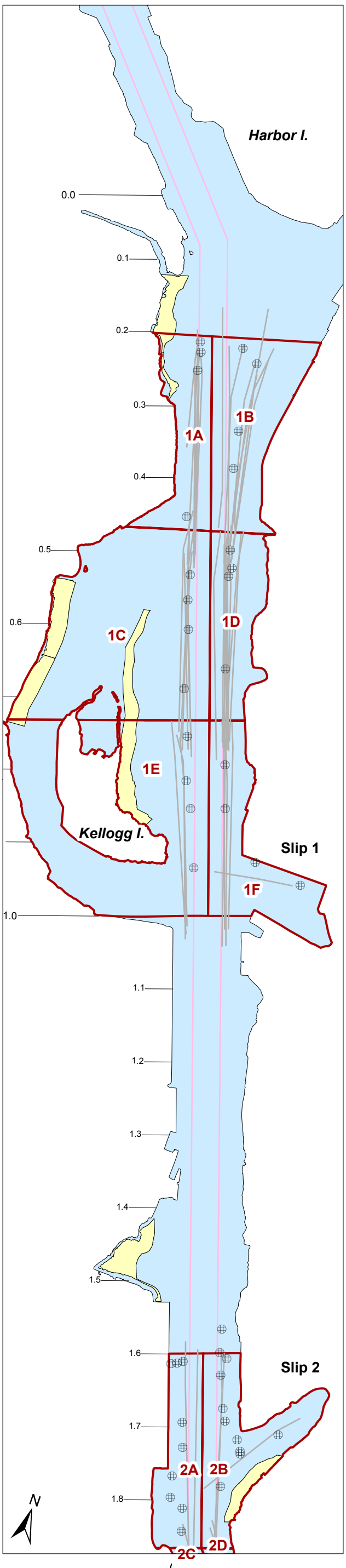
0 0.1 0.2 Miles

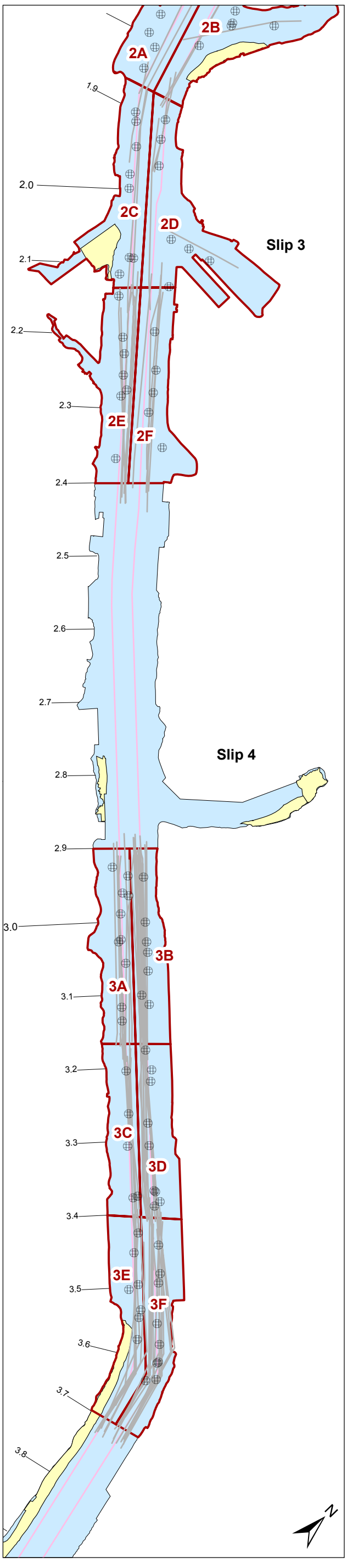
0 0.1 0.2 Kilometers

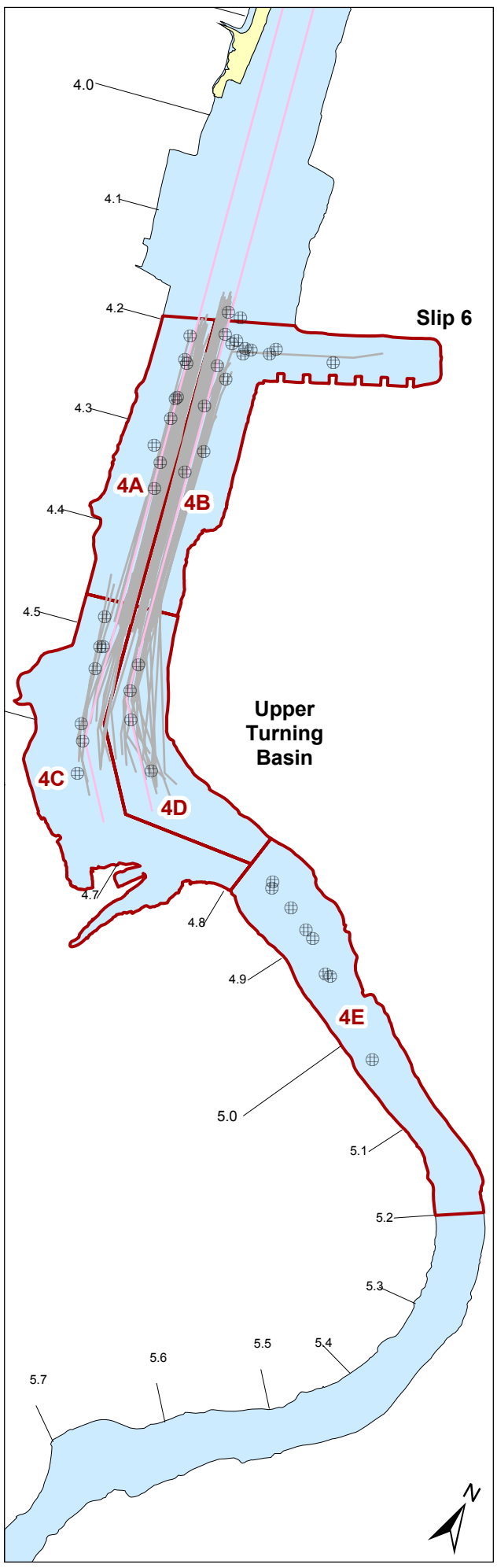
Scale is the same for each inset map

Map B.2-1. LDW surface sediment sampling locations (subtidal and intertidal)

Prepared by CEH 11/12/07; Map 2308; \data\gis\HHRA







Crab trap location (2004 - 2005)
Trawl line (2004 - 2005)
Clam sampling area (2004)
Tissue sampling area
Navigation channel
River mile

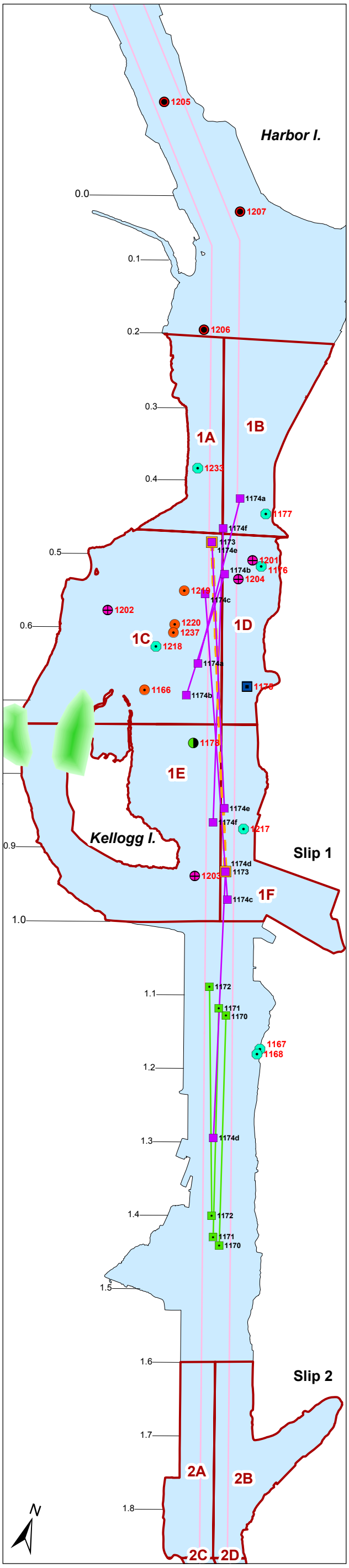
Wind Ward LLC environmental

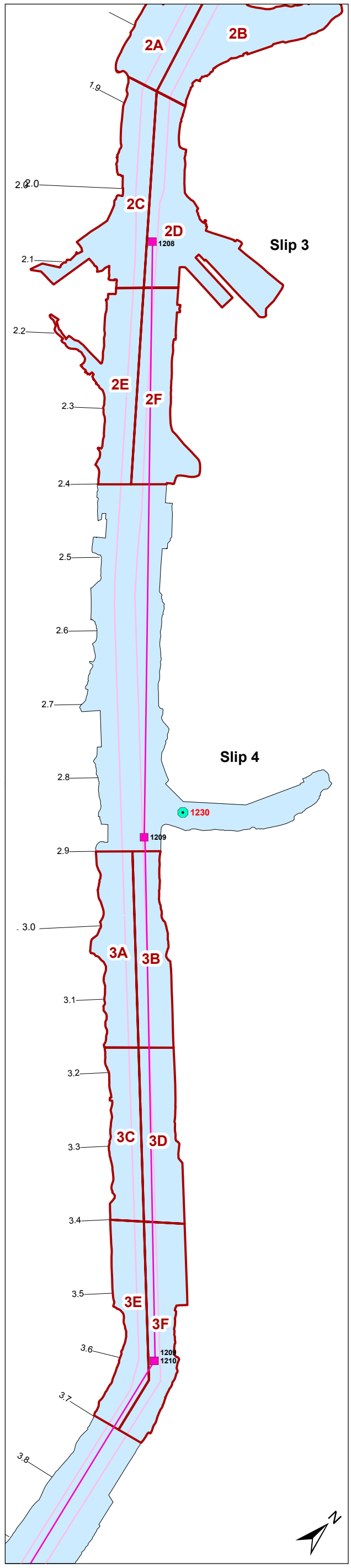
0 0.1 0.2 Miles
0 0.1 0.2 Kilometers

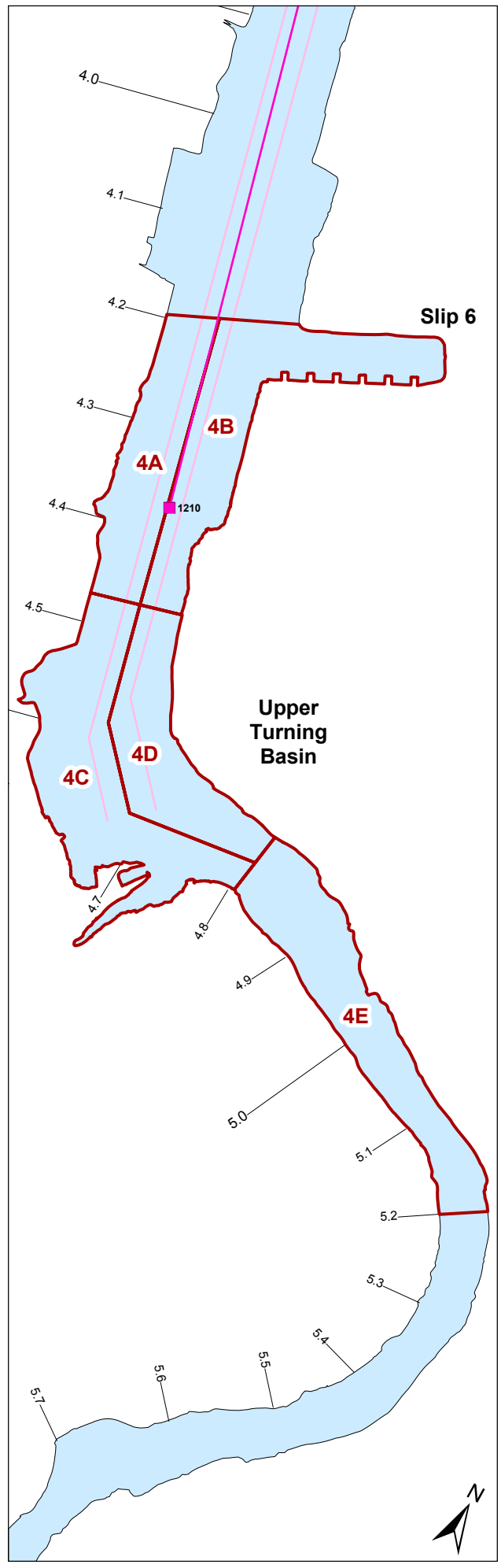
Scale is the same for each inset map

Map B.2-2a. LDW tissue sampling areas and subareas and individual locations where tissue samples were collected by LDWG in 2004-2005

Prepared by CEH, 11/12/07 Map 2152 \data\gis\HHRA







Tissue Transects
- EVS95-sole
- KCWQA-multiple species
- PSAMP-sole
- WSOU-sole

Transect start/end
- EVS95-sole
- KCWQA-multiple species
- PSAMP-sole
- WSOU-sole

Point Samples
- KCWQA-amphipods
- Benthic community samples
- KCWQA-mussels
- NOAA-salmon
- PSAMP-salmon
- WSOU-crab
- WSOU-perch
- Tissue sampling area
- Navigation channel
- River mile

Wind Ward environmental LLC

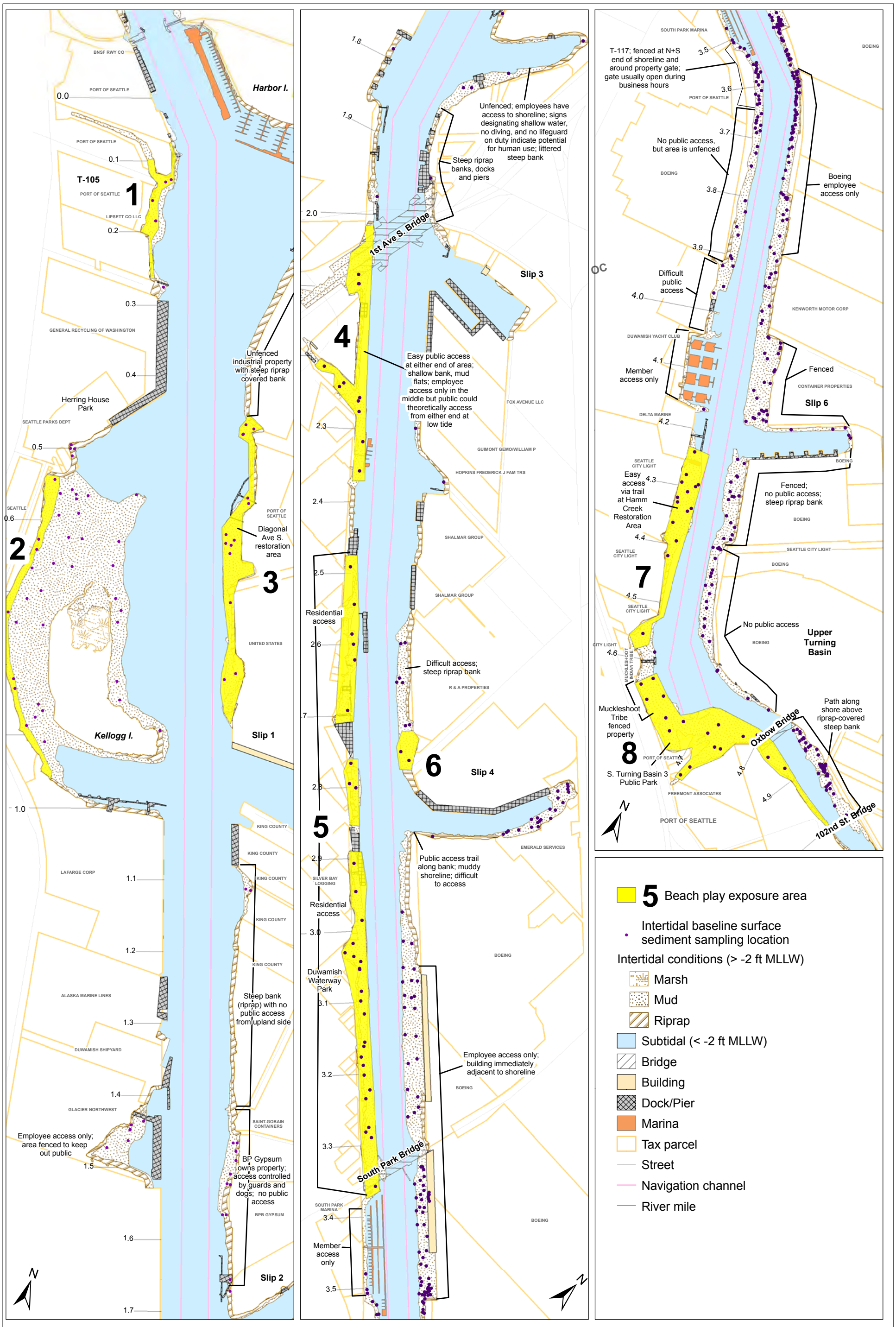
0 0.1 0.2 Miles

0 0.1 0.2 Kilometers

Scale is the same for each inset map

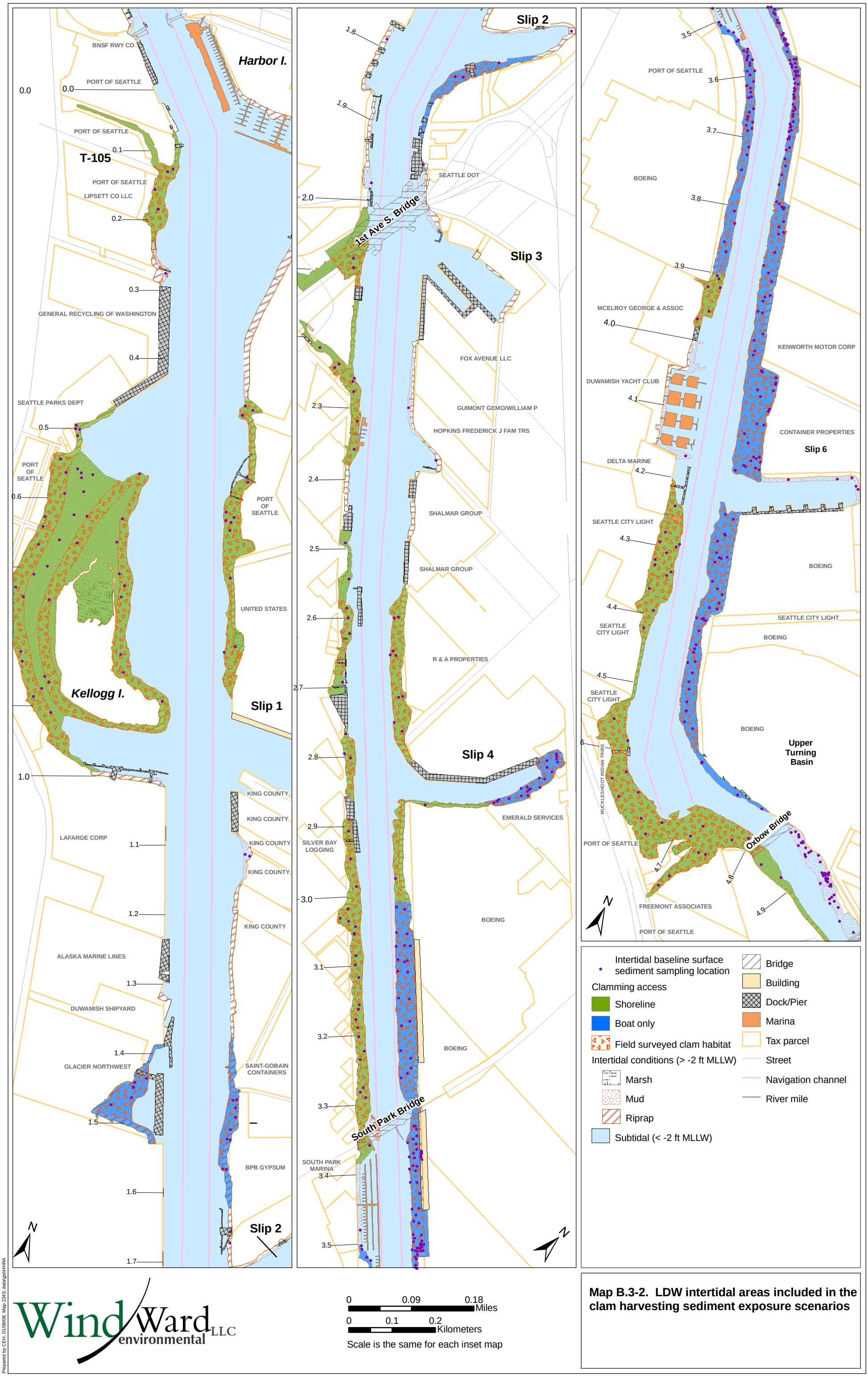
Map B.2-2b. LDW tissue sampling areas and subareas and individual locations where tissue samples were collected by non-LDWG parties prior to 2004

Prepared by CEH, 11/12/07; Map 2152; \data\gis\HHRA



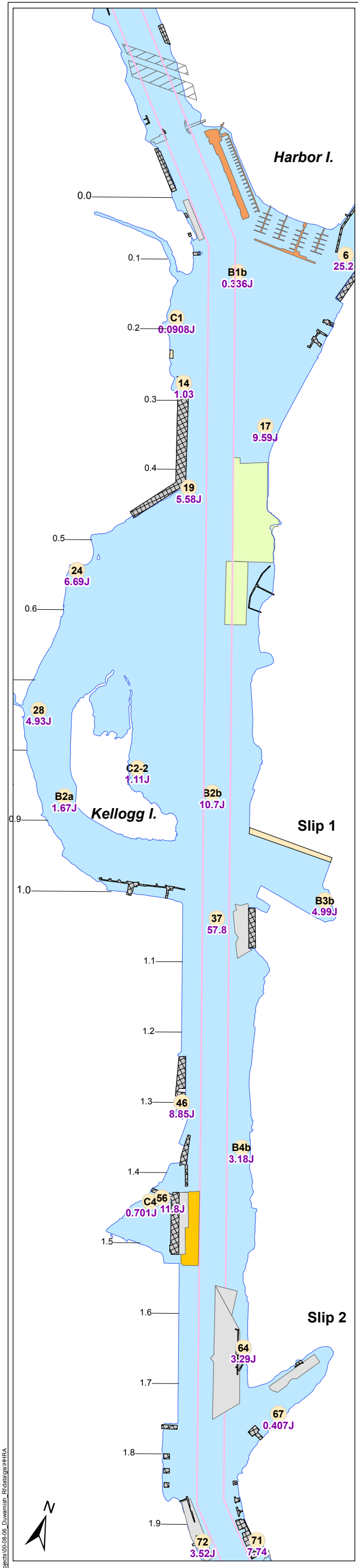
Map B.3-1. LDW intertidal areas included in the beach play sediment exposure scenarios

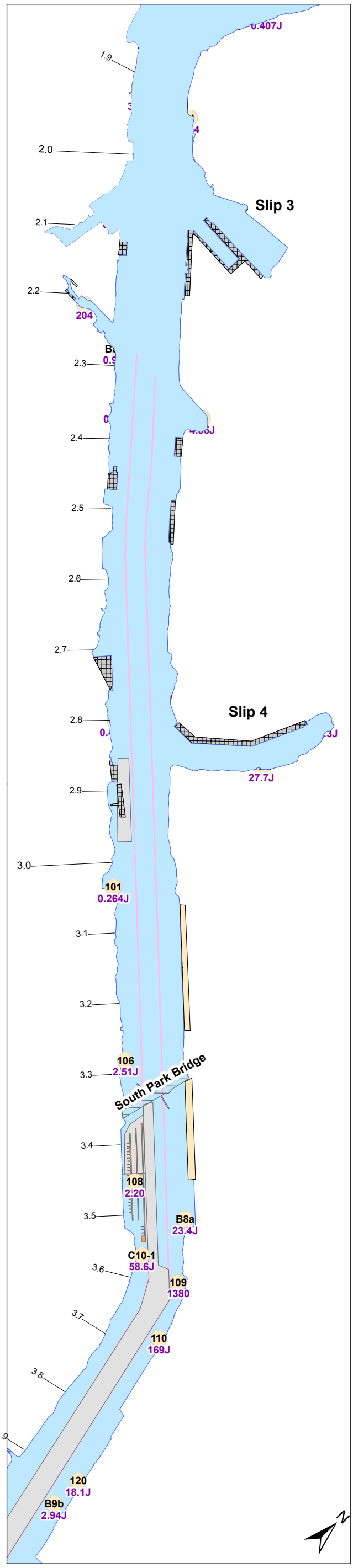
Prepared by CEH, 11/12/07; Map 2602; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA\

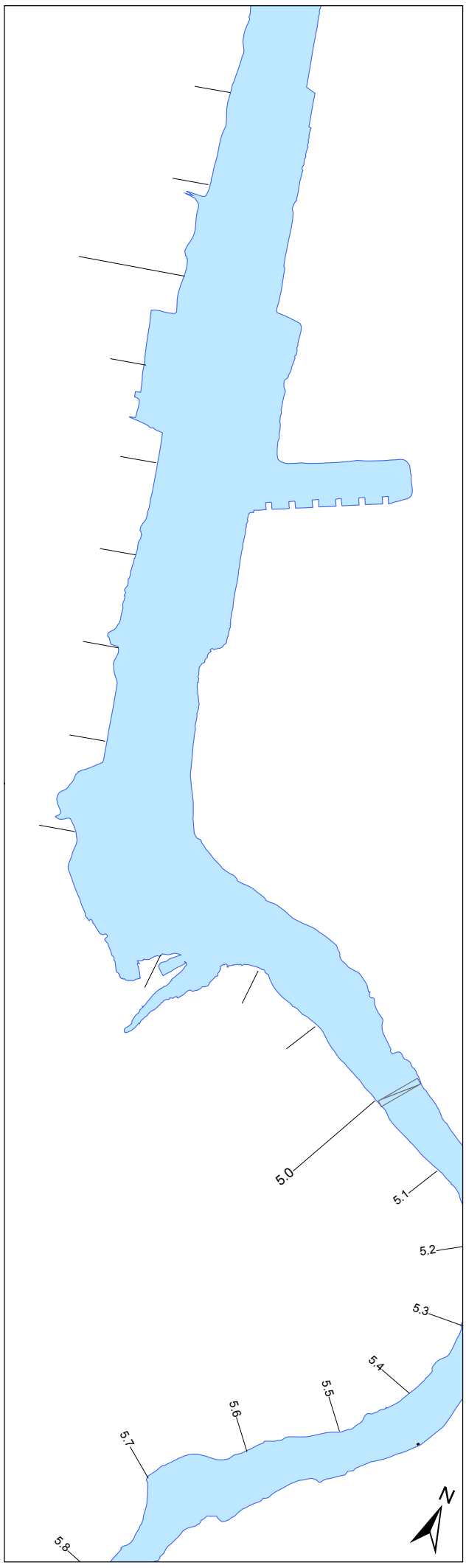


Map B.3-2. LDW intertidal areas included in the clam harvesting sediment exposure scenarios

Prepared by CEH, 01/09/08; Map 2245; data\gis\HHRA







Surface sediment sampling location ID
PCB TEQ value (ng/kg dw)*
J: estimated
56 19.6 J

- Dredged area
- Dredged and capped area
- Dredged and thin-layer placement
- Thin-layer placement
- Bridge
- Building
- Dock/Pier
- Marina
- Navigation channel
- River mile

* PCB TEQ values were calculated with mammalian TEFs for individual PCB congeners using ½ the reporting limit for undetected congeners.

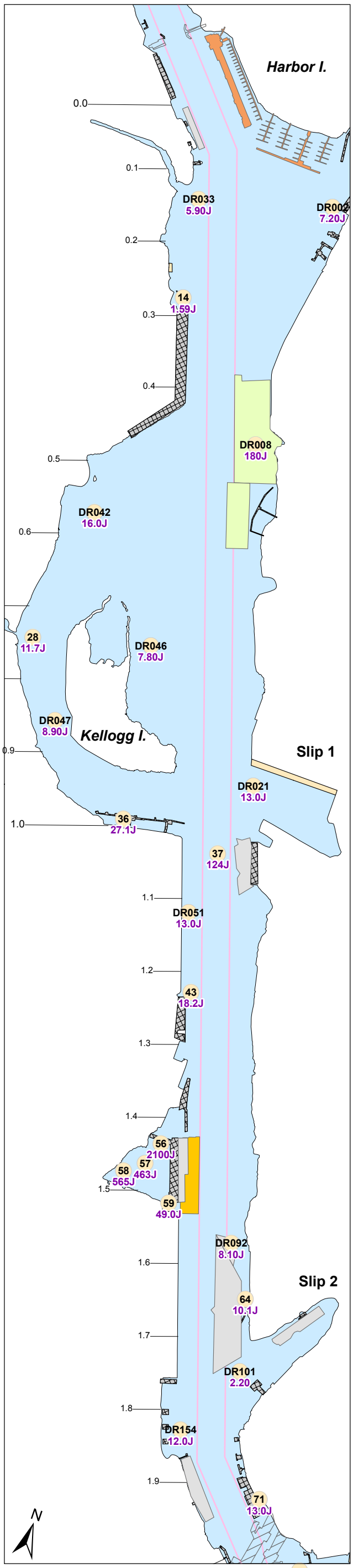
Wind Ward environmental LLC

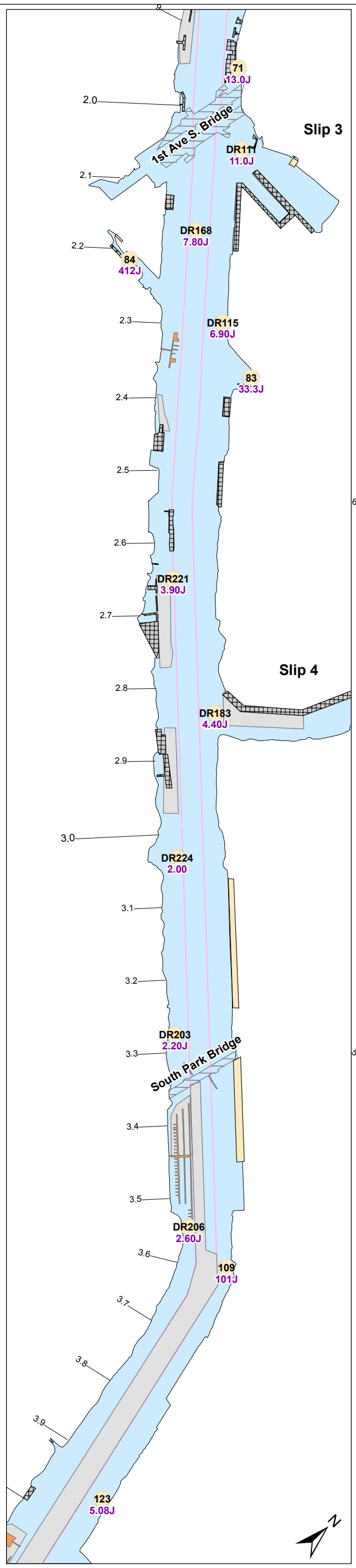
0 0.1 0.2 Miles
0 0.1 0.2 Kilometers

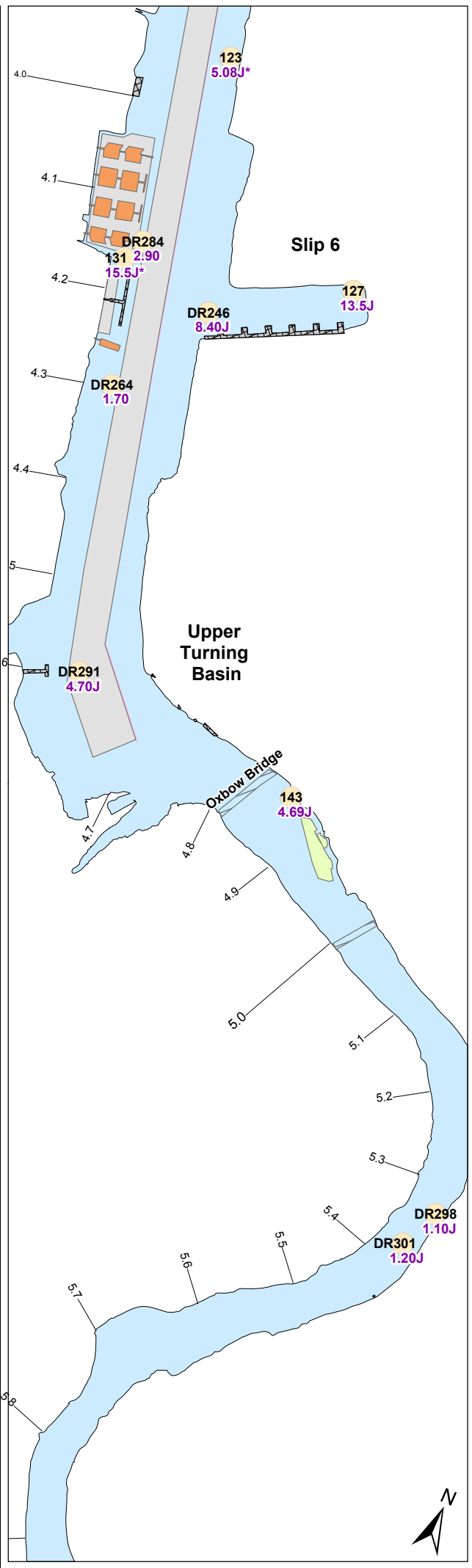
Scale is the same for each inset map

Map B.3-3. PCB TEQ values calculated from individual PCB congeners analyzed in LDW surface sediment using high-resolution methods

Prepared by CEH, 11/12/07; Map 2309; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Surface sediment sampling location ID
Dioxin/furan TEQ value (ng/kg dw)**
J: estimated
* Value is average of the original sample value and the field duplicate sample value.

123 5.15 J*

- Dredged area
- Dredged and capped area
- Dredged and thin-layer placement
- Thin-layer placement
- Bridge
- Building
- Dock/Pier
- Marina
- Navigation channel
- River mile

** Dioxin/furan TEQ values were calculated with mammalian TEFs for individual dioxin/furan congeners using ½ the reporting limit for undetected congeners.

Wind Ward LLC environmental

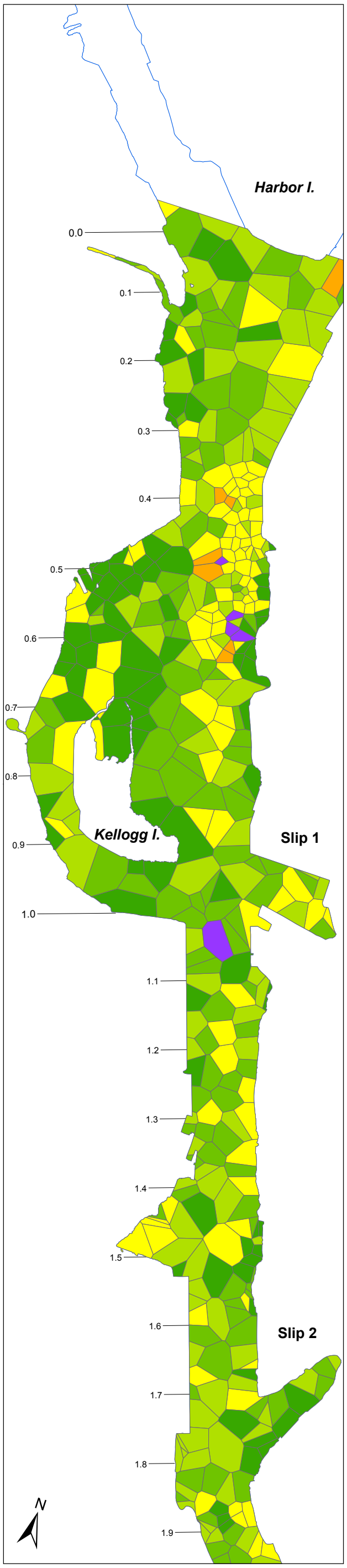
0 0.1 0.2 Miles

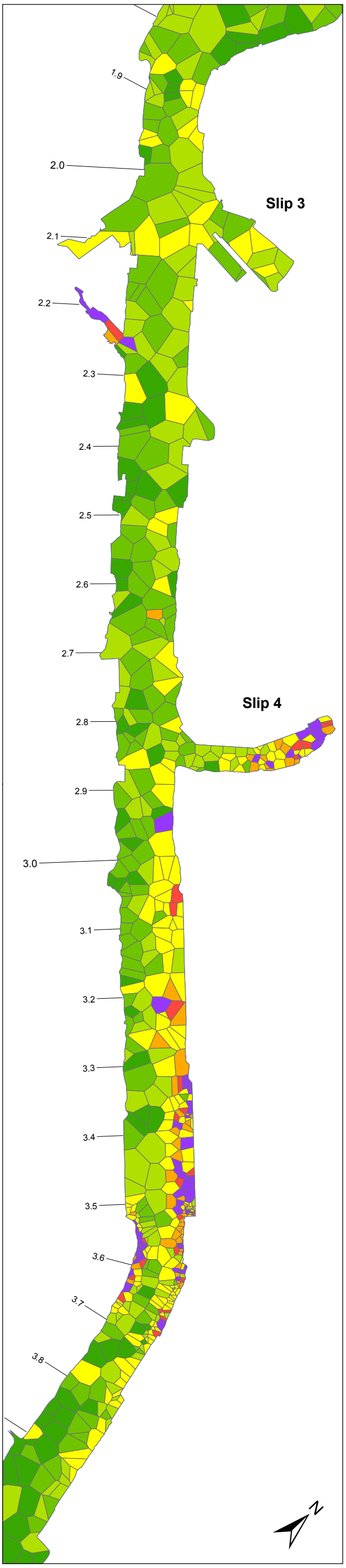
0 0.1 0.2 Kilometers

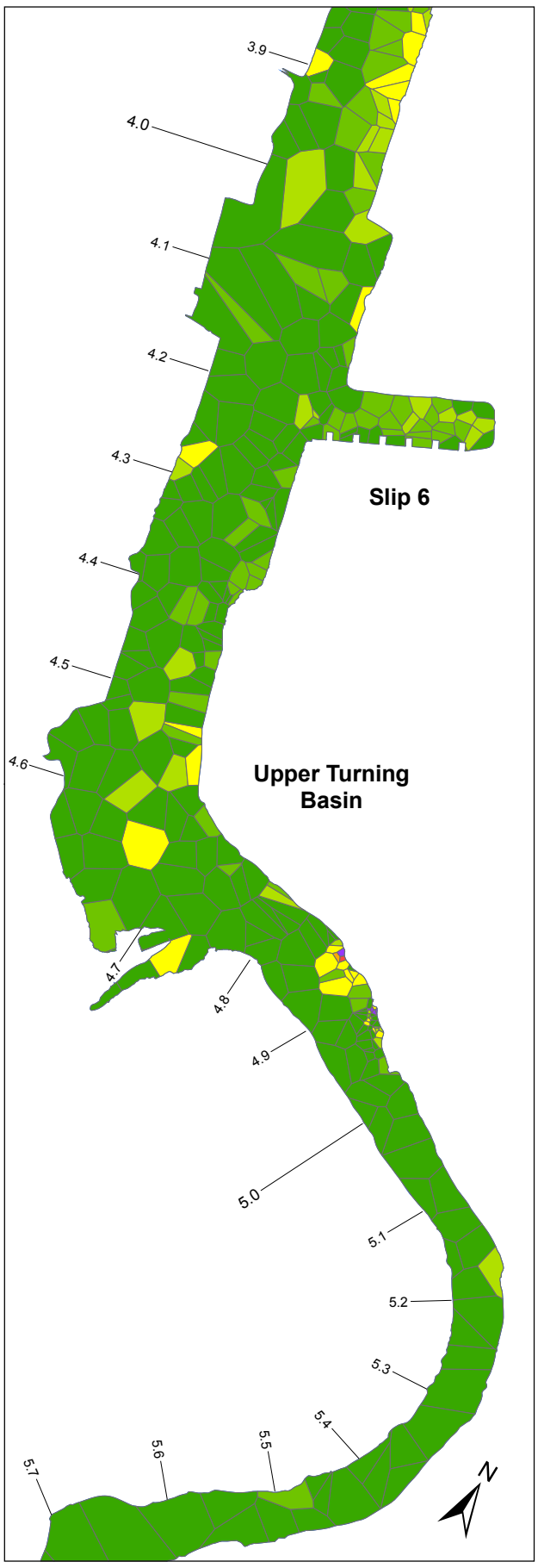
Scale is the same for each inset map

Map B.3-4. Dioxin/furan TEQ values calculated from dioxin/furan concentrations in LDW surface sediment samples

Prepared by CEH, 11/12/07, Map 2246; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Total PCBs (µg/kg dw)

- 0 - 60
- 61 - 120
- 121 - 240
- 241 - 1,300
- 1,301 - 2,600
- 2,601 - 3,900
- 3,901 - 22,0000
- River mile

Wind Ward environmental LLC

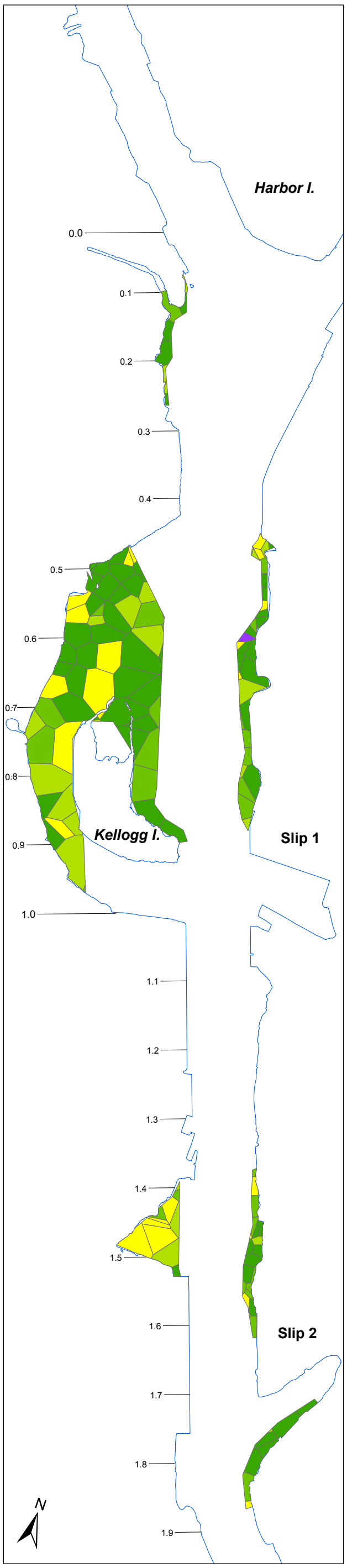
0 0.1 0.2 Miles

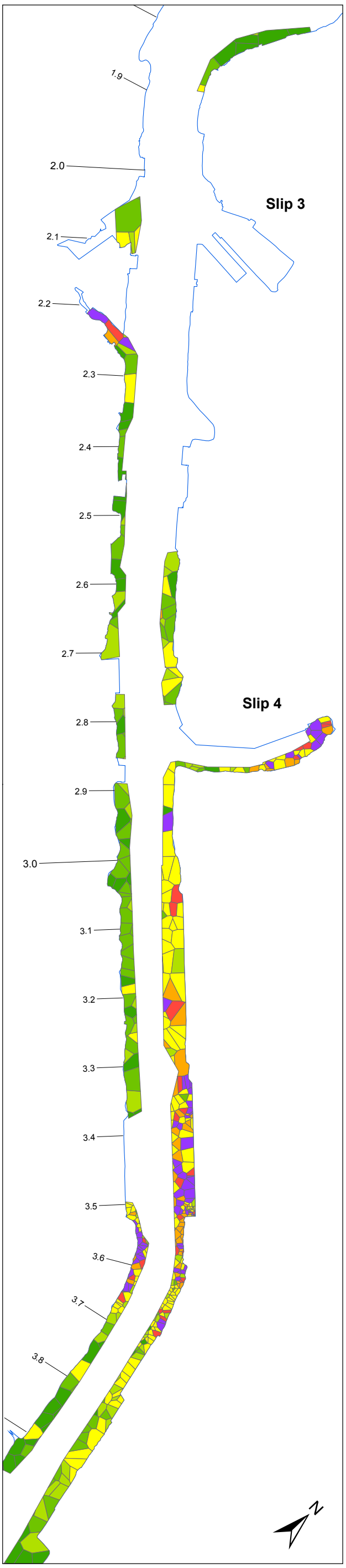
0 0.1 0.2 Kilometers

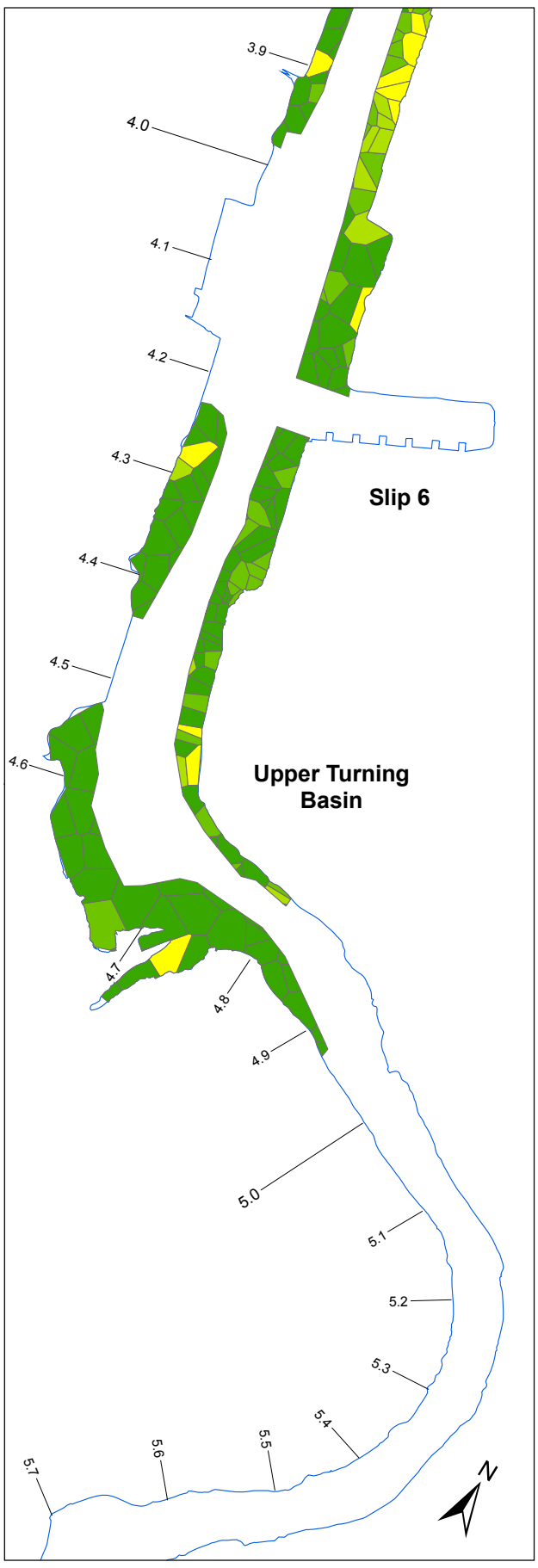
Scale is the same for each inset map

Map B.6-1. Total PCBs as Thiessen polygons for the LDW netfishing exposure area

Prepared by CEJ 11/08/07, MAP 3058; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Total PCBs (µg/kg dw)

- 0 - 60
- 61 - 120
- 121 - 240
- 241 - 1,300
- 1,301 - 2,600
- 2,601 - 3,900
- 3,901 - 22,0000
- River mile

Wind Ward environmental LLC

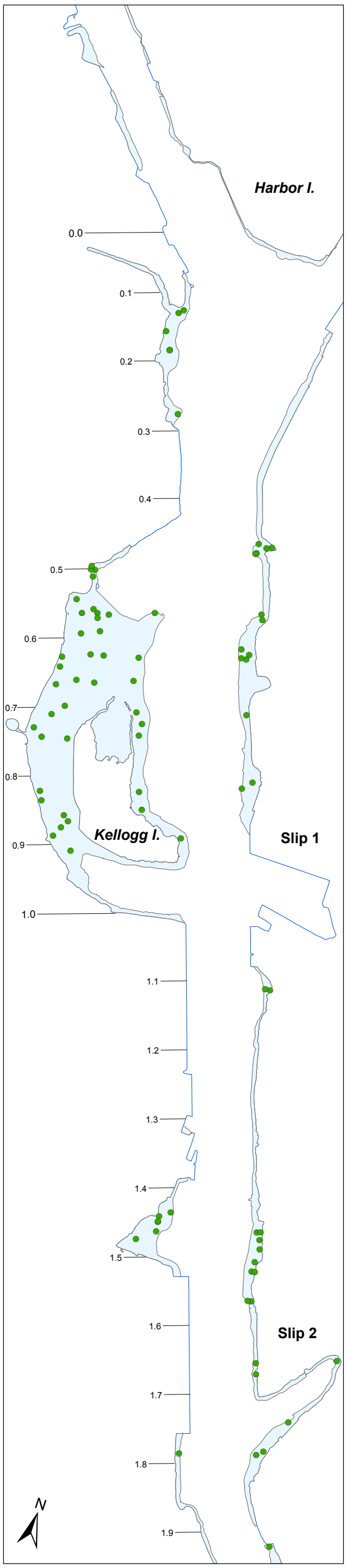
0 0.1 0.2 Miles

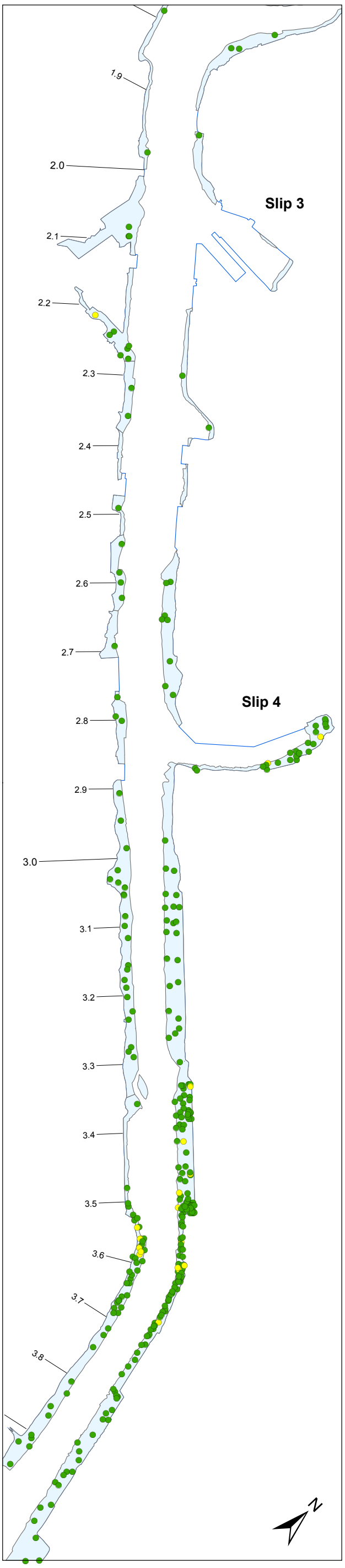
0 0.1 0.2 Kilometers

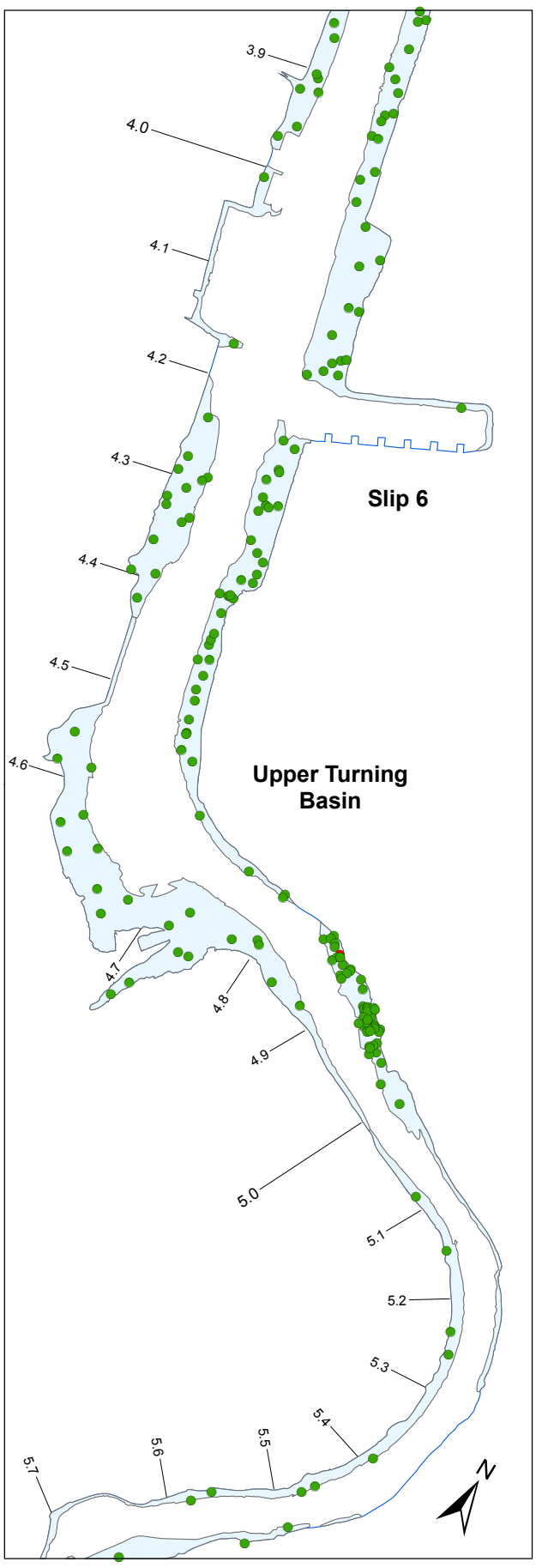
Scale is the same for each inset map

Map B.6-2. Total PCBs as Thiessen polygons for the LDW tribal clamming exposure areas

Prepared by CEJ 11/08/07, MAP 3059, W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6} (≤11 mg/kg dw)
- >10^{-6} and ≤10^{-5} (>11 and ≤110 mg/kg dw)
- >10^{-5} and ≤10^{-4} (>110 and ≤1,100 mg/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a habitat biologist who works only at that specific location for 15 days/yr over a 20-yr period. Each risk estimate is based on the total PCB concentration at that specific location only, without consideration of data for any neighboring locations.

Wind Ward environmental LLC

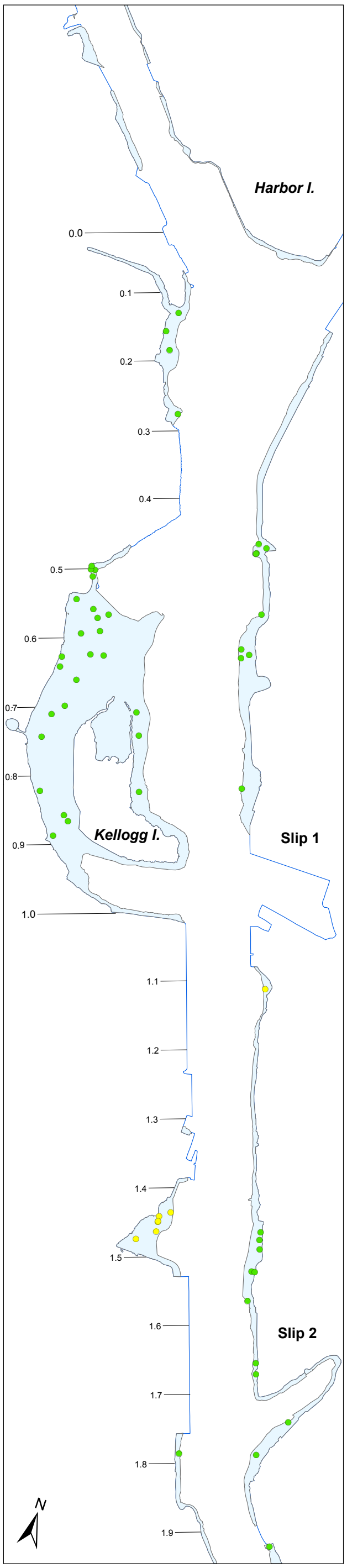
0 0.1 0.2 Miles

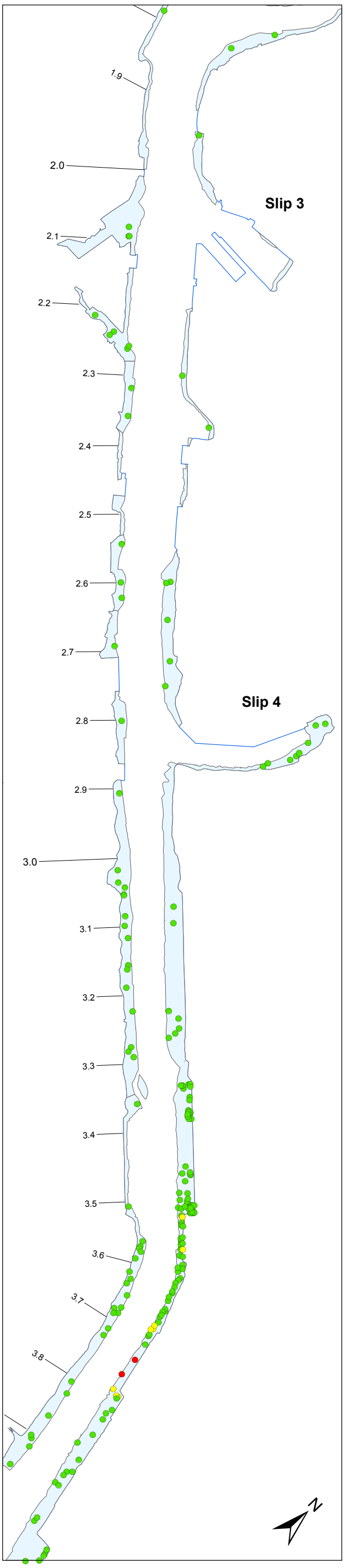
0 0.1 0.2 Kilometers

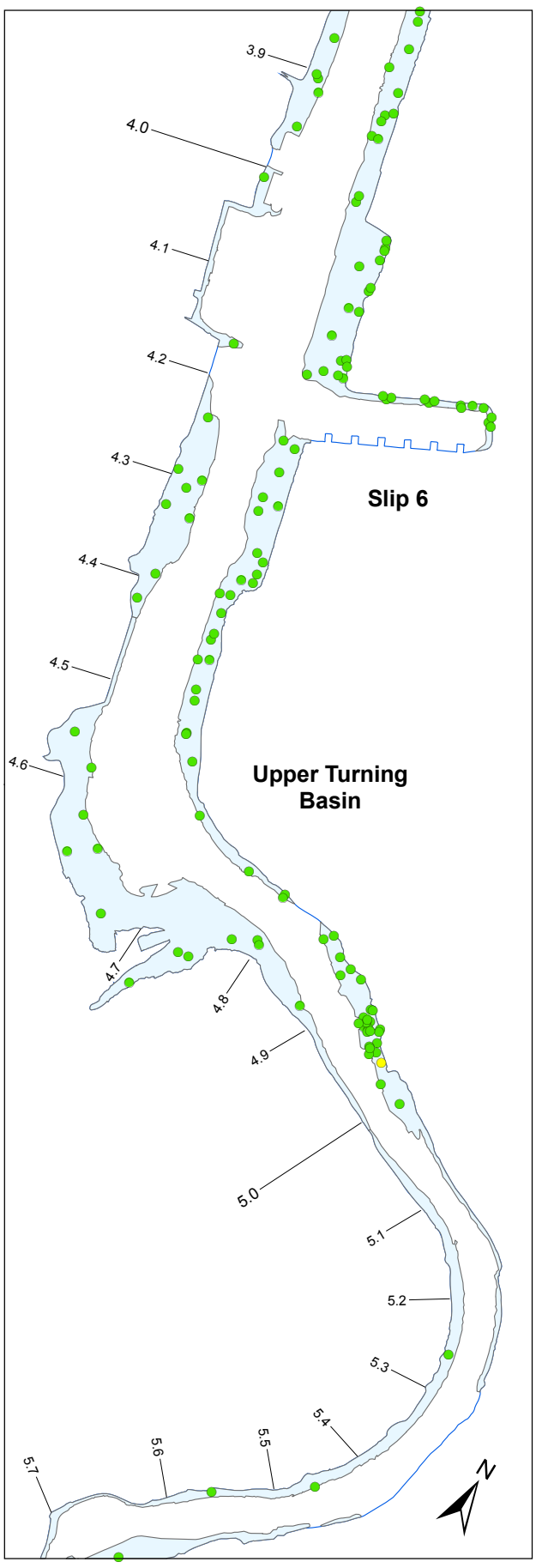
Scale is the same for each inset map

Map B.6-3. Risk-based concentration comparisons for total PCBs using the habitat biologist exposure scenario

Prepared by CEJ, 01/08/07; MAP # 2870; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6} (≤30 mg/kg dw)
- >10^{-6} and ≤10^{-5} (>30 and ≤300 mg/kg dw)
- >10^{-5} and ≤10^{-4} (>300 and ≤3,000mg/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a habitat biologist who works only at that specific location for 15 days/yr over a 20-yr period. Each risk estimate is based on the arsenic concentration at that specific location only, without consideration of data for any neighboring locations.

Wind Ward environmental LLC

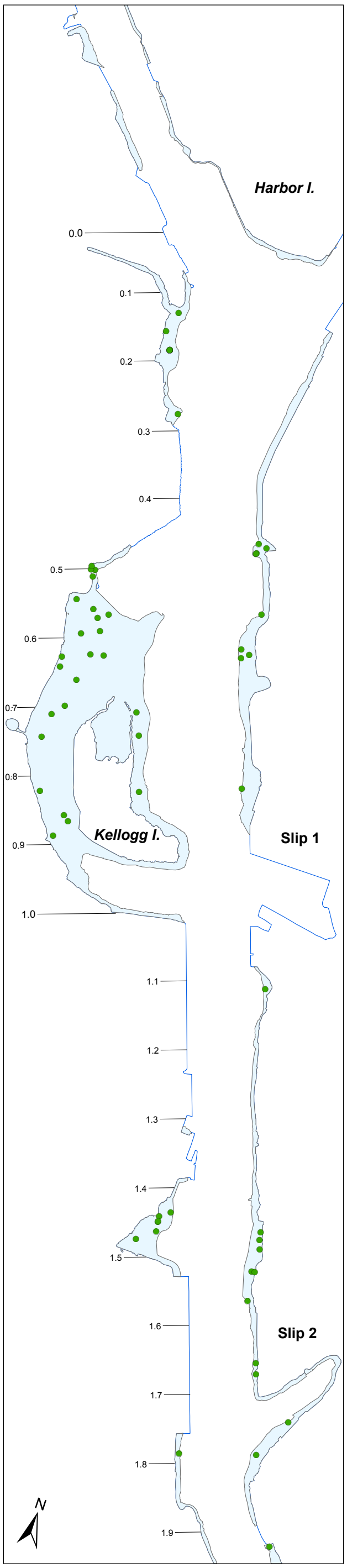
0 0.1 0.2 Miles

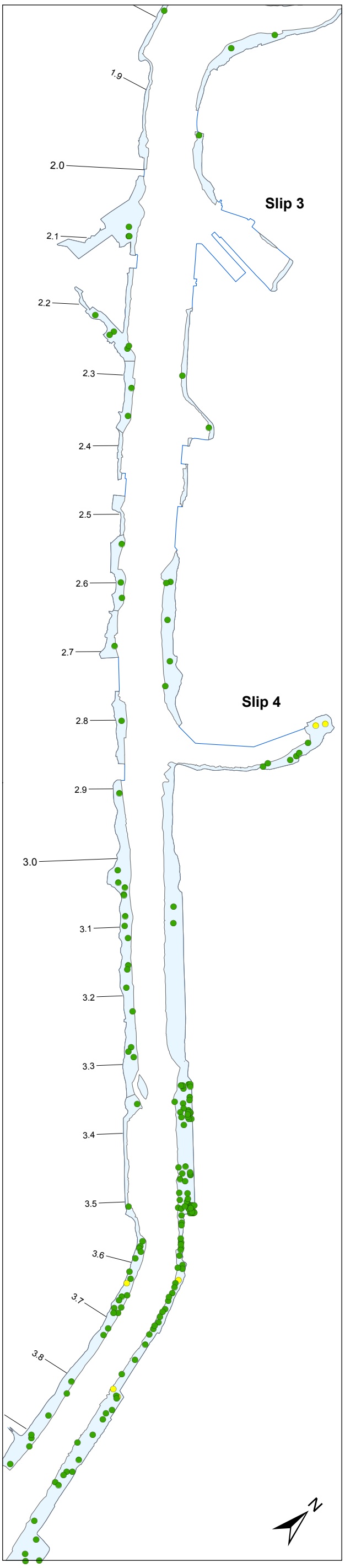
0 0.1 0.2 Kilometers

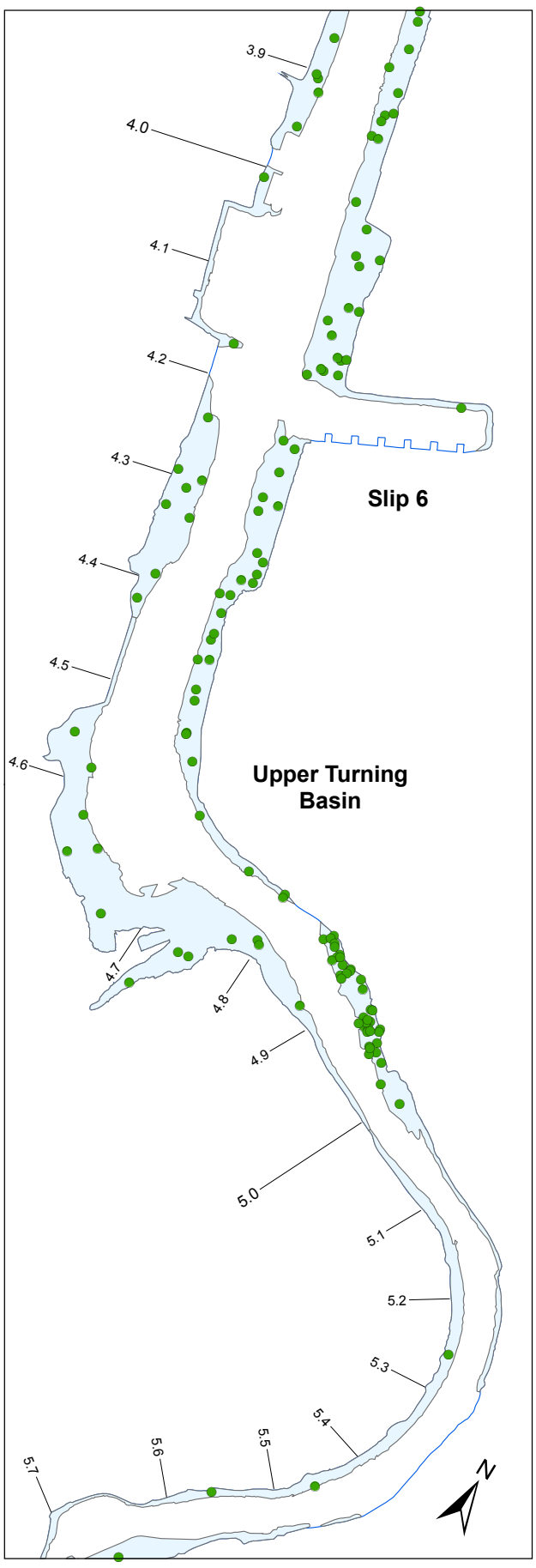
Scale is the same for each inset map

Map B.6-4. Risk-based concentration comparisons for arsenic using the habitat biologist exposure scenario

Prepared by CEJ, 11/08/07 MAP # 2871; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6} (≤3.3 mg/kg dw)
- >10^{-6} (>3.3 and ≤33 mg/kg dw)
- >10^{-6} and ≤10^{-5} (>33 and ≤330 mg/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a habitat biologist who works only at that specific location for 15 days/yr over a 20-yr period. Each risk estimate is based on the cPAH concentration at that specific location only, without consideration of data for any neighboring locations.

Wind Ward environmental LLC

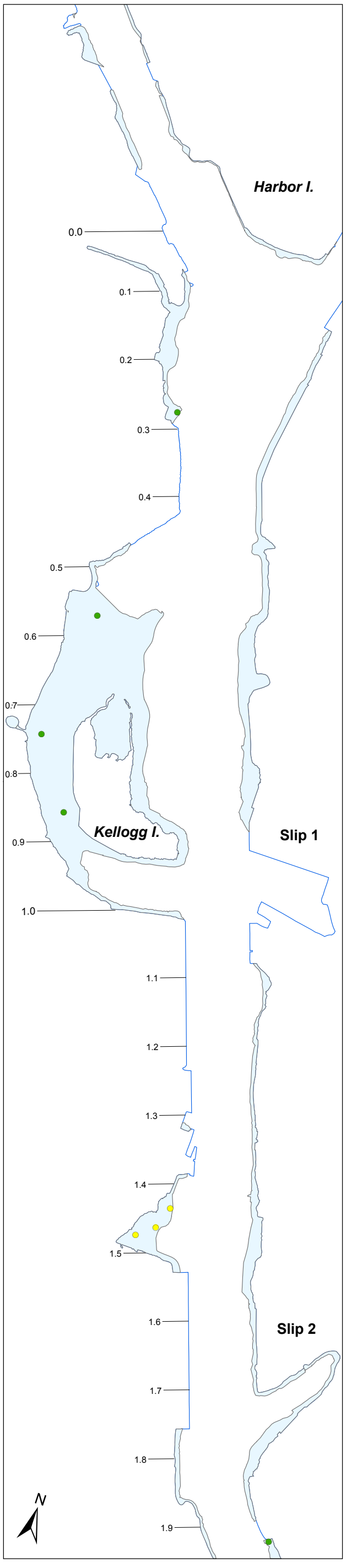
0 0.1 0.2 Miles

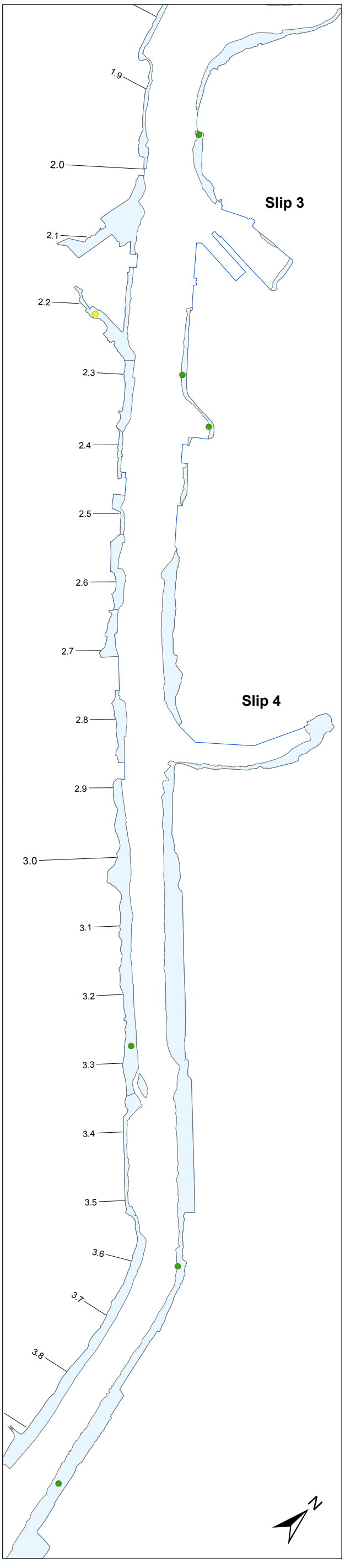
0 0.1 0.2 Kilometers

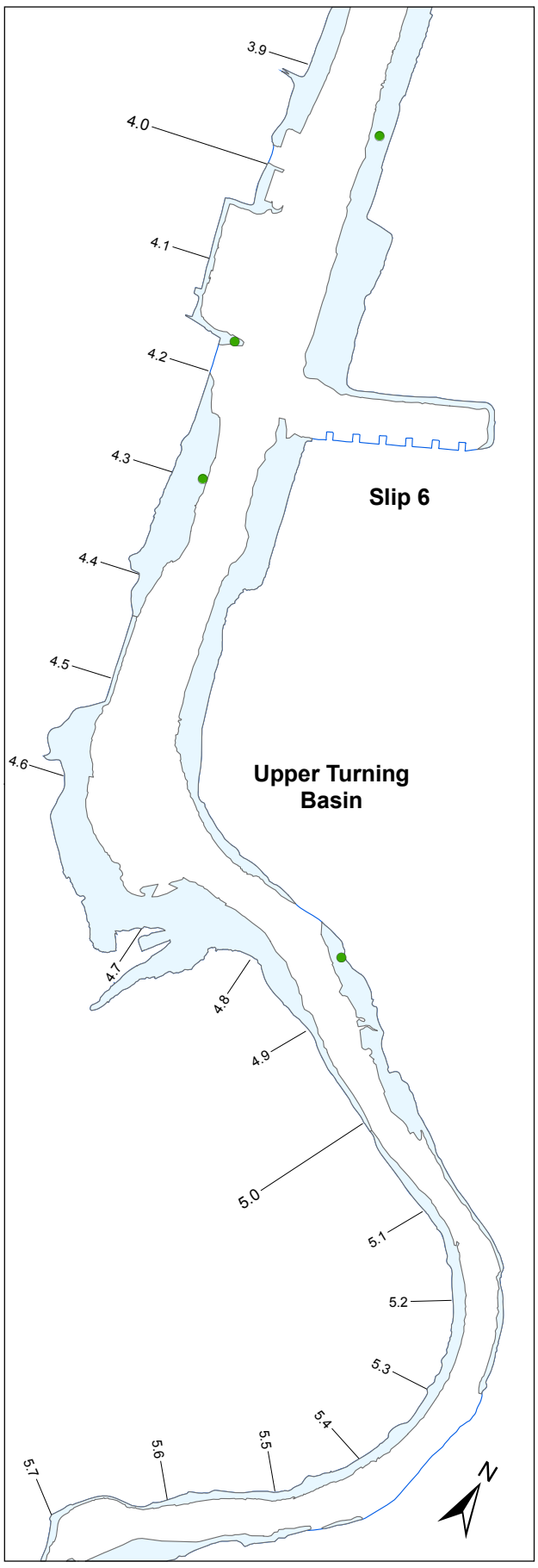
Scale is the same for each inset map

Map B.6-5. Risk-based concentration comparisons for cPAHs using the habitat biologist exposure scenario

Prepared by CEJ, 11/06/07 MAP # 2869; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6} (≤299 ng/kg dw)
- >10^{-6} and ≤10^{-5} (>299 and ≤2,990 ng/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a habitat biologist who works only at that specific location for 15 days/yr over a 20-yr period. Each risk estimate is based on the dioxin/furan TEQ at that specific location only, without consideration of data for any neighboring locations.

Wind Ward environmental LLC

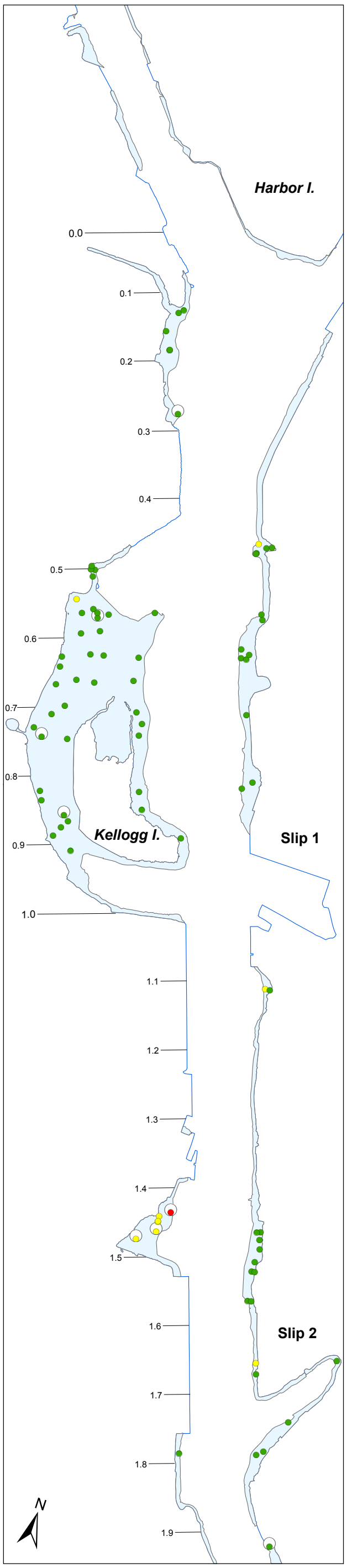
0 0.1 0.2 Miles

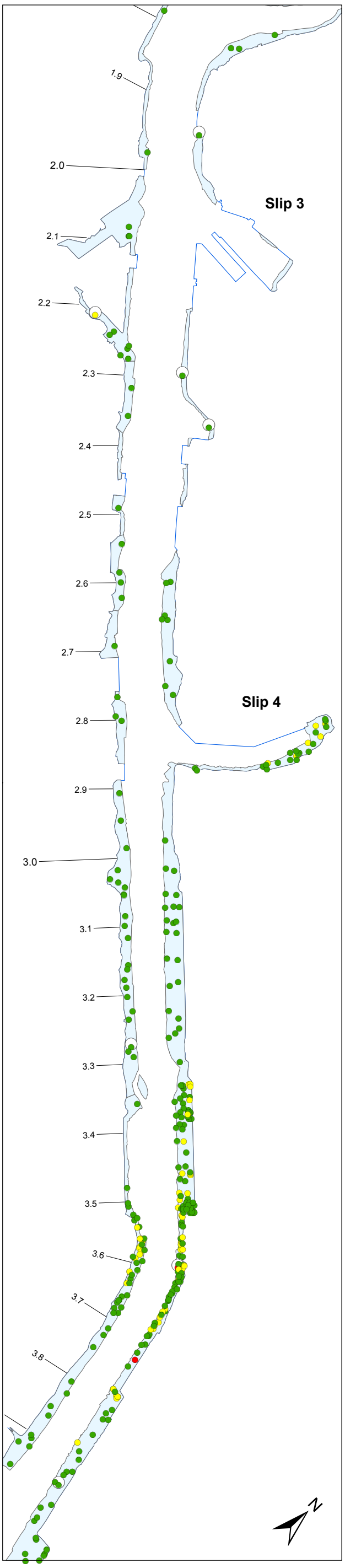
0 0.1 0.2 Kilometers

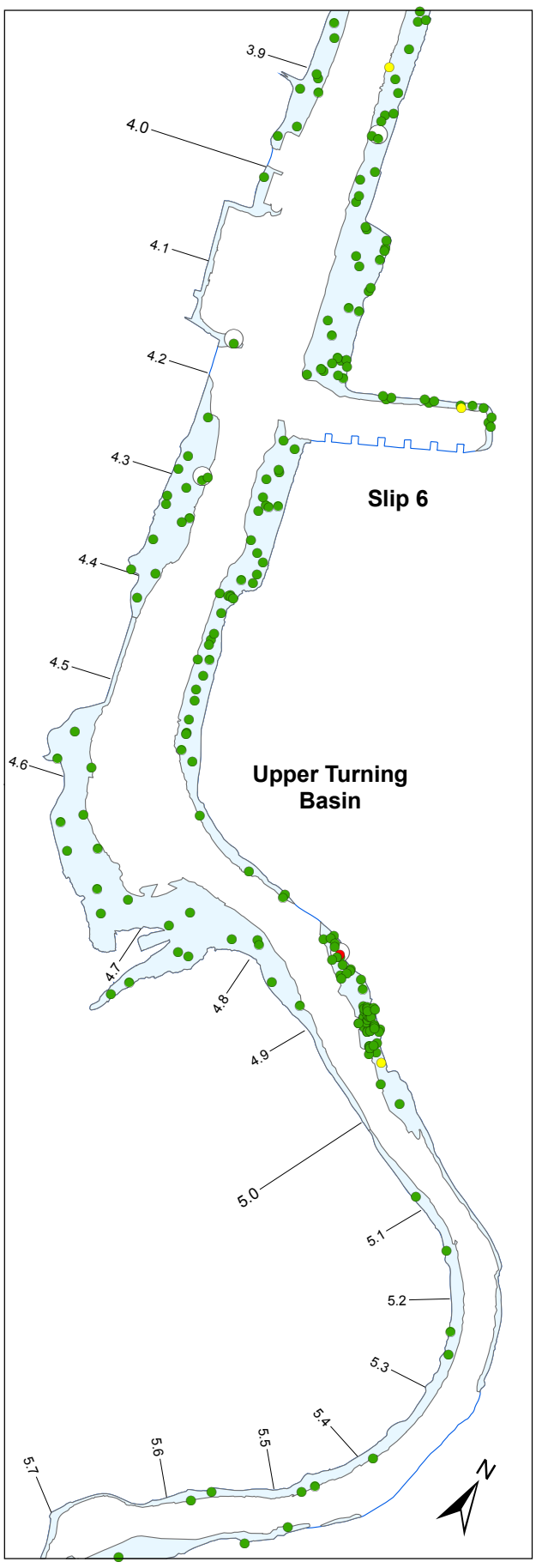
Scale is the same for each inset map

Map B.6-6. Risk-based concentration comparisons for dioxins/furans using the habitat biologist exposure scenario

Prepared by CEJ, 11/08/07 MAP # 2995; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6}
- >10^{-6} and ≤10^{-5}
- >10^{-5} and ≤10^{-4}
- Dioxin/Furan sample location
- Intertidal area
- River mile

* Risks may be underestimated at some locations because not all chemicals were analyzed.

Each point represents the hypothetical excess cancer risk for a habitat biologist who works only at that specific location for 15 days/yr over a 20-yr period. Each risk estimate is based on the total PCB, arsenic, cPAH, and dioxin/furan TEQ (if available) concentrations at that specific location only, without consideration of data for any neighboring locations.

Wind Ward environmental LLC

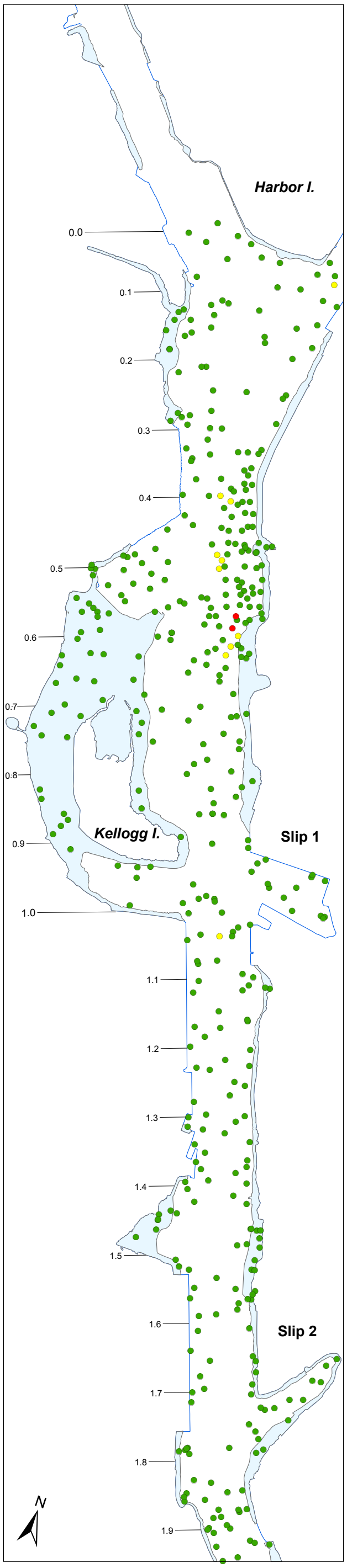
0 0.1 0.2 Miles

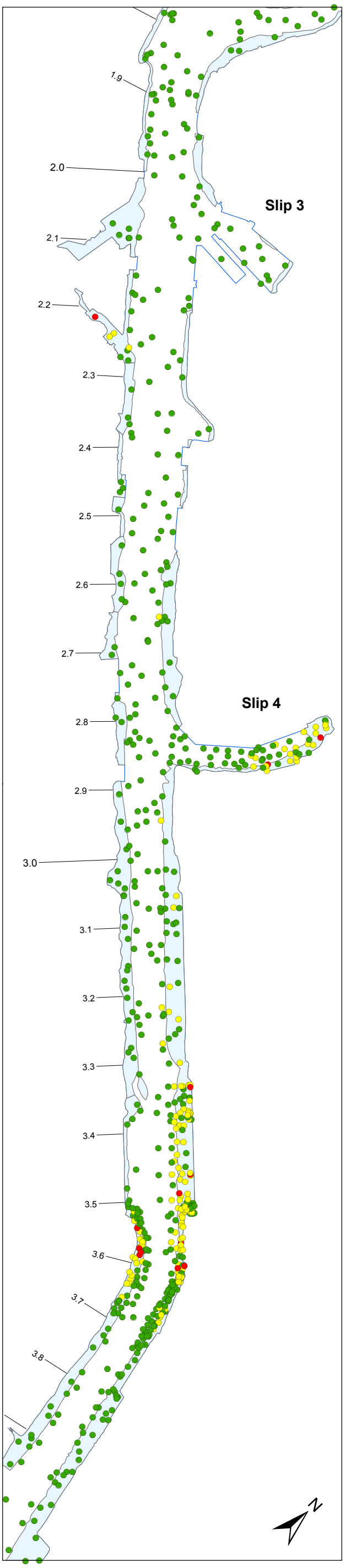
0 0.1 0.2 Kilometers

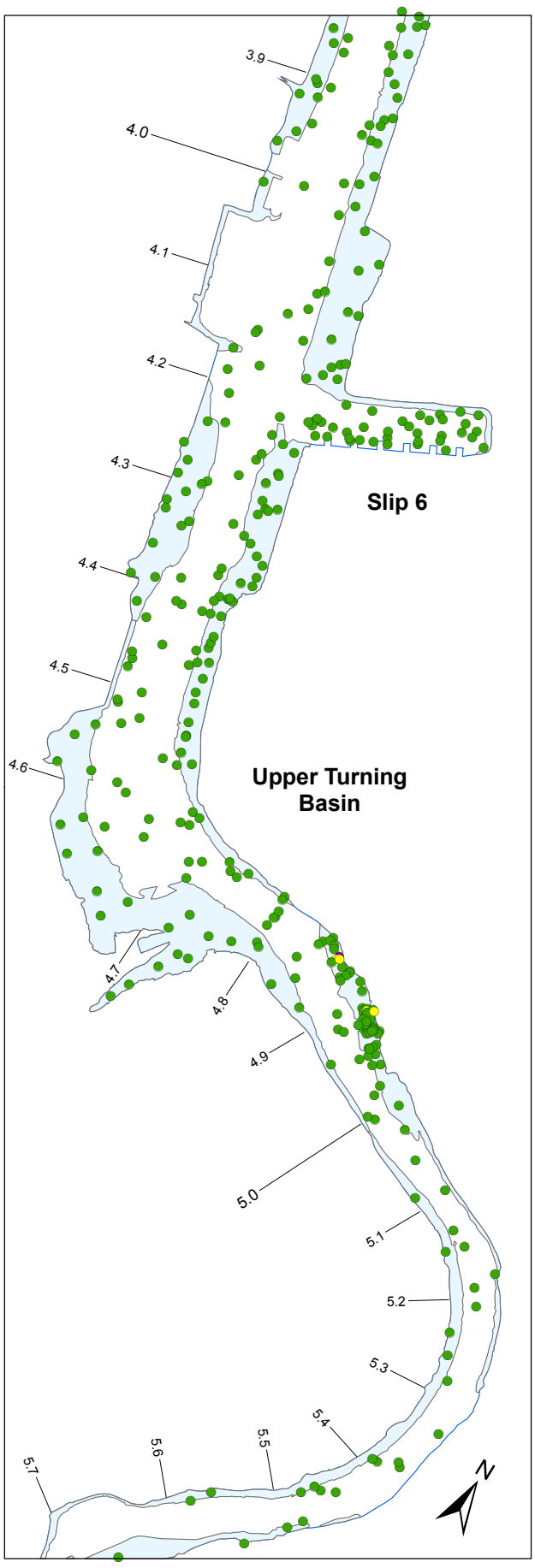
Scale is the same for each inset map

Map B.6-7. Combination of risks associated with total PCBs, arsenic, dioxins/furans, and cPAHs using the habitat biologist scenario

Prepared by CEJ, 11/08/07; MAP # 2868; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6} (≤1,320 µg/kg dw)
- >10^{-6} and ≤10^{-5} (>1,320 and ≤13,200 µg/kg dw)
- >10^{-5} and ≤10^{-4} (>13,200 and ≤132,000 µg/kg dw)
- >10^{-4} and ≤10^{-3} (>132,000 and ≤1,320,000 µg/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a netfisher who works only at that specific location for 119 days/yr over a 44-yr period (i.e., the assumptions from the netfishing RME scenario). Each risk estimate is based on a weighted mean of the total PCB concentration at that specific location only (25% of total) and the LDW-wide UCL on the spatially-weighted mean (470 µg/kg dw; 75% of the total). Mathematically, this is equivalent to assuming that netfishing occurs at a single location for 11 years and throughout the LDW for the other 33 years of the 44-yr exposure duration assumed for this scenario.

Wind Ward environmental LLC

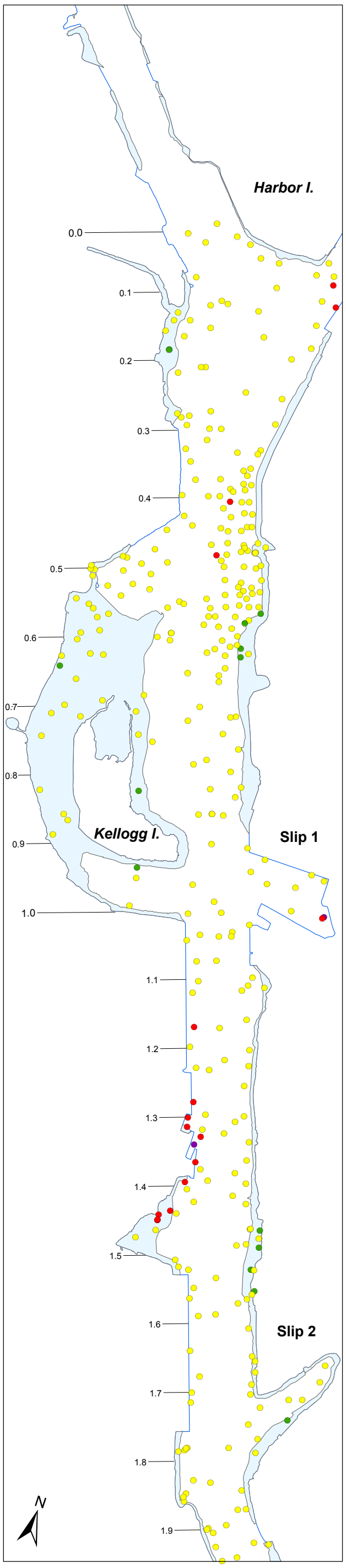
0 0.1 0.2 Miles

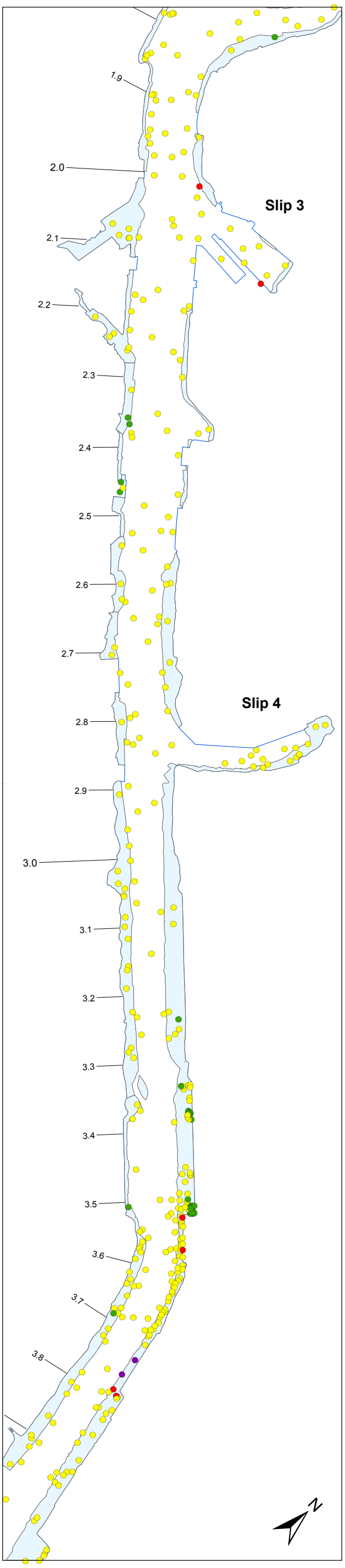
0 0.1 0.2 Kilometers

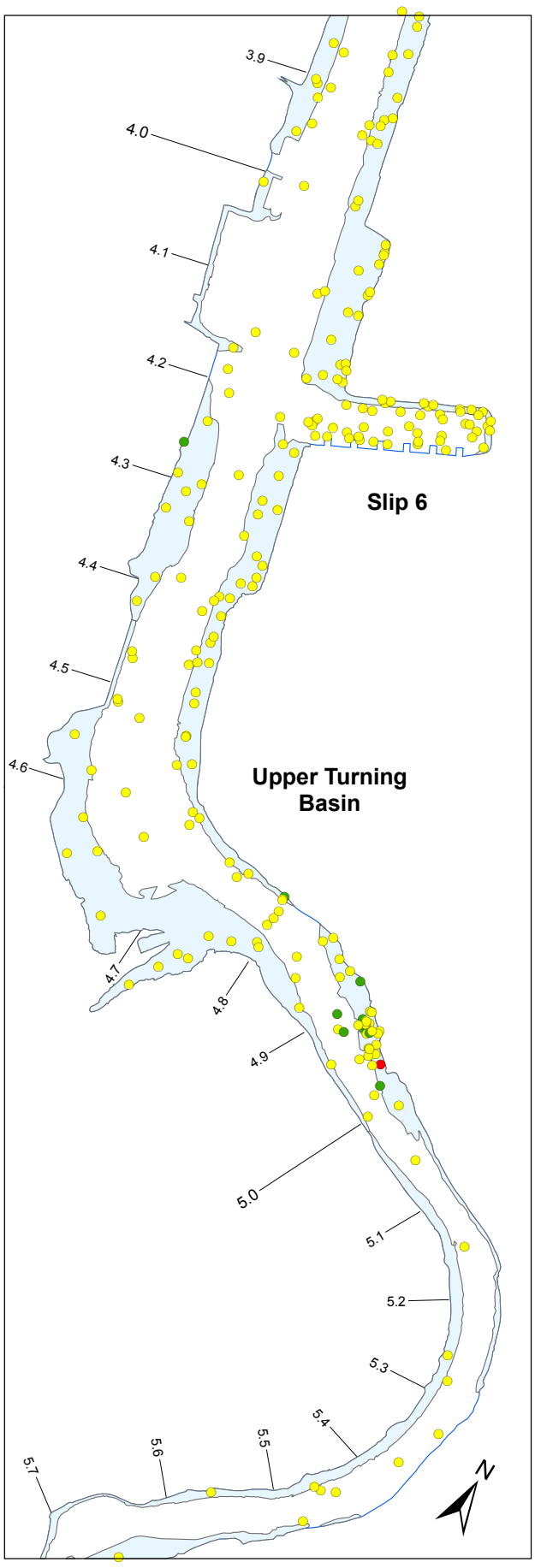
Scale is the same for each inset map

Map B.6-8. Risk-based concentration comparisons for total PCBs using the netfishing exposure scenario

Prepared by CEJ, 11/08/07 MAP # 2751; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6} (≤ 3.7 mg/kg dw)
- >10^{-6} and ≤10^{-5} (>3.7 and ≤37 mg/kg dw)
- >10^{-5} and ≤10^{-4} (>37 and ≤370 mg/kg dw)
- >10^{-4} and ≤10^{-3} (>370 and ≤ 3,700 mg/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a netfisher who works only at that specific location for 119 days/yr over a 44-yr period (i.e., the assumptions from the netfishing RME scenario). Each risk estimate is based on a weighted mean of the arsenic concentration at that specific location only (25% of total) and the LDW-wide EPC (21 mg/kg dw; 75% of the total). Mathematically, this is equivalent to assuming that netfishing occurs at a single location for 11 years and throughout the LDW for the other 33 years of the 44-yr exposure duration assumed for this scenario.

Wind Ward environmental LLC

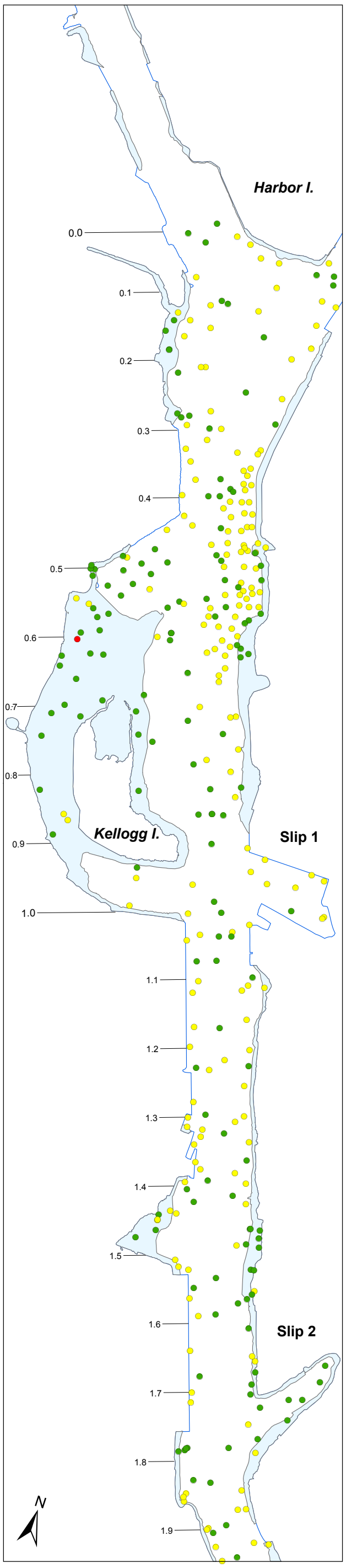
0 0.1 0.2 Miles

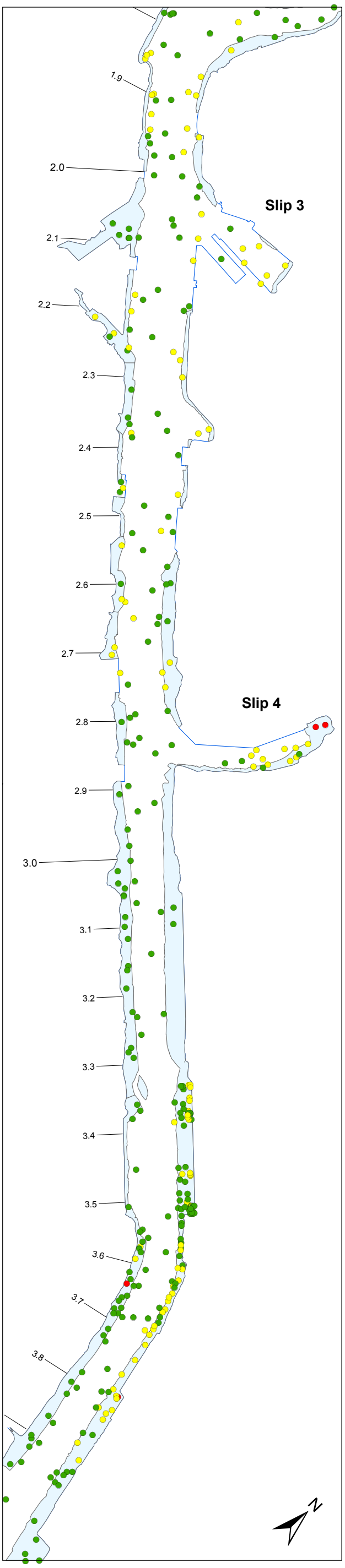
0 0.1 0.2 Kilometers

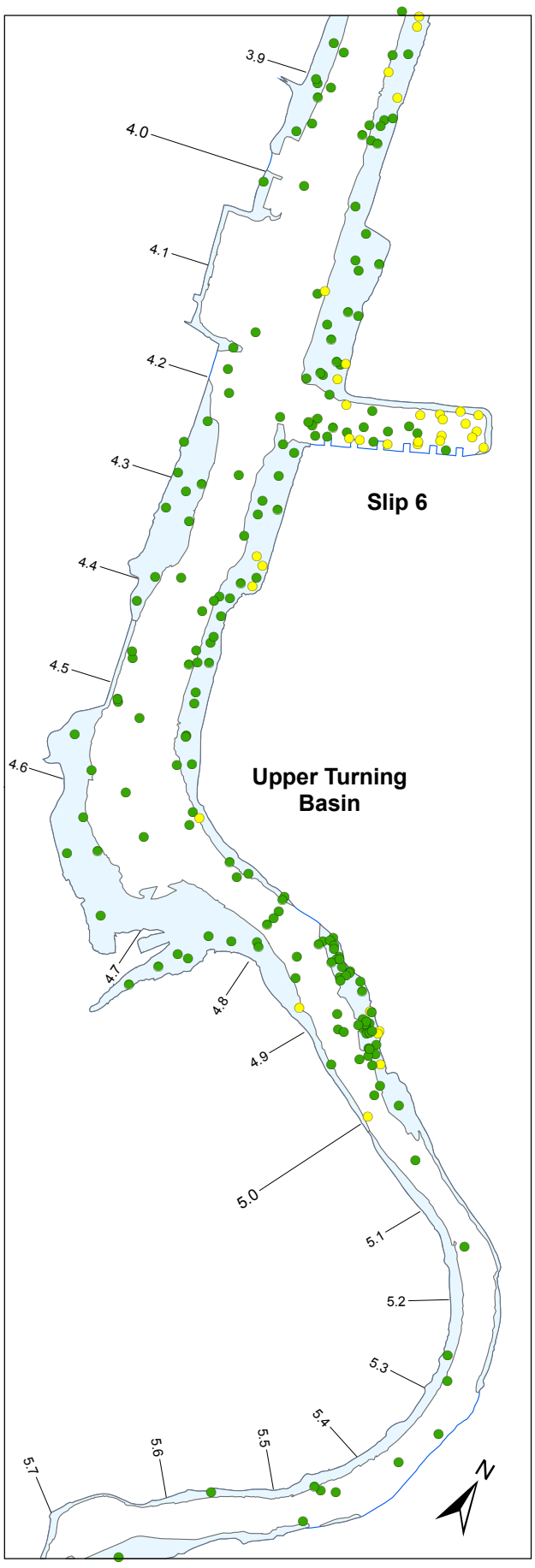
Scale is the same for each inset map

Map B.6-9. Risk-based concentration comparisons for arsenic using the netfishing exposure scenario

Prepared by CEJ, 11/06/07 MAP # 2752; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk *

- ≤10^{-6} (380 µg/kg dw)
- >10^{-6} and ≤10^{-5} (>380 and ≤3,800 µg/kg dw)
- >10^{-5} and ≤10^{-4} (>3,800 and ≤38,000 µg/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a netfisher who works only at that specific location for 119 days/yr over a 44-yr period (i.e., the assumptions from the netfishing RME scenario). Each risk estimate is based on a weighted mean of the cPAH concentration at that specific location only (25% of total) and the LDW-wide EPC (570 µg/kg dw; 75% of the total). Mathematically, this is equivalent to assuming that netfishing occurs at a single location for 11 years and throughout the LDW for the other 33 years of the 44-yr exposure duration assumed for this scenario.

Wind Ward environmental LLC

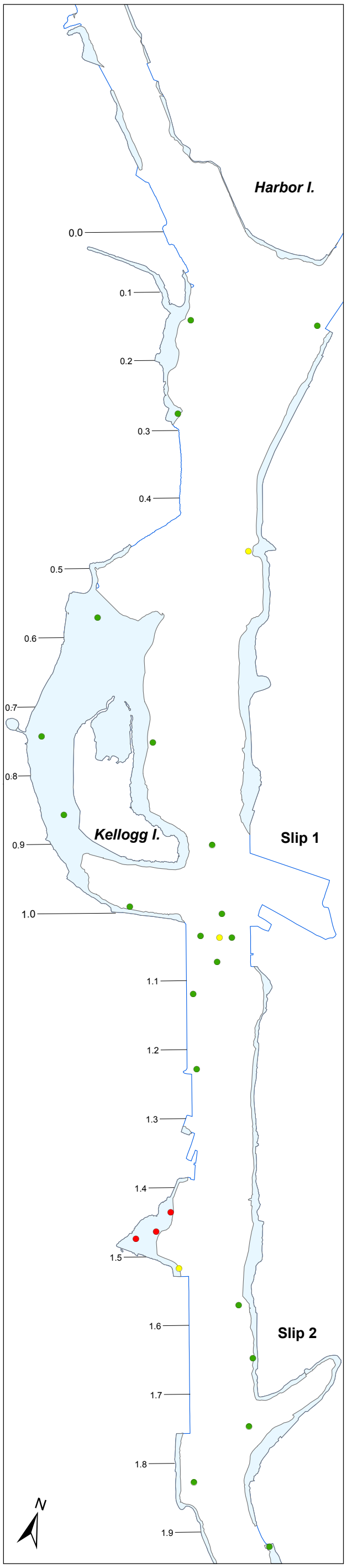
0 0.1 0.2 Miles

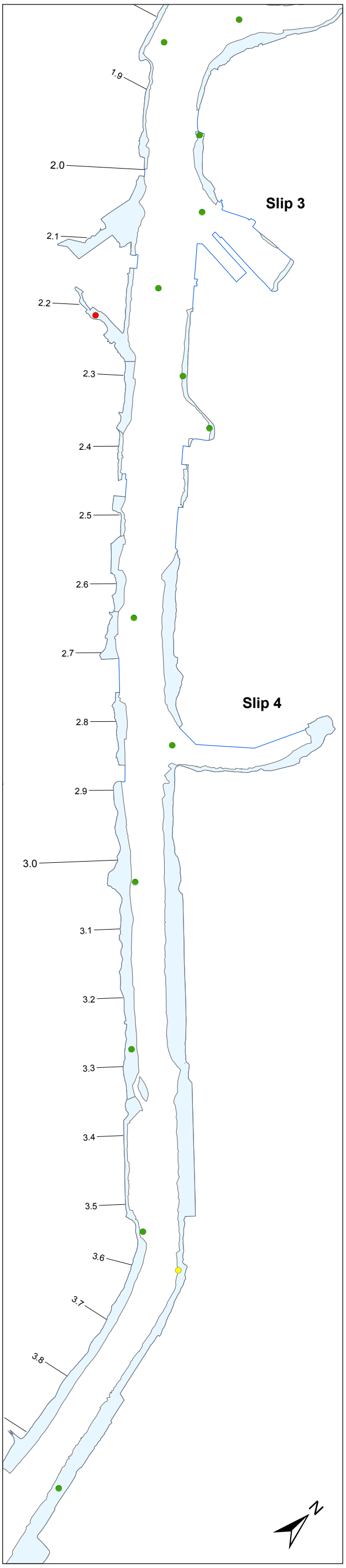
0 0.1 0.2 Kilometers

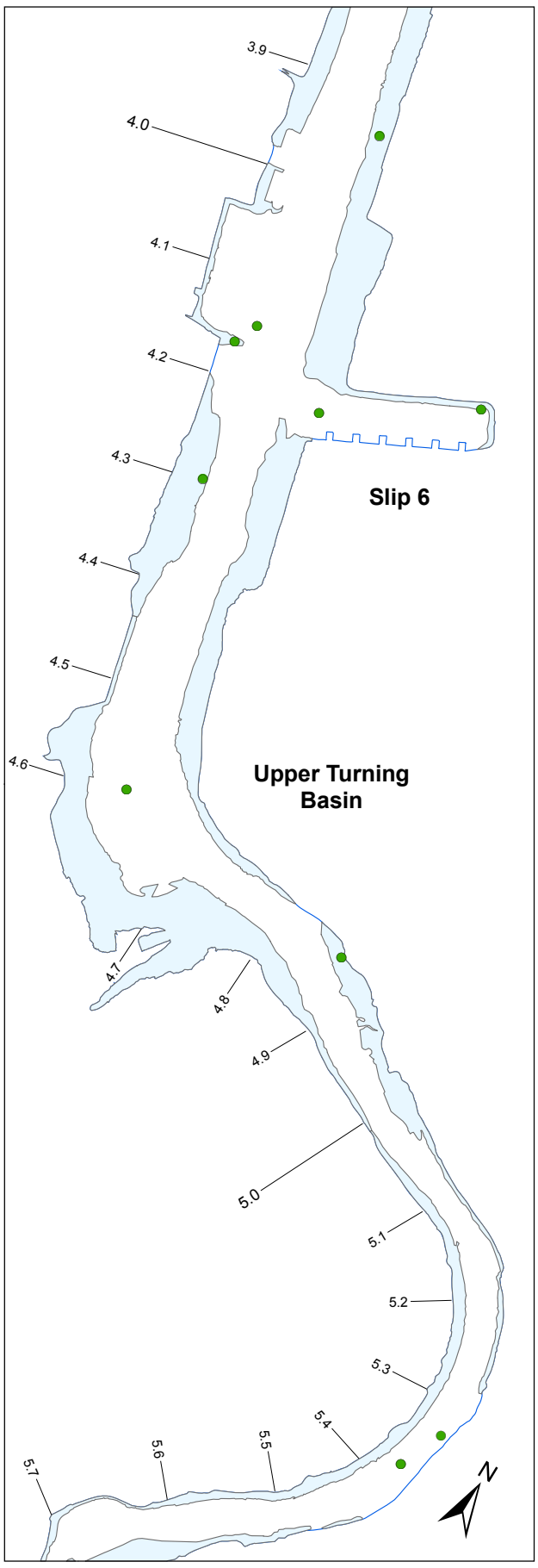
Scale is the same for each inset map

Map B.6-10. Risk-based concentration comparisons for cPAHs using the netfishing exposure scenario

Prepared by CEJ, 11/06/07 MAP # 2753; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6} (≤37 ng/kg dw)
- >10^{-6} and ≤10^{-5} (>37 and ≤370 ng/kg dw)
- >10^{-5} and ≤10^{-4} (>370 and ≤3,700 ng/kg dw)
- >10^{-4} and ≤10^{-3} (>3,700 and ≤37,000 ng/kg dw)
- Intertidal area
- River mile

* Each point represents the hypothetical excess cancer risk for a netfisher who works only at that specific location for 119 days/yr over a 44-yr period (i.e., the assumptions from the netfishing RME scenario). Each risk estimate is based on a weighted mean of the dioxin/furan TEQ at that specific location only (25% of total) and the LDW-wide EPC (610 ng/kg dw; 75% of the total). Mathematically, this is equivalent to assuming that netfishing occurs at a single location for 11 years and throughout the LDW for the other 33 years of the 44-yr exposure duration assumed for this scenario.

Wind Ward environmental LLC

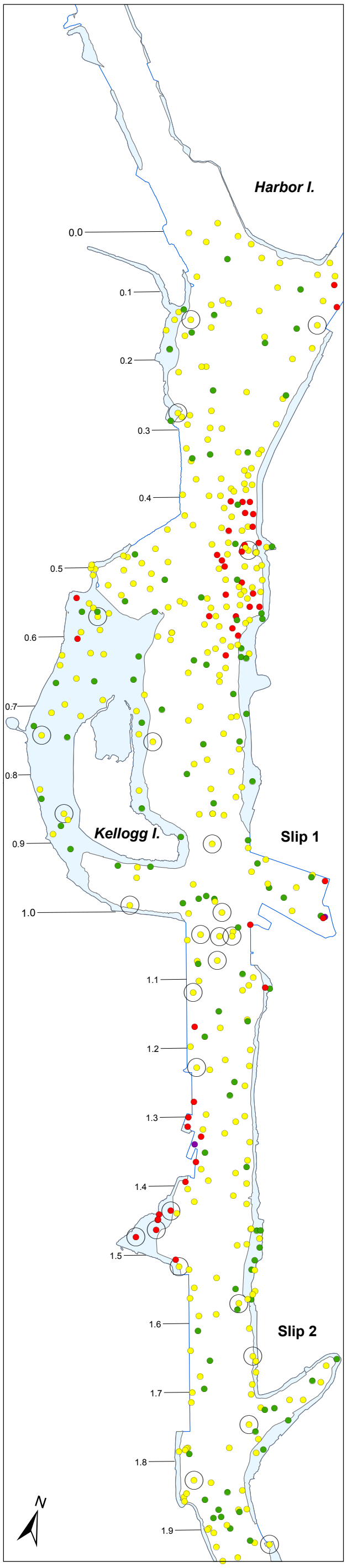
0 0.1 0.2 Miles

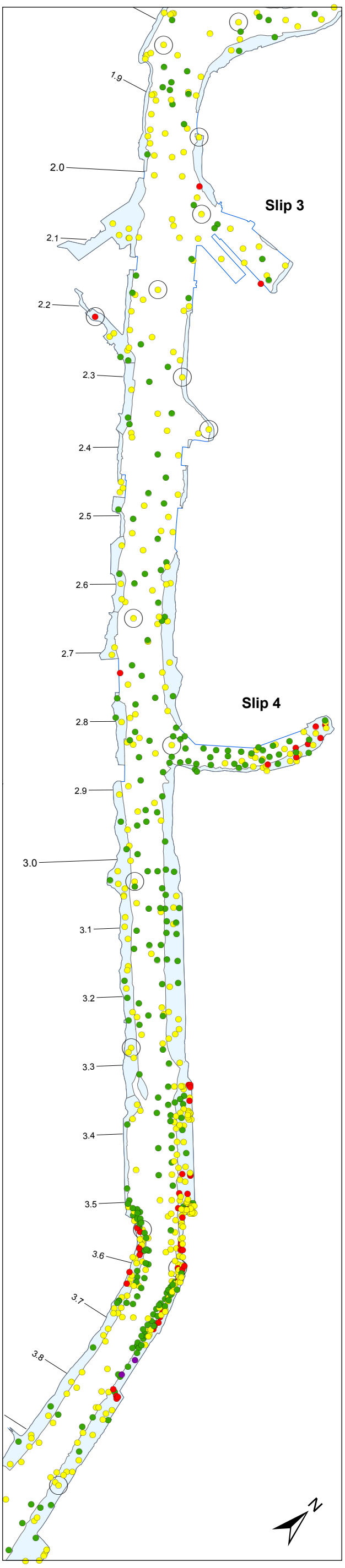
0 0.1 0.2 Kilometers

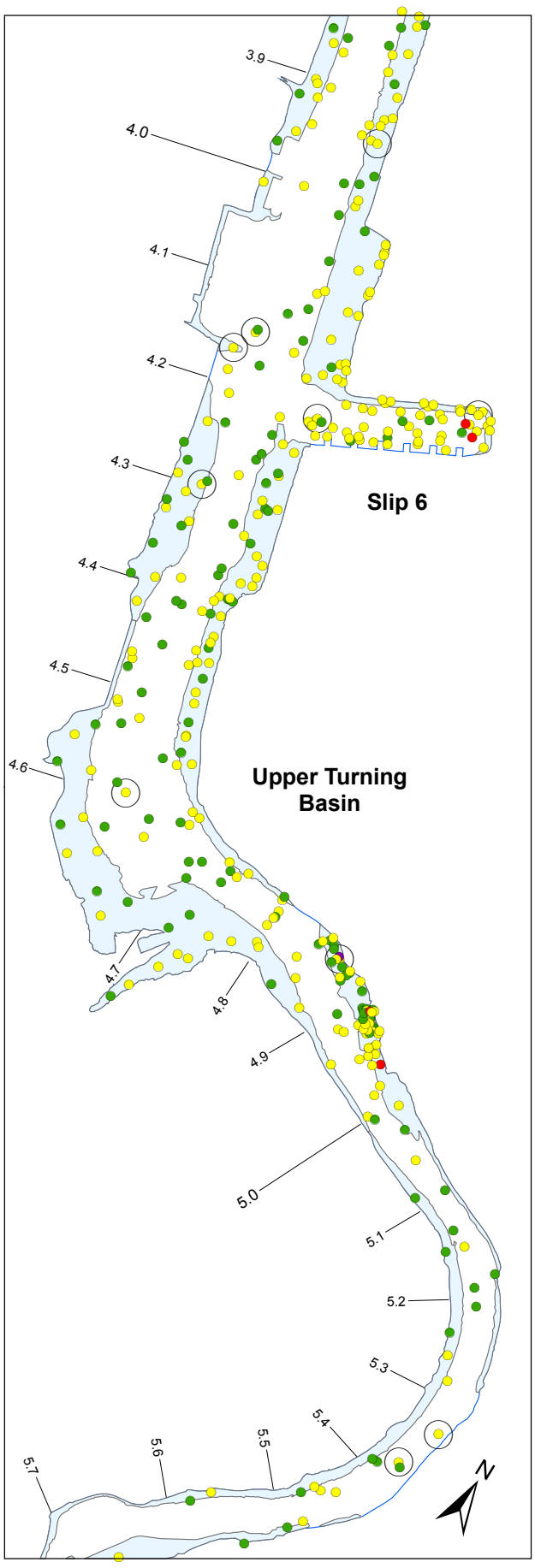
Scale is the same for each inset map

Map B.6-11. Risk-based concentration comparisons for dioxins/furans using the netfishing exposure scenario

Prepared by CEJ, 11/06/07 MAP # 3025; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Direct sediment contact risk*

- ≤10^{-6}
- >10^{-6} and ≤10^{-5}
- >10^{-5} and ≤10^{-4}
- >10^{-4} and ≤10^{-3}
- Dioxin/Furan sample location
- River mile

* Risks may be underestimated at some locations because not all chemicals were analyzed.

Each point represents the hypothetical excess cancer risk for a netfisher who works only at that specific location for 119 days/yr over a 44-yr period (i.e., the assumptions from the netfishing RME scenario). Each risk estimate reflects the cumulative results from Maps B.6-6 to B.6-9 based on weighted concentrations of total PCBs, arsenic, cPAHs, and dioxin/furan TEQs. Mathematically, this is equivalent to assuming that netfishing occurs at a single location for 11 years and throughout the LDW for the other 33 years of the 44-yr exposure duration assumed for this scenario.

Wind Ward environmental LLC

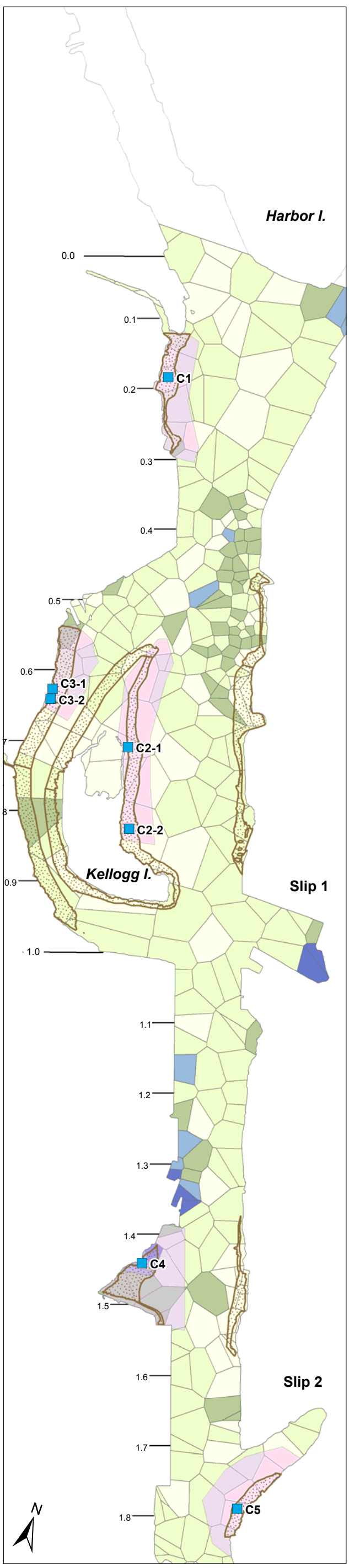
0 0.1 0.2 Miles

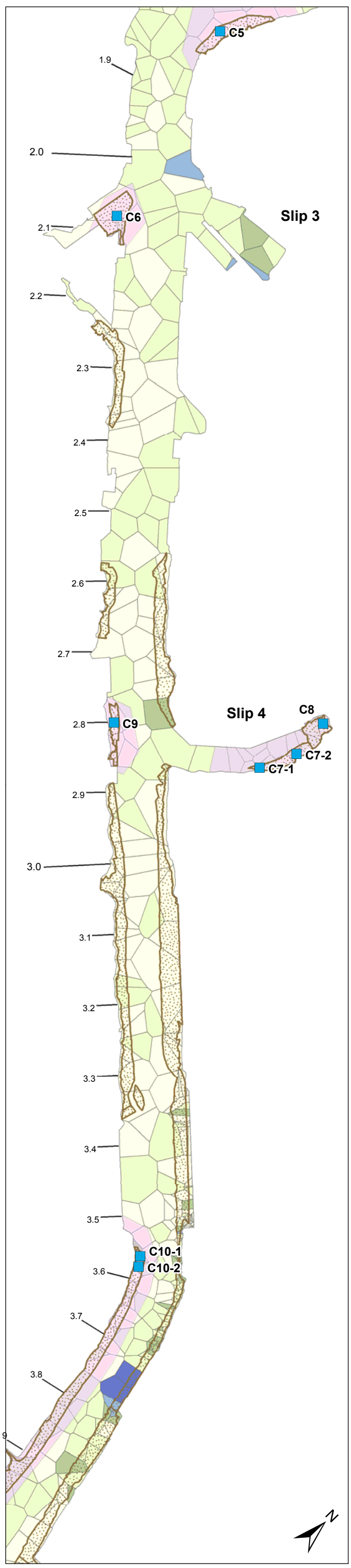
0 0.1 0.2 Kilometers

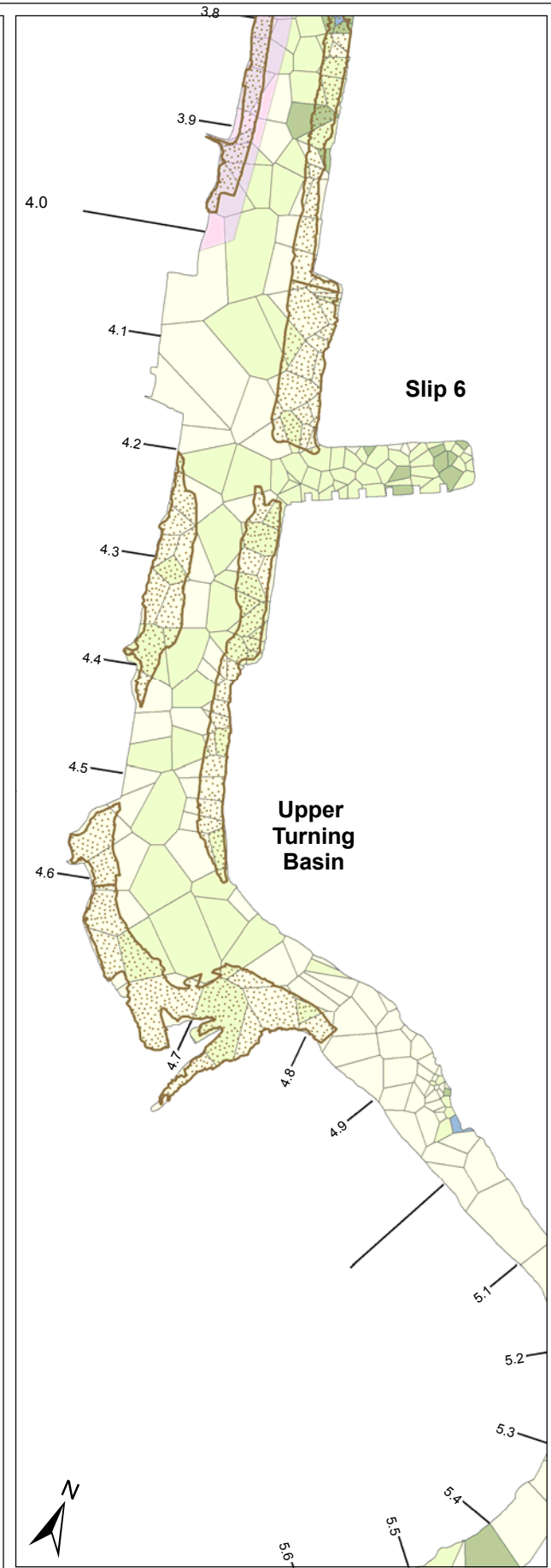
Scale is the same for each inset map

Map B.6-12. Combination of risks associated with total PCBs, arsenic, dioxins/furans, and cPAHs using the netfishing scenario

Prepared by CEJ, 11/06/07 MAP # 3026; W:\Projects\00-08-06_Duwamish_RI\data\gis\HHRA







Clam sampling location with location ID

Field surveyed clam habitat

Arsenic (mg/kg dw)

- 0 - 10
- 10 - 20
- 20 - 40
- 40 - 100
- > 100

Areas over which sediments were assumed to potentially serve as sources of particles for the clams

River mile

Location ID	Inorganic arsenic concentration in clams (mg/kg ww)	Arsenic concentration in sediment (mg/kg dw)
C1	0.132	3.53
C2-1	0.648	5.79
C3-1	0.885	4.63
C4	3.27	49
C5	0.795	4.72
C6	1.85	5.52
C7-2	2.11	6.8
C9	0.233	3.94

Arsenic in upstream sediments
Range: 3.2 to 10.9 mg/kg dw
UCL: 6.8 mg/kg dw

Wind Ward environmental LLC

0 0.1 0.2 Miles

0 0.1 0.2 Kilometers

Scale is the same for each inset map

Map B.6-13. Clam habitats, clam sampling locations, areas over which sediments were assumed to potentially serve as sources of particles for the clams, and the distribution of arsenic in surface sediments

Prepared by CEJ 11/08/07; Map 2611 (2465); data\gis\HHRA

ATTACHMENT 1. OCCURRENCE AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Table 1. Occurrence and selection of chemicals of potential concern for intertidal and subtidal sediment in the netfishing exposure scenario 2

Table 2. Occurrence and selection of chemicals of potential concern for intertidal sediment in the beach play and clamming exposure scenarios 15

Table 3. Occurrence and selection of chemicals of potential concern for tissue in the seafood consumption exposure scenario 27

Table 4. Chemicals analyzed in sediment but not in tissue samples evaluated in HHRA 40

Attachment 1. Occurrence and Selection of Chemicals of Potential Concern

This attachment to the LDW baseline HHRA presents the occurrence of chemicals in the LDW and documents the selection process for COPCs using the table format suggested in RAGS Part D (EPA 1998). The COPC selection process is summarized in Section B.3.3 and is based on a comparison of the maximum concentration of a chemical in a medium (or a sample reporting limit, for chemicals never detected) with an appropriate risk-based concentration (RBC).

The selection of COPCs for the netfishing scenario (Table 1) is based on comparisons of both intertidal and subtidal surface sediment chemical concentrations with soil RBCs for an industrial exposure scenario from EPA Region 9 (EPA 2004). The selection of COPCs for the beach play and clamming scenarios (Table 2) is based on comparisons of intertidal surface sediment chemical concentrations with soil RBCs for a residential exposure scenario from EPA Region 9 (EPA 2004). The selection of seafood ingestion COPCs (Table 3) is based on comparisons of chemical concentrations in composite tissue samples of English sole, starry flounder, perch, crab, clams, and mussels summarized in Table B.2-4 in the main document with tissue RBCs from EPA Region 3 (EPA 2005), as modified for site-specific conditions (see Section B.3.3.2 for a discussion of tissue RBC derivation). Chemicals that were analyzed in sediment (shown in Table 1), but not in tissue (tissue data are in Table 3) are listed in Table 4 and are screened for bioaccumulative potential. Sediment data in Tables 1 and 2 are summarized by location (i.e., multiple samples at a single location were averaged according to the project data rules summarized in Section B.2.2.1). Tissue data in Table 3 are summarized by sample. Non-chemical parameters (e.g., salinity, pH, clay, coarse sand, TOC, total solids) are not included in these tables.

Table 1. Occurrence and selection of chemicals of potential concern for intertidal and subtidal sediment in the netfishing exposure scenario

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
1,1,1,2-Tetrachloroethane	630-20-6	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	7,300 ca	no	bsl
1,1,1-Trichloroethane	71-55-6	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	120,000 nc	no	bsl
1,1,2,2-Tetrachloroethane	79-34-5	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	930 ca	no	bsl
1,1,2-Trichloroethane	79-00-5	0 / 42	nd	nd	ug/kg dw	1.4 - 1,100	1,100	not eval	1,600 ca	no	bsl
1,1,2-Trichlorotrifluoroethane	76-13-1	0 / 40	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	560,000 nc	no	bsl
1,1-Dichloroacetone	513-88-2	0 / 35	nd	nd	ug/kg dw	3.0 - 2,700	2,700	not eval	na	no	ntx
1,1-Dichloroethane	75-34-3	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	170,000 nc	no	bsl
1,1-Dichloroethene	75-35-4	0 / 42	nd	nd	ug/kg dw	1.4 - 1,100	1,100	not eval	41,000 nc	no	bsl
1,1-Dichloropropene	563-58-6	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	na	no	ntx
1,2,3,4,6,7,8-HpCDD	35822-46-9	41 / 43	41.4 J	73,700	ng/kg dw	0.99 - 1.1	74,000	not eval	na	no	ntx
1,2,3,4,6,7,8-HpCDF	67562-39-4	40 / 43	6.71	40,300	ng/kg dw	0.62 - 7.7	40,000	not eval	na	no	ntx
1,2,3,4,7,8,9-HpCDF	55673-89-7	27 / 43	0.421 J	3,720	ng/kg dw	0.77 - 4.2	3,700	not eval	na	no	ntx
1,2,3,4,7,8-HxCDD	39227-28-6	19 / 43	0.382 J	124	ng/kg dw	0.72 - 5.4	120	not eval	na	no	ntx
1,2,3,4,7,8-HxCDF	70648-26-9	30 / 43	0.694 J	2,530	ng/kg dw	0.29 - 4.2	2,500	not eval	na	no	ntx
1,2,3,6,7,8-HxCDD	57653-85-7	35 / 43	1.73 J	3,400	ng/kg dw	0.74 - 4.1	3,400	not eval	na	no	ntx
1,2,3,6,7,8-HxCDF	57117-44-9	19 / 43	0.335 J	365	ng/kg dw	0.22 - 4.3	370	not eval	na	no	ntx
1,2,3,7,8,9-HxCDD	19408-74-3	31 / 43	1.19 J	315	ng/kg dw	0.84 - 4.8	320	not eval	na	no	ntx
1,2,3,7,8,9-HxCDF	72918-21-9	18 / 43	0.0730 J	33.8 J	ng/kg dw	0.12 - 2.4	34	not eval	na	no	ntx
1,2,3,7,8-PeCDD	40321-76-4	19 / 43	0.284 J	57.1	ng/kg dw	0.53 - 4.1	57	not eval	na	no	ntx
1,2,3,7,8-PeCDF	57117-41-6	18 / 43	0.214 J	69.3	ng/kg dw	0.28 - 5.0	69	not eval	na	no	ntx
1,2,3-Trichlorobenzene	87-61-6	0 / 37	nd	nd	ug/kg dw	2.4 - 1,100	1,100	not eval	na	no	ntx
1,2,3-Trichloropropane	96-18-4	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	76 ca	no	ifd[c]
1,2,4-Trichlorobenzene	120-82-1	5 / 780	1.6 J	72 J	ug/kg dw	0.33 - 4,100	4,100	not eval	22,000 nc	no	bsl
1,2,4-Trimethylbenzene	95-63-6	1 / 37	0.54 J	0.54 J	ug/kg dw	1.5 - 530	530	not eval	17,000 nc	no	bsl
1,2-Dibromo-3-chloropropane	96-12-8	0 / 37	nd	nd	ug/kg dw	4.6 - 1,100	1,100	not eval	2,000 ca	no	bsl
1,2-Dibromoethane (EDB)	106-93-4	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	73 ca	no	ifd[d]
1,2-Dichlorobenzene	95-50-1	18 / 780	1.3 J	520 J	ug/kg dw	0.33 - 4,100	4,100	not eval	60,000 nc	no	bsl

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
1,2-Dichloroethane	107-06-2	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	600 ca	no	bsl
1,2-Dichloroethene (total)	540-59-0	0 / 2	nd	nd	ug/kg dw	23 - 24	24	not eval	na	no	ntx
1,2-Dichloropropane	78-87-5	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	740 ca	no	bsl
1,2-Diphenylhydrazine	122-66-7	0 / 111	nd	nd	ug/kg dw	13 - 880	880	not eval	2,200 ca	no	bsl
1,3,5-Trimethylbenzene	108-67-8	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	7,000 nc	no	bsl
1,3-Dichlorobenzene	541-73-1	3 / 769	2.0 J	11 J	ug/kg dw	0.33 - 4,100	4,100	not eval	60,000 nc	no	bsl
1,3-Dichloropropane	142-28-9	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	36,000 nc	no	bsl
1,4-Dichlorobenzene	106-46-7	35 / 780	0.74 J	1,600 J	ug/kg dw	0.16 - 4,100	4,100	not eval	7,900 ca	no	bsl
1-Chlorobutane	109-69-3	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	48,000 nc	no	bsl
1-Methylnaphthalene	90-12-0	20 / 20	1.9 J	32	ug/kg dw	na	32	not eval	na	no	ntx
2,2-Dichloropropane	594-20-7	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	na	no	ntx
2,3,4,6,7,8-HxCDF	60851-34-5	19 / 43	0.307 J	302 J	ng/kg dw	0.29 - 2.5	300	not eval	na	no	ntx
2,3,4,7,8-PeCDF	57117-31-4	19 / 43	0.392 J	230	ng/kg dw	0.44 - 5.4	230	not eval	na	no	ntx
2,3,7,8-TCDD	1746-01-6	17 / 43	0.0890 J	30.6	ng/kg dw	0.27 - 1.1	31	not eval	16 ca	no	bsl
2,3,7,8-TCDF	51207-31-9	34 / 43	0.426 J	397	ng/kg dw	0.18 - 1.4	400	not eval	na	no	ntx
2,4,5-Trichlorophenol	95-95-4	0 / 735	nd	nd	ug/kg dw	8.6 - 10,000	10,000	not eval	6,200,000 nc	no	bsl
2,4,6-Trichlorophenol	88-06-2	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	6,200 nc	no	bsl
2,4'-DDD	53-19-0	5 / 93	1.6 J	10 J	ug/kg dw	0.97 - 34	34	not eval	na	no	ntx
2,4'-DDE	3424-82-6	2 / 93	2.8 J	11	ug/kg dw	0.97 - 34	34	not eval	na	no	ntx
2,4'-DDT	789-02-6	29 / 93	0.24 J	11	ug/kg dw	1.9 - 460	460	not eval	na	no	ntx
2,4-Dichlorophenol	120-83-2	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	180,000 nc	no	bsl
2,4-Dimethylphenol	105-67-9	1 / 773	290 J	290 J	ug/kg dw	6.3 - 4,100	4,100	not eval	1,200,000 nc	no	bsl
2,4-Dinitrophenol	51-28-5	0 / 721	nd	nd	ug/kg dw	16 - 10,000	10,000	not eval	120,000 nc	no	bsl
2,4-Dinitrotoluene	121-14-2	0 / 735	nd	nd	ug/kg dw	3.8 - 4,100	4,100	not eval	120,000 nc	no	bsl
2,6-Dinitrotoluene	606-20-2	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	62,000 nc	no	bsl
2-Chloroethyl vinyl ether	110-75-8	0 / 3	nd	nd	ug/kg dw	7.0 - 12	12	not eval	na	no	ntx
2-Chloronaphthalene	91-58-7	0 / 745	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	2,300,000 nc	no	bsl
2-Chlorophenol	95-57-8	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	24,000 nc	no	bsl
2-Chlorotoluene	95-49-8	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	56,000 nc	no	bsl
2-Hexanone	591-78-6	0 / 42	nd	nd	ug/kg dw	3.0 - 2,100	2,100	not eval	na	no	ntx

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
2-Methylnaphthalene	91-57-6	139 / 782	1.0 J	3,300	ug/kg dw	1.0 - 4,100	4,100	not eval	na[e]	no	ntx
2-Methylphenol	95-48-7	3 / 785	21	58 J	ug/kg dw	6.3 - 4,100	4,100	not eval	3,100,000 nc	no	bsl
2-Nitroaniline	88-74-4	0 / 721	nd	nd	ug/kg dw	16 - 10,000	10,000	not eval	180,000 nc	no	bsl
2-Nitrophenol	88-75-5	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	na	no	ntx
2-Nitropropane	79-46-9	0 / 37	nd	nd	ug/kg dw	7.6 - 2,700	2,700	not eval	na	no	ntx
3,3'-Dichlorobenzidine	91-94-1	0 / 692	nd	nd	ug/kg dw	31 - 4,100	4,100	not eval	3,800 ca	no	ifd[f]
3-Nitroaniline	99-09-2	0 / 709	nd	nd	ug/kg dw	18 - 10,000	10,000	not eval	82,000 ca	no	bsl
4,4'-DDD	72-54-8	67 / 197	0.29 J	840	ug/kg dw	0.75 - 540	840	not eval	10,000 ca	no	sum
4,4'-DDE	72-55-9	30 / 197	0.28 J	370 J	ug/kg dw	0.81 - 800	800	not eval	7,000 ca	no	sum
4,4'-DDT	50-29-3	41 / 197	0.48 J	1,700	ug/kg dw	0.81 - 56	1,700	not eval	7,000 ca	no	sum
4,6-Dinitro-o-cresol	534-52-1	0 / 721	nd	nd	ug/kg dw	16 - 10,000	10,000	not eval	6,200 nc	no	ifd[g]
4-Bromophenyl phenyl ether	101-55-3	1 / 735	31	31	ug/kg dw	8.6 - 4,100	4,100	not eval	na	no	ntx
4-Chloro-3-methylphenol	59-50-7	1 / 721	6.4 J	6.4 J	ug/kg dw	8.6 - 4,100	4,100	not eval	na	no	ntx
4-Chloroaniline	106-47-8	0 / 686	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	250,000 nc	no	bsl
4-Chlorophenyl phenyl ether	7005-72-3	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	na	no	ntx
4-Chlorotoluene	106-43-4	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	na	no	ntx
4-Methylphenol	106-44-5	78 / 795	4.8 J	4,600 J	ug/kg dw	8.6 - 4,100	4,600	not eval	310,000 nc	no	bsl
4-Nitroaniline	100-01-6	0 / 709	nd	nd	ug/kg dw	16 - 10,000	10,000	not eval	82,000 ca	no	bsl
4-Nitrophenol	100-02-7	0 / 721	nd	nd	ug/kg dw	16 - 10,000	10,000	not eval	na	no	ntx
Acenaphthene	83-32-9	301 / 792	1.0 J	5,200	ug/kg dw	1.8 - 4,100	5,200	not eval	2,900,000 nc	no	bsl
Acenaphthylene	208-96-8	121 / 782	1.3 J	240	ug/kg dw	1.8 - 4,100	4,100	not eval	2,900,000 nc[h]	no	bsl
Acetone	67-64-1	3 / 42	110 J	1,000 J	ug/kg dw	11 - 21,000	21,000	not eval	5,400,000 nc	no	bsl
Acid volatile sulfides	na	41 / 52	88 J	6,100 J	mg/kg dw	48 - 89	6,100	not eval	na	no	ntx
Aldrin	309-00-2	4 / 197	0.014 J	1.6	ug/kg dw	0.37 - 56	56	not eval	100 ca	no	bsl
Allyl chloride	107-05-1	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	18,000 nc	no	bsl
alpha-BHC	319-84-6	3 / 197	0.14 J	1.8 J	ug/kg dw	0.37 - 56	56	not eval	360 ca	no	bsl
alpha-Chlordane	5103-71-9	13 / 153	0.10 J	36	ug/kg dw	0.72 - 43	43	not eval	6,500 ca[i]	no	bsl
alpha-Endosulfan	959-98-8	10 / 151	0.18 J	71 J	ug/kg dw	0.40 - 100	100	not eval	370,000 nc[J]	no	bsl
Aluminum	7429-90-5	455 / 455	2,800	110,000	mg/kg dw	na	110,000	12,000/21,000	10,000 nc	yes	asl
Ammonia	7664-41-7	14 / 14	5.40	20.3	mg/kg dw	na	20.3	not eval	na	no	ntx

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Ammonia (total as nitrogen)	7664-41-7	156 / 160	0.18	39.1	mg-N/kg	0.10 - 0.12	39.1	not eval	na	no	ntx
Aniline	62-53-3	1 / 250	13 J	13 J	ug/kg dw	18 - 290	290	not eval	300,000 ca	no	bsl
Anthracene	120-12-7	553 / 792	2.0	10,000	ug/kg dw	5 - 2,000	10,000	not eval	10,000,000 nc	no	bsl
Antimony	7440-36-0	139 / 552	0.09 J	122 J	mg/kg dw	0.2 - 31	122	0.23 / 0.44	41 nc	yes	asl
Aroclor-1016	12674-11-2	0 / 983	nd	nd	ug/kg dw	0.87 - 3,400	3,400	not eval	21,000 ca	no	sum, teq
Aroclor-1221	11104-28-2	0 / 857	nd	nd	ug/kg dw	1.9 - 5,500	5,500	not eval	na	no	ntx
Aroclor-1232	11141-16-5	0 / 857	nd	nd	ug/kg dw	0.87 - 3,400	3,400	not eval	na	no	ntx
Aroclor-1242	53469-21-9	103 / 984	7.8 J	2,700	ug/kg dw	0.87 - 6,100	6,100	not eval	na	no	ntx
Aroclor-1248	12672-29-6	205 / 993	13	220,000	ug/kg dw	0.87 - 4,300	220,000	not eval	na	no	ntx
Aroclor-1254	11097-69-1	779 / 985	2.2	110,000	ug/kg dw	1.3 - 4,300	110,000	not eval	740 ca	no	sum, teq
Aroclor-1254/1260	na	8 / 8	37	800	ug/kg dw	na	800	not eval	na	no	ntx
Aroclor-1260	11096-82-5	784 / 985	1.2 J	38,000	ug/kg dw	2.5 - 15,000	38,000	not eval	na	no	ntx
Aroclor-1262	37324-23-5	2 / 12	270	840	ug/kg dw	3.3 - 20	840	not eval	na	no	ntx
Aroclor-1268	11100-14-4	1 / 11	460 J	460 J	ug/kg dw	3.3 - 20	460	not eval	na	no	ntx
Arsenic	7440-38-2	754 / 816	1.2	1,100	mg/kg dw	3 - 31	1,100	5.03 / 10.4	1.6 ca	yes	asl
Barium	7440-39-3	418 / 418	9.40	7,400	mg/kg dw	na	7,400	24 / 55.5	6,700 nc	yes	asl
Benzaldehyde	100-52-7	6 / 10	120 J	380	ug/kg dw	170 - 170	380	not eval	6,200,000 nc	no	bsl
Benzene	71-43-2	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	1,400 ca	no	bsl
Benzidine	92-87-5	0 / 7	nd	nd	ug/kg dw	310 - 1,700	1,700	not eval	7.5 ca	yes	asl
Benzo(a)anthracene	56-55-3	719 / 792	3.6 J	8,400	ug/kg dw	6.4 - 200	8,400	not eval	2,100 ca	no	teq
Benzo(a)pyrene	50-32-8	718 / 786	5.8 J	7,900	ug/kg dw	6.4 - 350	7,900	not eval	210 ca	no	teq
Benzo(b)fluoranthene	205-99-2	725 / 786	4.6	8,200	ug/kg dw	6.4 - 450	8,200	not eval	2,100 ca	no	teq
Benzo(e)pyrene	192-97-2	20 / 20	16	1,300	ug/kg dw	na	1,300	not eval	na	no	ntx
Benzo(g,h,i)perylene	191-24-2	649 / 787	6.1	3,800	ug/kg dw	10 - 4,100	4,100	not eval	na	no	ntx
Benzo(k)fluoranthene	207-08-9	698 / 786	8.4	8,800	ug/kg dw	3.8 - 450	8,800	not eval	21,000 ca	no	teq
Benzofluoranthenes (total)	56832-73-6	727 / 786	4.6	17,000	ug/kg dw	19 - 450	17,000	not eval	na	no	ntx
Benzoic acid	65-85-0	69 / 783	54 J	4,500	ug/kg dw	13 - 3,000	4,500	not eval	10,000,000 nc	no	bsl
Benzyl alcohol	100-51-6	14 / 773	8.2 J	670	ug/kg dw	7.5 - 690	690	not eval	10,000,000 nc	no	bsl
Beryllium	7440-41-7	453 / 465	0.10	0.730	mg/kg dw	0.10 - 0.70	0.73	not eval	1,900 ca	no	bsl
beta-BHC	319-85-7	4 / 197	0.087 J	13	ug/kg dw	0.37 - 56	56	not eval	1,300 ca	no	bsl

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
beta-Endosulfan	33213-65-9	4 / 153	0.47 J	10 J	ug/kg dw	0.81 - 200	200	not eval	370,000 nc[k]	no	bsl
Biphenyl	92-52-4	20 / 20	0.86 J	33	ug/kg dw	na	33	not eval	2,300,000 nc	no	bsl
bis(2-chloroethoxy)methane	111-91-1	1 / 735	40	40	ug/kg dw	8.6 - 4,100	4,100	not eval	na	no	ntx
bis(2-chloroethyl)ether	111-44-4	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	580 ca	no	ifd[l]
bis(2-chloroisopropyl)ether	108-60-1	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	7,400 ca	no	bsl
Bis(2-ethylhexyl)phthalate	117-81-7	636 / 796	5.4	14,000	ug/kg dw	15 - 1,500	14,000	not eval	120,000 ca	no	bsl
Bromobenzene	108-86-1	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	9,200 nc	no	bsl
Bromochloromethane	74-97-5	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	na	no	ntx
Bromodichloromethane	75-27-4	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	1,800 ca	no	bsl
Bromoform	75-25-2	0 / 42	nd	nd	ug/kg dw	1.4 - 2,700	2,700	not eval	220,000 ca	no	bsl
Bromomethane	74-83-9	0 / 42	nd	nd	ug/kg dw	2.8 - 5,300	5,300	not eval	1,300 nc	no	ifd[m]
Butyl benzyl phthalate	85-68-7	390 / 786	2.0	7,100	ug/kg dw	1.8 - 4,100	7,100	not eval	10,000,000 nc	no	bsl
Butyltin (total)	na	29 / 37	70.0	600	ug/kg dw	15 - 24	600	not eval	na	no	ntx
C1-Chrysenes	3001965	20 / 20	12	2,100	ug/kg dw	na	2,100	not eval	na	no	ntx
C1-Dibenzothiophenes	3001957	14 / 20	4.6 J	59	ug/kg dw	5.0 - 5.5	59	not eval	na	no	ntx
C1-Fluoranthene/Pyrene	3001964	19 / 20	18	4,900	ug/kg dw	4.9 - 4.9	4,900	not eval	na	no	ntx
C1-Fluorenes	3001954	10 / 20	4.5 J	150	ug/kg dw	5.0 - 7.8	150	not eval	na	no	ntx
C1-Phenanthrenes/anthracenes	3001960	20 / 20	8.0	1,700	ug/kg dw	na	1,700	not eval	na	no	ntx
C2-Chrysenes	3001966	20 / 20	8.1	680	ug/kg dw	na	680	not eval	na	no	ntx
C2-Dibenzothiophenes	3001958	15 / 20	4.3 J	190	ug/kg dw	5.0 - 5.4	190	not eval	na	no	ntx
C2-Fluorenes	3001955	16 / 20	3.4 J	250	ug/kg dw	5.0 - 5.4	250	not eval	na	no	ntx
C2-Naphthalenes	3001951	20 / 20	6.2	100	ug/kg dw	na	100	not eval	na	no	ntx
C2-Phenanthrenes/anthracenes	3001961	20 / 20	8.5	840	ug/kg dw	na	840	not eval	na	no	ntx
C3-Chrysenes	3001967	20 / 20	7.3	370	ug/kg dw	na	370	not eval	na	no	ntx
C3-Dibenzothiophenes	3001959	16 / 20	7.6	150	ug/kg dw	4.9 - 5.0	150	not eval	na	no	ntx
C3-Fluorenes	3001956	18 / 20	3.9 J	220	ug/kg dw	5.0 - 5.0	220	not eval	na	no	ntx
C3-Naphthalenes	3001952	20 / 20	4.4 J	310	ug/kg dw	na	310	not eval	na	no	ntx
C3-Phenanthrenes/anthracenes	3001962	20 / 20	6.8	420	ug/kg dw	na	420	not eval	na	no	ntx
C4-Chrysenes	3001968	17 / 20	7.7	130	ug/kg dw	4.9 - 5.0	130	not eval	na	no	ntx
C4-Naphthalenes	3001953	20 / 20	6.0	250	ug/kg dw	na	250	not eval	na	no	ntx

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
C4-Phenanthrenes/anthracenes	3001963	20 / 20	5.6	180	ug/kg dw	na	180	not eval	na	no	ntx
Cadmium	7440-43-9	565 / 799	0.030 J	120	mg/kg dw	0.040 - 2.5	120	0.36 / 1.12	45 nc	yes	asl
Caffeine	58-08-2	1 / 33	1,500 J	1,500 J	ug/kg dw	6.8 - 4,100	4,100	not eval	na	no	ntx
Calcium	7440-70-2	418 / 418	1,800	49,000	mg/kg dw	na	49,000	not eval	na	no	ntx
Caprolactam	105-60-2	1 / 10	27 J	27 J	ug/kg dw	830 - 1,500	1,500	not eval	10,000,000 nc	no	bsl
Carbazole	86-74-8	385 / 745	3.2 J	4,200	ug/kg dw	8.8 - 4,100	4,200	not eval	86,000 ca	no	bsl
Carbon disulfide	75-15-0	13 / 42	0.84 J	4.0 J	ug/kg dw	1.4 - 1,100	1,100	not eval	72,000 nc	no	bsl
Carbon tetrachloride	56-23-5	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	550 ca	no	bsl
Carcinogenic PAHs - Mammal - Half DL	na	749 / 792	9.1 J	11,000	ug/kg dw	na	11,000	not eval	210 ca[n]	yes	asl
Chlordane	57-74-9	5 / 44	25	62	ug/kg dw	7.7 - 330	330	not eval	na	no	ntx
Chloroacetonitrile	107-14-2	0 / 2	nd	nd	ug/kg dw	7.6 - 24	24	not eval	na	no	ntx
Chlorobenzene	108-90-7	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	53,000 nc	no	bsl
Chloroethane	75-00-3	0 / 42	nd	nd	ug/kg dw	2.8 - 11,000	11,000	not eval	6,500 ca	no	ifd[o]
Chloroform	67-66-3	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	470 ca	no	ifd[p]
Chloromethane	74-87-3	0 / 42	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	16,000 nc	no	bsl
Chromium	7440-47-3	813 / 813	4.8	1,100 J	mg/kg dw	na	1,100	not eval	6.5 ca[q]	yes	asl
Chromium VI	18540-29-9	1 / 7	14 J	14 J	mg/kg dw	1.1 - 10	14	not eval	64 ca	no	bsl
Chrysene	218-01-9	741 / 792	12	7,700	ug/kg dw	5 - 170	7,700	not eval	210,000 ca	no	bsl
cis-1,2-Dichloroethene	156-59-2	0 / 40	nd	nd	ug/kg dw	1.4 - 530	530	not eval	15,000 nc	no	bsl
cis-1,3-Dichloropropene	10061-01-5	0 / 42	nd	nd	ug/kg dw	1.4 - 560	560	not eval	na	no	ntx
cis-Nonachlor	5103-73-1	0 / 58	nd	nd	ug/kg dw	1.9 - 330	330	not eval	na	no	ntx
Cobalt	7440-48-4	556 / 556	2.82	140	mg/kg dw	na	140	not eval	1,900 ca	no	bsl
Copper	7440-50-8	816 / 816	5	12,000 J	mg/kg dw	na	12,000	21.3 / 50.8	4,100 nc	yes	asl
Coprostanol	360-68-9	43 / 109	260 J	50,000 J	ug/kg dw	18 - 4,100	50,000	not eval	na	no	ntx
Cyanide	57-12-5	0 / 6	nd	nd	mg/kg dw	0.37 - 0.53	0.53	not eval	1,200 nc	no	ntx
DDTs (total-calc'd)	na	78 / 197	0.72 J	2,900 J	ug/kg dw	0.81 - 800	2,900	not eval	7,000 ca[r]	no	bsl
delta-BHC	319-86-8	3 / 158	0.081 J	11	ug/kg dw	0.37 - 56	56	not eval	na	no	ntx
Dibenzo(a,h)anthracene	53-70-3	400 / 792	1.6 J	1,500	ug/kg dw	1.0 - 4,100	4,100	not eval	210 ca	no	teq
Dibenzofuran	132-64-9	246 / 791	1.0 J	4,200	ug/kg dw	1.7 - 4,100	4,200	not eval	160,000 nc	no	bsl
Dibenzothiophene	132-65-0	20 / 20	0.81 J	150	ug/kg dw	na	150	not eval	na	no	ntx

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Dibromochloromethane	124-48-1	0 / 42	nd	nd	ug/kg dw	1.4 - 2,700	2,700	not eval	2,600 ca	no	ifd[s]
Dibromomethane	74-95-3	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	23,000 nc	no	bsl
Dibutyltin as ion	1002-53-5	106 / 147	0.39 J	560	ug/kg dw	1.0 - 49	560	not eval	na	no	ntx
Dichlorodifluoromethane	75-71-8	0 / 7	nd	nd	ug/kg dw	1.5 - 3.3	3.3	not eval	31,000 nc	no	bsl
Dichloromethane	75-09-2	1 / 42	1,600	1,600	ug/kg dw	2.8 - 21	1,600	not eval	21,000 ca	no	bsl
Dieldrin	60-57-1	9 / 197	0.099 J	280	ug/kg dw	0.81 - 91	280	not eval	110 ca	yes	asl
Diethyl ether	60-29-7	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	180,000 nc	no	bsl
Diethyl phthalate	84-66-2	42 / 796	2.0 J	150	ug/kg dw	1.8 - 4,100	4,100	not eval	10,000,000 nc	no	bsl
Dimethyl phthalate	131-11-3	136 / 786	2.0 J	1,400 J	ug/kg dw	1.8 - 4,100	4,100	not eval	10,000,000 nc	no	bsl
Di-n-butyl phthalate	84-74-2	181 / 786	3.0 J	3,800	ug/kg dw	1.8 - 4,100	4,100	not eval	6,200,000 nc	no	bsl
Di-n-octyl phthalate	117-84-0	48 / 796	1.8	1,000	ug/kg dw	1.8 - 4,100	4,100	not eval	2,500,000 nc	no	bsl
Dioxin/furan TEQ - Mammal - Half DL	na	43 / 43	1.10 J	2,100 J	ng/kg dw	na	2,100	not eval	16 ca[t]	yes	asl
Endosulfan	115-29-7	1 / 46	0.11 J	0.11 J	ug/kg dw	0.81 - 56	56	not eval	370,000 nc	no	bsl
Endosulfan sulfate	1031-07-8	3 / 195	0.63 J	25	ug/kg dw	0.75 - 200	200	not eval	na	no	ntx
Endrin	72-20-8	4 / 197	0.99 J	9.1	ug/kg dw	0.75 - 200	200	not eval	18,000 nc	no	bsl
Endrin aldehyde	7421-93-4	6 / 186	0.28 J	130	ug/kg dw	0.75 - 250	250	not eval	18,000 nc[u]	no	bsl
Endrin ketone	53494-70-5	7 / 141	0.83 J	110 J	ug/kg dw	0.75 - 200	200	not eval	18,000 nc[v]	no	bsl
Ethyl methacrylate	97-63-2	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	14,000 nc	no	bsl
Ethylbenzene	100-41-4	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	40,000 nc	no	bsl
Fluoranthene	206-44-0	762 / 792	18	24,000	ug/kg dw	10 - 340	24,000	not eval	2,200,000 nc	no	bsl
Fluorene	86-73-7	373 / 792	1.4 J	6,800	ug/kg dw	1.8 - 2,000	6,800	not eval	2,600,000 nc	no	bsl
gamma-BHC	58-89-9	11 / 197	0.050 J	6.7 J	ug/kg dw	0.37 - 56	56	not eval	1,700 ca	no	bsl
gamma-Chlordane	5103-74-2	25 / 153	0.20 J	200	ug/kg dw	0.72 - 96	200	not eval	6,500 ca[w]	no	bsl
Gasoline	8006-61-9	2 / 10	130	260	mg/kg dw	10 - 10	260	not eval	na	no	ntx
Heptachlor	76-44-8	8 / 197	0.12 J	5.2	ug/kg dw	0.43 - 70	70	not eval	380 ca	no	bsl
Heptachlor epoxide	1024-57-3	5 / 197	0.47 J	4.9 J	ug/kg dw	0.37 - 510	510	not eval	190 ca	no	ifd[x]
Hexachlorobenzene	118-74-1	46 / 783	0.4 J	95 J	ug/kg dw	0.11 - 4,100	4,100	not eval	1,100 ca	no	ifd[y]
Hexachlorobutadiene	87-68-3	0 / 782	nd	nd	ug/kg dw	0.94 - 4,100	4,100	not eval	22,000 ca	no	bsl
Hexachlorocyclopentadiene	77-47-4	1 / 682	100 J	100 J	ug/kg dw	32 - 4,100	4,100	not eval	370,000 nc	no	bsl
Hexachloroethane	67-72-1	0 / 763	nd	nd	ug/kg dw	1.5 - 4,100	4,100	not eval	120,000 ca	no	bsl

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Indeno(1,2,3-cd)pyrene	193-39-5	694 / 787	6.5	4,300	ug/kg dw	6.4 - 4,100	4,300	not eval	2,100 ca	no	teq
Iodomethane	74-88-4	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	na	no	ntx
Iron	7439-89-6	453 / 453	8,100	160,000	mg/kg dw	na	160,000	17,500/28,700	10,000 nc	yes	asl
Isophorone	78-59-1	2 / 745	26	430	ug/kg dw	8.6 - 4,100	4,100	not eval	510,000 ca	no	bsl
Isopropylbenzene	98-82-8	0 / 37	nd	nd	ug/kg dw	2.3 - 530	530	not eval	200,000 nc	no	bsl
Lead	7439-92-1	816 / 816	2	23,000	mg/kg dw	na	23,000	15 / 45	80 nc	yes	asl
Lube oils	na	0 / 7	nd	nd	mg/kg dw	10 - 10	10	not eval	na	no	ntx
Magnesium	7439-95-4	428 / 428	2,000	17,000	mg/kg dw	na	17,000	not eval	na	no	ntx
Manganese	7439-96-5	450 / 450	78.0	3,300	mg/kg dw	na	3,300	279 / 1,010	1,900 nc	yes	asl
Mercury	7439-97-6	717 / 833	0.021	4.6 J	mg/kg dw	0.020 - 0.10	4.6	0.0981 / 0.327	31 nc	no	bsl
Methacrylonitrile	126-98-7	0 / 37	nd	nd	ug/kg dw	4.6 - 1,100	1,100	not eval	840 nc	no	ifd[z]
Methoxychlor	72-43-5	11 / 197	0.34 J	99	ug/kg dw	0.97 - 330	330	not eval	310,000 nc	no	bsl
Methyl acrylate	96-33-3	0 / 37	nd	nd	ug/kg dw	2.3 - 530	530	not eval	23,000 nc	no	bsl
Methyl ethyl ketone	78-93-3	15 / 42	5.3	35	ug/kg dw	3.0 - 1,100	1,100	not eval	11,000,000 nc	no	bsl
Methyl isobutyl ketone	108-10-1	0 / 42	nd	nd	ug/kg dw	3.0 - 1,100	1,100	not eval	4,700,000 nc	no	bsl
Methyl methacrylate	80-62-6	0 / 37	nd	nd	ug/kg dw	2.3 - 530	530	not eval	270,000 nc	no	bsl
Methylmercury	22967-92-6	20 / 20	0.040 J	5.6	ug/kg dw	na	5.60	not eval	6,200 nc	no	bsl
Mirex	2385-85-5	3 / 93	0.29 J	1.0 J	ug/kg dw	0.97 - 34	34	not eval	960 ca	no	bsl
Moisture	na	5 / 5	34.4	48.8	% ww	na	48.8	not eval	na	no	ntx
Molybdenum	7439-98-7	199 / 257	0.390 J	75	mg/kg dw	0.6 - 5.3	75.0	not eval	510 nc	no	bsl
Monobutyltin as ion	78763-54-9	89 / 117	0.12 J	120	ug/kg dw	1.0 - 85	120	not eval	na	no	ntx
Naphthalene	91-20-3	148 / 782	3.0 J	5,300	ug/kg dw	1.0 - 4,100	5,300	not eval	19,000 nc	no	bsl
n-Butylbenzene	104-51-8	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	24,000 nc	no	bsl
Nickel	7440-02-0	775 / 775	5	910	mg/kg dw	na	910	26.8 / 41.7	2,000 nc	no	bsl
Nitrobenzene	98-95-3	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	10,000 nc	no	bsl
n-Nitrosodimethylamine	62-75-9	0 / 294	nd	nd	ug/kg dw	31 - 1,800	1,800	not eval	34 ca	yes	asl
n-Nitroso-di-n-propylamine	621-64-7	0 / 735	nd	nd	ug/kg dw	8.6 - 4,100	4,100	not eval	250 ca	no	ifd[aa]
n-Nitrosodiphenylamine	86-30-6	23 / 782	6.5	230	ug/kg dw	1.8 - 4,100	4,100	not eval	350,000 ca	no	bsl
n-Propylbenzene	103-65-1	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	24,000 nc	no	bsl
OCDD	3268-87-9	43 / 43	7.8 J	241,000	ng/kg dw	na	240,000	not eval	na	no	ntx

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
OCDF	39001-02-0	42 / 43	12.5	93,700	ng/kg dw	0.74 - 0.74	94,000	not eval	na	no	ntx
Oxychlordane	27304138	0 / 58	nd	nd	ug/kg dw	1.9 - 34	34	not eval	na	no	ntx
PCB TEQ - Mammal - Half DL	na	48 / 48	0.0908 J	1,380	ng/kg dw	na	1,380	not eval	16 ca[ab]	yes	asl
PCB-018	37680-65-2	73 / 237	1,000 J	170,000 J	ng/kg dw	1,000 - 24,000	170,000	not eval	na	no	ntx
PCB-028	7012-37-5	138 / 251	1,000 J	160,000 J	ng/kg dw	1,000 - 8,000	160,000	not eval	na	no	ntx
PCB-044	41464-39-5	165 / 251	1,000 J	190,000 J	ng/kg dw	1,000 - 2,000	190,000	not eval	na	no	ntx
PCB-055	74338-24-2	182 / 251	1,000 J	890,000 J	ng/kg dw	1,000 - 13,000	890,000	not eval	na	no	ntx
PCB-066	32598-10-0	217 / 299	73.6	3,060,000	ng/kg dw	1,000 - 250,000	3,060,000	not eval	na	no	ntx
PCB-077	32598-13-3	66 / 596	10.4	80,500	ng/kg dw	110 - 15,000	80,500	not eval	na	no	ntx
PCB-081	70362-50-4	48 / 299	0.396 J	6,970	ng/kg dw	1,000 - 10,000	10,000	not eval	na	no	ntx
PCB-090	68194-07-0	48 / 48	180 C	11,700,000 C	ng/kg dw	na	11,700,000	not eval	na	no	ntx
PCB-101[ac]	37680-73-2	538 / 594	na	5,600,000 J	ng/kg dw	120 - 10,000	5,600,000	not eval	na	no	ntx
PCB-105	32598-14-4	433 / 592	61.4	3,660,000	ng/kg dw	120 - 19,000	3,660,000	not eval	na	no	ntx
PCB-110	38380-03-9	310 / 345	220 J	14,500,000 C	ng/kg dw	120 - 6,600	14,500,000	not eval	na	no	ntx
PCB-113[ac]	68194-10-5	48 / 48	na	na	ng/kg dw	na	na	not eval	na	no	ntx
PCB-114	74472-37-0	53 / 299	2.75	207,000	ng/kg dw	1,000 - 12,000	207,000	not eval	na	no	ntx
PCB-115[ac]	74472-38-1	48 / 48	na	na	ng/kg dw	na	na	not eval	na	no	ntx
PCB-118	31508-00-6	492 / 595	154	12,000,000	ng/kg dw	120 - 8,300	12,000,000	not eval	na	no	ntx
PCB-123	65510-44-3	48 / 299	2.79	138,000	ng/kg dw	1,000 - 31,000	138,000	not eval	na	no	ntx
PCB-126	57465-28-8	56 / 595	0.758 J	7,980	ng/kg dw	100 - 50,000	50,000	not eval	na	no	ntx
PCB-128	38380-07-3	295 / 543	350 J	620,000 J	ng/kg dw	130 - 13,000	620,000	not eval	na	no	ntx
PCB-129	55215-18-4	48 / 48	320 C	14,000,000 C	ng/kg dw	na	14,000,000	not eval	na	no	ntx
PCB-138[ac]	35065-28-2	531 / 596	na	1,400,000	ng/kg dw	130 - 19,000	1,400,000	not eval	na	no	ntx
PCB-153	35065-27-1	543 / 593	258 C	9,090,000 C	ng/kg dw	120 - 11,000	9,090,000	not eval	na	no	ntx
PCB-156	38380-08-4	258 / 596	27.5 C	1,790,000 C	ng/kg dw	80 - 10,000	1,790,000	not eval	na	no	ntx
PCB-157[ac]	69782-90-7	109 / 594	na	56,000	ng/kg dw	80 - 27,000	56,000	not eval	na	no	ntx
PCB-160[ac]	41411-62-5	48 / 48	na	na	ng/kg dw	na	na	not eval	na	no	ntx
PCB-163[ac]	74472-44-9	48 / 48	na	na	ng/kg dw	na	na	not eval	na	no	ntx
PCB-167	52663-72-6	85 / 299	10.6	515,000	ng/kg dw	1,000 - 10,000	515,000	not eval	na	no	ntx
PCB-168[ac]	59291-65-5	48 / 48	na	na	ng/kg dw	na	na	not eval	na	no	ntx

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-169	32774-16-6	0 / 596	nd	nd	ng/kg dw	0.399 - 10,000	10,000	not eval	na	no	ntx
PCB-170	35065-30-6	417 / 548	190 J	460,000	ng/kg dw	80 - 14,000	460,000	not eval	na	no	ntx
PCB-180	35065-29-3	498 / 596	155 C	1,600,000 C	ng/kg dw	110 - 9,500	1,600,000	not eval	na	no	ntx
PCB-187	52663-68-0	209 / 251	1,000 J	360,000 J	ng/kg dw	1,000 - 6,000	360,000	not eval	na	no	ntx
PCB-189	39635-31-9	72 / 596	3.06	65,700	ng/kg dw	110 - 10,000	65,700	not eval	na	no	ntx
PCB-193 [ac]	69782-91-8	48 / 48	na	na	ng/kg dw	na	na	not eval	na	no	ntx
PCB-195	52663-78-2	32 / 251	1,000 J	49,000 J	ng/kg dw	1,000 - 10,000	49,000	not eval	na	no	ntx
PCB-206	40186-72-9	42 / 251	1,000	27,000	ng/kg dw	1,000 - 10,000	27,000	not eval	na	no	ntx
PCB-209	2051-24-3	7 / 251	1,000	2,000	ng/kg dw	1,000 - 10,000	10,000	not eval	na	no	ntx
Total PCBs (calc'd)	na	1205 / 1290	1.6 J	220,000	ug/kg dw	0.56 - 50	220,000	not eval	740 ca[ad]	yes	asl
PCBs + PCTs (total)	na	294 / 297	1.6	26,000	ug/kg dw	0.56 - 0.63	26,000	not eval	na	no	ntx
PCTs (total)	na	258 / 299	1.8 J	5,600	ug/kg dw	1.6 - 8.1	5,600	not eval	na	no	ntx
p-Cymene	99-87-6	3 / 37	1.6 J	25	ug/kg dw	1.5 - 530	530	not eval	na	no	ntx
Pentachloroethane	76-01-7	0 / 37	nd	nd	ug/kg dw	1.5 - 1,100	1,100	not eval	na	no	ntx
Pentachlorophenol	87-86-5	12 / 749	14 J	410	ug/kg dw	6.3 - 10,000	10,000	not eval	9,000 ca	no	ifd[ae]
Perylene	198-55-0	20 / 20	9.0	350	ug/kg dw	na	350	not eval	na	no	ntx
Phenanthrene	85-01-8	727 / 792	7.1	28,000	ug/kg dw	5 - 200	28,000	not eval	na	no	ntx
Phenol	108-95-2	254 / 795	10 J	2,800	ug/kg dw	7.3 - 790	2,800	not eval	10,000,000 nc	no	bsl
Potassium	7440-09-7	429 / 429	380	11,000	mg/kg dw	na	11,000	not eval	na	no	ntx
Pyrene	129-00-0	755 / 792	7 J	16,000	ug/kg dw	18 - 170	16,000	not eval	2,900,000 nc	no	bsl
Pyridine	110-86-1	0 / 12	nd	nd	ug/kg dw	160 - 2,400	2,400	not eval	62,000 nc	no	bsl
Retene	483-65-8	10 / 19	99 J	1,500 J	ug/kg dw	290 - 4,100	4,100	not eval	na	no	ntx
sec-Butylbenzene	135-98-8	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	na	no	ntx
Selenium	7782-49-2	277 / 631	0.2 J	28	mg/kg dw	0.20 - 40	40	not eval	510 nc	no	bsl
Silver	7440-22-4	481 / 784	0.020	270	mg/kg dw	0.046 - 5	270	0.28 / 0.74	510 nc	no	bsl
Sodium	7440-23-5	418 / 418	580	23,000	mg/kg dw	na	23,000	not eval	na	no	ntx
Styrene	100-42-5	0 / 42	nd	nd	ug/kg dw	1.4 - 1,100	1,100	not eval	170,000 nc	no	bsl
Sulfides (total)	na	136 / 231	2.0 J	7,700	mg/kg dw	0.68 - 46	7,700	not eval	na	no	ntx
tert-Butyl methyl ether	1634-04-4	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	70,000 ca	no	bsl
tert-Butylbenzene	98-06-6	0 / 37	nd	nd	ug/kg dw	1.5 - 530	530	not eval	39,000 nc	no	bsl

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Tetrabutyltin as ion	1461-25-2	15 / 119	0.27 J	58	ug/kg dw	0.60 - 20	58	not eval	na	no	ntx
Tetrachloroethene	127-18-4	2 / 42	0.21 J	0.52 J	ug/kg dw	1.4 - 530	530	not eval	1,300 ca	no	bsl
Thallium	7440-28-0	325 / 637	0.010 J	32 J	mg/kg dw	0.030 - 53	53	0.252 / 1.79	6.7 nc	yes	asl
Tin	7440-31-5	163 / 251	1.0 J	350	mg/kg dw	1.0 - 8.0	350	not eval	10,000 nc	no	bsl
Toluene	108-88-3	4 / 42	1.0 J	6.4	ug/kg dw	1.4 - 530	530	not eval	52,000 nc	no	bsl
Total aldrin/dieldrin (calc'd)	na	12 / 197	0.113 J	280	ug/kg dw	0.81 - 91	280	not eval	na	no	ntx
Total chlordane (calc'd)	na	28 / 153	0.20 J	230	ug/kg dw	0.72 - 330	330	not eval	6,500 ca[af]	no	bsl
Total HPAH (calc'd)	na	769 / 792	20	85,000	ug/kg dw	19 - 120	85,000	not eval	na	no	ntx
Total HpCDD	37871-00-4	23 / 25	120	11,000	ng/kg dw	0.99 - 1.9	11,000	not eval	na	no	ntx
Total HpCDF	38998-75-3	23 / 25	18	3,900	ng/kg dw	0.84 - 2.2	3,900	not eval	na	no	ntx
Total HxCDD	34465-46-8	23 / 25	7.5	1,100	ng/kg dw	1.1 - 1.7	1,100	not eval	na	no	ntx
Total HxCDF	55684-94-1	23 / 25	6.2	1,200	ng/kg dw	0.36 - 0.45	1,200	not eval	na	no	ntx
Total LPAH (calc'd)	na	731 / 792	9.1	44,000	ug/kg dw	19 - 200	44,000	not eval	na	no	ntx
Total PAH (calc'd)	na	771 / 792	20	128,000	ug/kg dw	19 - 120	128,000	not eval	na	no	ntx
Total PeCDD	36088-22-9	1 / 25	49	49	ng/kg dw	1.4 - 8.5	49	not eval	na	no	ntx
Total PeCDF	30402-15-4	22 / 25	4.9	180	ng/kg dw	0.71 - 3.9	180	not eval	na	no	ntx
Total petroleum hydrocarbons	na	2 / 2	68	81	mg/kg dw	na	81	not eval	na	no	ntx
Total TCDD	na	20 / 25	0.95	18	ng/kg dw	0.34 - 1.1	18	not eval	na	no	ntx
Total TCDF	30402-14-3	23 / 25	3.0	95	ng/kg dw	0.28 - 0.32	95	not eval	na	no	ntx
Total xylenes (calc'd)	na	0 / 40	nd	nd	ug/kg dw	1.4 - 1,100	1,100	not eval	na	no	bsl
Toxaphene	8001-35-2	2 / 195	340 J	6,300 J	ug/kg dw	1.0 - 4,300	6,300	not eval	1,600 ca	yes	asl
TPH	na	49 / 55	23	23,000	mg/kg dw	20 - 20	23,000	not eval	na	no	ntx
TPH - diesel #2 range	68334-30-5	0 / 8	nd	nd	mg/kg dw	10 - 10	10	not eval	na	no	ntx
TPH - diesel range	na	2 / 2	68	81	mg/kg dw	na	81	not eval	na	no	ntx
TPH - gasoline range	na	0 / 2	nd	nd	mg/kg dw	20 - 20	20	not eval	na	no	ntx
TPH - heavy fuel oil range	8001-58-9	2 / 3	250	370	mg/kg dw	10 - 10	370	not eval	na	no	ntx
trans-1,2-Dichloroethene	156-60-5	0 / 40	nd	nd	ug/kg dw	1.4 - 530	530	not eval	23,000 nc	no	bsl
trans-1,3-Dichloropropene	10061-02-6	0 / 42	nd	nd	ug/kg dw	1.4 - 500	500	not eval	na	no	ntx
trans-1,4-Dichloro-2-butene	110-57-6	0 / 35	nd	nd	ug/kg dw	7.6 - 2,700	2,700	not eval	na	no	ntx
trans-Nonachlor	39765-80-5	0 / 58	nd	nd	ug/kg dw	1.9 - 34	34	not eval	na	no	ntx

Table 1, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Industrial RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Tributyltin as ion	688-73-3	143 / 159	0.28 J	3,000	ug/kg dw	1.0 - 5.3	3,000	not eval	9,100 nc[ag]	no	bsl
Trichloroethene	79-01-6	0 / 42	nd	nd	ug/kg dw	1.4 - 530	530	not eval	110 ca	no	ifd[ah]
Trichlorofluoromethane	75-69-4	0 / 40	nd	nd	ug/kg dw	1.5 - 5,300	5,300	not eval	200,000 nc	no	bsl
Vanadium	7440-62-2	556 / 556	15	150	mg/kg dw	na	150	36 / 59.6	100 nc	yes	asl
Vinyl acetate	108-05-4	0 / 3	nd	nd	ug/kg dw	7.0 - 12	12	not eval	140,000 nc	no	bsl
Vinyl chloride	75-01-4	0 / 42	nd	nd	ug/kg dw	1.5 - 2,700	2,700	not eval	750 ca	no	ifd[ai]
Xylene (meta & para)	108-38-3/ 106-42-3	0 / 40	nd	nd	ug/kg dw	1.4 - 1,100	1,100	not eval	na	no	ntx
Xylene (ortho)	95-47-6	0 / 40	nd	nd	ug/kg dw	1.4 - 530	530	not eval	na	no	ntx
Xylene (total)	1330-20-7	0 / 2	nd	nd	ug/kg dw	23 - 24	24	not eval	42,000 nc	no	bsl
Zinc	7440-66-6	813 / 813	16	9,700	mg/kg dw	na	9,700	52.6 / 98.5	10,000 nc	no	bsl

[a] Background concentrations obtained from joint Ecology/PSAMP 1998 study entitled "Sediment Quality in Puget Sound. Year 2- Central Puget Sound" (Ecology 2000). Reported concentrations are mean and maximum from 52 sediment samples collected from the following areas: South Port Townsend, Port Townsend, North Admiralty Inlet, South Admiralty Inlet, Possession Sound, Central Basin, Port Madison, West Point, East Passage, Liberty Bay, Keyport, Northwest Bainbridge Island, Southwest Bainbridge Island, Rich Passage, Port Orchard, and Port Washington Narrows

[b] Risk-based concentrations (RBCs) are derived from EPA Region 9 Preliminary Remediation Goals (PRGs) for industrial soil (last updated October 1999). PRGs associated with a non-cancer endpoint (abbreviated "nc") were divided by 10 for this screening, reflecting the different target hazard quotients used in Region 9 (HQ = 1) and Region 10 (HQ = 0.1). All other RBCs were not modified for this screening. Abbreviations: ca = cancer endpoint, nc = non-cancer endpoint.

[c] Less than 10% (3 of 53) of reporting limits for 1,2,3-trichloropropane exceed the RBC.

[d] Less than 10% (3 of 53) of reporting limits for 1,2-dibromoethane exceed the RBC.

[e] While a reference dose of 0.004 is provided in IRIS for 2-methylnaphthalene, no RBC is available from the EPA Region 9 RBC tables. It should also be noted that if an RBC was calculated using the guidance provided by EPA, the maximum value for 2-methylnaphthalene would be well under the RBC and thus would not screen in as a COPC.

[f] Less than 10% (1 of 940) of reporting limits for 3,3-dichlorobenzidine exceed the RBC.

[g] Less than 10% (1 of 1,013) of reporting limits for 4,6-dinitro-o-cresol exceed the RBC.

[h] RBC for acenaphthylene is from acenaphthene.

[i] RBC for alpha-chlordane is from chlordane.

[j] RBC for alpha-endosulfan is from endosulfan.

[k] RBC for beta-endosulfan is from endosulfan.

[l] Less than 10% (55 of 1,066) of reporting limits for bis(2-chloroethyl)ether exceed the RBC.

[m] Less than 10% (3 of 72) of reporting limits for bromomethane exceed the RBC.

[n] RBC for carcinogenic PAHs is from benzo(a)pyrene.

[o] Less than 10% (3 of 72) of reporting limits for chloroethane exceed the RBC.

[p] Less than 10% (3 of 72) of reporting limits for chloroform exceed the RBC.

Table 1, continued

[q] RBC for chromium is from chromium VI.

[r] RBC for DDTs (total calc'd) is from 4,4-DDT.

[s] Less than 10% (3 of 72) of reporting limits for dibromochloromethane exceed the RBC.

[t] RBC for Dioxin/Furan TEQ is from 2,3,7,8-TCDD.

[u] RBC for endrin aldehyde is from endrin.

[v] RBC for endrin ketone is from endrin.

[w] RBC for gamma-chlordane is from chlordane.

[x] Less than 10% (1 of 405) of reporting limits for heptachlor epoxide exceed the RBC.

[y] Less than 10% (12 of 1,142) of reporting limits for hexachlorobenzene exceed the RBC.

[z] Less than 10% (3 of 53) of reporting limits for methacrylonitrile exceed the RBC.

[aa] Less than 10% (100 of 1,066) of reporting limits for n-nitroso-di-n-propylamine exceed the RBC.

[ab] RBC for PCB TEQ is from 2,3,7,8-TCDD.

[ac] Statistics for this congener reflect only those samples that were determined not to co-elute with other congeners. When no statistics are given, "na" will appear in place of statistics, meaning that all samples co-eluted with another congener.

[ad] RBC for PCB (total calc'd) is from Aroclor 1254.

[ae] Less than 10% (1 of 1,118) of reporting limits for pentachlorophenol exceed the RBC.

[af] RBC for total chlordane (calc'd) is from chlordane.

[ag] RBC for tributyltin as ion is from RBC for tributyltin oxide multiplied by 0.49 to account for differences in molecular weight.

[ah] Less than 10% (3 of 72) of reporting limits for trichloroethene exceed the RBC.

[ai] Less than 10% (3 of 72) of reporting limits for vinyl chloride exceed the RBC.

Abbreviations:

COPC – chemical of potential concern

nd – not detected

na – not applicable

HPAH – high-molecular-weight polycyclic aromatic hydrocarbon

LPAH – low-molecular-weight polycyclic aromatic hydrocarbon

C – concentration represents coelution

J – estimated value

asl – above screening level (selected as COPC)

bsl – below screening level (not selected as COPC)

ifd – infrequent detection (not selected as COPC)

ntx – no toxicity information (not selected as COPC)

sum – chemical included in sum and is not evaluated separately

teq – chemical included in TEQ calculation and is not evaluated separately

Table 2. Occurrence and selection of chemicals of potential concern for intertidal sediment in the beach play and clamming exposure scenarios

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
1,1,1,2-Tetrachloroethane	630-20-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	3,200 ca	no	bsl
1,1,1-Trichloroethane	71-55-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	120,000 nc	no	bsl
1,1,2,2-Tetrachloroethane	79-34-5	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	410 ca	no	bsl
1,1,2-Trichloroethane	79-00-5	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	730 ca	no	bsl
1,1,2-Trichlorotrifluoroethane	76-13-1	0 / 7	nd	nd	ug/kg dw	2.4 - 74	74	not eval.	560,000 nc	no	bsl
1,1-Dichloroacetone	513-88-2	0 / 6	nd	nd	ug/kg dw	4.7 - 37	37	not eval.	na	no	ntx
1,1-Dichloroethane	75-34-3	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	51,000 nc	no	bsl
1,1-Dichloroethene	75-35-4	0 / 7	nd	nd	ug/kg dw	2.4 - 7.4	7.4	not eval.	12,000 nc	no	bsl
1,1-Dichloropropene	563-58-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
1,2,3,4,6,7,8-HpCDD	35822-46-9	17 / 17	41.4 J	73,700	ng/kg dw	na - na	74,000	not eval.	na	no	ntx
1,2,3,4,6,7,8-HpCDF	67562-39-4	16 / 17	6.71	40,300	ng/kg dw	7.7 - 7.7	40,000	not eval.	na	no	ntx
1,2,3,4,7,8,9-HpCDF	55673-89-7	14 / 17	0.421 J	3,720	ng/kg dw	0.77 - 2.8	3,700	not eval.	na	no	ntx
1,2,3,4,7,8-HxCDD	39227-28-6	12 / 17	0.382 J	124	ng/kg dw	0.86 - 4.4	120	not eval.	na	no	ntx
1,2,3,4,7,8-HxCDF	70648-26-9	14 / 17	0.694 J	2,530	ng/kg dw	1.2 - 3.6	2,500	not eval.	na	no	ntx
1,2,3,6,7,8-HxCDD	57653-85-7	15 / 17	1.73 J	3,400	ng/kg dw	2.4 - 2.6	3,400	not eval.	na	no	ntx
1,2,3,6,7,8-HxCDF	57117-44-9	12 / 17	0.335 J	365	ng/kg dw	0.51 - 4.3	370	not eval.	na	no	ntx
1,2,3,7,8,9-HxCDD	19408-74-3	15 / 17	1.19 J	315	ng/kg dw	2.1 - 2.2	320	not eval.	na	no	ntx
1,2,3,7,8,9-HxCDF	72918-21-9	12 / 17	0.0730 J	33.8 J	ng/kg dw	0.15 - 2.4	34	not eval.	na	no	ntx
1,2,3,7,8-PeCDD	40321-76-4	12 / 17	0.284 J	57.1	ng/kg dw	0.53 - 4.1	57	not eval.	na	no	ntx
1,2,3,7,8-PeCDF	57117-41-6	12 / 17	0.214 J	69.3	ng/kg dw	0.28 - 2.8	69	not eval.	na	no	ntx
1,2,3-Trichlorobenzene	87-61-6	0 / 7	nd	nd	ug/kg dw	3.7 - 6.6	6.6	not eval.	na	no	ntx
1,2,3-Trichloropropane	96-18-4	0 / 7	nd	nd	ug/kg dw	2.4 - 18	18	not eval.	34 ca	no	bsl
1,2,4-Trichlorobenzene	120-82-1	0 / 312	nd	nd	ug/kg dw	0.33 - 290	290	not eval.	6,200 nc	no	bsl
1,2,4-Trimethylbenzene	95-63-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	5,200 nc	no	bsl
1,2-Dibromo-3-chloropropane	96-12-8	0 / 7	nd	nd	ug/kg dw	7.4 - 17	17	not eval.	460 ca	no	bsl
1,2-Dibromoethane (EDB)	106-93-4	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	32 ca	no	bsl
1,2-Dichlorobenzene	95-50-1	1 / 312	22 J	22 J	ug/kg dw	0.33 - 290	290	not eval.	60,000 nc	no	bsl

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
1,2-Dichloroethane	107-06-2	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	280 ca	no	bsl
1,2-Dichloropropane	78-87-5	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	340 ca	no	bsl
1,2-Diphenylhydrazine	122-66-7	0 / 33	nd	nd	ug/kg dw	13 - 170	170	not eval.	610 ca	no	bsl
1,3,5-Trimethylbenzene	108-67-8	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	2,100 nc	no	bsl
1,3-Dichlorobenzene	541-73-1	1 / 306	2.5 J	2.5 J	ug/kg dw	0.33 - 290	290	not eval.	53,000 nc	no	bsl
1,3-Dichloropropane	142-28-9	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	10,000 nc	no	bsl
1,4-Dichlorobenzene	106-46-7	10 / 312	3.4 J	1,300	ug/kg dw	0.16 - 290	1,300	not eval.	3,400 ca	no	bsl
1-Chlorobutane	109-69-3	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	48,000 nc	no	bsl
1-Methylnaphthalene	90-12-0	13 / 13	1.9 J	32	ug/kg dw	na - na	32	not eval.	na	no	ntx
2,2-Dichloropropane	594-20-7	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
2,3,4,6,7,8-HxCDF	60851-34-5	12 / 17	0.307 J	302 J	ng/kg dw	0.44 - 2.5	300	not eval.	na	no	ntx
2,3,4,7,8-PeCDF	57117-31-4	12 / 17	0.392 J	230	ng/kg dw	0.44 - 5.4	230	not eval.	na	no	ntx
2,3,7,8-TCDD	1746-01-6	11 / 17	0.0890 J	30.6	ng/kg dw	0.46 - 1.1	31	not eval.	3.9 ca	no	bsl
2,3,7,8-TCDF	51207-31-9	15 / 17	0.426 J	89.6	ng/kg dw	0.81 - 0.91	90	not eval.	na	no	ntx
2,4,5-Trichlorophenol	95-95-4	0 / 282	nd	nd	ug/kg dw	8.6 - 2,000	2,000	not eval.	610,000 nc	no	bsl
2,4,6-Trichlorophenol	88-06-2	0 / 282	nd	nd	ug/kg dw	8.6 - 2,000	2,000	not eval.	610 nc	no	ifd[c]
2,4'-DDD	53-19-0	4 / 53	1.6 J	10 J	ug/kg dw	0.97 - 34	34	not eval.	na	no	ntx
2,4'-DDE	3424-82-6	2 / 53	2.8 J	11	ug/kg dw	0.97 - 34	34	not eval.	na	no	ntx
2,4'-DDT	789-02-6	23 / 53	0.24 J	11	ug/kg dw	1.9 - 460	460	not eval.	na	no	ntx
2,4-Dichlorophenol	120-83-2	0 / 282	nd	nd	ug/kg dw	8.6 - 1,400	1,400	not eval.	18,000 nc	no	bsl
2,4-Dimethylphenol	105-67-9	1 / 310	290 J	290 J	ug/kg dw	6.3 - 500	500	not eval.	120,000 nc	no	bsl
2,4-Dinitrophenol	51-28-5	0 / 280	nd	nd	ug/kg dw	16 - 2,900	2,900	not eval.	12,000 nc	no	bsl
2,4-Dinitrotoluene	121-14-2	0 / 282	nd	nd	ug/kg dw	3.8 - 1,400	1,400	not eval.	12,000 nc	no	bsl
2,6-Dinitrotoluene	606-20-2	0 / 282	nd	nd	ug/kg dw	8.6 - 1,400	1,400	not eval.	6,100 nc	no	bsl
2-Chloronaphthalene	91-58-7	0 / 286	nd	nd	ug/kg dw	8.6 - 290	290	not eval.	490,000 nc	no	bsl
2-Chlorophenol	95-57-8	0 / 282	nd	nd	ug/kg dw	8.6 - 290	290	not eval.	6,300 nc	no	bsl
2-Chlorotoluene	95-49-8	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	16,000 nc	no	bsl
2-Hexanone	591-78-6	0 / 7	nd	nd	ug/kg dw	4.7 - 7.4	7.4	not eval.	na	no	ntx
2-Methylnaphthalene	91-57-6	67 / 314	1.0 J	1,400	ug/kg dw	1.0 - 290	1,400	not eval.	na	no	ntx
2-Methylphenol	95-48-7	1 / 317	32	32	ug/kg dw	6.3 - 290	290	not eval.	310,000 nc	no	bsl

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
2-Nitroaniline	88-74-4	0 / 280	nd	nd	ug/kg dw	16 - 2,000	2,000	not eval.	18,000 nc	no	bsl
2-Nitrophenol	88-75-5	0 / 282	nd	nd	ug/kg dw	8.6 - 2,000	2,000	not eval.	na	no	ntx
2-Nitropropane	79-46-9	0 / 7	nd	nd	ug/kg dw	12 - 18	18	not eval.	na	no	ntx
3,3'-Dichlorobenzidine	91-94-1	0 / 268	nd	nd	ug/kg dw	31 - 1,400	1,400	not eval.	1,100 ca	no	ifd[d]
3-Nitroaniline	99-09-2	0 / 275	nd	nd	ug/kg dw	18 - 1,400	1,400	not eval.	1,800 nc	no	bsl
4,4'-DDD	72-54-8	28 / 75	0.30 J	840	ug/kg dw	0.98 - 540	840	not eval.	2,400 ca	no	sum
4,4'-DDE	72-55-9	19 / 75	0.28 J	370 J	ug/kg dw	0.97 - 800	800	not eval.	1,700 ca	no	sum
4,4'-DDT	50-29-3	27 / 75	0.48 J	1,700	ug/kg dw	1.0 - 34	1,700	not eval.	1,700 ca	no	sum
4,6-Dinitro-o-cresol	534-52-1	0 / 280	nd	nd	ug/kg dw	16 - 2,900	2,900	not eval.	610 nc	yes	asl
4-Bromophenyl phenyl ether	101-55-3	0 / 282	nd	nd	ug/kg dw	8.6 - 290	290	not eval.	na	no	ntx
4-Chloro-3-methylphenol	59-50-7	1 / 280	6.4 J	6.4 J	ug/kg dw	8.6 - 1,400	1,400	not eval.	na	no	ntx
4-Chloroaniline	106-47-8	0 / 275	nd	nd	ug/kg dw	8.6 - 1,400	1,400	not eval.	24,000 nc	no	bsl
4-Chlorophenyl phenyl ether	7005-72-3	0 / 282	nd	nd	ug/kg dw	8.6 - 290	290	not eval.	na	no	ntx
4-Chlorotoluene	106-43-4	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
4-Methylphenol	106-44-5	37 / 321	8.7 J	444	ug/kg dw	8.6 - 290	440	not eval.	31,000 nc	no	bsl
4-Nitroaniline	100-01-6	0 / 275	nd	nd	ug/kg dw	16 - 1,400	1,400	not eval.	23,000 ca	no	bsl
4-Nitrophenol	100-02-7	0 / 280	nd	nd	ug/kg dw	16 - 1,400	1,400	not eval.	na	no	ntx
Acenaphthene	83-32-9	107 / 318	1.0 J	3,900	ug/kg dw	1.8 - 290	3,900	not eval.	370,000 nc	no	bsl
Acenaphthylene	208-96-8	58 / 314	1.3 J	240	ug/kg dw	1.8 - 290	290	not eval.	370,000 nc[e]	no	bsl
Acetone	67-64-1	3 / 7	110 J	1,000 J	ug/kg dw	24 - 150	1,000	not eval.	1,400,000 nc	no	bsl
Acid volatile sulfides	na	6 / 11	97 J	4,800	mg/kg dw	48 - 55	4,800	not eval.	na	no	ntx
Aldrin	309-00-2	3 / 75	0.014 J	0.81 J	ug/kg dw	0.81 - 17	17	not eval.	29 ca	no	bsl
Allyl chloride	107-05-1	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	1,700 nc	no	bsl
alpha-BHC	319-84-6	3 / 75	0.14 J	1.8 J	ug/kg dw	0.81 - 17	17	not eval.	90 ca	no	bsl
alpha-Chlordane	5103-71-9	10 / 62	0.10 J	36	ug/kg dw	0.81 - 17	36	not eval.	1,600 ca[f]	no	bsl
alpha-Endosulfan	959-98-8	9 / 60	0.18 J	71 J	ug/kg dw	0.96 - 100	100	not eval.	37,000 nc[g]	no	bsl
Aluminum	7429-90-5	130 / 130	3,800	110,000	mg/kg dw	na - na	110,000	12,000 / 21,000	7,600 nc	yes	asl
Ammonia (total as nitrogen)	7664-41-7	75 / 77	0.73	29.8	mg-N/kg	0.11 - 0.12	29.8	not eval.	na	no	ntx
Aniline	62-53-3	0 / 121	nd	nd	ug/kg dw	18 - 290	290	not eval.	85,000 ca	no	bsl
Anthracene	120-12-7	170 / 318	2.0	4,400	ug/kg dw	5 - 290	4,400	not eval.	2,200,000 nc	no	bsl

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Antimony	7440-36-0	53 / 177	0.09 J	110 J	mg/kg dw	0.2 - 31	110	0.23 / 0.44	3.1 nc	yes	asl
Aroclor-1016	12674-11-2	0 / 415	nd	nd	ug/kg dw	0.87 - 3,400	3,400	not eval.	390 nc	no	sum, teq
Aroclor-1221	11104-28-2	0 / 333	nd	nd	ug/kg dw	1.9 - 5,500	5,500	not eval.	na	no	ntx
Aroclor-1232	11141-16-5	0 / 333	nd	nd	ug/kg dw	0.87 - 3,400	3,400	not eval.	na	no	ntx
Aroclor-1242	53469-21-9	27 / 414	7.8 J	2,700	ug/kg dw	0.87 - 6,100	6,100	not eval.	na	no	ntx
Aroclor-1248	12672-29-6	66 / 419	13	220,000	ug/kg dw	0.87 - 3,400	220,000	not eval.	na	no	ntx
Aroclor-1254	11097-69-1	286 / 417	2.2	110,000	ug/kg dw	1.3 - 4,300	110,000	not eval.	220 ca	no	sum, teq
Aroclor-1254/1260	na	2 / 2	37	800	ug/kg dw	na - na	800	not eval.	na	no	ntx
Aroclor-1260	11096-82-5	294 / 417	1.2 J	38,000	ug/kg dw	2.5 - 15,000	38,000	not eval.	na	no	ntx
Aroclor-1262	37324-23-5	2 / 6	270	840	ug/kg dw	3.3 - 20	840	not eval.	na	no	ntx
Aroclor-1268	11100-14-4	1 / 5	460 J	460 J	ug/kg dw	3.3 - 20	460	not eval.	na	no	ntx
Arsenic	7440-38-2	302 / 330	1.2	1,100	mg/kg dw	3 - 31	1,100	5.03 / 10.4	0.39 ca	yes	asl
Barium	7440-39-3	116 / 116	9.40	3,500	mg/kg dw	na - na	3,500	24 / 55.5	540 nc	yes	asl
Benzaldehyde	100-52-7	0 / 4	nd	nd	ug/kg dw	170 - 170	170	not eval.	610,000 nc	no	bsl
Benzene	71-43-2	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	640 ca	no	bsl
Benzidine	92-87-5	0 / 5	nd	nd	ug/kg dw	760 - 1,700	1,700	not eval.	2.1 ca	yes	asl
Benzo(a)anthracene	56-55-3	275 / 318	3.6 J	8,400	ug/kg dw	6.6 - 200	8,400	not eval.	620 ca	no	teq
Benzo(a)pyrene	50-32-8	275 / 314	5.8 J	7,900	ug/kg dw	6.6 - 350	7,900	not eval.	62 ca	no	teq
Benzo(b)fluoranthene	205-99-2	283 / 313	4.6	8,200	ug/kg dw	6.6 - 140	8,200	not eval.	620 ca	no	teq
Benzo(e)pyrene	192-97-2	13 / 13	16	1,300	ug/kg dw	na - na	1,300	not eval.	na	no	ntx
Benzo(g,h,i)perylene	191-24-2	235 / 316	6.1	3,800	ug/kg dw	10 - 350	3,800	not eval.	na	no	ntx
Benzo(k)fluoranthene	207-08-9	269 / 313	8.4	8,800	ug/kg dw	3.8 - 200	8,800	not eval.	6,200 ca	no	teq
Benzofluoranthenes (total)	56832-73-6	284 / 313	4.6	17,000	ug/kg dw	19 - 140	17,000	not eval.	na	no	ntx
Benzoic acid	65-85-0	40 / 318	54 J	4,500	ug/kg dw	13 - 2,900	4,500	not eval.	10,000,000 nc	no	bsl
Benzyl alcohol	100-51-6	9 / 314	8.2 J	670	ug/kg dw	7.5 - 690	690	not eval.	1,800,000 nc	no	bsl
Beryllium	7440-41-7	122 / 132	0.10	0.60 J	mg/kg dw	0.10 - 0.70	0.70	not eval.	15 nc	no	bsl
beta-BHC	319-85-7	4 / 75	0.087 J	13	ug/kg dw	0.81 - 17	17	not eval.	320 ca	no	bsl
beta-Endosulfan	33213-65-9	3 / 62	0.47 J	10 J	ug/kg dw	0.81 - 200	200	not eval.	37,000 nc[h]	no	sum
Biphenyl	92-52-4	13 / 13	0.86 J	33	ug/kg dw	na - na	33	not eval.	300,000 nc	no	bsl
bis(2-chloroethoxy)methane	111-91-1	0 / 282	nd	nd	ug/kg dw	8.6 - 400	400	not eval.	na	no	ntx

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
bis(2-chloroethyl)ether	111-44-4	0 / 282	nd	nd	ug/kg dw	8.6 - 290	290	not eval.	220 ca	yes	asl
bis(2-chloroisopropyl)ether	108-60-1	0 / 282	nd	nd	ug/kg dw	8.6 - 400	400	not eval.	2,900 ca	no	bsl
Bis(2-ethylhexyl)phthalate	117-81-7	260 / 318	5.4	14,000	ug/kg dw	15 - 870	14,000	not eval.	35,000 ca	no	bsl
Bromobenzene	108-86-1	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	2,800 nc	no	bsl
Bromochloromethane	74-97-5	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
Bromodichloromethane	75-27-4	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	820 ca	no	bsl
Bromoform	75-25-2	0 / 7	nd	nd	ug/kg dw	4.7 - 7.4	7.4	not eval.	62,000 ca	no	bsl
Bromomethane	74-83-9	0 / 7	nd	nd	ug/kg dw	12 - 18	18	not eval.	390 nc	no	bsl
Butyl benzyl phthalate	85-68-7	116 / 314	2.4	6,900 J	ug/kg dw	1.8 - 290	6,900	not eval.	1,200,000 nc	no	bsl
Butyltin (total)	na	0 / 4	nd	nd	ug/kg dw	15 - 20	20	not eval.	na	no	ntx
C1-Chrysenes	3001965	13 / 13	12	980	ug/kg dw	na - na	980	not eval.	na	no	ntx
C1-Dibenzothiophenes	3001957	11 / 13	4.6 J	59	ug/kg dw	5.0 - 5.0	59	not eval.	na	no	ntx
C1-Fluoranthene/Pyrene	3001964	12 / 13	18	2,600	ug/kg dw	4.9 - 4.9	2,600	not eval.	na	no	ntx
C1-Fluorenes	3001954	5 / 13	4.5 J	150	ug/kg dw	5.0 - 7.8	150	not eval.	na	no	ntx
C1-Phenanthrenes/anthracenes	3001960	13 / 13	8.0	1,700	ug/kg dw	na - na	1,700	not eval.	na	no	ntx
C2-Chrysenes	3001966	13 / 13	8.1	370	ug/kg dw	na - na	370	not eval.	na	no	ntx
C2-Dibenzothiophenes	3001958	10 / 13	4.3 J	190	ug/kg dw	5.0 - 5.4	190	not eval.	na	no	ntx
C2-Fluorenes	3001955	9 / 13	5.1	250	ug/kg dw	5.0 - 5.4	250	not eval.	na	no	ntx
C2-Naphthalenes	3001951	13 / 13	6.2	100	ug/kg dw	na - na	100	not eval.	na	no	ntx
C2-Phenanthrenes/anthracenes	3001961	13 / 13	8.5	840	ug/kg dw	na - na	840	not eval.	na	no	ntx
C3-Chrysenes	3001967	13 / 13	7.3	210	ug/kg dw	na - na	210	not eval.	na	no	ntx
C3-Dibenzothiophenes	3001959	11 / 13	7.6	130	ug/kg dw	4.9 - 5.0	130	not eval.	na	no	ntx
C3-Fluorenes	3001956	11 / 13	5.2	220	ug/kg dw	5.0 - 5.0	220	not eval.	na	no	ntx
C3-Naphthalenes	3001952	13 / 13	4.4 J	310	ug/kg dw	na - na	310	not eval.	na	no	ntx
C3-Phenanthrenes/anthracenes	3001962	13 / 13	6.8	420	ug/kg dw	na - na	420	not eval.	na	no	ntx
C4-Chrysenes	3001968	10 / 13	7.8	78	ug/kg dw	4.9 - 5.0	78	not eval.	na	no	ntx
C4-Naphthalenes	3001953	13 / 13	6.0	250	ug/kg dw	na - na	250	not eval.	na	no	ntx
C4-Phenanthrenes/anthracenes	3001963	13 / 13	5.6	150	ug/kg dw	na - na	150	not eval.	na	no	ntx
Cadmium	7440-43-9	195 / 321	0.030 J	120	mg/kg dw	0.040 - 2.5	120	0.36 / 1.12	3.7 nc	yes	asl
Caffeine	58-08-2	0 / 13	nd	nd	ug/kg dw	6.8 - 12	12	not eval.	na	no	ntx

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Calcium	7440-70-2	116 / 116	2,000	30,000 J	mg/kg dw	na - na	30,000	not eval.	na	no	ntx
Caprolactam	105-60-2	0 / 4	nd	nd	ug/kg dw	830 - 870	870	not eval.	3,100,000 nc	no	bsl
Carbazole	86-74-8	124 / 286	3.2 J	2,100	ug/kg dw	8.8 - 290	2,100	not eval.	24,000 ca	no	bsl
Carbon disulfide	75-15-0	1 / 7	4.0 J	4.0 J	ug/kg dw	4.7 - 7.4	7.4	not eval.	36,000 nc	no	bsl
Carbon tetrachloride	56-23-5	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	250 ca	no	bsl
Carcinogenic PAHs - Mammal - Half DL	na	297 / 318	9.1 J	11,000	ug/kg dw	na - na	11,000	not eval.	62 ca[i]	yes	asl
Chlordane	57-74-9	2 / 13	40.0	62	ug/kg dw	7.7 - 21	62	not eval.	na	no	ntx
Chlorobenzene	108-90-7	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	15,000 nc	no	bsl
Chloroethane	75-00-3	0 / 7	nd	nd	ug/kg dw	4.7 - 18	18	not eval.	3,000 ca	no	bsl
Chloroform	67-66-3	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	220 ca	no	bsl
Chloromethane	74-87-3	0 / 7	nd	nd	ug/kg dw	2.4 - 18	18	not eval.	4,700 nc	no	bsl
Chromium	7440-47-3	330 / 330	4.8	1,100 J	mg/kg dw	na - na	1,100	not eval.	1.6 ca[j]	yes	asl
Chrysene	218-01-9	290 / 318	12 J	7,700	ug/kg dw	5 - 170	7,700	not eval.	62,000 ca	no	bsl
cis-1,2-Dichloroethene	156-59-2	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	4,300 nc	no	bsl
cis-1,3-Dichloropropene	10061-01-5	0 / 7	nd	nd	ug/kg dw	2.5 - 3.9	3.9	not eval.	na	no	ntx
cis-Nonachlor	5103-73-1	0 / 25	nd	nd	ug/kg dw	1.9 - 330	330	not eval.	na	no	ntx
Cobalt	7440-48-4	206 / 206	2.82	37 J	mg/kg dw	na - na	37	not eval.	900 ca	no	bsl
Copper	7440-50-8	330 / 330	5	12,000 J	mg/kg dw	na - na	12,000	21.3 / 50.8	310 nc	yes	asl
Coprostanol	360-68-9	8 / 29	367 J	5,700	ug/kg dw	18 - 450	5,700	not eval.	na	no	ntx
Total DDTs	na	34 / 75	0.72 J	2,900 J	ug/kg dw	1.5 - 800	2,900	not eval.	1,700 ca[k]	yes	asl
delta-BHC	319-86-8	3 / 66	0.081 J	11	ug/kg dw	0.81 - 17	17	not eval.	na	no	ntx
Dibenzo(a,h)anthracene	53-70-3	133 / 318	1.6 J	1,500	ug/kg dw	1.0 - 850	1,500	not eval.	62 ca	no	teq
Dibenzofuran	132-64-9	88 / 317	1.0 J	4,200	ug/kg dw	1.7 - 290	4,200	not eval.	15,000 nc	no	bsl
Dibenzothiophene	132-65-0	13 / 13	0.81 J	150	ug/kg dw	na - na	150	not eval.	na	no	ntx
Dibromochloromethane	124-48-1	0 / 7	nd	nd	ug/kg dw	2.4 - 7.4	7.4	not eval.	1,100 ca	no	bsl
Dibromomethane	74-95-3	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	6,700 nc	no	bsl
Dibutyltin as ion	1002-53-5	43 / 52	0.39 J	57	ug/kg dw	1.0 - 10	57	not eval.	na	no	ntx
Dichlorodifluoromethane	75-71-8	0 / 2	nd	nd	ug/kg dw	3.0 - 3.3	3.3	not eval.	9,400 nc	no	bsl
Dichloromethane	75-09-2	0 / 7	nd	nd	ug/kg dw	12 - 18	18	not eval.	9,100 ca	no	bsl
Dieldrin	60-57-1	6 / 75	0.099 J	280	ug/kg dw	0.81 - 91	280	not eval.	30 ca	yes	asl

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Diethyl ether	60-29-7	0 / 7	nd	nd	ug/kg dw	2.4 - 7.4	7.4	not eval.	180,000 nc	no	bsl
Diethyl phthalate	84-66-2	23 / 318	2.0 J	130	ug/kg dw	1.8 - 290	290	not eval.	4,900,000 nc	no	bsl
Dimethyl phthalate	131-11-3	49 / 314	2.0 J	200	ug/kg dw	1.8 - 290	290	not eval.	10,000,000 nc	no	bsl
Di-n-butyl phthalate	84-74-2	85 / 314	3.0 J	3,600	ug/kg dw	1.8 - 210	3,600	not eval.	610,000 nc	no	bsl
Di-n-octyl phthalate	117-84-0	21 / 318	1.8	798	ug/kg dw	1.8 - 870	870	not eval.	240,000 nc	no	bsl
Dioxin/furan TEQ - Mammal - Half DL	na	17 / 17	1.59 J	2,100 J	ng/kg dw	na - na	2,100	not eval.	3.9 ca[l]	yes	asl
Endosulfan	115-29-7	1 / 15	0.11 J	0.11 J	ug/kg dw	0.81 - 3.4	3.4	not eval.	37,000 nc	no	bsl
Endosulfan sulfate	1031-07-8	3 / 73	0.63 J	25	ug/kg dw	0.97 - 200	200	not eval.	na	no	ntx
Endrin	72-20-8	4 / 75	0.99 J	9.1	ug/kg dw	0.81 - 200	200	not eval.	1,800 nc	no	bsl
Endrin aldehyde	7421-93-4	6 / 69	0.28 J	130	ug/kg dw	0.97 - 250	250	not eval.	1,800 nc[m]	no	bsl
Endrin ketone	53494-70-5	5 / 62	1.2 J	110 J	ug/kg dw	0.97 - 200	200	not eval.	1,800 nc[n]	no	bsl
Ethyl methacrylate	97-63-2	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	14,000 nc	no	bsl
Ethylbenzene	100-41-4	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	40,000 nc	no	bsl
Fluoranthene	206-44-0	304 / 318	18	24,000	ug/kg dw	10 - 340	24,000	not eval.	230,000 nc	no	bsl
Fluorene	86-73-7	114 / 318	1.4 J	5,500	ug/kg dw	1.8 - 290	5,500	not eval.	270,000 nc	no	bsl
gamma-BHC	58-89-9	7 / 75	0.050 J	6.7 J	ug/kg dw	0.81 - 17	17	not eval.	440 ca	no	bsl
gamma-Chlordane	5103-74-2	18 / 62	0.20 J	200	ug/kg dw	0.96 - 96	200	not eval.	1,600 ca[o]	no	bsl
Heptachlor	76-44-8	2 / 75	0.12 J	0.89 J	ug/kg dw	0.81 - 70	70	not eval.	110 ca	no	bsl
Heptachlor epoxide	1024-57-3	2 / 75	1.0	1.0	ug/kg dw	0.62 - 510	510	not eval.	53 ca	no	ifd[p]
Hexachlorobenzene	118-74-1	20 / 315	0.4 J	63	ug/kg dw	0.11 - 200	200	not eval.	300 ca	no	bsl
Hexachlorobutadiene	87-68-3	0 / 314	nd	nd	ug/kg dw	0.94 - 270	270	not eval.	6,200 ca	no	bsl
Hexachlorocyclopentadiene	77-47-4	0 / 273	nd	nd	ug/kg dw	34 - 2,000	2,000	not eval.	37,000 nc	no	bsl
Hexachloroethane	67-72-1	0 / 301	nd	nd	ug/kg dw	2.4 - 290	290	not eval.	35,000 ca	no	bsl
Indeno(1,2,3-cd)pyrene	193-39-5	260 / 316	6.5	4,300	ug/kg dw	6.4 - 200	4,300	not eval.	620 ca	no	teq
Iodomethane	74-88-4	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
Iron	7439-89-6	128 / 128	8,100	160,000	mg/kg dw	na - na	160,000	17,500/28,700	2,300 nc	yes	asl
Isophorone	78-59-1	1 / 286	430	430	ug/kg dw	8.6 - 290	430	not eval.	510,000 ca	no	bsl
Isopropylbenzene	98-82-8	0 / 7	nd	nd	ug/kg dw	3.7 - 17	17	not eval.	57,000 nc	no	bsl
Lead	7439-92-1	330 / 330	2	23,000	mg/kg dw	na - na	23,000	15 / 45	40 nc	yes	asl
Magnesium	7439-95-4	117 / 117	2,000	12,000 J	mg/kg dw	na - na	12,000	not eval.	na	no	ntx

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Manganese	7439-96-5	130 / 130	78.0	3,300	mg/kg dw	na - na	3,300	279 / 1,010	180 nc	yes	asl
Mercury	7439-97-6	257 / 325	0.021	4.6 J	mg/kg dw	0.020 - 0.1	4.6	0.0981 / 0.327	2.3 nc	yes	asl
Methacrylonitrile	126-98-7	0 / 7	nd	nd	ug/kg dw	7.4 - 17	17	not eval.	210 nc	no	bsl
Methoxychlor	72-43-5	8 / 75	0.34 J	99	ug/kg dw	0.97 - 170	170	not eval.	31,000 nc	no	bsl
Methyl acrylate	96-33-3	0 / 7	nd	nd	ug/kg dw	7.4 - 17	17	not eval.	7,000 nc	no	bsl
Methyl ethyl ketone	78-93-3	1 / 7	17	17	ug/kg dw	4.7 - 7.4	17	not eval.	2,200,000 nc	no	bsl
Methyl isobutyl ketone	108-10-1	0 / 7	nd	nd	ug/kg dw	4.7 - 7.4	7.4	not eval.	530,000 nc	no	bsl
Methyl methacrylate	80-62-6	0 / 7	nd	nd	ug/kg dw	3.7 - 6.6	6.6	not eval.	220,000 nc	no	bsl
Methylmercury	22967-92-6	4 / 4	0.040 J	5.6	ug/kg dw	na - na	5.60	not eval.	610 nc	no	bsl
Mirex	2385-85-5	1 / 53	1.0 J	1.0 J	ug/kg dw	0.97 - 34	34	not eval.	270 ca	no	bsl
Molybdenum	7439-98-7	105 / 119	0.390 J	49	mg/kg dw	1.2 - 5.3	49	not eval.	39 nc	yes	asl
Monobutyltin as ion	78763-54-9	36 / 42	0.12 J	17 J	ug/kg dw	3.8 - 45	45	not eval.	na	no	ntx
Naphthalene	91-20-3	69 / 314	3.0 J	1,300	ug/kg dw	1.0 - 290	1,300	not eval.	5,600 nc	no	bsl
n-Butylbenzene	104-51-8	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	24,000 nc	no	bsl
Nickel	7440-02-0	300 / 300	5	910	mg/kg dw	na - na	910	26.8 / 41.7	160 nc	yes	asl
Nitrobenzene	98-95-3	0 / 282	nd	nd	ug/kg dw	8.6 - 290	290	not eval.	2,000 nc	no	bsl
n-Nitrosodimethylamine	62-75-9	0 / 132	nd	nd	ug/kg dw	31 - 1,000	1,000	not eval.	9.5 ca	yes	asl
n-Nitroso-di-n-propylamine	621-64-7	0 / 282	nd	nd	ug/kg dw	8.6 - 1,400	1,400	not eval.	69 ca	yes	asl
n-Nitrosodiphenylamine	86-30-6	11 / 314	6.6	95	ug/kg dw	1.8 - 290	290	not eval.	99,000 ca	no	bsl
n-Propylbenzene	103-65-1	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	24,000 nc	no	bsl
OCDD	3268-87-9	17 / 17	297 J	241,000	ng/kg dw	na - na	240,000	not eval.	na	no	ntx
OCDF	39001-02-0	17 / 17	12.5	93,700	ng/kg dw	na - na	94,000	not eval.	na	no	ntx
Oxychlordane	27304138	0 / 25	nd	nd	ug/kg dw	1.9 - 34	34	not eval.	na	no	ntx
PCB TEQ - Mammal - Half DL	na	34 / 34	0.0908 J	1,380	ng/kg dw	na - na	1,380	not eval.	3.9 ca[q]	yes	asl
PCB-018	37680-65-2	5 / 34	1,000 J	170,000 J	ng/kg dw	1,000 - 24,000	170,000	not eval.	na	no	ntx
PCB-028	7012-37-5	12 / 43	1,000 J	160,000 J	ng/kg dw	1,000 - 2,000	160,000	not eval.	na	no	ntx
PCB-044	41464-39-5	17 / 43	1,000 J	190,000 J	ng/kg dw	1,000 - 1,000	190,000	not eval.	na	no	ntx
PCB-055	74338-24-2	25 / 43	1,000 J	890,000 J	ng/kg dw	1,000 - 2,000	890,000	not eval.	na	no	ntx
PCB-066	32598-10-0	69 / 77	73.6	3,060,000	ng/kg dw	1,000 - 250,000	3,100,000	not eval.	na	no	ntx
PCB-077	32598-13-3	48 / 182	10.4	80,500	ng/kg dw	110 - 15,000	81,000	not eval.	na	no	ntx

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-081	70362-50-4	34 / 77	0.396 J	6,970	ng/kg dw	1,000 - 1,000	7,000	not eval.	na	no	ntx
PCB-090	68194-07-0	34 / 34	180 C	11,700,000 C	ng/kg dw	na - na	11,700,000	not eval.	na	no	ntx
PCB-101[r]	37680-73-2	160 / 181	na	5,600,000 J	ng/kg dw	160 - 5,000	5,600,000	not eval.	na	no	ntx
PCB-105	32598-14-4	125 / 181	61.4	3,660,000	ng/kg dw	130 - 1,000	3,700,000	not eval.	na	no	ntx
PCB-110	38380-03-9	124 / 139	220 J	14,500,000 C	ng/kg dw	130 - 1,200	14,500,000	not eval.	na	no	ntx
PCB-113[r]	68194-10-5	34 / 34	na	na	ng/kg dw	na - na	na	not eval.	na	no	ntx
PCB-114	74472-37-0	38 / 77	2.75	207,000	ng/kg dw	1,000 - 12,000	210,000	not eval.	na	no	ntx
PCB-115[r]	74472-38-1	34 / 34	na	na	ng/kg dw	na - na	na	not eval.	na	no	ntx
PCB-118	31508-00-6	131 / 181	154	12,000,000	ng/kg dw	120 - 8,300	12,000,000	not eval.	na	no	ntx
PCB-123	65510-44-3	34 / 77	2.79	138,000	ng/kg dw	1,000 - 31,000	140,000	not eval.	na	no	ntx
PCB-126	57465-28-8	34 / 181	0.758 J	7,980	ng/kg dw	100 - 4,000	8,000	not eval.	na	no	ntx
PCB-128	38380-07-3	71 / 147	350 J	620,000 J	ng/kg dw	140 - 13,000	620,000	not eval.	na	no	ntx
PCB-129	55215-18-4	34 / 34	320 C	14,000,000 C	ng/kg dw	na - na	14,000,000	not eval.	na	no	ntx
PCB-138[r]	35065-28-2	163 / 182	na	1,400,000	ng/kg dw	130 - 5,000	1,400,000	not eval.	na	no	ntx
PCB-153	35065-27-1	158 / 181	258 C	9,090,000 C	ng/kg dw	130 - 4,400	9,100,000	not eval.	na	no	ntx
PCB-156	38380-08-4	80 / 182	27.5 C	1,790,000 C	ng/kg dw	80 - 1,000	1,800,000	not eval.	na	no	ntx
PCB-157[r]	69782-90-7	59 / 180	na	56,000	ng/kg dw	80 - 27,000	56,000	not eval.	na	no	ntx
PCB-160[r]	41411-62-5	34 / 34	na	na	ng/kg dw	na - na	na	not eval.	na	no	ntx
PCB-163[r]	74472-44-9	34 / 34	na	na	ng/kg dw	na - na	na	not eval.	na	no	ntx
PCB-167	52663-72-6	41 / 77	10.6	515,000	ng/kg dw	1,000 - 1,000	520,000	not eval.	na	no	ntx
PCB-168[r]	59291-65-5	34 / 34	na	na	ng/kg dw	na - na	na	not eval.	na	no	ntx
PCB-169	32774-16-6	0 / 182	nd	nd	ng/kg dw	0.399 - 1,900	1,900	not eval.	na	no	ntx
PCB-170	35065-30-6	90 / 148	190 J	360,000	ng/kg dw	80 - 12,000	360,000	not eval.	na	no	ntx
PCB-180	35065-29-3	138 / 182	155 C	1,600,000 C	ng/kg dw	110 - 4,900	1,600,000	not eval.	na	no	ntx
PCB-187	52663-68-0	29 / 43	1,000	360,000 J	ng/kg dw	1,000 - 1,000	360,000	not eval.	na	no	ntx
PCB-189	39635-31-9	48 / 182	3.06	65,700	ng/kg dw	110 - 5,000	66,000	not eval.	na	no	ntx
PCB-193[r]	69782-91-8	34 / 34	na	na	ng/kg dw	na - na	na	not eval.	na	no	ntx
PCB-195	52663-78-2	8 / 43	1,000 J	49,000 J	ng/kg dw	1,000 - 1,000	49,000	not eval.	na	no	ntx
PCB-206	40186-72-9	6 / 43	1,000 J	21,000 J	ng/kg dw	1,000 - 1,000	21,000	not eval.	na	no	ntx
PCB-209	2051-24-3	3 / 43	1,000	1,000	ng/kg dw	1,000 - 1,000	1,000	not eval.	na	no	ntx

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCBs (total calc'd)	na	481 / 524	2.2 J	220,000	ug/kg dw	5 - 40	220,000	not eval.	220 ca[s]	yes	asl
PCBs + PCTs (total)	na	105 / 105	2.2	26,000	ug/kg dw	na - na	26,000	not eval.	na	no	ntx
PCTs (total)	na	81 / 107	2.2 J	5,600	ug/kg dw	1.7 - 8.1	5,600	not eval.	na	no	ntx
p-Cymene	99-87-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
Pentachloroethane	76-01-7	0 / 7	nd	nd	ug/kg dw	2.4 - 7.4	7.4	not eval.	na	no	ntx
Pentachlorophenol	87-86-5	8 / 291	14 J	410	ug/kg dw	6.3 - 1,700	1,700	not eval.	3,000 ca	no	bsl
Perylene	198-55-0	13 / 13	9.0	230	ug/kg dw	na - na	230	not eval.	na	no	ntx
Phenanthrene	85-01-8	275 / 318	7.1	28,000	ug/kg dw	5 - 200	28,000	not eval.	na	no	ntx
Phenol	108-95-2	84 / 321	10 J	2,800	ug/kg dw	7.3 - 300	2,800	not eval.	1,800,000 nc	no	bsl
Potassium	7440-09-7	119 / 119	380	11,000	mg/kg dw	na - na	11,000	not eval.	na	no	ntx
Pyrene	129-00-0	300 / 318	7 J	16,000	ug/kg dw	18 - 170	16,000	not eval.	230,000 nc	no	bsl
Retene	483-65-8	2 / 4	99 J	100 J	ug/kg dw	340 - 350	350	not eval.	na	no	ntx
sec-Butylbenzene	135-98-8	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
Selenium	7782-49-2	69 / 225	0.2 J	20	mg/kg dw	1.0 - 34	34	not eval.	39 nc	no	bsl
Silver	7440-22-4	158 / 318	0.020	270	mg/kg dw	0.046 - 5	270	0.28 / 0.74	39 nc	yes	asl
Sodium	7440-23-5	114 / 114	580	21,000	mg/kg dw	na - na	21,000	not eval.	na	no	ntx
Styrene	100-42-5	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	170,000 nc	no	bsl
Sulfides (total)	na	42 / 102	4.0 J	1,500 J	mg/kg dw	0.68 - 46	1,500	not eval.	na	no	ntx
tert-Butyl methyl ether	1634-04-4	0 / 7	nd	nd	ug/kg dw	2.4 - 7.4	7.4	not eval.	32,000 ca	no	bsl
tert-Butylbenzene	98-06-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	39,000 nc	no	bsl
Tetrabutyltin as ion	1461-25-2	3 / 43	0.56 J	2.0 J	ug/kg dw	1.0 - 15	15	not eval.	na	no	ntx
Tetrachloroethene	127-18-4	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	480 ca	no	bsl
Thallium	7440-28-0	87 / 225	0.010 J	30	mg/kg dw	0.032 - 53	53	0.252 / 1.79	0.52 nc	yes	asl
Tin	7440-31-5	15 / 43	2.0 J	11 J	mg/kg dw	1.0 - 7.0	11	not eval.	4,700 nc	no	bsl
Toluene	108-88-3	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	52,000 nc	no	bsl
Total aldrin/dieldrin (calc'd)	na	8 / 75	0.113 J	280	ug/kg dw	0.81 - 91	280	not eval.	na	no	ntx
Total chlordane (calc'd)	na	20 / 62	0.20 J	230	ug/kg dw	1.0 - 330	330	not eval.	1,600 ca[t]	no	bsl
Total HPAH (calc'd)	na	310 / 318	20	85,000	ug/kg dw	19 - 120	85,000	not eval.	na	no	ntx
Total HpCDD	37871-00-4	5 / 5	120	1,300	ng/kg dw	na - na	1,300	not eval.	na	no	ntx
Total HpCDF	38998-75-3	5 / 5	18	430	ng/kg dw	na - na	430	not eval.	na	no	ntx

Table 2, continued

Chemical	CAS Number	Detection Frequency	Min imum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	Residential RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Total HxCDD	34465-46-8	5 / 5	12	200	ng/kg dw	na - na	200	not eval.	na	no	ntx
Total HxCDF	55684-94-1	5 / 5	9.7	170	ng/kg dw	na - na	170	not eval.	na	no	ntx
Total LPAH (calc'd)	na	278 / 318	9.1 J	43,000	ug/kg dw	19 - 200	43,000	not eval.	na	no	ntx
Total PAH (calc'd)	na	311 / 318	20	128,000	ug/kg dw	19 - 120	128,000	not eval.	na	no	ntx
Total PeCDD	36088-22-9	0 / 5	nd	nd	ng/kg dw	1.4 - 8.5	8.5	not eval.	na	no	ntx
Total PeCDF	30402-15-4	4 / 5	5.3	64	ng/kg dw	3.9 - 3.9	64	not eval.	na	no	ntx
Total TCDD	na	4 / 5	0.95	16	ng/kg dw	0.76 - 0.76	16	not eval.	na	no	ntx
Total TCDF	30402-14-3	5 / 5	3.0	51	ng/kg dw	na - na	51	not eval.	na	no	ntx
Total Xylenes (calc'd)	na	0 / 7	nd	nd	ug/kg dw	4.7 - 7.4	7.4	not eval.	na	no	bsl
Toxaphene	8001-35-2	2 / 73	340 J	6,300 J	ug/kg dw	10 - 4,300	6,300	not eval.	440 ca	yes	asl
TPH	na	29 / 29	130	23,000	mg/kg dw	na - na	23,000	not eval.	na	no	ntx
trans-1,2-Dichloroethene	156-60-5	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	6,900 nc	no	bsl
trans-1,3-Dichloropropene	10061-02-6	0 / 7	nd	nd	ug/kg dw	3.5 - 6.2	6.2	not eval.	na	no	ntx
trans-1,4-Dichloro-2-butene	110-57-6	0 / 6	nd	nd	ug/kg dw	12 - 18	18	not eval.	na	no	ntx
trans-Nonachlor	39765-80-5	0 / 25	nd	nd	ug/kg dw	1.9 - 34	34	not eval.	na	no	ntx
Tributyltin as ion	688-73-3	47 / 53	0.28 J	99	ug/kg dw	3.7 - 5.0	99	not eval.	900 nc[U]	no	bsl
Trichloroethene	79-01-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	53 ca	no	bsl
Trichlorofluoromethane	75-69-4	0 / 7	nd	nd	ug/kg dw	2.4 - 74	74	not eval.	39,000 nc	no	bsl
Vanadium	7440-62-2	206 / 206	15	87	mg/kg dw	na - na	87	36 / 59.6	7.8 nc	yes	asl
Vinyl chloride	75-01-4	0 / 7	nd	nd	ug/kg dw	2.4 - 18	18	not eval.	79 ca	no	bsl
Xylene (meta & para)	108-38-3/106-42-3	0 / 7	nd	nd	ug/kg dw	4.7 - 7.4	7.4	not eval.	na	no	ntx
Xylene (ortho)	95-47-6	0 / 7	nd	nd	ug/kg dw	2.4 - 3.7	3.7	not eval.	na	no	ntx
Zinc	7440-66-6	328 / 328	16	9,700	mg/kg dw	na - na	9,700	52.6 / 98.5	2,300 nc	yes	asl

[a] Background concentrations obtained from joint Ecology/PSAMP 1998 study entitled "Sediment Quality in Puget Sound. Year 2- Central Puget Sound" (Ecology 2000). Reported concentrations are mean and maximum from 52 sediment samples collected from the following areas: South Port Townsend, Port Townsend, North Admiralty Inlet, South Admiralty Inlet, Possession Sound, Central Basin, Port Madison, West Point, East Passage, Liberty Bay, Keyport, Northwest Bainbridge Island, Southwest Bainbridge Island, Rich Passage, Port Orchard, and Port Washington Narrows

[b] Risk-based concentrations (RBCs) are derived from EPA Region 9 Preliminary Remediation Goals (PRGs) for residential soil (last updated October 2004). PRGs associated with a non-cancer endpoint (abbreviated "nc") were divided by 10 for this screening, reflecting the different target hazard quotients used in Region 9 (HQ = 1) and Region 10 (HQ = 0.1). All other RBCs were not modified for this screening. Abbreviations: ca = cancer endpoint, nc = non-cancer endpoint.

[c] Less than 10% (29 of 414) of reporting limits for 2,4,6-trichlorophenol exceed the RBC.

[d] Less than 10% (1 of 390) of reporting limits for 3,3-dichlorobenzidine exceed the RBC.

Table 2, continued

[e] RBC for acenaphthylene is from acenaphthene.

[f] RBC for alpha-chlordane is from chlordane.

[g] RBC for alpha-endosulfan is from endosulfan.

[h] RBC for beta-endosulfan is from endosulfan.

[i] RBC for carcinogenic PAHs is from benzo(a)pyrene.

[j] RBC for chromium is from chromium VI.

[k] RBC for DDTs (total calc'd) is from 4,4-DDT.

[l] RBC for Dioxin/Furan TEQ is from 2,3,7,8-TCDD.

[m] RBC for endrin aldehyde is from endrin.

[n] RBC for endrin ketone is from endrin.

[o] RBC for gamma-chlordane is from chlordane.

[p] Less than 10% (2 of 171) of reporting limits for heptachlor epoxide exceed the RBC.

[q] RBC for PCB TEQ is from 2,3,7,8-TCDD.

[r] Statistics for this congener reflect only those samples that were determined not to co-elute with other congeners. When no statistics are given, "na" will appear in place of statistics, meaning that all samples co-eluted with another congener.

[s] RBC for PCBs (total calc'd) is from Aroclor 1254.

[t] RBC for total chlordane (calc'd) is from chlordane.

[u] RBC for tributyltin ion is the RBC for tribultytin oxide multiplied by 0.49 to account for differences in molecular weight.

Abbreviations:

COPC – chemical of potential concern
nd – not detected
n/a – not applicable
HPAH – high-molecular-weight polycyclic aromatic hydrocarbon
LPAH – low-molecular-weight polycyclic aromatic hydrocarbon
C – concentration represents coelution
J – estimated value
asl – above screening level (selected as COPC)
bsl – below screening level (not selected as COPC)
ifd – infrequent detection (not selected as COPC)
nsd – no significant difference between mean background and mean detected concentrations
ntx – no toxicity information (not selected as COPC)
sum – chemical included in sum and is not evaluated separately
teq – chemical included in TEQ calculation and is not evaluated separately

Table 3. Occurrence and selection of chemicals of potential concern for tissue in the seafood consumption exposure scenario

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
1,2,4-Trichlorobenzene	120-82-1	0 / 145	nd	nd	µg/kg ww	3.6 – 580	580	not eval.	870 nc	no	bsl
1,2-Dichlorobenzene	95-50-1	0 / 145	nd	nd	µg/kg ww	9 – 580	580	not eval.	7,800 nc	no	bsl
1,2-Diphenylhydrazine	122-66-7	0 / 35	nd	nd	µg/kg ww	3.6 – 80	80	not eval.	1.0 ca	yes	asl
1,3-Dichlorobenzene	541-73-1	0 / 145	nd	nd	µg/kg ww	9 – 580	580	not eval.	260 nc	yes	asl
1,4-Dichlorobenzene	106-46-7	0 / 145	nd	nd	µg/kg ww	9 – 580	580	not eval.	34 ca	yes	asl
2,4,5-Trichlorophenol	95-95-4	0 / 145	nd	nd	µg/kg ww	18 – 15,000	15,000	not eval.	8,700 nc	no	ifd[c]
2,4,6-Trichlorophenol	88-06-2	0 / 145	nd	nd	µg/kg ww	18 – 15,000	15,000	not eval.	75 ca	yes	asl
2,4'-DDD	na	30 / 110	1.6 JN	57 JN	µg/kg ww	1.0 – 88	88	not eval.	na	no	ntx
2,4'-DDE	na	3 / 110	3.2 JN	110 JN	µg/kg ww	0.8 – 18	110	not eval.	na	no	ntx
2,4'-DDT	na	108 / 110	2.1 JN	440 JN	µg/kg ww	3.2 – 3.5	440	not eval.	na	no	ntx
2,4-Dichlorophenol	120-83-2	0 / 145	nd	nd	µg/kg ww	3.6 – 1,200	1,200	not eval.	260 nc	yes	asl
2,4-Dimethylphenol	105-67-9	0 / 145	nd	nd	µg/kg ww	3.6 – 1,200	1,200	not eval.	1,700 nc	no	bsl
2,4-Dinitrophenol	51-28-5	0 / 130	nd	nd	µg/kg ww	31 – 29,000	29,000	not eval.	170 nc	yes	asl
2,4-Dinitrotoluene	121-14-2	0 / 145	nd	nd	µg/kg ww	6 – 15,000	15,000	not eval.	170 nc	yes	asl
2,6-Dinitrotoluene	606-20-2	0 / 145	nd	nd	µg/kg ww	6 – 15,000	15,000	not eval.	87 nc	yes	asl
2-Chloronaphthalene	91-58-7	0 / 145	nd	nd	µg/kg ww	9 – 2,900	2,900	not eval.	6,900 nc	no	bsl
2-Chlorophenol	95-57-8	0 / 145	nd	nd	µg/kg ww	3.6 – 1,200	1,200	not eval.	430 nc	yes	asl
2-Methylnaphthalene	91-57-6	87 / 145	0.41 J	10	µg/kg ww	0.24 – 64	64	not eval.	350 nc	no	bsl
2-Methylphenol	95-48-7	19 / 145	28	1,100 J	µg/kg ww	3.6 – 1,200	1,200	not eval.	4,300 nc	no	bsl
2-Nitroaniline	na	0 / 145	nd	nd	µg/kg ww	7.1 – 15,000	15,000	not eval.	na	no	ntx
2-Nitrophenol	na	0 / 145	nd	nd	µg/kg ww	3.6 – 5,200	5,200	not eval.	na	no	ntx
3,3'-Dichlorobenzidine	91-94-1	0 / 112	nd	nd	µg/kg ww	27 – 29,000	29,000	not eval.	1.8 ca	yes	asl
3-Nitroaniline	99-09-2	0 / 124	nd	nd	µg/kg ww	3.6 – 29,000	29,000	not eval.	41 ca	yes	asl
4,4'-DDD	72-54-8	82 / 130	0.23 JN	20 JN	µg/kg ww	0.8 – 7.2	20	not eval.	3.4 ca	no	sum
4,4'-DDE	72-55-9	100 / 130	0.70 JN	20 JN	µg/kg ww	1.0 – 14	20	not eval.	2.4 ca	no	sum
4,4'-DDT	50-29-3	110 / 130	0.84 JN	470 JN	µg/kg ww	1.3 – 2.0	470	not eval.	2.4 ca	no	sum
4,6-Dinitro-o-cresol	534-52-1	0 / 145	nd	nd	µg/kg ww	31 – 29,000	29,000	not eval.	8.7 nc	yes	asl

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
4-Bromophenyl phenyl ether	na	0 / 143	nd	nd	µg/kg ww	6 – 580	580	not eval.	na	no	ntx
4-Chloro-3-methylphenol	na	0 / 145	nd	nd	µg/kg ww	3.6 – 2,900	2,900	not eval.	na	no	ntx
4-Chloroaniline	106-47-8	0 / 113	nd	nd	µg/kg ww	36 – 2,900	2,900	not eval.	350 nc	yes	asl
4-Chlorophenyl phenyl ether	na	0 / 145	nd	nd	µg/kg ww	3.6 – 2,900	2,900	not eval.	na	no	ntx
4-Methylphenol	106-44-5	8 / 145	15 J	1,500	µg/kg ww	3.6 – 1,200	1,500	not eval.	430 nc	yes	asl
4-Nitroaniline	100-01-6	0 / 133	nd	nd	µg/kg ww	18 – 29,000	29,000	not eval.	41 ca	yes	asl
4-Nitrophenol	na	2 / 145	530 J	530 J	µg/kg ww	31 – 29,000	29,000	not eval.	na	no	ntx
Acenaphthene	83-32-9	104 / 145	0.13 J	22	µg/kg ww	0.72 – 16	22	not eval.	5,200 nc	no	bsl
Acenaphthylene	208-96-8	88 / 145	0.13 J	2.8	µg/kg ww	0.26 – 24	24	not eval.	5,200 nc[d]	no	bsl
Aldrin	309-00-2	5 / 130	0.77 JN	6.2 JN	µg/kg ww	0.50 – 10	10	not eval.	0.048 ca	yes	asl
alpha-BHC	319-84-6	7 / 130	0.35 JN	1.2 JNM	µg/kg ww	0.50 – 10	10	not eval.	0.13 ca	yes	asl
alpha-Chlordane	5103-71-9	31 / 119	0.60 JN	6.6 JN	µg/kg ww	0.50 – 10	10	not eval.	2.3 ca[e]	no	sum
alpha-Endosulfan	959-98-8	34 / 130	0.34 JN	6.6 JN	µg/kg ww	0.50 – 10	10	not eval.	520 nc[f]	no	bsl
Aniline	62-53-3	0 / 132	nd	nd	µg/kg ww	53 – 12,000	12,000	not eval.	140 ca	yes	asl
Anthracene	120-12-7	103 / 145	0.090 J	9.0	µg/kg ww	0.41 – 24	24	not eval.	26,000 nc	no	bsl
Antimony	7440-36-0	102 / 142	0.0009 J	0.252	mg/kg ww	0.0079 – 0.020	0.25	not eval.	0.035 nc	yes	asl
Aroclor-1016	12674-11-2	0 / 212	nd	nd	µg/kg ww	5.3 – 200	200	not eval.	12 ca	no	sum
Aroclor-1016/1242	na	9 / 9	5.6 J	16 J	µg/kg ww	na	16	not eval.	na	no	ntx
Aroclor-1221	11104-28-2	0 / 212	nd	nd	µg/kg ww	5.3 – 290	290	not eval.	0.41 ca	no	sum
Aroclor-1232	11141-16-5	0 / 212	nd	nd	µg/kg ww	5.3 – 200	200	not eval.	0.41 ca	no	sum
Aroclor-1242	53469-21-9	0 / 212	nd	nd	µg/kg ww	5.3 – 200	200	not eval.	0.41 ca	no	sum
Aroclor-1248	12672-29-6	97 / 221	9.0	4,400	µg/kg ww	0.21 – 200	4,400	not eval.	0.41 ca	no	sum
Aroclor-1254	11097-69-1	214 / 221	16	7,600	µg/kg ww	13 – 20	7,600	not eval.	0.41 ca	no	sum
Aroclor-1260	11096-82-5	184 / 221	22	7,100	µg/kg ww	10 – 20	7,100	not eval.	0.41 ca	no	sum
Arsenic	7440-38-2	148 / 148	0.274	15	mg/kg ww	na	15	7.7	0.00055 ca	no	other[g]
Arsenic (inorganic)	na	42 / 46	0.003	3.27	mg/kg ww	0.003 – 0.010	3.3	not eval.	0.00055 ca[h]	yes	asl
Benzidine	92-87-5	0 / 77	nd	nd	µg/kg ww	5,000 – 72,000	72,000	not eval.	0.0036 ca	yes	asl
Benzo(a)anthracene	56-55-3	101 / 145	0.069 J	42	µg/kg ww	0.36 – 24	42	not eval.	1.1 ca	no	teq
Benzo(a)pyrene	50-32-8	58 / 145	0.13 J	26	µg/kg ww	0.36 – 40	40	not eval.	0.11 ca	no	teq
Benzo(b)fluoranthene	205-99-2	73 / 145	0.12 J	44	µg/kg ww	0.36 – 64	64	not eval.	1.1 ca	no	teq

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Benzo(g,h,i)perylene	na	56 / 145	0.16 J	32	µg/kg ww	0.16 – 40	40	not eval.	na	no	ntx
Benzo(k)fluoranthene	207-08-9	70 / 145	0.16	38	µg/kg ww	0.36 – 64	64	not eval.	11 ca	no	teq
Benzofluoranthenes (total-calc'd)	na	73 / 145	0.12 J	82	µg/kg ww	0.36 – 64	82	not eval.	na	no	ntx
Benzoic acid	65-85-0	78 / 145	340 J	54,000	µg/kg ww	36 – 12,000	54,000	65	350,000 nc	no	bsl
Benzyl alcohol	100-51-6	23 / 145	12 J	610	µg/kg ww	3.6 – 1,200	1,200	14	26,000 nc	no	bsl
beta-BHC	319-85-7	37 / 130	0.41 JN	15 JN	µg/kg ww	0.50 – 10	15	not eval.	0.46 ca	yes	asl
beta-Endosulfan	33213-65-9	23 / 130	2.0 JN	44 JN	µg/kg ww	0.8 – 11	44	not eval.	520 nc[i]	no	bsl
bis(2-chloroethoxy)methane	na	1 / 145	240 J	240 J	µg/kg ww	3.6 – 2,900	2,900	not eval.	na	no	ntx
bis(2-chloroethyl)ether	111-44-4	0 / 145	nd	nd	µg/kg ww	3.6 – 1,200	1,200	not eval.	0.75 ca	yes	asl
bis(2-chloroisopropyl)ether	108-60-1	0 / 145	nd	nd	µg/kg ww	11 – 580	580	not eval.	12 ca	yes	asl
Bis(2-ethylhexyl)phthalate	117-81-7	22 / 145	28	2,100 J	µg/kg ww	3.6 – 3,600	3,600	140	59 ca	yes	asl
Butyl benzyl phthalate	85-68-7	20 / 142	300	1,700 M	µg/kg ww	9 – 1,200	1,700	not eval.	430 ca	yes	asl
Cadmium	7440-43-9	130 / 142	0.0013 J	0.84	mg/kg ww	0.0042 – 0.0079	0.84	not eval.	0.087 nc	yes	asl
Caffeine	na	0 / 32	nd	nd	µg/kg ww	3.1 – 8.0	8.0	not eval.	na	no	ntx
Carbazole	86-74-8	2 / 145	6,000	14,000	µg/kg ww	3.6 – 2,900	14,000	not eval.	41 ca	yes	asl
Carcinogenic PAHs – mammal – full DL	na	104 / 145	0.33	59	µg/kg ww	na	85	not eval.	na	no	ntx
Carcinogenic PAHs – mammal – half DL	na	104 / 145	0.33	44	µg/kg ww	na	44	not eval.	0.11 ca[j]	yes	asl
Carcinogenic PAHs – mammal – zero DL	na	104 / 145	0.0 JM	44	µg/kg ww	na	44	not eval.	na	no	ntx
Chlordane	57-74-9	0 / 11	nd	nd	µg/kg ww	6.7 – 6.7	6.7	not eval.	2.3 ca	no	sum
Chromium	7440-47-3	92 / 142	0.054	3.74	mg/kg ww	0.03 – 0.14	3.7	not eval.	0.26 nc[k]	yes	asl
Chrysene	218-01-9	102 / 145	0.12 J	85	µg/kg ww	0.36 – 24	85	not eval.	110 ca	no	bsl
Cobalt	na	121 / 121	0.0037 J	0.7110	mg/kg ww	na	0.7110	not eval.	na	no	ntx
Copper	7440-50-8	148 / 148	0.18	24 M	mg/kg ww	na	24	0.31	3.5 nc	yes	asl
Coprostanol	na	0 / 35	nd	nd	µg/kg ww	60 – 180	180	not eval.	na	no	ntx
DDTs (total-calc'd)	na	117 / 130	1.1	1,020 JN	µg/kg ww	1.3 – 2.0	1,000	0.77	2.4 ca[l]	yes	asl
delta-BHC	na	3 / 130	0.51 JN	2.2 JN	µg/kg ww	0.50 – 10	10	not eval.	na	no	ntx
Dibenzo(a,h)anthracene	53-70-3	28 / 145	0.12 J	5.1	µg/kg ww	0.18 – 64	64	not eval.	0.11 ca	no	teq
Dibenzofuran	na	106 / 145	0.097 J	9.5	µg/kg ww	0.11 – 40	40	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Dibutyltin as ion	na	94 / 120	0.53 J	17	µg/kg ww	1.5 – 3.9	17	not eval.	na	no	ntx
Dieldrin	60-57-1	6 / 130	1.3 JN	5.0 JN	µg/kg ww	1.0 – 24	24	not eval.	0.051 ca	yes	asl
Diethyl phthalate	84-66-2	37 / 145	9.5 J	900 J	µg/kg ww	3.6 – 1,200	1,200	not eval.	69,000 nc	no	bsl
Dimethyl phthalate	na	5 / 145	7.6 J	424 JM	µg/kg ww	3.6 – 2,900	2,900	not eval.	na	no	ntx
Di-n-butyl phthalate	84-74-2	9 / 145	19 J	2,300	µg/kg ww	3.6 – 1,200	2,300	not eval.	8,700 nc	no	bsl
Di-n-octyl phthalate	na	0 / 145	nd	nd	µg/kg ww	3.6 – 2,900	2,900	not eval.	na	no	ntx
Endosulfan sulfate	na	0 / 130	nd	nd	µg/kg ww	0.8 – 10	10	not eval.	33 nc[m]	no	bsl
Endrin	72-20-8	26 / 130	0.10 JN	40 JN	µg/kg ww	0.8 – 72	72	not eval.	26 nc	yes	asl
Endrin aldehyde	7421-93-4	18 / 130	0.42 JN	78 JN	µg/kg ww	0.8 – 10	78	not eval.	26 nc[n]	yes	asl
Endrin ketone	53494-70-5	0 / 110	nd	nd	µg/kg ww	0.9 – 15	15	not eval.	26 nc[o]	no	bsl
Fluoranthene	206-44-0	126 / 145	0.18 J	120	µg/kg ww	0.87 – 24	120	not eval.	3,500 nc	no	bsl
Fluorene	86-73-7	110 / 145	0.094 J	7.1	µg/kg ww	3.6 – 24	24	not eval.	3,500 nc	no	bsl
gamma-BHC	58-89-9	14 / 130	0.51 JN	7.4 JNM	µg/kg ww	0.50 – 10	10	not eval.	0.63 ca	yes	asl
gamma-Chlordane	5103-74-2	110 / 119	0.52 J	330 JN	µg/kg ww	0.50 – 56	330	not eval.	2.3 ca[p]	no	sum
Heptachlor	76-44-8	3 / 130	5.7 JN	9.7 JN	µg/kg ww	0.50 – 10	10	not eval.	0.18 ca	yes	asl
Heptachlor epoxide	1024-57-3	53 / 130	0.93 JN	45 JN	µg/kg ww	0.50 – 10	45	not eval.	0.090 ca	yes	asl
Hexachlorobenzene	118-74-1	20 / 145	0.38 JN	6.6 JN	µg/kg ww	0.8 – 24	24	not eval.	0.51 ca	yes	asl
Hexachlorobutadiene	87-68-3	0 / 145	nd	nd	µg/kg ww	11 – 580	580	not eval.	11 ca	yes	asl
Hexachlorocyclopentadiene	77-47-4	0 / 142	nd	nd	µg/kg ww	16 – 360,000	360,000	not eval.	520 nc	yes	asl
Hexachloroethane	67-72-1	0 / 144	nd	nd	µg/kg ww	11 – 580	580	not eval.	59 ca	yes	asl
Indeno(1,2,3-cd)pyrene	193-39-5	55 / 145	0.11 J	28	µg/kg ww	0.13 – 40	40	not eval.	1.1 ca	no	teq
Isophorone	78-59-1	0 / 145	nd	nd	µg/kg ww	3.6 – 580	580	not eval.	860 ca	no	bsl
Lead	na	139 / 148	0.006	6.370	mg/kg ww	0.020 – 0.030	6.4	not eval.	na	no	ntx
Mercury	7439-97-6	159 / 159	0.005	0.11 J	mg/kg ww	na	0.11	0.051	0.0087 nc[q]	yes	asl
Methoxychlor	72-43-5	7 / 130	0.63 JN	130 JN	µg/kg ww	0.8 – 23	130	not eval.	430 nc	no	bsl
Methylmercury	22967-92-6	3 / 3	18	25	µg/kg ww	na	25	not eval.	8.7 nc	no	sum
Mirex	2385-85-5	0 / 110	nd	nd	µg/kg ww	0.8 – 10	10	not eval.	17 nc	no	bsl
Molybdenum	7439-98-7	120 / 120	0.0025 J	0.4100	mg/kg ww	na	0.410	not eval.	0.43 nc	no	bsl
Monobutyltin as ion	na	72 / 120	0.57 J	4.9 J	µg/kg ww	0.8 – 1.7	4.9	not eval.	na	no	ntx
Naphthalene	91-20-3	41 / 145	0.80 J	12	µg/kg ww	0.57 – 64	64	not eval.	1,700 nc	no	bsl

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
Nickel	7440-02-0	139 / 142	0.015 J	2.060	mg/kg ww	0.020 – 0.020	2.1	not eval.	1.7 nc	yes	asl
Nitrobenzene	98-95-3	0 / 145	nd	nd	µg/kg ww	11 – 580	580	not eval.	43 nc	yes	asl
n-Nitrosodimethylamine	62-75-9	0 / 145	nd	nd	µg/kg ww	3.6 – 12,000	12,000	not eval.	0.016 ca	yes	asl
n-Nitroso-di-n-propylamine	621-64-7	1 / 145	270 JN	270 JN	µg/kg ww	3.6 – 580	580	not eval.	0.12 ca	yes	asl
n-Nitrosodiphenylamine	86-30-6	0 / 145	nd	nd	µg/kg ww	3.6 – 2,900	2,900	not eval.	170 ca	yes	asl
PCB congeners (total calc'd)	na	49 / 49	41,050 J	12,228,000 J	ng/kg ww	na	12,200,000	not eval.	410 ca[r]	no	sum
PCB TEQ – mammal – half DL	na	52 / 52	0.440	73.0 J	ng/kg ww	na	73.0	not eval.	0.0055 ca[s]	yes	asl
PCB-001	na	27 / 48	1.04 J	74.8	ng/kg ww	0.686 – 23.7	74.8	not eval.	na	no	ntx
PCB-002	na	26 / 49	0.263 J	13.3	ng/kg ww	0.269 – 13.2	13.3	not eval.	na	no	ntx
PCB-003	na	26 / 49	0.401 J	39.3 J	ng/kg ww	0.466 – 20.5	39.3	not eval.	na	no	ntx
PCB-004	na	49 / 49	4.68	1,700	ng/kg ww	na	1,700	not eval.	na	no	ntx
PCB-005	na	32 / 49	0.222 J	34.1	ng/kg ww	0.257 – 16.2	34.1	not eval.	na	no	ntx
PCB-006	na	49 / 49	1.25 J	3,240	ng/kg ww	na	3,240	not eval.	na	no	ntx
PCB-007	na	46 / 49	0.391 J	698	ng/kg ww	6.48 – 13.3	698	not eval.	na	no	ntx
PCB-008	na	49 / 49	1.69 J	2,570	ng/kg ww	na	2,570	not eval.	na	no	ntx
PCB-009	na	49 / 49	0.490 J	222	ng/kg ww	na	222	not eval.	na	no	ntx
PCB-010	na	37 / 49	0.165 J	80.7	ng/kg ww	0.238 – 15.7	80.7	not eval.	na	no	ntx
PCB-011	na	29 / 49	1.51 J	263	ng/kg ww	2.05 – 31.7	263	not eval.	na	no	ntx
PCB-012	na	40 / 49	1.49 CJ	532 C	ng/kg ww	7.99 – 28.8	532	not eval.	na	no	ntx
PCB-013[t]	na	40 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-014	na	4 / 49	0.0980 J	2.05	ng/kg ww	0.0570 – 16.7	16.7	not eval.	na	no	ntx
PCB-015	na	47 / 49	13.6	1,320	ng/kg ww	88.5 – 96.2	1,320	not eval.	na	no	ntx
PCB-016	na	49 / 49	8.90	3,780	ng/kg ww	na	3,780	not eval.	na	no	ntx
PCB-017	na	49 / 49	19.2	8,700	ng/kg ww	na	8,700	not eval.	na	no	ntx
PCB-018	na	49 / 49	114 C	24,900 C	ng/kg ww	na	24,900	not eval.	na	no	ntx
PCB-019	na	49 / 49	2.01 J	2,330	ng/kg ww	na	2,330	not eval.	na	no	ntx
PCB-020	na	49 / 49	630 C	75,100 C	ng/kg ww	na	75,100	not eval.	na	no	ntx
PCB-021	na	44 / 49	16.13 CM	9,010 C	ng/kg ww	0.658 – 1.09	9,010	not eval.	na	no	ntx
PCB-022	na	49 / 49	123	8,300	ng/kg ww	na	8,300	not eval.	na	no	ntx
PCB-023	na	20 / 49	0.371 J	32.8	ng/kg ww	0.649 – 16.4	32.8	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-024	na	47 / 49	0.237 J	232	ng/kg ww	0.858 – 1.53	232	not eval.	na	no	ntx
PCB-025	na	49 / 49	7.62	55,000	ng/kg ww	na	55,000	not eval.	na	no	ntx
PCB-026	na	49 / 49	129 C	151,000 C	ng/kg ww	na	151,000	not eval.	na	no	ntx
PCB-027	na	49 / 49	0.0855 J	7,670	ng/kg ww	na	7,670	not eval.	na	no	ntx
PCB-028[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-029[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-030[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-031	na	49 / 49	391	55,100	ng/kg ww	na	55,100	not eval.	na	no	ntx
PCB-032	na	48 / 49	14.1	9,340	ng/kg ww	8.66 – 8.66	9,340	not eval.	na	no	ntx
PCB-033[t]	na	44 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-034	na	46 / 49	1.02 J	681	ng/kg ww	0.747 – 0.970	681	not eval.	na	no	ntx
PCB-035	na	15 / 49	0.350 J	49.6	ng/kg ww	0.357 – 17.7	49.6	not eval.	na	no	ntx
PCB-036	na	8 / 49	0.646 J	63.7	ng/kg ww	0.197 – 15.6	63.7	not eval.	na	no	ntx
PCB-037	na	49 / 49	60.6	6,560	ng/kg ww	na	6,560	not eval.	na	no	ntx
PCB-038	na	31 / 49	0.839 J	223 J	ng/kg ww	0.206 – 8.27	223	not eval.	na	no	ntx
PCB-039	na	42 / 49	1.95 J	184	ng/kg ww	3.26 – 7.49	184	not eval.	na	no	ntx
PCB-040	na	49 / 49	172 C	32,600 C	ng/kg ww	na	32,600	not eval.	na	no	ntx
PCB-041[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-042	na	49 / 49	186	27,900	ng/kg ww	na	27,900	not eval.	na	no	ntx
PCB-043	na	43 / 49	34.0	2,320	ng/kg ww	0.138 – 0.469	2,320	not eval.	na	no	ntx
PCB-044	na	49 / 49	994 C	328,000 C	ng/kg ww	na	328,000	not eval.	na	no	ntx
PCB-045	na	49 / 49	18.5 CJ	8,600 C	ng/kg ww	na	8,600	not eval.	na	no	ntx
PCB-046	na	49 / 49	2.89	3,830	ng/kg ww	na	3,830	not eval.	na	no	ntx
PCB-047[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-048	na	49 / 49	87.6	8,050	ng/kg ww	na	8,050	not eval.	na	no	ntx
PCB-049	na	49 / 49	781 C	396,000 C	ng/kg ww	na	396,000	not eval.	na	no	ntx
PCB-050	na	49 / 49	13.8 C	23,000 C	ng/kg ww	na	23,000	not eval.	na	no	ntx
PCB-051[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-052	na	52 / 52	1,680	770,000	ng/kg ww	na	770,000	not eval.	na	no	ntx
PCB-053[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-054	na	40 / 49	0.65 JM	255	ng/kg ww	0.0799 – 0.37	255	not eval.	na	no	ntx
PCB-055	na	23 / 49	18.1	1,050	ng/kg ww	0.496 – 1,000	1,050	not eval.	na	no	ntx
PCB-056	na	48 / 49	153	15,800	ng/kg ww	2.23 – 2.23	15,800	not eval.	na	no	ntx
PCB-057	na	49 / 49	5.07	11,500	ng/kg ww	na	11,500	not eval.	na	no	ntx
PCB-058	na	48 / 49	5.77	2,700	ng/kg ww	5.40 – 5.40	2,700	not eval.	na	no	ntx
PCB-059	na	49 / 49	106 C	20,500 C	ng/kg ww	na	20,500	not eval.	na	no	ntx
PCB-060	na	49 / 49	197	16,100	ng/kg ww	na	16,100	not eval.	na	no	ntx
PCB-061	na	49 / 49	1,480 C	335,000 C	ng/kg ww	na	335,000	not eval.	na	no	ntx
PCB-062[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-063	na	49 / 49	36.5	13,500	ng/kg ww	na	13,500	not eval.	na	no	ntx
PCB-064	na	49 / 49	346	65,900	ng/kg ww	na	65,900	not eval.	na	no	ntx
PCB-065[s]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-066	na	49 / 49	1,040	217,000	ng/kg ww	na	217,000	not eval.	na	no	ntx
PCB-067	na	48 / 49	31.5	9,160	ng/kg ww	18.2 – 18.2	9,160	not eval.	na	no	ntx
PCB-068	na	49 / 49	15.1	12,700	ng/kg ww	na	12,700	not eval.	na	no	ntx
PCB-069[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-070[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-071[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-072	na	49 / 49	33.2	21,100	ng/kg ww	na	21,100	not eval.	na	no	ntx
PCB-073	na	19 / 49	51.6	1,010	ng/kg ww	0.0251 – 4.99	1,010	not eval.	na	no	ntx
PCB-074[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-075	na	52 / 52	1,800	3,600	ng/kg ww	na	3,600	not eval.	na	no	ntx
PCB-076[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-077	na	49 / 52	71.5	5,940	ng/kg ww	110 – 120	5,940	not eval.	na	no	ntx
PCB-078	na	0 / 49	nd	nd	ng/kg ww	0.436 – 1,020	1,020	not eval.	na	no	ntx
PCB-079	na	49 / 49	17.9 J	9,580	ng/kg ww	na	9,580	not eval.	na	no	ntx
PCB-080	na	1 / 49	321	321	ng/kg ww	0.362 – 910	910	not eval.	na	no	ntx
PCB-081	na	47 / 52	3.04	187	ng/kg ww	10.8 – 180	187	not eval.	na	no	ntx
PCB-082	na	49 / 49	134	14,700	ng/kg ww	na	14,700	not eval.	na	no	ntx
PCB-083	na	49 / 49	1,390 C	686,000 C	ng/kg ww	na	686,000	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-084	na	49 / 49	315	155,000	ng/kg ww	na	155,000	not eval.	na	no	ntx
PCB-085	na	49 / 49	313 C	80,800 C	ng/kg ww	na	80,800	not eval.	na	no	ntx
PCB-086	na	49 / 49	1,050 C	423,000 C	ng/kg ww	na	423,000	not eval.	na	no	ntx
PCB-087[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-088	na	49 / 49	286 C	105,000 C	ng/kg ww	na	105,000	not eval.	na	no	ntx
PCB-089	na	43 / 49	4.35 J	1,680	ng/kg ww	0.256 – 5.1	1,680	not eval.	na	no	ntx
PCB-090	na	49 / 49	2,240 C	886,000 C	ng/kg ww	na	886,000	not eval.	na	no	ntx
PCB-091[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-092	na	49 / 49	631	241,000	ng/kg ww	na	241,000	not eval.	na	no	ntx
PCB-093	na	49 / 49	1,820 C	705,000 C	ng/kg ww	na	705,000	not eval.	na	no	ntx
PCB-094	na	47 / 49	4.58 J	2,090	ng/kg ww	0.256 – 29.6	2,090	not eval.	na	no	ntx
PCB-095[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-096	na	48 / 49	1.04	2,510	ng/kg ww	0.695 – 0.695	2,510	not eval.	na	no	ntx
PCB-097[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-098[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-099[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-100[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-101	na	52 / 52	9,000	14,000	ng/kg ww	na	14,000	not eval.	na	no	ntx
PCB-102[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-103	na	49 / 49	36.8	13,500	ng/kg ww	na	13,500	not eval.	na	no	ntx
PCB-104	na	47 / 49	0.222 J	73.3	ng/kg ww	0.197 – 4.66	73.3	not eval.	na	no	ntx
PCB-105	na	52 / 52	478	195,000	ng/kg ww	na	195,000	not eval.	na	no	ntx
PCB-106	na	0 / 49	nd	nd	ng/kg ww	0.246 – 77.6	77.6	not eval.	na	no	ntx
PCB-107	na	49 / 49	64.3 C	19,000 C	ng/kg ww	na	19,000	not eval.	na	no	ntx
PCB-108[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-109	na	49 / 49	116	80,300	ng/kg ww	na	80,300	not eval.	na	no	ntx
PCB-110	na	49 / 49	1,980 C	906,000 C	ng/kg ww	na	906,000	not eval.	na	no	ntx
PCB-111	na	49 / 49	3.36	1,310	ng/kg ww	na	1,310	not eval.	na	no	ntx
PCB-112	na	2 / 49	46.5	93.6	ng/kg ww	0.0879 – 22.4	93.6	not eval.	na	no	ntx
PCB-113[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-114	na	49 / 49	29.8	11,400	ng/kg ww	na	11,400	not eval.	na	no	ntx
PCB-115[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-116[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-117[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-118	na	52 / 52	1,290	812,000	ng/kg ww	na	812,000	not eval.	na	no	ntx
PCB-119[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-120	na	49 / 49	5.69	8,790	ng/kg ww	na	8,790	not eval.	na	no	ntx
PCB-121	na	33 / 49	1.20 J	275	ng/kg ww	0.179 – 6.63	275	not eval.	na	no	ntx
PCB-122	na	45 / 49	23.9	3,650	ng/kg ww	3.98 – 45.1	3,650	not eval.	na	no	ntx
PCB-123	na	49 / 49	32.6	7,750	ng/kg ww	na	7,750	not eval.	na	no	ntx
PCB-124[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-125[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-126	na	49 / 52	3.31	370 J	ng/kg ww	160 – 180	370	not eval.	na	no	ntx
PCB-127	na	49 / 49	2.42	1,550	ng/kg ww	na	1,550	not eval.	na	no	ntx
PCB-128	na	52 / 52	295 C	124,000 C	ng/kg ww	na	124,000	not eval.	na	no	ntx
PCB-129	na	49 / 49	2,350 C	854,000 C	ng/kg ww	na	854,000	not eval.	na	no	ntx
PCB-130	na	49 / 49	189	56,100	ng/kg ww	na	56,100	not eval.	na	no	ntx
PCB-131	na	49 / 49	22.6	6,270	ng/kg ww	na	6,270	not eval.	na	no	ntx
PCB-132	na	49 / 49	652	209,000	ng/kg ww	na	209,000	not eval.	na	no	ntx
PCB-133	na	49 / 49	87.4	15,000	ng/kg ww	na	15,000	not eval.	na	no	ntx
PCB-134	na	49 / 49	141 C	44,500 C	ng/kg ww	na	44,500	not eval.	na	no	ntx
PCB-135	na	49 / 49	1,200 C	334,000 C	ng/kg ww	na	334,000	not eval.	na	no	ntx
PCB-136	na	49 / 49	309	73,800	ng/kg ww	na	73,800	not eval.	na	no	ntx
PCB-137	na	49 / 49	100	47,800	ng/kg ww	na	47,800	not eval.	na	no	ntx
PCB-138	na	52 / 52	6,600	12,000	ng/kg ww	na	12,000	not eval.	na	no	ntx
PCB-139	na	49 / 49	48.7 C	17,600 C	ng/kg ww	na	17,600	not eval.	na	no	ntx
PCB-140[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-141	na	52 / 52	257	183,000	ng/kg ww	na	183,000	not eval.	na	no	ntx
PCB-142	na	0 / 49	nd	nd	ng/kg ww	0.383 – 96.8	96.8	not eval.	na	no	ntx
PCB-143[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-144	na	49 / 49	112	41,700	ng/kg ww	na	41,700	not eval.	na	no	ntx
PCB-145	na	36 / 49	0.732 J	224	ng/kg ww	0.0821 – 3.22	224	not eval.	na	no	ntx
PCB-146	na	49 / 49	627	158,000	ng/kg ww	na	158,000	not eval.	na	no	ntx
PCB-147	na	49 / 49	2,260 C	515,000 C	ng/kg ww	na	515,000	not eval.	na	no	ntx
PCB-148	na	49 / 49	9.13	1,380	ng/kg ww	na	1,380	not eval.	na	no	ntx
PCB-149	na	52 / 52	6,400	11,000	ng/kg ww	na	11,000	not eval.	na	no	ntx
PCB-150	na	49 / 49	5.78	988	ng/kg ww	na	988	not eval.	na	no	ntx
PCB-151	na	49 / 52	nd	nd	ng/kg ww	160 – 180	180	not eval.	na	no	ntx
PCB-152	na	49 / 49	0.996	639	ng/kg ww	na	639	not eval.	na	no	ntx
PCB-153	na	52 / 52	2,590 C	1,070,000 C	ng/kg ww	na	1,070,000	not eval.	na	no	ntx
PCB-154[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-155	na	49 / 49	0.475 J	60.4	ng/kg ww	na	60.4	not eval.	na	no	ntx
PCB-156	na	49 / 49	163 C	108,000 C	ng/kg ww	na	108,000	not eval.	na	no	ntx
PCB-157[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-158	na	49 / 49	207	81,200	ng/kg ww	na	81,200	not eval.	na	no	ntx
PCB-159	na	41 / 49	31.6	3,970	ng/kg ww	0.299 – 0.548	3,970	not eval.	na	no	ntx
PCB-160[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-161	na	0 / 49	nd	nd	ng/kg ww	0.282 – 66.0	66.0	not eval.	na	no	ntx
PCB-162	na	49 / 49	7.25	2,670	ng/kg ww	na	2,670	not eval.	na	no	ntx
PCB-163[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-164	na	49 / 49	214	46,300	ng/kg ww	na	46,300	not eval.	na	no	ntx
PCB-165	na	40 / 49	2.31	370	ng/kg ww	2.71 – 66.9	370	not eval.	na	no	ntx
PCB-166[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-167	na	49 / 49	85.8	34,900	ng/kg ww	na	34,900	not eval.	na	no	ntx
PCB-168[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-169	na	16 / 52	0.468	22.4	ng/kg ww	0.378 – 180	180	not eval.	na	no	ntx
PCB-170	na	52 / 52	232	366,000	ng/kg ww	na	366,000	not eval.	na	no	ntx
PCB-171	na	49 / 49	184 C	109,000 C	ng/kg ww	na	109,000	not eval.	na	no	ntx
PCB-172	na	49 / 49	43.1	59,700	ng/kg ww	na	59,700	not eval.	na	no	ntx
PCB-173[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-174	na	52 / 52	296	90,000	ng/kg ww	na	90,000	not eval.	na	no	ntx
PCB-175	na	49 / 49	37.8	14,700	ng/kg ww	na	14,700	not eval.	na	no	ntx
PCB-176	na	49 / 49	86.7	27,200	ng/kg ww	na	27,200	not eval.	na	no	ntx
PCB-177	na	52 / 52	547	223,000	ng/kg ww	na	223,000	not eval.	na	no	ntx
PCB-178	na	49 / 49	276	78,800	ng/kg ww	na	78,800	not eval.	na	no	ntx
PCB-179	na	49 / 49	318	119,000	ng/kg ww	na	119,000	not eval.	na	no	ntx
PCB-180	na	52 / 52	1,040 C	1,080,000 C	ng/kg ww	na	1,080,000	not eval.	na	no	ntx
PCB-181	na	49 / 49	3.45	2,070	ng/kg ww	na	2,070	not eval.	na	no	ntx
PCB-182	na	42 / 49	7.47	927	ng/kg ww	0.221 – 0.762	927	not eval.	na	no	ntx
PCB-183	na	52 / 52	476 C	310,000 C	ng/kg ww	na	310,000	not eval.	na	no	ntx
PCB-184	na	48 / 49	0.638 J	86.9	ng/kg ww	2.42 – 2.42	86.9	not eval.	na	no	ntx
PCB-185[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-186	na	3 / 49	1.14 J	5.24 J	ng/kg ww	0.0681 – 9.39	9.39	not eval.	na	no	ntx
PCB-187	na	52 / 52	1,110	549,000	ng/kg ww	na	549,000	not eval.	na	no	ntx
PCB-188	na	49 / 49	1.98	273	ng/kg ww	na	273	not eval.	na	no	ntx
PCB-189	na	49 / 49	7.52	9,590	ng/kg ww	na	9,590	not eval.	na	no	ntx
PCB-190	na	49 / 49	82.5	76,800	ng/kg ww	na	76,800	not eval.	na	no	ntx
PCB-191	na	49 / 49	25.8	15,000	ng/kg ww	na	15,000	not eval.	na	no	ntx
PCB-192	na	0 / 49	nd	nd	ng/kg ww	0.0864 – 10.7	10.7	not eval.	na	no	ntx
PCB-193[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-194	na	49 / 49	43.8	97,600	ng/kg ww	na	97,600	not eval.	na	no	ntx
PCB-195	na	52 / 52	21.9	50,700	ng/kg ww	na	50,700	not eval.	na	no	ntx
PCB-196	na	49 / 49	94.9	63,600	ng/kg ww	na	63,600	not eval.	na	no	ntx
PCB-197	na	49 / 49	17.5 C	9,590 C	ng/kg ww	na	9,590	not eval.	na	no	ntx
PCB-198	na	49 / 49	221 C	98,800 C	ng/kg ww	na	98,800	not eval.	na	no	ntx
PCB-199[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-200[t]	na	49 / 49	na	na	ng/kg ww	na	na	not eval.	na	no	ntx
PCB-201	na	49 / 49	52.6	15,300	ng/kg ww	na	15,300	not eval.	na	no	ntx
PCB-202	na	49 / 49	116	21,500	ng/kg ww	na	21,500	not eval.	na	no	ntx
PCB-203	na	49 / 49	68.5	77,100	ng/kg ww	na	77,100	not eval.	na	no	ntx

Table 3, continued

Chemical	CAS Number	Detection Frequency	Minimum Detected Conc.	Maximum Detected Conc.	Units	Range of Reporting Limits	Value Used for Screening	Background Conc.[a]	RBC[b]	COPC Flag?	Rationale for Selection or Exclusion
PCB-204	na	45 / 49	0.0540 J	15.2 J	ng/kg ww	0.0210 – 0.457	15.2	not eval.	na	no	ntx
PCB-205	na	49 / 49	3.88	4,700	ng/kg ww	na	4,700	not eval.	na	no	ntx
PCB-206	na	52 / 52	10.3	9,710	ng/kg ww	na	9,710	not eval.	na	no	ntx
PCB-207	na	49 / 49	1.15 J	1,680	ng/kg ww	na	1,680	not eval.	na	no	ntx
PCB-208	na	49 / 49	2.88	1,640	ng/kg ww	na	1,640	not eval.	na	no	ntx
PCB-209	na	49 / 49	2.23	456	ng/kg ww	na	456	not eval.	na	no	ntx
PCBs (total calc'd)	na	214 / 221	16	18,400 J	µg/kg ww	13 – 20	18,000	18	0.41 ca[r]	yes	asl
Pentachlorophenol	87-86-5	8 / 145	1.1 J	2,400 J	µg/kg ww	1.7 – 5,800	5,800	not eval.	6.8 ca	yes	asl
Phenanthrene	na	92 / 145	0.49	26	µg/kg ww	0.31 – 24	26	not eval.	na	no	ntx
Phenol	108-95-2	27 / 145	18 J	670 J	µg/kg ww	3.6 – 1,500	1,500	not eval.	26,000 nc	no	bsl
Pyrene	129-00-0	117 / 145	0.16 J	130	µg/kg ww	0.36 – 24	130	not eval.	2,600 nc	no	bsl
Selenium	7782-49-2	110 / 110	0.10	0.373	mg/kg ww	na	0.37	not eval.	0.43 nc	no	bsl
Silver	7440-22-4	104 / 142	0.0013 J	0.29 M	mg/kg ww	0.0042 – 0.012	0.29	not eval.	0.43 nc	no	bsl
Tetrabutyltin as ion	na	0 / 109	nd	nd	µg/kg ww	0.8 – 1.5	1.5	not eval.	na	no	ntx
Thallium	7440-28-0	60 / 110	0.0005 J	0.0042	mg/kg ww	0.0015 – 0.0055	0.0055	not eval.	0.0061 nc	no	bsl
Total aldrin/dieldrin (calc'd)	na	10 / 130	0.95 JN	6.2 JN	µg/kg ww	1.0 – 24	24	not eval.	na	no	ntx
Total chlordane (calc'd)	na	113 / 119	0.86 JN	330 JN	µg/kg ww	0.50 – 0.50	330	not eval.	2.3 ca[u]	yes	asl
Total HPAH (calc'd)	na	128 / 145	0.21 J	550	µg/kg ww	0.87 – 64	550	not eval.	na	no	ntx
Total LPAH (calc'd)	na	111 / 145	0.094 J	54	µg/kg ww	3.6 – 64	64	not eval.	na	no	ntx
Total PAH (calc'd)	na	132 / 145	0.43 J	600	µg/kg ww	18 – 64	600	not eval.	na	no	ntx
Toxaphene	8001-35-2	0 / 130	nd	nd	µg/kg ww	10 – 4,800	4,800	not eval.	0.75 ca	yes	asl
Tributyltin as ion	688-73-3	119 / 153	0.81 J	660	µg/kg ww	0.74 – 2.1	660	not eval.	26 nc[v]	yes	asl
Vanadium	7440-62-2	80 / 118	0.058	2.65	mg/kg ww	0.08 – 0.25	3	not eval.	0.087 nc	yes	asl
Zinc	7440-66-6	142 / 142	3.8	44	mg/kg ww	na	44	not eval.	26 nc	yes	asl

[a] Background concentration is an average from up to 229 English sole filet samples collected from non-urban areas by the PSAMP (West et al. 2001) from 1989-1999. Multiple samples were collected from some locations, but averaging by location was not conducted prior to calculating the overall average concentration.

[b] Risk-based concentrations (RBCs) are derived from EPA Region 3 RBCs for fish tissue (last updated October 2005). Site-specific modifications were made to the Region 3 RBCs to reflect differences in body weight (70 kg for Region 3, 79 kg for this site), seafood consumption rate (54 g/day for Region 3, 84 g/day for this site), and exposure frequency (350 d/yr for Region 3, 365 d/yr for this site), and exposure duration (30 yrs for Region 3, 55 yrs for this site). For chemicals with cancer endpoints, the Region 3 RBCs were multiplied by 0.38 to reflect the site-specific modifications; RBCs for chemicals with non-cancer endpoints were multiplied by 0.70 to reflect the site-specific modifications. RBCs associated with a non-cancer endpoint (abbreviated "nc") were divided by 10 for this screening, reflecting the different target hazard quotients used in Region 3 (HQ = 1) and Region 10 (HQ = 0.1). Abbreviations: ca = cancer endpoint, nc = non-cancer endpoint.

Table 3, continued

[c] Less than 10% (1 of 145) reporting limits for 2,4,5-trichlorophenol exceed the RBC.
[d] RBC for acenaphthylene is from acenaphthene.
[e] RBC for alpha-chlordane is from chlordane.
[f] RBC for alpha-endosulfan is from endosulfan.
[g] Inorganic arsenic was evaluated instead of arsenic for this scenario.
[h] RBC for arsenic (inorganic) is from arsenic.
[i] RBC for beta-endosulfan is from endosulfan.
[j] RBC for carcinogenic PAHs is from benzo(a)pyrene.
[k] RBC for chromium is from chromium VI.
[l] RBC for DDTs (total calc'd) is from 4,4-DDT.
[m] RBC for endosulfan sulfate is from endosulfan.
[n] RBC for endrin aldehyde is from endrin.
[o] RBC for endrin ketone is from endrin.
[p] RBC for gamma-chlordane is from chlordane.
[q] RBC for mercury is from methylmercury.
[r] RBC for PCB congeners and PCBs (total calc'd) is from Aroclor 1254.
[s] RBC for PCB TEQ is from 2,3,7,8-TCDD.
[t] Statistics for this congener reflect only those samples that were determined not to co-elute with other congeners. When no statistics are given, "na" will appear in place of statistics, meaning that all samples co-eluted with another congener.
[u] RBC for total chlordane (calc'd) is from chlordane.
[v] RBC for tributyltin ion is the value for tributyltin oxide multiplied by 0.49 to account for differences in molecular weight.

Abbreviations:

COPC – chemical of potential concern
nd – not detected
na – not applicable
C – concentration represents coelution
J – estimated value
M – calculated value
N – tentative identification
asl – above screening level (selected as COPC)
bsl – below screening level (not selected as COPC)
ifd – infrequent detection (not selected as COPC)
ntx – no toxicity information (not selected as COPC)
sum – chemical included in sum and is not evaluated separately
teq – chemical included in TEQ calculation and is not evaluated separately

Table 4. Chemicals analyzed in sediment but not in tissue samples evaluated in HHRA

Chemical	CAS Number	Detection Frequency	Detected in Surface Sediment	Identified as COPC in Sediment	Important Bioaccumulative Compound (EPA 2000)
1,1,1,2-Tetrachloroethane	630-20-6	0 / 53	no	no	no
1,1,1-Trichloroethane	71-55-6	0 / 72	no	no	no
1,1,2,2-Tetrachloroethane	79-34-5	0 / 72	no	no	no
1,1,2-Trichloroethane	79-00-5	0 / 72	no	no	no
1,1,2-Trichlorotrifluoroethane	76-13-1	0 / 58	no	no	no
1,1-Dichloroacetone	513-88-2	0 / 51	no	no	no
1,1-Dichloroethane	75-34-3	0 / 72	no	no	no
1,1-Dichloroethene	75-35-4	0 / 72	no	no	no
1,1-Dichloropropene	563-58-6	0 / 53	no	no	no
1,2,3,4,6,7,8-HpCDD	35822-46-9	58 / 62	yes	no	yes
1,2,3,4,6,7,8-HpCDF	67562-39-4	57 / 62	yes	no	no
1,2,3,4,7,8,9-HpCDF	55673-89-7	34 / 62	yes	no	no
1,2,3,4,7,8-HxCDD	39227-28-6	24 / 62	yes	no	yes
1,2,3,4,7,8-HxCDF	70648-26-9	37 / 62	yes	no	yes
1,2,3,6,7,8-HxCDD	57653-85-7	44 / 62	yes	no	yes
1,2,3,6,7,8-HxCDF	57117-44-9	24 / 62	yes	no	no
1,2,3,7,8,9-HxCDD	19408-74-3	40 / 62	yes	no	no
1,2,3,7,8,9-HxCDF	72918-21-9	21 / 62	yes	no	no
1,2,3,7,8-PeCDD	40321-76-4	24 / 62	yes	no	no
1,2,3,7,8-PeCDF	57117-41-6	21 / 62	yes	no	yes
1,2,3-Trichlorobenzene	87-61-6	0 / 53	no	no	no
1,2,3-Trichloropropane	96-18-4	0 / 53	no	no	no
1,2,4-Trimethylbenzene	95-63-6	3 / 53	yes	no	no
1,2-Dibromo-3-chloropropane	96-12-8	0 / 53	no	no	no
1,2-Dibromoethane (EDB)	106-93-4	0 / 53	no	no	no
1,2-Dichloroethane	107-06-2	0 / 72	no	no	no
1,2-Dichloroethene (total)	540-59-0	0 / 14	no	no	no
1,2-Dichloropropane	78-87-5	0 / 72	no	no	no
1,3,5-Trimethylbenzene	108-67-8	0 / 53	no	no	no

Table 4, continued

Chemical	CAS Number	Detection Frequency	Detected in Surface Sediment	Identified as COPC in Sediment	Important Bioaccumulative Compound (EPA 2000)
1,3-Dichloropropane	142-28-9	0 / 53	no	no	no
1-Chlorobutane	109-69-3	0 / 53	no	no	no
1-Methylnaphthalene	90-12-0	45 / 45	yes	no	no
2,2-Dichloropropane	594-20-7	0 / 53	no	no	no
2,3,4,6,7,8-HxCDF	60851-34-5	24 / 62	yes	no	no
2,3,4,7,8-PeCDF	57117-31-4	24 / 62	yes	no	yes
2,3,7,8-TCDD	1746-01-6	21 / 62	yes	no	yes
2,3,7,8-TCDF	51207-31-9	43 / 62	yes	no	yes
2-Chloroethyl vinyl ether	110-75-8	0 / 5	no	no	no
2-Chlorotoluene	95-49-8	0 / 53	no	no	no
2-Hexanone	591-78-6	0 / 72	no	no	no
2-Nitropropane	79-46-9	0 / 53	no	no	no
4-Chlorotoluene	106-43-4	0 / 53	no	no	no
Acetone	67-64-1	3 / 72	yes	no	no
Acid volatile sulfides	n/a	71 / 83	yes	no	no
Allyl chloride	107-05-1	0 / 53	no	no	no
Aluminum	7429-90-5	727 / 727	yes	yes	no
Ammonia	7664-41-7	57 / 57	yes	no	no
Ammonia (total as nitrogen)	7664-41-7	167 / 171	yes	no	no
Aroclor-1254/1260	n/a	16 / 16	yes	no	no
Aroclor-1262	37324-23-5	2 / 25	yes	no	no
Aroclor-1268	11100-14-4	1 / 24	yes	no	yes
Barium	7440-39-3	590 / 590	yes	yes	no
Benzaldehyde	100-52-7	12 / 23	yes	no	no
Benzene	71-43-2	0 / 72	no	no	no
Benzo(e)pyrene	192-97-2	45 / 45	yes	no	no
Beryllium	7440-41-7	734 / 746	yes	no	no
Biphenyl	92-52-4	45 / 45	yes	no	no
Bromobenzene	108-86-1	0 / 53	no	no	no
Bromochloromethane	74-97-5	0 / 53	no	no	no
Bromodichloromethane	75-27-4	0 / 72	no	no	no

Table 4, continued

Chemical	CAS Number	Detection Frequency	Detected in Surface Sediment	Identified as COPC in Sediment	Important Bioaccumulative Compound (EPA 2000)
Bromoform	75-25-2	0 / 72	no	no	no
Bromomethane	74-83-9	0 / 72	no	no	no
Butyltin (total)	n/a	45 / 54	yes	no	no
C1-Chrysenes	3001965	45 / 45	yes	no	no
C1-Dibenzothiophenes	3001957	33 / 45	yes	no	no
C1-Fluoranthene/Pyrene	3001964	43 / 45	yes	no	no
C1-Fluorenes	3001954	22 / 45	yes	no	no
C1-Phenanthrenes/anthracenes	3001960	45 / 45	yes	no	no
C2-Chrysenes	3001966	45 / 45	yes	no	no
C2-Dibenzothiophenes	3001958	31 / 45	yes	no	no
C2-Fluorenes	3001955	33 / 45	yes	no	no
C2-Naphthalenes	3001951	45 / 45	yes	no	no
C2-Phenanthrenes/anthracenes	3001961	45 / 45	yes	no	no
C3-Chrysenes	3001967	45 / 45	yes	no	no
C3-Dibenzothiophenes	3001959	37 / 45	yes	no	no
C3-Fluorenes	3001956	37 / 45	yes	no	no
C3-Naphthalenes	3001952	45 / 45	yes	no	no
C3-Phenanthrenes/anthracenes	3001962	45 / 45	yes	no	no
C4-Chrysenes	3001968	35 / 45	yes	no	no
C4-Naphthalenes	3001953	45 / 45	yes	no	no
C4-Phenanthrenes/anthracenes	3001963	45 / 45	yes	no	no
Calcium	7440-70-2	619 / 619	yes	no	no
Caprolactam	105-60-2	2 / 23	yes	no	no
Carbon disulfide	75-15-0	19 / 72	yes	no	no
Carbon tetrachloride	56-23-5	0 / 72	no	no	no
Chloroacetonitrile	107-14-2	0 / 2	no	no	no
Chlorobenzene	108-90-7	0 / 72	no	no	no
Chloroethane	75-00-3	0 / 72	no	no	no
Chloroform	67-66-3	0 / 72	no	no	no
Chloromethane	74-87-3	0 / 72	no	no	no
Chromium VI	18540-29-9	4 / 20	yes	no	yes

Table 4, continued

Chemical	CAS Number	Detection Frequency	Detected in Surface Sediment	Identified as COPC in Sediment	Important Bioaccumulative Compound (EPA 2000)
cis-1,2-Dichloroethene	156-59-2	0 / 58	no	no	no
cis-1,3-Dichloropropene	10061-01-5	0 / 72	no	no	no
cis-Nonachlor	5103-73-1	0 / 63	no	no	no
Cyanide	57-12-5	0 / 25	no	no	no
Dibenzothiophene	132-65-0	45 / 45	yes	no	no
Dibromochloromethane	124-48-1	0 / 72	no	no	no
Dibromomethane	74-95-3	0 / 53	no	no	no
Dichlorodifluoromethane	75-71-8	0 / 9	no	no	no
Dichloromethane	75-09-2	3 / 72	yes	no	no
Diethyl ether	60-29-7	0 / 53	no	no	no
Dioxin/furan TEQ – mammal – half DL	n/a	62 / 62	yes	yes	yes[a]
Endosulfan	115-29-7	3 / 80	yes	no	no
Ethyl methacrylate	97-63-2	0 / 53	no	no	no
Ethylbenzene	100-41-4	0 / 72	no	no	no
Gasoline	8006-61-9	2 / 29	yes	no	no
Iodomethane	74-88-4	0 / 53	no	no	no
Iron	7439-89-6	725 / 725	yes	yes	no
Isopropylbenzene	98-82-8	0 / 53	no	no	no
Magnesium	7439-95-4	631 / 631	yes	no	no
Manganese	7439-96-5	688 / 688	yes	yes	no
Methacrylonitrile	126-98-7	0 / 53	no	no	no
Methyl acrylate	96-33-3	0 / 53	no	no	no
Methyl ethyl ketone	78-93-3	35 / 72	yes	no	no
Methyl isobutyl ketone	108-10-1	0 / 72	no	no	no
Methyl methacrylate	80-62-6	0 / 53	no	no	no
n-Butylbenzene	104-51-8	0 / 53	no	no	no
n-Propylbenzene	103-65-1	0 / 53	no	no	no
OCDD	3268-87-9	62 / 62	yes	no	no
OCDF	39001-02-0	60 / 62	yes	no	no
Oxychlordane	27304138	0 / 63	no	no	no
PCTs (total)	n/a	258 / 299	yes	no	no

Table 4, continued

Chemical	CAS Number	Detection Frequency	Detected in Surface Sediment	Identified as COPC in Sediment	Important Bioaccumulative Compound (EPA 2000)
p-Cymene	99-87-6	3 / 53	yes	no	no
Pentachloroethane	76-01-7	0 / 53	no	no	no
Perylene	198-55-0	45 / 45	yes	no	no
Potassium	7440-09-7	633 / 633	yes	no	no
Pyridine	110-86-1	0 / 49	no	no	no
Retene	483-65-8	28 / 61	yes	no	no
sec-Butylbenzene	135-98-8	0 / 53	no	no	no
Sodium	7440-23-5	617 / 617	yes	no	no
Styrene	100-42-5	0 / 72	no	no	no
Sulfides (total)	n/a	208 / 320	yes	no	no
tert-Butyl methyl ether	1634-04-4	0 / 53	no	no	no
tert-Butylbenzene	98-06-6	0 / 53	no	no	no
Tetrachloroethene	127-18-4	3 / 72	yes	no	no
Tin	7440-31-5	188 / 294	yes	no	no
Toluene	108-88-3	6 / 72	yes	no	no
Total HpCDD	37871-00-4	37 / 41	yes	no	no
Total HpCDF	38998-75-3	37 / 41	yes	no	no
Total HxCDD	34465-46-8	37 / 41	yes	no	no
Total HxCDF	55684-94-1	37 / 41	yes	no	no
Total PeCDD	36088-22-9	3 / 41	yes	no	no
Total PeCDF	30402-15-4	36 / 41	yes	no	no
Total petroleum hydrocarbons (calc'd)	n/a	2 / 2	yes	no	no
Total TCDD	n/a	32 / 41	yes	no	no
Total TCDF	30402-14-3	37 / 41	yes	no	no
Total Xylenes (calc'd)	n/a	0 / 58	no	yes	no
TPH	n/a	60 / 70	yes	no	no
TPH – diesel #2 range	68334-30-5	0 / 27	no	no	no
TPH – diesel range	n/a	2 / 2	yes	no	no
TPH – gasoline range	n/a	0 / 2	no	no	no
TPH – heavy fuel oil range	8001-58-9	2 / 9	yes	no	no
trans-1,2-Dichloroethene	156-60-5	0 / 58	no	no	no

Table 4, continued

Chemical	CAS Number	Detection Frequency	Detected in Surface Sediment	Identified as COPC in Sediment	Important Bioaccumulative Compound (EPA 2000)
trans-1,3-Dichloropropene	10061-02-6	0 / 72	no	no	no
trans-1,4-Dichloro-2-butene	110-57-6	0 / 51	no	no	no
trans-Nonachlor	39765-80-5	0 / 63	no	no	no
Trichloroethene	79-01-6	0 / 72	no	no	no
Trichlorofluoromethane	75-69-4	0 / 58	no	no	no
Vinyl acetate	108-05-4	0 / 5	no	no	no
Vinyl chloride	75-01-4	0 / 72	no	no	no
Xylene (meta and para)	108-38-3 106-42-3	0 / 58	no	no	no
Xylene (ortho)	95-47-6	0 / 58	no	no	no
Xylene (total)	1330-20-7	0 / 14	no	no	no

COPC – chemical of potential concern

n/a – not applicable

[a] Although the chemical group dioxin/furan TEQ was not listed in EPA (2000) as an important bioaccumulative chemical, the chemical benchmark for this group – 2,3,7,8-TCDD – was considered an important bioaccumulative chemical. Therefore, in keeping with the intent of the TEQ calculation, the chemical group was also designated as an important bioaccumulative chemical.

REFERENCES

Ecology. 2000. Sediment quality in Puget Sound. Year 2 - central Puget Sound. No. 00-03-055. Washington Department of Ecology, Olympia, WA.

EPA. 1998. Risk assessment guidance for Superfund. Volume 1. Human health evaluation manual. Part D, Standardized planning, reporting, and review of Superfund risk assessments. Interim Publication No. 9825.7-01D. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 2000. Bioaccumulation testing and interpretation for the purpose of sediment quality assessment: status and needs. EPA-823-R-00-001. Bioaccumulation Analysis Workgroup, US Environmental Protection Agency, Washington, DC.

EPA. 2004. EPA Region IX preliminary remediation goals: 2004 PRG table [online]. US Environmental Protection Agency Region IX, San Francisco, CA. Updated October 2004. Available from: http://www.epa.gov/region09/waste/sfund/prg/index.html.

EPA. 2005. EPA Region III risk-based concentration table [online]. US Environmental Protection Agency Region III, Philadelphia, PA. Updated October 2005. Available from: http://www.epa.gov/reg3hwmd/risk/human/index.htm.

West JE, O'Neill SM, Lippert G, Quinnell S. 2001. Toxic contaminants in marine and anadromous fishes from Puget Sound, Washington. Results of the Puget Sound ambient monitoring program fish component 1989-1999. Washington Department of Fish and Wildlife, Olympia, WA.

ATTACHMENT 2. MODEL TOXICS CONTROL ACT-CONFORMING EXPOSURE POINT CONCENTRATIONS

Table 1. Summary statistics for seafood consumption and sediment exposure EPCs selected for re-analysis using MTCA-conforming data evaluation methods 6

Table 2. Comparison of EPCs used in this HHRA and MTCA-conforming EPCs 9

Attachment 2. Model Toxics Control Act-Conforming Exposure Point Concentrations

This attachment to the Lower Duwamish Waterway (LDW) baseline human health risk assessment (HHRA) presents Model Toxics Control Act (MTCA)-conforming exposure point concentrations (EPCs) and compares them to the EPCs used in this HHRA. EPCs in this HHRA were calculated according to guidance from US Environmental Protection Agency (EPA) statisticians following the most current statistical methods and software (i.e., ProUCL 4.0). This attachment explores the potential differences that could occur if MTCA methods[1] had been used instead. Rather than recalculating all of the EPCs used in the HHRA, a subset of the chemical of potential concern (COPC)-scenario combinations that represent a variety of datasets were selected. A total of 31 seafood consumption scenario EPCs and 23 sediment exposure EPCs were re-analyzed as a part of this exercise; summary statistics for these EPCs are presented in Table 1.

MTCA-conforming EPCs are presented together with the EPCs calculated for the HHRA using ProUCL software in Table 2. Methods for calculating the MTCA-conforming EPCs provided in Table 2 were reviewed by Gregory Glass on behalf of the Washington State Department of Ecology (Ecology). Mr. Glass provided instructions for calculating MTCA-conforming EPCs for several scenarios that were not possible to calculate using MTCAStat statistical analysis software (Ecology 1997). In addition, Mr. Glass suggested that when the distribution of a dataset was determined to be neither lognormal nor normal, the results of several statistical approaches should be shown for comparison purposes (Glass 2007a).

Methods for Determination of Distribution

Goodness of fit to a lognormal or normal distribution was assessed using MTCAStat for all datasets with fewer than 500 values, with non-detected results entered at their reporting limit and coded as non-detected for calculations using a "censored probability plot" approach. For other datasets, the MTCA censored probability plot method was replicated in Microsoft® Excel® as follows:

1. Data values were sorted, with non-detect values equal to one-half the RL, to determine the rank ordering.

[1] As noted in the risk characterization, Section B.5.1.3, the exposures for scenarios that are evaluated under MTCA have not been specifically quantified in this HHRA. MTCA-conforming EPCs are calculated following current statistical guidance (Ecology 1992, 1997).

2. Percentiles were calculated from ranks according to the formula used in MTCA (Equation 1; see Ecology 1992, 1997) along with the normal z-score[2] for each percentile.

$$\text{Percentile} = [(\text{rank} - 3/8)/(n + 1/4)] \qquad \textbf{Equation 1}$$

3. Using only detected values, the coefficient of determination (R^2) was calculated between the z-scores of the detected values and their corresponding arithmetic and log-transformed concentrations.
4. If the R^2 between the z-scores and the log-transformed data was greater than 0.90, the dataset was considered to be lognormal (regardless of whether it was also normal). If the data were not lognormal, but the R^2 between the z-scores and the arithmetic data was greater than 0.90, the data were considered to be normal (Ecology 1992). Some datasets did not meet either criterion and were accordingly evaluated under MTCA as neither normal nor lognormal.

Methods for Calculation of Upper Confidence Limits

Upper confidence limits (UCLs) were calculated using both detect and non-detect data. When possible, UCLs were calculated using MTCAStat according to MTCA guidance (Ecology 1992). Calculations for datasets with more than 500 values were performed following MTCA statistical methods using ProUCL 4.0 or, for larger datasets, using unpublished BTNCTD.EXE software provided by Dr. Charles Land through Ecology's consultant, Gregory Glass (Glass 2007b; Land 1994). The latter software was used for datasets with more than 1,000 values because ProUCL will not calculate an h-statistic for such large datasets. According to the authors of ProUCL, the behavior of the h-statistic with such large datasets has not been adequately evaluated (EPA 2006).

Non-detect results were handled in accordance with MTCA guidance (Ecology 1992) as follows:

1. If 85% or more of the samples were detected, one-half of each RL was used in place of the non-detect results.
2. If ≥ 50% and < 85% of samples were detected, Cohen's method was applied.[3] Because only one reporting limit value can be entered into MTCAStat for calculations using Cohen's method, the maximum RL was generally used when multiple RLs were present. When non-detect reporting limits were greater than detect values, it was sometimes necessary to develop an alternative approach than the maximum RL. In most of these situations, the average RL was

[2] A z-score for each calculated percentile was determined from standard tables for the normal distribution.

[3] Cohen's method was used to calculate a corrected mean and standard deviation for use in determining a UCL (Ecology 2001).

substituted for all non-detect values. In one case, the value of the smallest detected value was substituted for all non-detected values.

3. If the detection frequency was less than 50%, the maximum detected value was used as the UCL.

Either Land's method (h-statistic) for lognormal data or the t-statistic for normal data was selected for calculating a UCL, according to the distribution of the data. If a dataset is neither normal nor lognormal, MTCA statistical guidance discusses several alternatives for UCL calculations (see Ecology (1992), Section 5.2.1 and Supplement S-6, Note 2). EPCs based on both maximum values and z-statistics are reported in this attachment for datasets that are neither normal nor lognormal. A z-statistic UCL can be appropriate when sample size is "sufficiently large" that the distribution of the mean approaches a normal distribution (Central Limit Theorem), but it is not well-supported for smaller sample sizes. The coverage (i.e., inclusion of the true mean with the stated probability) of the maximum value as an EPC depends on sample size and location (distribution percentile) of the true mean value. For comparison purposes, in cases where the R^2 for the lognormal distribution approached but did not quite meet the 0.90 acceptance criterion, the h-statistic was also calculated.

The MTCA statistical methods are more restricted than those available in ProUCL. A comparison of the MTCA-conforming EPCs and ProUCL EPCs as calculated in this HHRA (see Table 2) reveals no consistent pattern; the MTCA-conforming EPCs are for varying datasets less than, equal to, or greater than the ProUCL results. In some cases, different statistical approaches lead to practically identical results. Most datasets evaluated using non-parametric Chebyshev statistics in ProUCL have lower EPCs when calculated according to MTCA methods. The greatest MTCA exceedances of ProUCL results occur for MTCA EPCs calculated using Land's h-statistic together with small sample sizes.

Research has shown that the h-statistic UCL can result in unrealistically high values, even for some datasets that are technically lognormally distributed, especially as skewness increases and sample size decreases. In accordance with MTCA guidance (Ecology 1992), in this attachment, an h-statistic UCL was calculated for all datasets that passed the R^2 criterion for being lognormally distributed. It should be noted that, of the 54 EPCs recalculated, the h-statistic UCL was greater than the maximum detected value in seven cases.[4] All seven cases reflect small datasets, with between 6 and 14 values (Table 1). Because no alternative UCL calculation method is provided in MTCA guidance for these cases (without additional data collection), the maximum detected value is also presented for comparison (Table 2). EPCs calculated using

[4] The COPC-scenario combinations with h-statistic UCLs greater than the maximum detected value included arsenic (clams), arsenic (pelagic fish), carcinogenic polycyclic aromatic hydrocarbons (cPAHs) (beach play area 3), dioxin/furan toxic equivalent (TEQ) (tribal clamming and clamming 7 days per year), and total polychlorinated biphenyls (PCBs) (beach play area 3 and area 4).

ProUCL also infrequently exceed the maximum dataset value and are used in HHRA exposure calculations. The degree of exceedance of a maximum dataset value is occasionally much larger for MTCA EPCs based on an h-statistic than for ProUCL results.

For four of the seven cases discussed above, the h-statistic UCL was greater than the maximum detected value by a factor of 5 or less. For the other three cases, the difference was much larger. The most extreme examples were for cPAHs in beach play – Area 3 and dioxin/furans for the tribal clamming scenario. For cPAHs in beach play – Area 3, the h-statistic UCL was 71 mg/kg dw compared to a maximum detected value of 2.9 mg/kg dw for that exposure area. The maximum cPAH result in surface sediment for the entire LDW was 11 mg/kg dw (n = 793), so the value of 71 mg/kg dw was well outside the bounds of the existing data. Similarly, the h-statistic UCL for dioxin/furan TEQ for the tribal clamming scenario was 0.136 mg/kg dw (n = 11), which was much higher than the maximum detected value anywhere in the LDW of 0.0021 mg/kg dw (n = 43). These cases of high EPCs calculated using the h-statistic primarily reflect the small sample sizes and skewness for specific datasets and exposure areas. The additional LDW-wide data provide a broader context for the interpretation of these EPCs and indicate that they are biased high.

References

Ecology. 1992. Statistical guidance for Ecology site managers and supplement S-6. Publication no. 92-54. Toxics Cleanup Program, Washington State Department of Ecology, Olympia, WA.

Ecology. 1997. MTCAStat 3.0: statistical analysis tools for Model Toxics Control Act site cleanup work [online]. Toxics Cleanup, Washington State Department of Ecology, Olympia, WA Available from: http://www.ecy.wa.gov/programs/tcp/tools/toolmain.html.

Ecology. 2001. Model Toxics Control Act Cleanup Regulation, Chapter 173-340 WAC. Publication No. 94-06. Toxics Cleanup Program, Washington State Department of Ecology, Olympia, WA.

EPA. 2006. ProUCL Version 4.0 user guide. National Exposure Research Laboratory, US Environmental Protection Agency, Las Vegas, NV.

Glass G. 2007a. Personal communication (e-mail and attached Excel file to Lucinda Tear, Windward Environmental, regarding MTCA EPC calculations). Gregory L. Glass Environmental, Seattle, WA. November 13, 2007.

Glass G. 2007b. Personal communication (transmittal memo and attached instructions to Lucinda Tear, Windward Environmental, regarding BTNCTD software for Land's H-statistic). Gregory L. Glass Environmental, Seattle, WA. November 14, 2007.

Land CE. 1994. BTNCTD (software to calculate Land's H-statistic). C.E. Land, Ph.D., National Institutes of Health, Bethesda, MD.

Table 1. Summary statistics for seafood consumption and sediment exposure EPCs selected for re-analysis using MTCA-conforming data evaluation methods

Chemical	Consumption Category or Scenario	Detection Frequency		Concentration (mg/kg)[a]			Number of Different RLs
		Ratio	Percent	Mean Value	Range of Detected Values	RL or Range of RLs	
Seafood consumption scenarios							
Arsenic (inorganic)[b]	benthic fish, fillet	6/8	75	0.004	0.003 – 0.006 J	0.003	1
	benthic fish, whole body	8/8	100	0.056	0.02 – 0.09	na	na
	clams	8/8	100	1.24	0.132 – 3.27	na	na
	crab, edible meat	6/6	100	0.023	0.01 – 0.03	na	na
	crab, whole body	6/6	100	0.075	0.022 JM – 0.123 M	na	na
	pelagic fish, whole body	8/10	80	0.057	0.02 – 0.16	0.01	1
Carcinogenic PAHs[b, c]	benthic fish, fillet	5/8	63	0.00039	0.00037 J – 0.00064 J	0.00045	1
	benthic fish, whole body	21/24	88	0.0014	0.00045 J – 0.0028 J	0.00045	1
	clams	14/14	100	0.015	0.0068 – 0.044	na	na
	crab, edible meat	8/19	42	0.00044	0.00033 – 0.00084 J	0.00065	1
	crab, whole body	19/19	100	0.00075	0.00045 M – 0.0024 JM	na	na
	pelagic fish, whole body	26/26	100	0.00078	0.00037 J – 0.0022	na	na
Copper	benthic fish, whole body	24/24	100	1.73	0.494 – 3.47	na	na
	crab, edible meat	21/21	100	7.5	4.43 – 16	na	na
	crab, whole body	21/21	100	14	6.9 M – 24 M	na	na
Mercury	benthic fish, fillet	23/23	100	0.041	0.013 – 0.083	na	na
	benthic fish, whole body	24/24	100	0.01	0.005 – 0.027	na	na
	crab, edible meat	25/25	100	0.057	0.023 – 0.11	na	na
	crab, whole body	21/21	100	0.046	0.022 M – 0.097 M	na	na
	mussels	21/21	100	0.013	0.0088 – 0.023	na	na
	pelagic fish, whole body	31/31	100	0.033	0.018 – 0.088	na	na

Chemical	Consumption Category or Scenario	Detection Frequency		Concentration (mg/kg)[a]			Number of Different RLs
		Ratio	Percent	Mean Value	Range of Detected Values	RL or Range of RLs	
Total PCBs	benthic fish, fillet	33/33	100	0.7	0.079 – 2.0	na	na
	benthic fish, whole body	45/45	100	2.2	0.45 – 4.7	na	na
	clams	14/14	100	0.14	0.024 – 0.58 J	na	na
	crab, edible meat	26/29	90	0.17	0.060 – 0.39 J	0.020	1
	crab, whole body	25/25	100	0.89	0.25 – 1.9 JM	na	na
	mussels	18/22	82	0.034	0.016 – 0.060	0.013	1
	pelagic fish, whole body	53/53	100	1.7	0.164 – 18.4 J	na	na
Vanadium	clams	14/14	100	1.3	0.68 – 2.65	na	na
	mussels	8/8	100	0.15	0.058 – 0.26	na	na
	pelagic fish, whole body	22/26	85	0.4	0.21 – 1.23	0.2 – 0.25	2
Sediment exposure scenarios							
Arsenic	beach play RME, Area 3	6/9	67	8.5	7.2 – 18	3.1 – 6.6	3
	beach play RME, Area 5	22/22	100	8.1	3.94 – 11.8	na	na
	beach play RME, Area 7	9/9	100	8.9	5.05 J – 14	na	na
	tribal clamming – RME and 183 days per year	254/275	92	20	1.2 – 1,100	3.1 – 31	11
	clamming –7 days per year	100/103	97	8.8	2.7 – 20.7	3.1 – 6.6	3
	netfishing	755/817	92	20	1.2 – 1,100	3 – 31	18
Carcinogenic PAHs	beach play RME, Area 3	7/9	78	0.66	0.038 – 2.9 J	0.035 – 0.036	2
	beach play RME, Area 5	22/22	100	0.21	0.015 J – 1.0 J	na	na
	beach play RME, Area 7	8/9	89	0.077	0.024 J – 0.15	0.0094	1
	beach play RME, Area 8	11/11	100	0.23	0.049 – 0.62	na	na
	tribal clamming – RME and 183 days per year	255/264	97	0.5	0.0097 – 11	0.0091 – 0.11	8
	clamming – 7 days per year	97/103	94	0.27	0.0097 J – 3.0	0.0091 – 0.036	5

Chemical	Consumption Category or Scenario	Detection Frequency		Concentration (mg/kg)[a]			Number of Different RLs
		Ratio	Percent	Mean Value	Range of Detected Values	RL or Range of RLs	
	netfishing	749/793	94	0.5	0.0091 J – 11	0.009 – 0.13	22
Dioxin/furan TEQ	tribal clamming – RME and 183 days per year	11/11	100	0.00034	0.0000017 – 0.0021 J	na	na
	clamming – 7 days per year	6/6	100	0.000075	0.0000017 – 0.000412 J	na	na
	netfishing	43/43	100	0.00010	0.0000011 – 0.0021 J	na	na
Total PCBs	beach play RME, Area 3	11/14	79	0.089	0.0022 – 0.42 J	0.016 – 0.017	2
	beach play RME, Area 4	12/12	100	2.8	0.011 J – 23	na	na
	beach play RME, Area 5	31/32	97	0.1	0.024 – 0.66	0.020	1
	beach play RME, Area 7	10/14	71	0.063	0.0098 – 0.34	0.019 – 0.040	3
	tribal clamming – RME and 183 days per year	415/440	94	2.0	0.0022 – 110	0.016 – 0.040	5
	clamming – 7 days per year	142/161	88	0.43	0.0022 – 23	0.016 – 0.040	5
	netfishing	1205/1291	93	1.0	0.0016 J – 220	0.00056 – 0.05	25

[a] Summary statistics are presented in mg/kg ww for seafood consumption scenarios and in mg/kg dw for sediment exposure scenarios.

[b] No mussel data were available for this chemical. When calculating the CDI and risk values, the proportion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization section of this document are from only 2004 because of high RLs in historical data. All cPAH data are analyzed in the uncertainty analysis (Section B.6).

dw – dry weight

EPC – exposure point concentration

J – Analyte was positively identified and detected; however, concentration is an estimated value because the result is less than the quantitation limit or QC criteria were not met.

M – value is a weighted mean (described in Table B.2-4).

MTCA – Model Toxics Control Act

na – not applicable; no analytical data for that chemical in that seafood category

PAH – polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

RL – reporting limit

RME – reasonable maximum exposure

TEQ – toxic equivalent

ww – wet weight

Table 2. Comparison of EPCs used in this HHRA and MTCA-conforming EPCs

Chemical	Consumption Category or Scenario	DF (%)	HHRA ProUCL 4.0 EPCs		Distribution Test		MTCA-Conforming EPCs	
			Statistic Used	EPC (mg/kg)[a]	Lognormal R^2	Normal R^2	Statistic Used	EPC (mg/kg)[a]
Seafood Consumption Scenarios								
Arsenic (inorganic)	benthic fish, fillet	75	95% Chebyshev, pooled one-half RL	0.0062	0.902	0.938	Land's h-statistic (Cohen's method)	0.0053
	benthic fish, whole body	100	Student's-t UCL	0.073	0.911	0.924	Land's h-statistic	0.091
	clams	100	Student's-t UCL	2.0	0.955	0.902	Land's h-statistic [maximum detected value][b]	6.8 [3.3]
	crab, edible meat	100	95% Chebyshev (mean, SD) UCL	0.042	0.663	0.663	z-statistic maximum detected value	0.030 0.030
	crab, whole body	100	Student's-t UCL	0.11	0.884	0.861	z-statistic maximum detected value [Land's h-statistic][c]	0.11 0.12 [0.23]
	pelagic fish, whole body	80	95% KM (t) UCL	0.088	0.930	0.962	Land's h-statistic (Cohen's method) [maximum detected value][b]	0.27 [0.16]
Carcinogenic PAHs	benthic fish, fillet	63	maximum detected value	0.00064	0.982	0.985	Land's h-statistic (Cohen's method)[d]	0.00053
	benthic fish, whole body	88	95% KM (Chebyshev) UCL	0.0023	0.847	0.901	t-statistic (one-half RL substitution)	0.0018
	clams	100	approximate gamma UCL	0.020	0.920	0.733	Land's h-statistic	0.020
	crab, edible meat	42	95% KM (t) UCL	0.00065	0.663	0.738	maximum detected value[e]	0.00084
	crab, whole body	100	95% modified-t UCL	0.00092	0.435	0.319	z-statistic maximum detected value	0.00090 0.0024
	pelagic fish, whole body	100	95% modified-t UCL	0.00095	0.872	0.701	z-statistic maximum detected value [Land's h-statistic][c]	0.00094 0.0022 [0.00093]

Chemical	Consumption Category or Scenario	DF (%)	HHRA ProUCL 4.0 EPCs		Distribution Test		MTCA-Conforming EPCs	
			Statistic Used	EPC (mg/kg)[a]	Lognormal R^2	Normal R^2	Statistic Used	EPC (mg/kg)[a]
Copper	benthic fish, whole body	100	95% Chebyshev (mean, SD) UCL	2.6	0.886	0.879	z-statistic maximum detected value [Land's h-statistic][c]	2.1 3.5 [2.4]
	crab, edible meat	100	Student's-t UCL	8.5	0.884	0.745	z-statistic maximum detected value [Land's h-statistic][c]	8.5 16 [8.5]
	crab, whole body	100	Student's-t UCL	16	0.954	0.926	Land's h-statistic	16
Mercury	benthic fish, fillet	100	95% Chebyshev, pooled RL	0.058	0.921	0.923	Land's h-statistic	0.056
	benthic fish, whole body	100	Student's-t UCL	0.020	0.962	0.939	Land's h-statistic	0.017
	crab, edible meat	100	approximate gamma UCL	0.064	0.951	0.870	Land's h-statistic	0.065
	crab, whole body	100	Student's-t UCL	0.052	0.889	0.757	z-statistic maximum detected value [Land's h-statistic][c]	0.052 0.097 [0.052]
	mussels	100	approximate gamma UCL	0.014	0.929	0.834	Land's h-statistic	0.014
	pelagic fish, whole body	100	Student's-t UCL	0.039	0.876	0.712	z-statistic maximum detected value [Land's h-statistic][c]	0.038 0.088 [0.038]
Total PCBs	benthic fish, fillet	100	95% Chebyshev, pooled RL	1.2	0.962	0.894	Land's h-statistic	1.1
	benthic fish, whole body	100	approximate gamma UCL	2.6	0.962	0.932	Land's h-statistic	2.7
	clams	100	99% Chebyshev (mean, SD) UCL	0.60	0.820	0.728	z-statistic maximum detected value [Land's h-statistic][c]	0.22 0.58 [0.40]
	crab, edible meat	90	95% KM (t) UCL	0.20	0.850	0.950	t-statistic (one-half RL substitution)	0.20
	crab, whole body	100	95% H-UCL	1.1	0.934	0.866	Land's h-statistic	1.1
	mussels	82	95% KM (percentile bootstrap) UCL	0.041	0.870	0.958	t-statistic (Cohen's method)	0.040
	pelagic fish, whole body	100	95% H-UCL	1.9	0.926	0.394	Land's h-statistic	1.9

Chemical	Consumption Category or Scenario	DF (%)	HHRA ProUCL 4.0 EPCs		Distribution Test		MTCA-Conforming EPCs	
			Statistic Used	EPC (mg/kg)[a]	Lognormal R^2	Normal R^2	Statistic Used	EPC (mg/kg)[a]
Vanadium	clams	100	approximate gamma UCL	1.5	0.902	0.787	Land's h-statistic	1.5
	mussels	100	Student's-t UCL	0.19	0.894	0.941	t-statistic	0.19
	pelagic fish, whole body	85	95% KM (Chebyshev) UCL	0.62	0.927	0.827	Land's h-statistic (Cohen's method)[f]	0.48
Sediment Exposure Scenarios								
Arsenic	beach play RME, Area 3	67	95% KM (percentile bootstrap) UCL	13	0.934	0.955	Land's h-statistic (Cohen's method)	14[f] 16[g]
	beach play RME, Area 5	100	95% Students-t UCL	8.9	0.943	0.973	Land's h-statistic	9.1
	beach play RME, Area 7	100	95% Students-t UCL	11	0.949	0.945	Land's h-statistic	12
	tribal clamming – RME and 183 days per year	92	95% KM (BCA) UCL	27	0.941	0.987	Land's h-statistic (one-half RL substitution)[h]	14
	clamming – 7 days per year	97	95% KM (percentile bootstrap) UCL	9.5	0.985	0.958	Land's h-statistic (one-half RL substitution)	9.7
	netfishing	92	95% KM (BCA) UCL	21	0.980	0.985	Land's h-statistic using ProUCL (one-half RL substitution)[h, i]	15
Carcinogenic PAHs	beach play RME, Area 3	78	95% KM (Chebyshev) UCL	2.1	0.914	0.843	Land's h-statistic (Cohen's method)[f] [maximum detected value][b]	71 [2.9]
	beach play RME, Area 5	100	95% Chebyshev (MVUE) UCL	0.41	0.919	0.570	Land's h-statistic	0.37
	beach play RME, Area 7	89	95% KM (t) UCL	0.11	0.854	0.921	t-statistic (one-half RL substitution)	0.11
	beach play RME, Area 8	100	95% Students-t UCL	0.32	0.951	0.842	Land's h-statistic	0.43
	tribal clamming – RME and 183 days per year	97	95% KM (Chebyshev) UCL	0.77	0.993	0.795	Land's h-statistic (one-half RL substitution)	0.55
	clamming – 7 days per year	94	95% KM (Chebyshev) UCL	0.48	0.989	0.552	Land's h-statistic (one-half RL substitution)	0.38
	netfishing	94	95% KM (Chebyshev) UCL	0.57	0.984	0.925	Land's h-statistic using ProUCL (one-half RL substitution)[i]	0.56

Chemical	Consumption Category or Scenario	DF (%)	HHRA ProUCL 4.0 EPCs		Distribution Test		MTCA-Conforming EPCs	
			Statistic Used	EPC (mg/kg)[a]	Lognormal R^2	Normal R^2	Statistic Used	EPC (mg/kg)[a]
Dioxin/furan TEQ	tribal clamming – RME and 183 days per year	100	95% adjusted gamma UCL	0.00142	0.935	0.579	Land's h-statistic [maximum detected value][b]	0.136 [0.00210]
	clamming – 7 days per year	100	99% Chebyshev (MVUE) UCL	0.000365	0.866	na	z-statistic maximum detected value [Land's h-statistic][c]	0.000186 0.000412 [0.0644]
	netfishing	100	99% Chebyshev (mean, SD) UCL	0.000610	0.899[j]	0.290	Land's h-statistic	0.000139
Total PCBs	beach play RME, Area 3	79	95% KM (Chebyshev) UCL	0.24	0.972	0.751	Land's h-statistic (Cohen's method)[f] [maximum detected value][b]	1.8 [0.42]
	beach play RME, Area 4	100	95% adjusted gamma UCL	11	0.986	0.460	Land's h-statistic [maximum detected value][b]	207 [23]
	beach play RME, Area 5	97	95% KM (Chebyshev) UCL	0.19	0.925	0.505	Land's h-statistic (one-half RL substitution)	0.13
	beach play RME, Area 7	71	97.5% KM (Chebyshev) UCL	0.23	0.866	0.555	maximum detected value [Land's h-statistic (Cohen's method)][c, g]	0.34 [0.19]
	tribal clamming – RME and 183 days per year	94	97.5% KM (Chebyshev) UCL	4.0	0.983	0.690	Land's h-statistic (one-half RL substitution)	3.9
	clamming – 7 days per year	88	97.5% KM (Chebyshev) UCL	1.5	0.970	0.152	Land's h-statistic (one-half RL substitution)	0.34
	netfishing	93	97.5% KM (Chebyshev) UCL	2.5	0.993	0.867	Land's h-statistic (one-half RL substitution)[k]	0.98

[a] EPCs are presented in mg/kg ww for seafood consumption scenarios and in mg/kg dw for sediment exposure scenarios.

[b] For some recalculated EPCs, the Land's h-statistic UCL is substantially greater than the maximum detected value. For comparison purposes, the maximum detected value is shown in brackets.

[c] The Land's h-statistic is presented for comparison purposes only, as indicated by the brackets around the UCL. While the lognormal R^2 value is less than the 0.9 cutoff, it approaches this threshold.

[d] To calculate the UCL, the lowest detected value was used in place of the RL for the non-detect values because the mean of the detected data (0.49) was only slightly higher than the RLs (0.45).

[e] Detection frequency less than 50%, so no UCL was calculated.

[f] To calculate the UCL in MTCAStat, the highest RL was used to represent the non-detect data.

[g] To calculate the UCL in MTCAStat, the average RL for the non-detect data was used.

[h] An additional UCL calculation was performed without the two highest RLs, but the EPC remained the same.

[i] Several datasets were evaluated in both MTCAStat and ProUCL to ensure that the same h-statistic was calculated in both programs.

[j] Equal to 0.90 when rounded to two significant figures.

[k] UCL calculated using BTNCTD.EXE program supplied by Gregory Glass (Glass 2007b; Land 1994) because ProUCL will not calculate an h-statistic for datasets with more than 1,000 values.

BCA – bias-corrected accelerated

DF – detection frequency

dw – dry weight

EPC – exposure point concentration

HHRA – human health risk assessment

H-UCL – UCL based on Land's h-statistic

KM – Kaplan-Meier method for calculating a UCL

MTCA – Model Toxics Control Act

MVUE – minimum-variance unbiased eliminator

na – not applicable; no analytical data for that chemical in that seafood category

nd – not detected

PAH – polycyclic aromatic hydrocarbon

PCB – polychlorinated biphenyl

RL – reporting limit

RME – reasonable maximum exposure

SD – standard deviation

t (t-distribution) – statistical method used to estimate the mean for a normally distributed set of samples

TEQ – toxic equivalent

UCL – upper confidence limit on the mean

ww – wet weight

ATTACHMENT 3. CHRONIC DAILY INTAKE RATES FOR CHEMICALS OF POTENTIAL CONCERN

Table 1. Chronic daily intake rates for RME and Suquamish seafood ingestion scenarios 2

Table 2. Chronic daily intake rates for CT seafood ingestion scenarios 5

Table 3. Chronic daily intake rates for one-meal-per-month seafood ingestion scenarios 7

Table 4. Chronic daily intake rates for additional child seafood ingestion scenarios (presented in the uncertainty analysis) 10

Table 5. Chronic daily intake rates for netfishing sediment exposure scenarios 13

Table 6. Chronic daily intake rates for clamming sediment exposure scenarios 15

Table 7a. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 1 and 2 17

Table 7b. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 3 and 4 19

Table 7c. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 5 and 6 21

Table 7d. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 7 and 8 23

Attachment 3. Chronic Daily Intake Rates for Chemicals of Potential Concern

This attachment to the LDW baseline HHRA presents the chronic daily intake (CDI) rates for chemicals of potential concern identified for the seafood ingestion and sediment exposure scenarios. CDI rates represent the estimated daily chemical dose for an individual averaged over the exposure duration for each scenario. Separate CDIs are calculated for chemicals with carcinogenic and non-carcinogenic effects because the averaging time over which the doses are calculated are different.

Tables 1 through 7 of this attachment present the results of CDI calculations performed using Equations 1 and 2 in Section B.3.4 and the exposure parameters given in Tables B.3-7 through B.3-24 of the main document. The CDI results are used in the risk characterization and uncertainty analysis (Sections B.5 and B.6, respectively). Risk estimates for COPCs that were never detected are presented in the uncertainty analysis. The CDIs are expressed in scientific notation (e.g., 1.0×10^{-5} is equivalent to 0.000010).

Table 1. Chronic daily intake rates for RME and Suquamish seafood ingestion scenarios

Chemical	Adult Tribal RME (Tulalip Data)		Child Tribal RME (Tulalip Data)		API – RME		Adult Tribal (Suquamish Data)	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Detected Chemicals								
4-Methylphenol	5.0×10^{-4}	5.0×10^{-4}	9.2×10^{-5}	1.1×10^{-3}	1.0×10^{-4}	2.3×10^{-4}	1.8×10^{-3}	1.8×10^{-3}
Aldrin	2.8×10^{-6}	2.8×10^{-6}	5.2×10^{-7}	6.1×10^{-6}	6.0×10^{-7}	1.4×10^{-6}	1.2×10^{-5}	1.2×10^{-5}
alpha-BHC	2.6×10^{-6}	2.6×10^{-6}	4.8×10^{-7}	5.6×10^{-6}	5.0×10^{-7}	1.2×10^{-6}	8.9×10^{-6}	8.9×10^{-6}
Antimony	4.7×10^{-5}	4.7×10^{-5}	8.7×10^{-6}	1.0×10^{-4}	1.9×10^{-5}	4.4×10^{-5}	5.1×10^{-4}	5.1×10^{-4}
Arsenic[a,b]	9.7×10^{-4}	9.7×10^{-4}	1.8×10^{-4}	2.1×10^{-3}	4.4×10^{-4}	1.0×10^{-3}	1.1×10^{-2}	1.1×10^{-2}
beta-BHC	3.5×10^{-6}	3.5×10^{-6}	6.5×10^{-7}	7.6×10^{-6}	8.0×10^{-7}	1.9×10^{-6}	1.6×10^{-5}	1.6×10^{-5}
Bis(2-ethylhexyl)phthalate	4.5×10^{-4}	4.5×10^{-4}	8.3×10^{-5}	9.7×10^{-4}	1.3×10^{-4}	3.1×10^{-4}	2.8×10^{-3}	2.8×10^{-3}
Butyl benzyl phthalate	5.4×10^{-4}	5.4×10^{-4}	1.0×10^{-4}	1.2×10^{-3}	1.1×10^{-4}	2.5×10^{-4}	1.4×10^{-3}	1.4×10^{-3}
Cadmium	9.3×10^{-5}	9.3×10^{-5}	1.7×10^{-5}	2.0×10^{-4}	4.7×10^{-5}	1.1×10^{-4}	7.0×10^{-4}	7.0×10^{-4}
Carbazole	2.3×10^{-3}	2.3×10^{-3}	4.2×10^{-4}	4.9×10^{-3}	5.9×10^{-4}	1.4×10^{-3}	1.2×10^{-2}	1.2×10^{-2}
Carcinogenic PAHs[a,c]	9.8×10^{-6}	9.8×10^{-6}	1.8×10^{-6}	2.1×10^{-5}	4.4×10^{-6}	1.0×10^{-5}	1.1×10^{-4}	1.1×10^{-4}
Total chlordane	1.7×10^{-5}	1.7×10^{-5}	3.1×10^{-6}	3.6×10^{-5}	5.0×10^{-6}	1.2×10^{-5}	1.0×10^{-4}	1.0×10^{-4}
Chromium	4.7×10^{-4}	4.7×10^{-4}	8.6×10^{-5}	1.0×10^{-3}	1.8×10^{-4}	4.4×10^{-4}	4.7×10^{-3}	4.7×10^{-3}
Copper	8.6×10^{-3}	8.6×10^{-3}	1.6×10^{-3}	1.8×10^{-2}	2.2×10^{-3}	5.1×10^{-3}	4.2×10^{-2}	4.2×10^{-2}
Total DDTs	6.2×10^{-5}	6.2×10^{-5}	1.1×10^{-5}	1.3×10^{-4}	1.7×10^{-5}	4.0×10^{-5}	3.2×10^{-4}	3.2×10^{-4}
Dieldrin	8.2×10^{-6}	8.2×10^{-6}	1.5×10^{-6}	1.8×10^{-5}	2.8×10^{-6}	6.6×10^{-6}	7.3×10^{-5}	7.3×10^{-5}
Endrin	3.0×10^{-6}	3.0×10^{-6}	5.5×10^{-7}	6.4×10^{-6}	6.5×10^{-7}	1.5×10^{-6}	1.2×10^{-5}	1.2×10^{-5}
Endrin aldehyde	1.1×10^{-5}	1.1×10^{-5}	2.1×10^{-6}	2.4×10^{-5}	3.4×10^{-6}	7.8×10^{-6}	7.1×10^{-5}	7.1×10^{-5}
gamma-BHC	4.2×10^{-6}	4.2×10^{-6}	7.8×10^{-7}	9.1×10^{-6}	1.0×10^{-6}	2.4×10^{-6}	2.0×10^{-5}	2.0×10^{-5}
Heptachlor	3.2×10^{-6}	3.2×10^{-6}	5.9×10^{-7}	6.9×10^{-6}	7.1×10^{-7}	1.7×10^{-6}	1.3×10^{-5}	1.3×10^{-5}
Heptachlor epoxide	3.4×10^{-6}	3.4×10^{-6}	6.2×10^{-7}	7.3×10^{-6}	9.8×10^{-7}	2.3×10^{-6}	1.9×10^{-5}	1.9×10^{-5}
Hexachlorobenzene	6.5×10^{-6}	6.5×10^{-6}	1.2×10^{-6}	1.4×10^{-5}	1.5×10^{-6}	3.4×10^{-6}	2.2×10^{-5}	2.2×10^{-5}

Chemical	Adult Tribal RME (Tulalip Data)		Child Tribal RME (Tulalip Data)		API – RME		Adult Tribal (Suquamish Data)	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Mercury	5.1×10^{-5}	5.1×10^{-5}	9.4×10^{-6}	1.1×10^{-4}	1.1×10^{-5}	2.5×10^{-5}	2.0×10^{-4}	2.0×10^{-4}
Nickel	4.0×10^{-4}	4.0×10^{-4}	7.5×10^{-5}	8.7×10^{-4}	1.6×10^{-4}	3.8×10^{-4}	4.3×10^{-3}	4.3×10^{-3}
PCB TEQ[a]	8.1×10^{-9}	8.1×10^{-9}	1.5×10^{-9}	1.8×10^{-8}	2.6×10^{-9}	6.1×10^{-9}	4.9×10^{-8}	4.9×10^{-8}
Total PCBs	8.0×10^{-4}	8.0×10^{-4}	1.5×10^{-4}	1.7×10^{-3}	2.5×10^{-4}	5.8×10^{-4}	5.5×10^{-3}	5.5×10^{-3}
Pentachlorophenol	7.3×10^{-4}	7.3×10^{-4}	1.3×10^{-4}	1.6×10^{-3}	1.8×10^{-4}	4.1×10^{-4}	4.0×10^{-3}	4.0×10^{-3}
TBT (as ion)	2.1×10^{-4}	2.1×10^{-4}	3.9×10^{-5}	4.5×10^{-4}	8.5×10^{-5}	2.0×10^{-4}	2.3×10^{-3}	2.3×10^{-3}
Vanadium	8.3×10^{-4}	8.3×10^{-4}	1.5×10^{-4}	1.8×10^{-3}	3.4×10^{-4}	7.8×10^{-4}	8.9×10^{-3}	8.9×10^{-3}
Zinc	3.4×10^{-2}	3.4×10^{-2}	6.3×10^{-3}	7.3×10^{-2}	9.5×10^{-3}	2.2×10^{-2}	1.9×10^{-1}	1.9×10^{-1}
Undetected Chemicals								
1,2-Diphenylhydrazine[d]	3.2×10^{-5}	3.2×10^{-5}	3.6×10^{-6}	4.2×10^{-5}	9.6×10^{-6}	2.2×10^{-5}	2.4×10^{-4}	2.4×10^{-4}
1,3-Dichlorobenzene	2.0×10^{-4}	2.0×10^{-4}	3.7×10^{-5}	4.3×10^{-4}	3.5×10^{-5}	8.1×10^{-5}	6.0×10^{-4}	6.0×10^{-4}
1,4-Dichlorobenzene	2.0×10^{-4}	2.0×10^{-4}	3.7×10^{-5}	4.3×10^{-4}	3.5×10^{-5}	8.1×10^{-5}	6.0×10^{-4}	6.0×10^{-4}
2,4,6-Trichlorophenol	1.6×10^{-3}	1.6×10^{-3}	3.0×10^{-4}	3.4×10^{-3}	3.7×10^{-4}	8.6×10^{-4}	7.0×10^{-3}	7.0×10^{-3}
2,4-Dichlorophenol	4.1×10^{-4}	4.1×10^{-4}	7.6×10^{-5}	8.9×10^{-4}	7.1×10^{-5}	1.7×10^{-4}	1.2×10^{-3}	1.2×10^{-3}
2,4-Dinitrophenol	5.0×10^{-3}	5.0×10^{-3}	9.3×10^{-4}	1.1×10^{-2}	1.0×10^{-3}	2.4×10^{-3}	1.9×10^{-2}	1.9×10^{-2}
2,4-Dinitrotoluene	1.5×10^{-3}	1.5×10^{-3}	2.8×10^{-4}	3.3×10^{-3}	3.5×10^{-4}	8.2×10^{-4}	6.7×10^{-3}	6.7×10^{-3}
2,6-Dinitrotoluene	1.5×10^{-3}	1.5×10^{-3}	2.8×10^{-4}	3.3×10^{-3}	3.5×10^{-4}	8.2×10^{-4}	6.6×10^{-3}	6.6×10^{-3}
2-Chlorophenol	4.1×10^{-4}	4.1×10^{-4}	7.6×10^{-5}	8.9×10^{-4}	7.2×10^{-5}	1.7×10^{-4}	1.2×10^{-3}	1.2×10^{-3}
3,3'-Dichlorobenzidine[a]	1.0×10^{-2}	1.0×10^{-2}	1.9×10^{-3}	2.2×10^{-2}	1.9×10^{-3}	4.5×10^{-3}	3.1×10^{-2}	3.1×10^{-2}
3-Nitroaniline	3.2×10^{-3}	3.2×10^{-3}	5.9×10^{-4}	6.9×10^{-3}	7.5×10^{-4}	1.8×10^{-3}	1.5×10^{-2}	1.5×10^{-2}
4,6-Dinitro-o-cresol	3.2×10^{-3}	3.2×10^{-3}	5.9×10^{-4}	6.9×10^{-3}	7.5×10^{-4}	1.7×10^{-3}	1.5×10^{-2}	1.5×10^{-2}
4-Chloroaniline[a]	1.0×10^{-3}	1.0×10^{-3}	1.9×10^{-4}	2.2×10^{-3}	1.9×10^{-4}	4.5×10^{-4}	3.1×10^{-3}	3.1×10^{-3}
4-Nitroaniline	3.0×10^{-3}	3.0×10^{-3}	5.6×10^{-4}	6.5×10^{-3}	7.1×10^{-4}	1.7×10^{-3}	1.4×10^{-2}	1.4×10^{-2}
Aniline	4.1×10^{-3}	4.1×10^{-3}	7.6×10^{-4}	8.9×10^{-3}	7.1×10^{-4}	1.7×10^{-3}	1.2×10^{-2}	1.2×10^{-2}
Benzidine[a]	2.4×10^{-2}	2.4×10^{-2}	4.4×10^{-3}	5.2×10^{-2}	4.5×10^{-3}	1.1×10^{-2}	7.2×10^{-2}	7.2×10^{-2}

Chemical	Adult Tribal RME (Tulalip Data)		Child Tribal RME (Tulalip Data)		API – RME		Adult Tribal (Suquamish Data)	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Bis(2-chloroethyl)ether	3.4×10^{-4}	3.4×10^{-4}	6.4×10^{-5}	7.4×10^{-4}	5.2×10^{-5}	1.2×10^{-4}	7.9×10^{-4}	7.9×10^{-4}
Bis(2-chloroisopropyl)ether	2.0×10^{-4}	2.0×10^{-4}	3.7×10^{-5}	4.3×10^{-4}	3.5×10^{-5}	8.3×10^{-5}	6.0×10^{-4}	6.0×10^{-4}
Hexachlorobutadiene	2.0×10^{-4}	2.0×10^{-4}	3.7×10^{-5}	4.3×10^{-4}	3.5×10^{-5}	8.2×10^{-5}	6.0×10^{-4}	6.0×10^{-4}
Hexachlorocyclopentadiene	3.9×10^{-2}	3.9×10^{-2}	7.2×10^{-3}	8.4×10^{-2}	9.1×10^{-3}	2.1×10^{-2}	1.8×10^{-1}	1.8×10^{-1}
Hexachloroethane	2.0×10^{-4}	2.0×10^{-4}	3.7×10^{-5}	4.3×10^{-4}	3.5×10^{-5}	8.2×10^{-5}	6.0×10^{-4}	6.0×10^{-4}
Nitrobenzene	2.0×10^{-4}	2.0×10^{-4}	3.7×10^{-5}	4.3×10^{-4}	3.5×10^{-5}	8.2×10^{-5}	6.0×10^{-4}	6.0×10^{-4}
N-Nitrosodimethylamine	3.4×10^{-3}	3.4×10^{-3}	6.2×10^{-4}	7.2×10^{-3}	4.8×10^{-4}	1.1×10^{-3}	6.9×10^{-3}	6.9×10^{-3}
N-Nitroso-di-n-propylamine[e]	2.0×10^{-4}	2.0×10^{-4}	3.7×10^{-5}	4.3×10^{-4}	3.5×10^{-5}	8.2×10^{-5}	6.0×10^{-4}	6.0×10^{-4}
N-Nitrosodiphenylamine	3.2×10^{-4}	3.2×10^{-4}	5.9×10^{-5}	6.9×10^{-4}	7.5×10^{-5}	1.8×10^{-4}	1.5×10^{-3}	1.5×10^{-3}
Toxaphene	4.2×10^{-4}	4.2×10^{-4}	7.8×10^{-5}	9.1×10^{-4}	1.3×10^{-4}	2.9×10^{-4}	2.7×10^{-3}	2.7×10^{-3}

[a] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally between the remaining consumption categories

[b] Arsenic CDI is based on inorganic arsenic.

[c] Carcinogenic PAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document is from 2004 only due to high reporting limits in historical data. All carcinogenic PAH data is analyzed in the uncertainty analysis (Section B.6).

[d] No benthic whole body or clam data were available for 1,2-diphenylhydrazine. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to benthic whole body and mussels was divided proportionally between the remaining consumption categories

[e] One composite sample of whole-body English sole contained a detected concentration of 0.27 mg/kg ww. However, this result was qualified as JN (estimated concentration, tentative identification). Given the uncertain quantification for this single result (all other results were undetected), the risks for this chemical are discussed in the uncertainty analysis (Section B.6).

API – Asian and Pacific Islander

RME – reasonable maximum exposure

Table 2. Chronic daily intake rates for CT seafood ingestion scenarios

Chemical	Adult Tribal CT (Tulalip Data)		Child Tribal CT (Tulalip Data)		API – CT	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Detected Chemicals						
4-Methylphenol	1.9×10^{-5}	4.5×10^{-5}	8.3×10^{-6}	9.7×10^{-5}	1.5×10^{-6}	1.2×10^{-5}
Aldrin	8.7×10^{-8}	2.0×10^{-7}	3.7×10^{-8}	4.4×10^{-7}	9.2×10^{-9}	7.2×10^{-8}
alpha-BHC	8.6×10^{-8}	2.0×10^{-7}	3.7×10^{-8}	4.3×10^{-7}	8.9×10^{-9}	6.9×10^{-8}
Antimony	1.7×10^{-6}	4.0×10^{-6}	7.3×10^{-7}	8.5×10^{-6}	3.3×10^{-7}	2.5×10^{-6}
Arsenic[a,b]	4.1×10^{-5}	9.7×10^{-5}	1.7×10^{-5}	2.0×10^{-4}	8.5×10^{-6}	6.6×10^{-5}
beta-BHC	1.4×10^{-7}	3.3×10^{-7}	6.0×10^{-8}	7.0×10^{-7}	1.7×10^{-8}	1.3×10^{-7}
Bis(2-ethylhexyl)phthalate	1.2×10^{-5}	2.8×10^{-5}	5.2×10^{-6}	6.1×10^{-5}	1.9×10^{-6}	1.4×10^{-5}
Butyl benzyl phthalate	2.3×10^{-5}	5.4×10^{-5}	9.9×10^{-6}	1.2×10^{-4}	2.1×10^{-6}	1.6×10^{-5}
Cadmium	5.4×10^{-6}	1.2×10^{-5}	2.3×10^{-6}	2.7×10^{-5}	1.3×10^{-6}	1.0×10^{-5}
Carbazole	5.0×10^{-5}	1.2×10^{-4}	2.2×10^{-5}	2.5×10^{-4}	4.2×10^{-6}	3.3×10^{-5}
Carcinogenic PAHs[a,c]	5.9×10^{-7}	1.4×10^{-6}	2.5×10^{-7}	3.0×10^{-6}	1.0×10^{-7}	8.0×10^{-7}
Total chlordane	5.7×10^{-7}	1.3×10^{-6}	2.5×10^{-7}	2.9×10^{-6}	7.9×10^{-8}	6.1×10^{-7}
Chromium	2.4×10^{-5}	5.5×10^{-5}	1.0×10^{-5}	1.2×10^{-4}	4.6×10^{-6}	3.6×10^{-5}
Copper	4.9×10^{-4}	1.1×10^{-3}	2.1×10^{-4}	2.5×10^{-3}	5.9×10^{-5}	4.6×10^{-4}
Total DDTs	3.1×10^{-6}	7.1×10^{-6}	1.3×10^{-6}	1.5×10^{-5}	4.0×10^{-7}	3.1×10^{-6}
Dieldrin	1.6×10^{-7}	3.7×10^{-7}	6.9×10^{-8}	8.0×10^{-7}	2.2×10^{-8}	1.7×10^{-7}
Endrin	1.1×10^{-7}	2.6×10^{-7}	4.8×10^{-8}	5.6×10^{-7}	1.2×10^{-8}	9.7×10^{-8}
Endrin aldehyde	1.3×10^{-7}	3.0×10^{-7}	5.6×10^{-8}	6.5×10^{-7}	1.4×10^{-8}	1.1×10^{-7}
gamma-BHC	9.8×10^{-8}	2.3×10^{-7}	4.2×10^{-8}	4.9×10^{-7}	1.1×10^{-8}	8.3×10^{-8}
Heptachlor	8.9×10^{-8}	2.1×10^{-7}	3.8×10^{-8}	4.5×10^{-7}	9.4×10^{-9}	7.3×10^{-8}
Heptachlor epoxide	1.3×10^{-7}	3.0×10^{-7}	5.6×10^{-8}	6.5×10^{-7}	1.6×10^{-8}	1.2×10^{-7}
Hexachlorobenzene	1.5×10^{-7}	3.6×10^{-7}	6.6×10^{-8}	7.7×10^{-7}	2.2×10^{-8}	1.7×10^{-7}
Mercury	3.0×10^{-6}	6.9×10^{-6}	1.3×10^{-6}	1.5×10^{-5}	3.0×10^{-7}	2.3×10^{-6}
Nickel	2.3×10^{-5}	5.3×10^{-5}	9.8×10^{-6}	1.1×10^{-4}	4.3×10^{-6}	3.4×10^{-5}
PCB TEQ[a]	3.8×10^{-10}	8.8×10^{-10}	1.5×10^{-10}	1.8×10^{-9}	5.0×10^{-11}	3.9×10^{-10}
Total PCBs	3.2×10^{-5}	7.5×10^{-5}	1.4×10^{-5}	1.6×10^{-4}	4.2×10^{-6}	3.3×10^{-5}
Pentachlorophenol	1.4×10^{-5}	3.2×10^{-5}	5.9×10^{-6}	6.9×10^{-5}	2.1×10^{-6}	1.7×10^{-5}
TBT (as ion)	1.0×10^{-5}	2.4×10^{-5}	4.5×10^{-6}	5.2×10^{-5}	2.0×10^{-6}	1.6×10^{-5}
Vanadium	4.6×10^{-5}	1.1×10^{-4}	2.0×10^{-5}	2.3×10^{-4}	8.8×10^{-6}	6.8×10^{-5}
Zinc	2.1×10^{-3}	4.8×10^{-3}	8.9×10^{-4}	1.0×10^{-2}	2.7×10^{-4}	2.1×10^{-3}
Undetected chemicals						
1,2-Diphenylhydrazine[d]	2.4×10^{-6}	5.7×10^{-6}	5.2×10^{-7}	6.1×10^{-6}	2.8×10^{-7}	2.2×10^{-6}
1,3-Dichlorobenzene	9.2×10^{-6}	2.1×10^{-5}	4.0×10^{-6}	4.6×10^{-5}	7.3×10^{-7}	5.6×10^{-6}

Chemical	Adult Tribal CT (Tulalip Data)		Child Tribal CT (Tulalip Data)		API – CT	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
1,4-Dichlorobenzene	9.2×10^{-6}	2.1×10^{-5}	4.0×10^{-6}	4.6×10^{-5}	7.3×10^{-7}	5.6×10^{-6}
2,4,6-Trichlorophenol	4.2×10^{-5}	9.9×10^{-5}	1.8×10^{-5}	2.1×10^{-4}	3.3×10^{-6}	2.5×10^{-5}
2,4-Dichlorophenol	1.9×10^{-5}	4.5×10^{-5}	8.3×10^{-6}	9.7×10^{-5}	1.5×10^{-6}	1.2×10^{-5}
2,4-Dinitrophenol	2.0×10^{-4}	4.7×10^{-4}	8.7×10^{-5}	1.0×10^{-3}	1.6×10^{-5}	1.3×10^{-4}
2,4-Dinitrotoluene	3.2×10^{-5}	7.4×10^{-5}	1.4×10^{-5}	1.6×10^{-4}	2.6×10^{-6}	2.0×10^{-5}
2,6-Dinitrotoluene	2.9×10^{-5}	6.7×10^{-5}	1.2×10^{-5}	1.4×10^{-4}	2.3×10^{-6}	1.8×10^{-5}
2-Chlorophenol	1.9×10^{-5}	4.5×10^{-5}	8.3×10^{-6}	9.7×10^{-5}	1.5×10^{-6}	1.2×10^{-5}
3,3'-Dichlorobenzidine[a]	5.4×10^{-4}	1.3×10^{-3}	2.3×10^{-4}	2.7×10^{-3}	4.4×10^{-5}	3.4×10^{-4}
3-Nitroaniline	1.1×10^{-4}	2.5×10^{-4}	4.6×10^{-5}	5.3×10^{-4}	8.5×10^{-6}	6.6×10^{-5}
4,6-Dinitro-o-cresol	9.7×10^{-5}	2.3×10^{-4}	4.2×10^{-5}	4.9×10^{-4}	8.0×10^{-6}	6.2×10^{-5}
4-Chloroaniline[a]	5.4×10^{-5}	1.3×10^{-4}	2.3×10^{-5}	2.7×10^{-4}	4.4×10^{-6}	3.4×10^{-5}
4-Nitroaniline	7.1×10^{-5}	1.7×10^{-4}	3.1×10^{-5}	3.6×10^{-4}	5.7×10^{-6}	4.4×10^{-5}
Aniline	2.1×10^{-4}	5.0×10^{-4}	9.1×10^{-5}	1.1×10^{-3}	1.6×10^{-5}	1.3×10^{-4}
Benzidine[a]	1.3×10^{-3}	3.0×10^{-3}	5.6×10^{-4}	6.5×10^{-3}	1.1×10^{-4}	8.2×10^{-4}
Bis(2-chloroethyl)ether	1.3×10^{-5}	2.9×10^{-5}	5.4×10^{-6}	6.3×10^{-5}	9.4×10^{-7}	7.3×10^{-6}
Bis(2-chloroisopropyl)ether	9.3×10^{-6}	2.2×10^{-5}	4.0×10^{-6}	4.7×10^{-5}	7.5×10^{-7}	5.9×10^{-6}
Hexachlorobutadiene	9.2×10^{-6}	2.1×10^{-5}	4.0×10^{-6}	4.6×10^{-5}	7.3×10^{-7}	5.7×10^{-6}
Hexachlorocyclopentadiene	1.3×10^{-3}	2.9×10^{-3}	5.4×10^{-4}	6.3×10^{-3}	1.0×10^{-4}	8.0×10^{-4}
Hexachloroethane	9.2×10^{-6}	2.1×10^{-5}	4.0×10^{-6}	4.6×10^{-5}	7.3×10^{-7}	5.7×10^{-6}
Nitrobenzene	9.2×10^{-6}	2.1×10^{-5}	4.0×10^{-6}	4.6×10^{-5}	7.3×10^{-7}	5.7×10^{-6}
N-Nitrosodimethylamine	9.0×10^{-5}	2.1×10^{-4}	3.9×10^{-5}	4.5×10^{-4}	6.1×10^{-6}	4.7×10^{-5}
N-Nitroso-di-n-propylamine[e]	9.3×10^{-6}	2.2×10^{-5}	4.0×10^{-6}	4.7×10^{-5}	7.4×10^{-7}	5.8×10^{-6}
N-Nitrosodiphenylamine	9.6×10^{-6}	2.2×10^{-5}	4.1×10^{-6}	4.8×10^{-5}	7.9×10^{-7}	6.2×10^{-6}
Toxaphene	7.7×10^{-6}	1.8×10^{-5}	3.3×10^{-6}	3.9×10^{-5}	9.4×10^{-7}	7.3×10^{-6}

[a] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally between the remaining consumption categories

[b] Arsenic CDI is based on inorganic arsenic.

[c] Carcinogenic PAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document is from 2004 only due to high reporting limits in historical data. All carcinogenic PAH data is analyzed in the uncertainty analysis (Section B.6).

[d] No benthic whole body or clam data were available for 1,2-diphenylhydrazine. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to benthic whole body and mussels was divided proportionally between the remaining consumption categories

[e] One composite sample of whole-body English sole contained a detected concentration of 0.27 mg/kg ww. However, this result was qualified as JN (estimated concentration, tentative identification). Given the uncertain quantification for this single result (all other results were undetected), the risks for this chemical are discussed in the uncertainty analysis (Section B.6).

API – Asian and Pacific Islander

CT – central tendency

Table 3. Chronic daily intake rates for one-meal-per-month seafood ingestion scenarios

Chemical	Benthic Fish		Pelagic Fish		Clam		Crab	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Detected Chemicals								
4-Methylphenol	2.7×10^{-5}	6.3×10^{-5}	6.7×10^{-5}	1.6×10^{-4}	1.4×10^{-6}	3.2×10^{-6}	2.7×10^{-5}	6.3×10^{-5}
Aldrin	1.6×10^{-7}	3.8×10^{-7}	1.6×10^{-7}	3.8×10^{-7}	4.5×10^{-8}	1.0×10^{-7}	1.6×10^{-7}	3.8×10^{-7}
alpha-BHC	1.6×10^{-7}	3.8×10^{-7}	1.6×10^{-7}	3.8×10^{-7}	2.2×10^{-8}	5.2×10^{-8}	1.6×10^{-7}	3.8×10^{-7}
Antimony	4.5×10^{-7}	1.0×10^{-6}	1.7×10^{-7}	4.1×10^{-7}	4.0×10^{-6}	9.4×10^{-6}	4.3×10^{-7}	9.9×10^{-7}
Arsenic[a]	2.8×10^{-7}	6.5×10^{-7}	3.9×10^{-6}	9.2×10^{-6}	9.0×10^{-5}	2.1×10^{-4}	1.9×10^{-6}	4.4×10^{-6}
beta-BHC	1.6×10^{-7}	3.8×10^{-7}	3.5×10^{-7}	8.1×10^{-7}	5.4×10^{-8}	1.3×10^{-7}	1.8×10^{-7}	4.3×10^{-7}
Bis(2-ethylhexyl)phthalate	5.8×10^{-5}	1.4×10^{-4}	9.4×10^{-5}	2.2×10^{-4}	5.8×10^{-6}	1.4×10^{-5}	5.8×10^{-6}	1.4×10^{-5}
Butyl benzyl phthalate	2.7×10^{-5}	6.3×10^{-5}	3.8×10^{-5}	8.8×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	2.7×10^{-5}	6.3×10^{-5}
Cadmium	1.8×10^{-7}	4.2×10^{-7}	7.2×10^{-7}	1.7×10^{-6}	4.9×10^{-6}	1.1×10^{-5}	1.2×10^{-6}	2.8×10^{-6}
Carbazole	6.7×10^{-5}	1.6×10^{-4}	6.3×10^{-4}	1.5×10^{-3}	4.5×10^{-6}	1.0×10^{-5}	6.7×10^{-5}	1.6×10^{-4}
Carcinogenic PAHs[b]	6.7×10^{-7}	1.6×10^{-6}	4.3×10^{-8}	9.9×10^{-8}	9.0×10^{-7}	2.1×10^{-6}	2.9×10^{-8}	6.8×10^{-8}
Total chlordane	9.0×10^{-7}	2.1×10^{-6}	3.8×10^{-6}	8.8×10^{-6}	2.1×10^{-7}	4.9×10^{-7}	2.0×10^{-7}	4.7×10^{-7}
Chromium	2.8×10^{-6}	6.5×10^{-6}	9.0×10^{-6}	2.1×10^{-5}	3.5×10^{-5}	8.3×10^{-5}	7.2×10^{-6}	1.7×10^{-5}
Copper	7.2×10^{-5}	1.7×10^{-4}	7.6×10^{-5}	1.8×10^{-4}	2.7×10^{-4}	6.3×10^{-4}	3.8×10^{-4}	8.9×10^{-4}
Total DDTs	3.8×10^{-6}	8.8×10^{-6}	1.1×10^{-5}	2.5×10^{-5}	6.7×10^{-7}	1.6×10^{-6}	1.0×10^{-6}	2.4×10^{-6}
Dieldrin	1.6×10^{-7}	3.8×10^{-7}	1.6×10^{-7}	3.8×10^{-7}	5.4×10^{-7}	1.3×10^{-6}	1.6×10^{-7}	3.8×10^{-7}
Endrin	1.6×10^{-7}	3.8×10^{-7}	3.0×10^{-7}	7.0×10^{-7}	2.5×10^{-8}	5.8×10^{-8}	1.6×10^{-7}	3.8×10^{-7}
Endrin aldehyde	3.6×10^{-7}	8.5×10^{-7}	3.5×10^{-6}	8.1×10^{-6}	8.1×10^{-8}	1.9×10^{-7}	1.6×10^{-7}	3.8×10^{-7}
gamma-BHC	1.6×10^{-7}	3.8×10^{-7}	9.0×10^{-8}	2.1×10^{-7}	1.1×10^{-7}	2.6×10^{-7}	1.8×10^{-7}	4.2×10^{-7}
Heptachlor	1.6×10^{-7}	3.8×10^{-7}	4.3×10^{-7}	1.0×10^{-6}	2.2×10^{-8}	5.2×10^{-8}	1.6×10^{-7}	3.8×10^{-7}
Heptachlor epoxide	1.6×10^{-7}	3.8×10^{-7}	4.5×10^{-7}	1.0×10^{-6}	6.7×10^{-8}	1.6×10^{-7}	9.4×10^{-8}	2.2×10^{-7}
Hexachlorobenzene	4.0×10^{-7}	9.4×10^{-7}	5.4×10^{-7}	1.3×10^{-6}	3.8×10^{-8}	9.0×10^{-8}	3.6×10^{-7}	8.4×10^{-7}

Chemical	Benthic Fish		Pelagic Fish		Clam		Crab	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Mercury	2.6×10^{-6}	6.1×10^{-6}	1.7×10^{-6}	4.1×10^{-6}	9.0×10^{-7}	2.1×10^{-6}	2.9×10^{-6}	6.7×10^{-6}
Nickel	2.8×10^{-6}	6.6×10^{-6}	2.1×10^{-5}	4.9×10^{-5}	3.1×10^{-5}	7.2×10^{-5}	2.5×10^{-6}	5.7×10^{-6}
PCB TEQ	5.4×10^{-10}	1.3×10^{-9}	1.5×10^{-9}	3.6×10^{-9}	1.4×10^{-10}	3.3×10^{-10}	1.1×10^{-10}	2.5×10^{-10}
Total PCBs	5.4×10^{-5}	1.3×10^{-4}	8.5×10^{-5}	2.0×10^{-4}	2.7×10^{-5}	6.3×10^{-5}	9.0×10^{-6}	2.1×10^{-5}
Pentachlorophenol	1.3×10^{-4}	3.0×10^{-4}	1.1×10^{-4}	2.5×10^{-4}	9.0×10^{-6}	2.1×10^{-5}	1.3×10^{-5}	3.0×10^{-5}
TBT (as ion)	1.5×10^{-7}	3.4×10^{-7}	3.6×10^{-6}	8.5×10^{-6}	1.8×10^{-5}	4.2×10^{-5}	1.3×10^{-6}	3.1×10^{-6}
Vanadium	5.8×10^{-6}	1.4×10^{-5}	2.8×10^{-5}	6.5×10^{-5}	6.7×10^{-5}	1.6×10^{-4}	4.9×10^{-6}	1.1×10^{-5}
Zinc	3.8×10^{-4}	9.0×10^{-4}	9.8×10^{-4}	2.3×10^{-3}	1.2×10^{-3}	2.7×10^{-3}	1.6×10^{-3}	3.8×10^{-3}
Undetected chemicals								
1,2-Diphenylhydrazine	1.2×10^{-6}	2.8×10^{-6}	1.8×10^{-6}	4.2×10^{-6}	na	na	1.2×10^{-6}	2.8×10^{-6}
1,3-Dichlorobenzene	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
1,4-Dichlorobenzene	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
2,4,6-Trichlorophenol	6.7×10^{-5}	1.6×10^{-4}	3.4×10^{-4}	7.8×10^{-4}	1.8×10^{-6}	4.2×10^{-6}	6.7×10^{-5}	1.6×10^{-4}
2,4-Dichlorophenol	2.7×10^{-5}	6.3×10^{-5}	2.7×10^{-5}	6.3×10^{-5}	1.8×10^{-6}	4.2×10^{-6}	2.7×10^{-5}	6.3×10^{-5}
2,4-Dinitrophenol	2.7×10^{-4}	6.3×10^{-4}	6.7×10^{-4}	1.6×10^{-3}	1.8×10^{-5}	4.2×10^{-5}	2.7×10^{-4}	6.3×10^{-4}
2,4-Dinitrotoluene	3.4×10^{-5}	7.8×10^{-5}	3.4×10^{-4}	7.8×10^{-4}	1.8×10^{-6}	4.2×10^{-6}	6.7×10^{-5}	1.6×10^{-4}
2,6-Dinitrotoluene	3.4×10^{-5}	7.8×10^{-5}	3.4×10^{-4}	7.8×10^{-4}	9.0×10^{-7}	2.1×10^{-6}	6.7×10^{-5}	1.6×10^{-4}
2-Chlorophenol	2.7×10^{-5}	6.3×10^{-5}	2.7×10^{-5}	6.3×10^{-5}	1.8×10^{-6}	4.2×10^{-6}	2.7×10^{-5}	6.3×10^{-5}
3,3'-Dichlorobenzidine	6.7×10^{-4}	1.6×10^{-3}	6.7×10^{-4}	1.6×10^{-3}	4.5×10^{-5}	1.0×10^{-4}	6.7×10^{-4}	1.6×10^{-3}
3-Nitroaniline	1.3×10^{-4}	3.0×10^{-4}	6.7×10^{-4}	1.6×10^{-3}	9.0×10^{-6}	2.1×10^{-5}	1.3×10^{-4}	3.0×10^{-4}
4,6-Dinitro-o-cresol	1.3×10^{-4}	3.0×10^{-4}	6.7×10^{-4}	1.6×10^{-3}	9.0×10^{-6}	2.1×10^{-5}	1.3×10^{-4}	3.0×10^{-4}
4-Chloroaniline	6.7×10^{-5}	1.6×10^{-4}	6.7×10^{-5}	1.6×10^{-4}	4.5×10^{-6}	1.0×10^{-5}	6.7×10^{-5}	1.6×10^{-4}
4-Nitroaniline	6.7×10^{-5}	1.6×10^{-4}	6.7×10^{-4}	1.6×10^{-3}	4.5×10^{-6}	1.0×10^{-5}	1.3×10^{-4}	3.0×10^{-4}
Aniline	2.7×10^{-4}	6.3×10^{-4}	2.7×10^{-4}	6.3×10^{-4}	1.8×10^{-5}	4.2×10^{-5}	2.7×10^{-4}	6.3×10^{-4}
Benzidine	1.1×10^{-3}	2.6×10^{-3}	1.6×10^{-3}	3.8×10^{-3}	1.1×10^{-4}	2.6×10^{-4}	1.6×10^{-3}	3.8×10^{-3}

Chemical	Benthic Fish		Pelagic Fish		Clam		Crab	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Bis(2-chloroethyl)ether	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	2.7×10^{-5}	6.3×10^{-5}
Bis(2-chloroisopropyl)ether	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
Hexachlorobutadiene	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
Hexachlorocyclopentadiene	1.6×10^{-3}	3.8×10^{-3}	8.1×10^{-3}	1.9×10^{-2}	1.1×10^{-4}	2.6×10^{-4}	1.6×10^{-3}	3.8×10^{-3}
Hexachloroethane	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
Nitrobenzene	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
N-Nitrosodimethylamine	1.3×10^{-4}	3.0×10^{-4}	1.3×10^{-4}	3.0×10^{-4}	9.0×10^{-7}	2.1×10^{-6}	2.7×10^{-4}	6.3×10^{-4}
N-Nitroso-di-n-propylamine[c]	1.3×10^{-5}	3.0×10^{-5}	1.3×10^{-5}	3.0×10^{-5}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
N-Nitrosodiphenylamine	1.3×10^{-5}	3.0×10^{-5}	6.7×10^{-5}	1.6×10^{-4}	9.0×10^{-7}	2.1×10^{-6}	1.3×10^{-5}	3.0×10^{-5}
Toxaphene	9.8×10^{-6}	2.3×10^{-5}	1.1×10^{-4}	2.5×10^{-4}	5.8×10^{-6}	1.4×10^{-5}	8.1×10^{-6}	1.9×10^{-5}

[a] Arsenic CDI is based on inorganic arsenic.

[b] Carcinogenic PAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document is from 2004 only due to high reporting limits in historical data. All carcinogenic PAH data is analyzed in the uncertainty analysis (Section B.6).

[c] One composite sample of whole-body English sole contained a detected concentration of 0.27 mg/kg ww. However, this result was qualified as JN (estimated concentration, tentative identification). Given the uncertain quantification for this single result (all other results were undetected), the risks for this chemical are discussed in the uncertainty analysis (Section B.6).

nd – no data

Table 4. Chronic daily intake rates for additional child seafood ingestion scenarios (presented in the uncertainty analysis)

Chemical	Child Tribal 95th percentile (Tulalip Data)		Child Tribal (Suquamish Data)	
	Cancer	Non-Cancer	Cancer	Non-Cancer
Detected Chemicals				
4-Methylphenol	5.0×10^{-5}	5.8×10^{-4}	2.2×10^{-4}	2.6×10^{-3}
Aldrin	2.1×10^{-7}	2.5×10^{-6}	1.2×10^{-6}	1.4×10^{-5}
alpha-BHC	2.0×10^{-7}	2.3×10^{-6}	1.0×10^{-6}	1.2×10^{-5}
Antimony	2.9×10^{-6}	3.4×10^{-5}	2.3×10^{-5}	2.6×10^{-4}
Arsenic[a,b]	6.1×10^{-5}	7.1×10^{-4}	4.7×10^{-4}	5.5×10^{-3}
beta-BHC	3.2×10^{-7}	3.7×10^{-6}	1.6×10^{-6}	1.8×10^{-5}
Bis(2-ethylhexyl)phthalate	5.3×10^{-5}	6.2×10^{-4}	2.1×10^{-4}	2.5×10^{-3}
Butyl benzyl phthalate	4.3×10^{-5}	5.0×10^{-4}	1.8×10^{-4}	2.1×10^{-3}
Cadmium	6.0×10^{-6}	6.9×10^{-5}	3.1×10^{-5}	3.6×10^{-4}
Carbazole	3.4×10^{-4}	4.0×10^{-3}	1.3×10^{-3}	1.5×10^{-2}
Carcinogenic PAHs[a,c]	6.1×10^{-7}	7.1×10^{-6}	4.7×10^{-6}	5.5×10^{-5}
Total chlordane	2.2×10^{-6}	2.5×10^{-5}	7.9×10^{-6}	9.2×10^{-5}
Chromium	3.1×10^{-5}	3.7×10^{-4}	2.2×10^{-4}	2.6×10^{-3}
Copper	5.4×10^{-4}	6.4×10^{-3}	3.0×10^{-3}	3.5×10^{-2}
Total DDTs	7.0×10^{-6}	8.1×10^{-5}	2.5×10^{-5}	2.9×10^{-4}
Dieldrin	5.4×10^{-7}	6.3×10^{-6}	3.7×10^{-6}	4.3×10^{-5}
Endrin	2.7×10^{-7}	3.1×10^{-6}	1.3×10^{-6}	1.5×10^{-5}
Endrin aldehyde	1.8×10^{-6}	2.1×10^{-5}	6.5×10^{-6}	7.5×10^{-5}
gamma-BHC	2.7×10^{-7}	3.2×10^{-6}	1.5×10^{-6}	1.7×10^{-5}
Heptachlor	3.2×10^{-7}	3.8×10^{-6}	1.5×10^{-6}	1.7×10^{-5}
Heptachlor epoxide	3.4×10^{-7}	4.0×10^{-6}	1.4×10^{-6}	1.7×10^{-5}
Hexachlorobenzene	5.4×10^{-7}	6.2×10^{-6}	2.6×10^{-6}	3.0×10^{-5}
Mercury	3.5×10^{-6}	4.0×10^{-5}	1.9×10^{-5}	2.3×10^{-4}
Nickel	3.1×10^{-5}	3.7×10^{-4}	2.0×10^{-4}	2.3×10^{-3}
PCB TEQ[a]	9.5×10^{-10}	1.1×10^{-8}	3.6×10^{-9}	4.2×10^{-8}
Total PCBs	7.2×10^{-5}	8.4×10^{-4}	3.2×10^{-4}	3.7×10^{-3}
Pentachlorophenol	6.7×10^{-5}	7.8×10^{-4}	3.0×10^{-4}	3.5×10^{-3}
TBT (as ion)	1.4×10^{-5}	1.6×10^{-4}	1.0×10^{-4}	1.2×10^{-3}
Vanadium	6.0×10^{-5}	6.9×10^{-4}	4.0×10^{-4}	4.7×10^{-3}
Zinc	2.4×10^{-3}	2.8×10^{-2}	1.4×10^{-2}	1.6×10^{-1}

Chemical	Child Tribal 95th Percentile (Tulalip Data)		Child Tribal (Suquamish Data)	
	Cancer	Non-Cancer	Cancer	Non-Cancer
Undetected chemicals				
1,2-Diphenylhydrazine[d]	1.7×10^{-6}	2.0×10^{-5}	7.9×10^{-6}	9.2×10^{-5}
1,3-Dichlorobenzene	1.5×10^{-5}	1.8×10^{-4}	8.0×10^{-5}	9.3×10^{-4}
1,4-Dichlorobenzene	1.5×10^{-5}	1.8×10^{-4}	8.0×10^{-5}	9.3×10^{-4}
2,4,6-Trichlorophenol	2.0×10^{-4}	2.4×10^{-3}	8.1×10^{-4}	9.4×10^{-3}
2,4-Dichlorophenol	3.2×10^{-5}	3.7×10^{-4}	1.6×10^{-4}	1.9×10^{-3}
2,4-Dinitrophenol	5.0×10^{-4}	5.9×10^{-3}	2.3×10^{-3}	2.6×10^{-2}
2,4-Dinitrotoluene	2.0×10^{-4}	2.4×10^{-3}	7.9×10^{-4}	9.3×10^{-3}
2,6-Dinitrotoluene	2.0×10^{-4}	2.3×10^{-3}	7.9×10^{-4}	9.2×10^{-3}
2-Chlorophenol	3.2×10^{-5}	3.7×10^{-4}	1.6×10^{-4}	1.9×10^{-3}
3,3'-Dichlorobenzidine[a]	8.0×10^{-4}	9.3×10^{-3}	4.1×10^{-3}	4.8×10^{-2}
3-Nitroaniline	4.1×10^{-4}	4.7×10^{-3}	1.6×10^{-3}	1.9×10^{-2}
4,6-Dinitro-o-cresol	4.1×10^{-4}	4.7×10^{-3}	1.6×10^{-3}	1.9×10^{-2}
4-Chloroaniline[a]	8.0×10^{-5}	9.3×10^{-4}	4.1×10^{-4}	4.8×10^{-3}
4-Nitroaniline	4.0×10^{-4}	4.7×10^{-3}	1.6×10^{-3}	1.8×10^{-2}
Aniline	3.2×10^{-4}	3.7×10^{-3}	1.6×10^{-3}	1.9×10^{-2}
Benzidine[a]	1.9×10^{-3}	2.2×10^{-2}	9.7×10^{-3}	1.1×10^{-1}
Bis(2-chloroethyl)ether	2.4×10^{-5}	2.8×10^{-4}	1.3×10^{-4}	1.6×10^{-3}
Bis(2-chloroisopropyl)ether	1.5×10^{-5}	1.8×10^{-4}	8.0×10^{-5}	9.3×10^{-4}
Hexachlorobutadiene	1.5×10^{-5}	1.8×10^{-4}	8.0×10^{-5}	9.3×10^{-4}
Hexachlorocyclopentadiene	4.9×10^{-3}	5.7×10^{-2}	2.0×10^{-2}	2.3×10^{-1}
Hexachloroethane	1.5×10^{-5}	1.8×10^{-4}	8.0×10^{-5}	9.3×10^{-4}
Nitrobenzene	1.5×10^{-5}	1.8×10^{-4}	8.0×10^{-5}	9.3×10^{-4}
N-Nitrosodimethylamine	2.4×10^{-4}	2.8×10^{-3}	1.3×10^{-3}	1.5×10^{-2}
N-Nitroso-di-n-propylamine[e]	1.5×10^{-5}	1.8×10^{-4}	8.0×10^{-5}	9.3×10^{-4}
N-Nitrosodiphenylamine	4.1×10^{-5}	4.7×10^{-4}	1.6×10^{-4}	1.9×10^{-3}
Toxaphene	6.0×10^{-5}	7.0×10^{-4}	2.3×10^{-4}	2.7×10^{-3}

[a] No mussel data were available for this chemical. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to mussels was divided proportionally between the remaining consumption categories.

[b] Arsenic CDI is based on inorganic arsenic.

[c] Carcinogenic PAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document is from 2004 only due to high reporting limits in historical data. All carcinogenic PAH data is analyzed in the uncertainty analysis (Section B.6).

[d] No benthic whole body or clam data were available for 1,2-diphenylhydrazine. When calculating the CDI and risk values, the portion of seafood consumption that had been assigned to benthic whole body and mussels was divided proportionally between the remaining consumption categories

[e] One composite sample of whole-body English sole contained a detected concentration of 0.27 mg/kg ww. However, this result was qualified as JN (estimated concentration, tentative identification). Given the uncertain quantification for this single result (all other results were undetected), the risks for this chemical are discussed in the uncertainty analysis (Section B.6).

Table 5. Chronic daily intake rates for netfishing sediment exposure scenarios

Chemical	Netfishing – RME				Netfishing – CT			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Detected Chemicals								
Aluminum[a]	2.4×10^{-3}	na	3.8×10^{-3}	na	8.3×10^{-4}	na	2.0×10^{-3}	na
Antimony[a]	4.3×10^{-7}	na	6.8×10^{-7}	na	1.5×10^{-7}	na	3.6×10^{-7}	na
Arsenic	2.6×10^{-6}	1.1×10^{-6}	4.2×10^{-6}	1.8×10^{-6}	9.2×10^{-7}	4.0×10^{-8}	2.2×10^{-6}	9.6×10^{-8}
Barium[a]	2.9×10^{-5}	na	4.6×10^{-5}	na	1.0×10^{-5}	na	2.4×10^{-5}	na
Cadmium[b]	1.9×10^{-7}	na	3.0×10^{-7}	na	6.6×10^{-8}	na	1.6×10^{-7}	na
Carcinogenic PAHs	7.1×10^{-8}	1.3×10^{-7}	1.1×10^{-7}	2.1×10^{-7}	2.5×10^{-8}	4.7×10^{-9}	6.0×10^{-8}	1.1×10^{-8}
Chromium[a]	6.4×10^{-6}	na	1.0×10^{-5}	na	2.2×10^{-6}	na	5.4×10^{-6}	na
Copper[a]	2.5×10^{-5}	na	4.0×10^{-5}	na	8.7×10^{-6}	na	2.1×10^{-5}	na
Dieldrin	5.6×10^{-10}	8.1×10^{-10}	9.0×10^{-10}	1.3×10^{-9}	2.0×10^{-10}	2.8×10^{-11}	4.7×10^{-10}	6.8×10^{-11}
Dioxin/furan TEQ	7.6×10^{-11}	3.3×10^{-11}	1.2×10^{-10}	5.3×10^{-11}	2.7×10^{-11}	1.2×10^{-12}	6.4×10^{-11}	2.8×10^{-12}
Iron[a]	3.6×10^{-3}	na	5.8×10^{-3}	na	1.3×10^{-3}	na	3.1×10^{-3}	na
Manganese[a]	4.5×10^{-5}	na	7.2×10^{-5}	na	1.6×10^{-5}	na	3.8×10^{-5}	na
PCB TEQ	9.0×10^{-12}	1.8×10^{-11}	1.4×10^{-11}	2.9×10^{-11}	3.1×10^{-12}	6.3×10^{-13}	7.6×10^{-12}	1.5×10^{-12}
Total PCBs	3.1×10^{-7}	6.3×10^{-7}	5.0×10^{-7}	1.0×10^{-6}	1.1×10^{-7}	2.2×10^{-8}	2.6×10^{-7}	5.3×10^{-8}
Thallium[a]	3.0×10^{-7}	na	4.8×10^{-7}	na	1.0×10^{-7}	na	2.5×10^{-7}	na
Toxaphene	7.9×10^{-7}	1.1×10^{-6}	1.3×10^{-6}	1.8×10^{-6}	2.8×10^{-7}	4.0×10^{-8}	6.6×10^{-7}	9.6×10^{-8}
Vanadium[a]	7.5×10^{-6}	na	1.2×10^{-5}	na	2.6×10^{-6}	na	6.3×10^{-6}	na
Undetected Chemicals								
Benzidine	1.1×10^{-7}	1.5×10^{-7}	1.7×10^{-7}	2.4×10^{-7}	3.7×10^{-8}	5.3×10^{-9}	9.0×10^{-8}	1.3×10^{-8}
N-Nitrosodimethylamine	1.1×10^{-7}	1.6×10^{-7}	1.8×10^{-7}	2.6×10^{-7}	3.9×10^{-8}	5.7×10^{-9}	9.5×10^{-8}	1.4×10^{-8}

[a] No absorption factor is available for this chemical. Dermal exposure for this chemical will be discussed in the uncertainty analysis (Section B.6).

[b] An oral adjustment factor was applied to the RfD to determine the non-cancer risk from dermal absorption of this chemical because dermal absorption data are not available. This adjustment was necessary in order to account for differences between absorption across the skin versus the gastrointestinal tract.

na – not applicable

Table 6. Chronic daily intake rates for clamming sediment exposure scenarios

Chemical	Boat and Shoreline Clamming – 183 days per Year				Boat and Shoreline Clamming – 120 days per year				Shoreline Clamming – 7 days per year			
	Cancer		Non-Cancer		Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Detected Chemicals												
Aluminum[a]	1.2×10^{-2}	na	1.2×10^{-2}	na	7.3×10^{-3}	na	8.0×10^{-3}	na	1.7×10^{-4}	na	4.0×10^{-4}	na
Antimony[a]	5.0×10^{-6}	na	5.0×10^{-6}	na	3.0×10^{-6}	na	3.3×10^{-6}	na	1.5×10^{-8}	na	3.5×10^{-8}	na
Arsenic	1.7×10^{-5}	6.0×10^{-6}	1.7×10^{-5}	6.0×10^{-6}	9.9×10^{-6}	3.3×10^{-6}	1.1×10^{-5}	3.9×10^{-6}	1.1×10^{-7}	3.9×10^{-8}	2.5×10^{-7}	9.2×10^{-8}
Barium[a]	1.9×10^{-4}	na	1.9×10^{-4}	na	1.1×10^{-4}	na	1.2×10^{-4}	na	6.0×10^{-7}	na	1.4×10^{-6}	na
Cadmium[b]	2.9×10^{-6}	na	2.9×10^{-6}	na	1.8×10^{-6}	na	1.9×10^{-6}	na	4.8×10^{-9}	na	1.1×10^{-8}	na
Carcinogenic PAHs	4.7×10^{-7}	7.4×10^{-7}	4.7×10^{-7}	7.4×10^{-7}	2.8×10^{-7}	4.4×10^{-7}	3.1×10^{-7}	4.9×10^{-7}	5.5×10^{-9}	8.6×10^{-9}	1.3×10^{-8}	2.0×10^{-8}
Chromium[a]	5.0×10^{-5}	na	5.0×10^{-5}	na	3.0×10^{-5}	na	3.3×10^{-5}	na	3.2×10^{-7}	na	7.5×10^{-7}	na
Copper[a]	2.8×10^{-4}	na	2.8×10^{-4}	na	1.7×10^{-4}	na	1.8×10^{-4}	na	5.6×10^{-7}	na	1.3×10^{-6}	na
Total DDTs	2.1×10^{-7}	7.8×10^{-8}	2.1×10^{-7}	7.8×10^{-8}	1.3×10^{-7}	4.7×10^{-8}	1.4×10^{-7}	5.1×10^{-8}	2.5×10^{-10}	9.1×10^{-11}	5.9×10^{-10}	2.1×10^{-10}
Dieldrin	8.0×10^{-9}	9.6×10^{-9}	8.0×10^{-9}	9.6×10^{-9}	4.8×10^{-9}	5.8×10^{-9}	5.2×10^{-9}	6.3×10^{-9}	1.9×10^{-10}	2.4×10^{-10}	4.5×10^{-10}	5.5×10^{-10}
Dioxin/furan TEQ	8.7×10^{-10}	3.2×10^{-10}	8.7×10^{-10}	3.2×10^{-10}	5.2×10^{-10}	1.9×10^{-10}	5.7×10^{-10}	2.1×10^{-10}	4.2×10^{-12}	1.5×10^{-12}	9.7×10^{-12}	3.5×10^{-12}
Iron[a]	2.0×10^{-2}	na	2.0×10^{-2}	na	1.2×10^{-2}	na	1.3×10^{-2}	na	2.7×10^{-4}	na	6.4×10^{-4}	na
Manganese[a]	4.0×10^{-4}	na	4.0×10^{-4}	na	2.4×10^{-4}	na	2.6×10^{-4}	na	3.5×10^{-6}	na	8.3×10^{-6}	na
Mercury[a]	1.4×10^{-7}	na	1.4×10^{-7}	na	8.5×10^{-8}	na	9.2×10^{-8}	na	2.3×10^{-9}	na	5.3×10^{-9}	na
Molybdenum[a]	2.3×10^{-6}	na	2.3×10^{-6}	na	1.4×10^{-6}	na	1.5×10^{-6}	na	2.4×10^{-8}	na	5.6×10^{-8}	na
PCB TEQ	1.1×10^{-10}	1.9×10^{-10}	1.1×10^{-10}	1.9×10^{-10}	6.8×10^{-11}	1.1×10^{-10}	$7.4 \times 10\text{-}^{11}$	1.3×10^{-10}	4.8×10^{-13}	8.1×10^{-13}	1.1×10^{-12}	1.9×10^{-12}
Total PCBs	2.5×10^{-6}	4.1×10^{-6}	2.5×10^{-6}	4.1×10^{-6}	1.5×10^{-6}	2.5×10^{-6}	1.6×10^{-6}	2.7×10^{-6}	1.7×10^{-8}	2.9×10^{-8}	4.0×10^{-8}	6.8×10^{-8}
Silver[a]	4.1×10^{-6}	na	4.1×10^{-6}	na	2.5×10^{-6}	na	2.7×10^{-6}	na	6.5×10^{-9}	na	1.5×10^{-8}	na
Thallium[a]	2.2×10^{-6}	na	2.2×10^{-6}	na	1.3×10^{-6}	na	1.4×10^{-6}	na	8.1×10^{-10}	na	1.9×10^{-9}	na
Toxaphene	3.9×10^{-6}	4.7×10^{-6}	3.9×10^{-6}	4.7×10^{-6}	2.3×10^{-6}	2.8×10^{-6}	2.5×10^{-6}	3.1×10^{-6}	9.7×10^{-9}	1.2×10^{-8}	2.3×10^{-8}	2.7×10^{-8}
Vanadium[a]	3.4×10^{-5}	na	3.4×10^{-5}	na	2.1×10^{-5}	na	2.3×10^{-5}	na	6.1×10^{-7}	na	1.4×10^{-6}	na
Zinc[a]	2.9×10^{-4}	na	2.9×10^{-4}	na	1.8×10^{-4}	na	1.9×10^{-4}	na	1.6×10^{-6}	na	3.7×10^{-6}	na

Chemical	Boat and Shoreline Clamming – 183 days per Year				Boat and Shoreline Clamming – 120 days per year				Shoreline Clamming – 7 days per year			
	Cancer		Non-Cancer		Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Undetected chemicals												
4,6-Dinitro-o-cresol	9.2×10^{-7}	1.1×10^{-6}	9.2×10^{-7}	1.1×10^{-6}	5.5×10^{-7}	6.7×10^{-7}	6.0×10^{-7}	7.3×10^{-7}	1.7×10^{-8}	2.1×10^{-8}	4.0×10^{-8}	4.8×10^{-8}
Benzidine	4.3×10^{-7}	5.2×10^{-7}	4.3×10^{-7}	5.2×10^{-7}	2.6×10^{-7}	3.1×10^{-7}	2.8×10^{-7}	3.4×10^{-7}	6.3×10^{-9}	7.6×10^{-9}	1.5×10^{-8}	1.8×10^{-8}
Bis(2-chloroethyl)ether	9.2×10^{-8}	1.1×10^{-7}	9.2×10^{-8}	1.1×10^{-7}	5.5×10^{-8}	6.7×10^{-8}	6.0×10^{-8}	7.3×10^{-8}	1.7×10^{-9}	2.1×10^{-9}	4.0×10^{-9}	4.8×10^{-9}
N-Nitrosodi methylamine	3.1×10^{-7}	3.7×10^{-7}	3.1×10^{-7}	3.7×10^{-7}	1.8×10^{-7}	2.2×10^{-7}	2.0×10^{-7}	2.4×10^{-7}	2.9×10^{-9}	3.5×10^{-9}	6.7×10^{-9}	8.1×10^{-9}
N-Nitroso-di-n-propylamine	4.3×10^{-7}	5.2×10^{-7}	4.3×10^{-7}	5.2×10^{-7}	2.6×10^{-7}	3.1×10^{-7}	2.8×10^{-7}	3.4×10^{-7}	8.0×10^{-9}	9.7×10^{-9}	1.9×10^{-8}	2.3×10^{-8}

[a] No absorption factor is available for this chemical. Dermal exposure for this chemical will be discussed in the uncertainty analysis (Section B.6).

[b] An oral adjustment factor was applied to the RfD to determine the non-cancer risk from dermal absorption of this chemical because dermal absorption data are not available. This adjustment was necessary in order to account for differences between absorption across the skin versus the gastrointestinal tract.

na – not applicable

Table 7a. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 1 and 2

Chemical	Beach Play – Area 1				Beach Play – Area 2			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Detected Chemicals								
Aluminum[a]	nd	nd	nd	nd	nd	nd	nd	nd
Antimony[a]	2.5×10^{-7}	na	2.9×10^{-6}	na	5.2×10^{-7}	na	6.1×10^{-6}	na
Arsenic	3.4×10^{-6}	2.0×10^{-7}	4.0×10^{-5}	2.4×10^{-6}	4.8×10^{-6}	2.9×10^{-7}	5.6×10^{-5}	3.3×10^{-6}
Barium[a]	nd	nd	nd	nd	nd	nd	nd	nd
Cadmium[b]	3.4×10^{-8}	na	4.0×10^{-7}	na	2.3×10^{-7}	na	2.7×10^{-6}	na
Carcinogenic PAHs	2.7×10^{-7}	7.1×10^{-8}	3.2×10^{-6}	8.3×10^{-7}	6.8×10^{-7}	1.8×10^{-7}	8.0×10^{-6}	2.1×10^{-6}
Chromium[a]	4.8×10^{-6}	na	5.6×10^{-5}	na	1.1×10^{-5}	na	1.3×10^{-4}	na
Copper[a]	1.1×10^{-5}	na	1.3×10^{-4}	na	3.9×10^{-5}	na	4.5×10^{-4}	na
Total DDTs	8.4×10^{-10}	5.0×10^{-11}	9.8×10^{-9}	5.9×10^{-10}	3.0×10^{-9}	1.8×10^{-10}	3.5×10^{-8}	2.1×10^{-9}
Dieldrin	3.0×10^{-10}	5.9×10^{-11}	3.5×10^{-9}	6.9×10^{-10}	1.1×10^{-10}	2.3×10^{-11}	1.3×10^{-9}	2.6×10^{-10}
Dioxin/furan TEQ	nd	nd	nd	nd	nd	nd	nd	nd
Iron[a]	nd	nd	nd	nd	nd	nd	nd	nd
Manganese[a]	nd	nd	nd	nd	nd	nd	nd	nd
Mercury[a]	3.9×10^{-8}	na	4.5×10^{-7}	na	1.4×10^{-7}	na	1.7×10^{-6}	na
Molybdenum[a]	4.1×10^{-7}	na	4.8×10^{-6}	na	6.8×10^{-7}	na	8.0×10^{-6}	na
PCB TEQ	2.1×10^{-14}	5.8×10^{-15}	2.4×10^{-13}	6.7×10^{-14}	1.5×10^{-12}	4.3×10^{-13}	1.8×10^{-11}	5.0×10^{-12}
Total PCBs	2.7×10^{-8}	7.6×10^{-9}	3.2×10^{-7}	8.9×10^{-8}	4.1×10^{-8}	1.1×10^{-8}	4.8×10^{-7}	1.3×10^{-7}
Silver[a]	4.6×10^{-8}	na	5.3×10^{-7}	na	1.1×10^{-7}	na	1.3×10^{-6}	na
Thallium[a]	3.4×10^{-8}	na	4.0×10^{-7}	na	4.6×10^{-8}	na	5.3×10^{-7}	na
Toxaphene	1.1×10^{-8}	2.2×10^{-9}	1.3×10^{-7}	2.6×10^{-8}	5.7×10^{-9}	1.1×10^{-9}	6.6×10^{-8}	1.3×10^{-8}
Vanadium[a]	1.1×10^{-5}	na	1.2×10^{-4}	na	1.5×10^{-5}	na	1.8×10^{-4}	na

Chemical	Beach Play – Area 1				Beach Play – Area 2			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Zinc[a]	3.2×10^{-5}	na	3.7×10^{-4}	na	1.0×10^{-4}	na	1.2×10^{-3}	na
Undetected chemicals								
4,6-Dinitro-o-cresol	2.3×10^{-8}	4.5×10^{-9}	2.7×10^{-7}	5.3×10^{-8}	5.7×10^{-8}	1.1×10^{-8}	6.6×10^{-7}	1.3×10^{-7}
Benzidine	nd	nd	nd	nd	nd	nd	nd	nd
Bis(2-chloroethyl)ether	2.3×10^{-9}	4.5×10^{-10}	2.7×10^{-8}	5.3×10^{-9}	5.7×10^{-9}	1.1×10^{-9}	6.6×10^{-8}	1.3×10^{-8}
N-Nitrosodimethylamine	1.1×10^{-8}	2.3×10^{-9}	1.3×10^{-7}	2.6×10^{-8}	1.5×10^{-8}	2.9×10^{-9}	1.7×10^{-7}	3.4×10^{-8}
N-Nitroso-di-n-propylamine	3.9×10^{-9}	7.7×10^{-10}	4.5×10^{-8}	9.0×10^{-9}	1.5×10^{-8}	2.9×10^{-9}	1.7×10^{-7}	3.4×10^{-8}

[a] No absorption factor is available for this chemical. Dermal exposure for this chemical will be discussed in the uncertainty analysis (Section B.6).

[b] An oral adjustment factor was applied to the RfD to determine the non-cancer risk from dermal absorption of this chemical because dermal absorption data are not available. This adjustment was necessary in order to account for differences between absorption across the skin versus the gastrointestinal tract.

na – not applicable

nd – no data were available for this chemical in this beach play area

Table 7b. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 3 and 4

Chemical	Beach Play – Area 3				Beach Play – Area 4			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Detected Chemicals								
Aluminum[a]	2.7×10^{-3}	na	3.2×10^{-2}	na	4.8×10^{-3}	na	5.6×10^{-2}	na
Antimony[a]	1.2×10^{-6}	na	1.4×10^{-5}	na	1.4×10^{-6}	na	1.6×10^{-5}	na
Arsenic	3.0×10^{-6}	1.8×10^{-7}	3.5×10^{-5}	2.1×10^{-6}	2.5×10^{-6}	1.5×10^{-7}	2.9×10^{-5}	1.7×10^{-6}
Barium[a]	1.7×10^{-5}	na	2.0×10^{-4}	na	1.8×10^{-5}	na	2.2×10^{-4}	na
Cadmium[b]	4.6×10^{-7}	na	5.3×10^{-6}	na	2.1×10^{-7}	na	2.5×10^{-6}	na
Carcinogenic PAHs	4.8×10^{-7}	1.2×10^{-7}	5.6×10^{-6}	1.4×10^{-6}	1.7×10^{-7}	4.3×10^{-8}	1.9×10^{-6}	5.0×10^{-7}
Chromium[a]	8.6×10^{-6}	na	1.0×10^{-4}	na	1.9×10^{-5}	na	2.2×10^{-4}	na
Copper[a]	1.8×10^{-5}	na	2.1×10^{-4}	na	1.7×10^{-5}	na	2.0×10^{-4}	na
Total DDTs	4.8×10^{-9}	2.9×10^{-10}	5.6×10^{-8}	3.3×10^{-9}	9.1×10^{-8}	5.4×10^{-9}	1.1×10^{-6}	6.3×10^{-8}
Dieldrin	2.3×10^{-9}	4.5×10^{-10}	2.7×10^{-8}	5.3×10^{-9}	3.9×10^{-9}	7.7×10^{-10}	4.5×10^{-8}	9.0×10^{-9}
Dioxin/furan TEQ	nd	nd	nd	nd	9.4×10^{-11}	5.6×10^{-12}	1.1×10^{-9}	6.5×10^{-11}
Iron[a]	3.9×10^{-3}	na	4.5×10^{-2}	na	7.3×10^{-3}	na	8.5×10^{-2}	na
Manganese[a]	5.5×10^{-5}	na	6.4×10^{-4}	na	6.4×10^{-5}	na	7.4×10^{-4}	na
Mercury[a]	7.1×10^{-8}	na	8.2×10^{-7}	na	3.9×10^{-7}	na	4.5×10^{-6}	na
Molybdenum[a]	1.3×10^{-6}	na	1.5×10^{-5}	na	7.3×10^{-7}	na	8.5×10^{-6}	na
PCB TEQ	nd	nd	nd	nd	4.6×10^{-11}	1.3×10^{-11}	5.3×10^{-10}	1.5×10^{-10}
Total PCBs	5.5×10^{-8}	1.5×10^{-8}	6.4×10^{-7}	1.8×10^{-7}	2.5×10^{-6}	7.0×10^{-7}	2.9×10^{-5}	8.1×10^{-6}
Silver[a]	1.8×10^{-7}	na	2.1×10^{-6}	na	1.8×10^{-7}	na	2.1×10^{-6}	na
Thallium[a]	3.9×10^{-6}	na	4.5×10^{-5}	na	2.1×10^{-8}	na	2.4×10^{-7}	na
Toxaphene	2.0×10^{-8}	4.1×10^{-9}	2.4×10^{-7}	4.8×10^{-8}	1.9×10^{-7}	3.9×10^{-8}	2.3×10^{-6}	4.5×10^{-7}
Vanadium[a]	1.1×10^{-5}	na	1.3×10^{-4}	na	1.3×10^{-5}	na	1.5×10^{-4}	na

Chemical	Beach Play – Area 3				Beach Play – Area 4			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Zinc[a]	8.0×10^{-5}	na	9.3×10^{-4}	na	5.5×10^{-5}	na	6.4×10^{-4}	na
Undetected chemicals								
4,6-Dinitro-o-cresol	2.3×10^{-8}	4.5×10^{-9}	2.7×10^{-7}	5.3×10^{-8}	3.4×10^{-7}	6.8×10^{-8}	4.0×10^{-6}	7.9×10^{-7}
Benzidine	1.3×10^{-7}	2.5×10^{-8}	1.5×10^{-6}	2.9×10^{-7}	nd	nd	nd	nd
Bis(2-chloroethyl)ether	3.2×10^{-9}	6.3×10^{-10}	3.7×10^{-8}	7.4×10^{-9}	3.4×10^{-8}	6.8×10^{-9}	4.0×10^{-7}	7.9×10^{-8}
N-Nitrosodimethylamine	2.0×10^{-8}	4.1×10^{-9}	2.4×10^{-7}	4.8×10^{-8}	3.4×10^{-8}	6.8×10^{-9}	4.0×10^{-7}	7.9×10^{-8}
N-Nitroso-di-n-propylamine	8.2×10^{-9}	1.6×10^{-9}	9.6×10^{-8}	1.9×10^{-8}	1.6×10^{-7}	3.2×10^{-8}	1.9×10^{-6}	3.7×10^{-7}

[a] No absorption factor is available for this chemical. Dermal exposure for this chemical will be discussed in the uncertainty analysis (Section B.6).

[b] An oral adjustment factor was applied to the RfD to determine the non-cancer risk from dermal absorption of this chemical because dermal absorption data are not available. This adjustment was necessary in order to account for differences between absorption across the skin versus the gastrointestinal tract.

na – not applicable

nd – no data were available for this chemical in this beach play area

Table 7c. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 5 and 6

Chemical	Beach Play – Area 5				Beach Play – Area 6			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Detected Chemicals								
Aluminum[a]	3.4×10^{-3}	na	4.0×10^{-2}	na	nd	nd	nd	nd
Antimony[a]	1.1×10^{-6}	na	1.3×10^{-5}	na	3.4×10^{-8}	na	4.0×10^{-7}	na
Arsenic	2.0×10^{-6}	1.2×10^{-7}	2.4×10^{-5}	1.4×10^{-6}	2.2×10^{-6}	1.3×10^{-7}	2.6×10^{-5}	1.6×10^{-6}
Barium[a]	9.6×10^{-6}	na	1.1×10^{-4}	na	nd	nd	nd	nd
Cadmium[b]	5.2×10^{-8}	na	6.1×10^{-7}	na	3.4×10^{-8}	na	4.0×10^{-7}	na
Carcinogenic PAHs	9.3×10^{-8}	2.4×10^{-8}	1.1×10^{-6}	2.8×10^{-7}	1.0×10^{-7}	2.6×10^{-8}	1.2×10^{-6}	3.0×10^{-7}
Chromium[a]	6.4×10^{-6}	na	7.4×10^{-5}	na	5.2×10^{-6}	na	6.1×10^{-5}	na
Copper[a]	1.7×10^{-5}	na	2.0×10^{-4}	na	8.0×10^{-6}	na	9.3×10^{-5}	na
Total DDTs	8.0×10^{-9}	4.8×10^{-10}	9.3×10^{-8}	5.6×10^{-9}	1.1×10^{-10}	6.8×10^{-12}	1.3×10^{-9}	7.9×10^{-11}
Dieldrin	5.9×10^{-10}	1.2×10^{-10}	6.9×10^{-9}	1.4×10^{-9}	2.3×10^{-10}	4.5×10^{-11}	2.7×10^{-9}	5.3×10^{-10}
Dioxin/furan TEQ	5.0×10^{-13}	3.0×10^{-14}	5.8×10^{-12}	3.5×10^{-13}	nd	nd	nd	nd
Iron[a]	5.9×10^{-3}	na	6.9×10^{-2}	na	nd	nd	nd	nd
Manganese[a]	6.4×10^{-5}	na	7.4×10^{-4}	na	nd	nd	nd	nd
Mercury[a]	2.5×10^{-8}	na	2.9×10^{-7}	na	2.0×10^{-8}	na	2.4×10^{-7}	na
Molybdenum[a]	4.6×10^{-7}	na	5.3×10^{-6}	na	3.4×10^{-7}	na	4.0×10^{-6}	na
PCB TEQ	5.7×10^{-13}	1.6×10^{-13}	6.7×10^{-12}	1.9×10^{-12}	1.2×10^{-12}	3.4×10^{-13}	1.4×10^{-11}	4.0×10^{-12}
Total PCBs	4.3×10^{-8}	1.2×10^{-8}	5.0×10^{-7}	1.4×10^{-7}	2.2×10^{-7}	6.2×10^{-8}	2.6×10^{-6}	7.2×10^{-7}
Silver[a]	3.4×10^{-8}	na	4.0×10^{-7}	na	4.6×10^{-8}	na	5.3×10^{-7}	na
Thallium[a]	1.4×10^{-8}	na	1.6×10^{-7}	na	3.4×10^{-8}	na	4.0×10^{-7}	na
Toxaphene	7.7×10^{-8}	1.5×10^{-8}	9.0×10^{-7}	1.8×10^{-7}	1.1×10^{-8}	2.2×10^{-9}	1.3×10^{-7}	2.6×10^{-8}
Vanadium[a]	1.3×10^{-5}	na	1.5×10^{-4}	na	1.1×10^{-5}	na	1.3×10^{-4}	na

Chemical	Beach Play – Area 5				Beach Play – Area 6			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Zinc[a]	2.5×10^{-5}	na	2.9×10^{-4}	na	2.1×10^{-5}	na	2.4×10^{-4}	na
Undetected chemicals								
4,6-Dinitro-o-cresol	1.1×10^{-7}	2.3×10^{-8}	1.3×10^{-6}	2.6×10^{-7}	6.6×10^{-8}	1.3×10^{-8}	7.7×10^{-7}	1.5×10^{-7}
Benzidine	nd	nd	nd	nd	nd	nd	nd	nd
Bis(2-chloroethyl)ether	1.1×10^{-8}	2.3×10^{-9}	1.3×10^{-7}	2.6×10^{-8}	6.6×10^{-9}	1.3×10^{-9}	7.7×10^{-8}	1.5×10^{-8}
N-Nitrosodimethylamine	1.1×10^{-8}	2.3×10^{-9}	1.3×10^{-7}	2.6×10^{-8}	3.6×10^{-9}	7.3×10^{-10}	4.2×10^{-8}	8.5×10^{-9}
N-Nitroso-di-n-propylamine	4.6×10^{-9}	9.1×10^{-10}	5.3×10^{-8}	1.1×10^{-8}	3.6×10^{-9}	7.3×10^{-10}	4.2×10^{-8}	8.5×10^{-9}

[a] No absorption factor is available for this chemical. Dermal exposure for this chemical will be discussed in the uncertainty analysis (Section B.6).

[b] An oral adjustment factor was applied to the RfD to determine the non-cancer risk from dermal absorption of this chemical because dermal absorption data are not available. This adjustment was necessary in order to account for differences between absorption across the skin versus the gastrointestinal tract.

na – not applicable

nd – no data were available for this chemical in this beach play area

Table 7d. Chronic daily intake rates for beachplay sediment exposure scenarios – areas 7 and 8

Chemical	Beach Play – Area 7				Beach Play – Area 8			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Detected Chemicals								
Aluminum[a]	5.2×10^{-3}	na	6.1×10^{-2}	na	3.9×10^{-3}	na	4.5×10^{-2}	na
Antimony[a]	1.1×10^{-6}	na	1.3×10^{-5}	na	1.6×10^{-6}	na	1.9×10^{-5}	na
Arsenic	2.5×10^{-6}	1.5×10^{-7}	2.9×10^{-5}	1.7×10^{-6}	2.3×10^{-6}	1.4×10^{-7}	2.7×10^{-5}	1.6×10^{-6}
Barium[a]	1.6×10^{-5}	na	1.9×10^{-4}	na	1.3×10^{-5}	na	1.5×10^{-4}	na
Cadmium[b]	1.8×10^{-7}	na	2.1×10^{-6}	na	4.6×10^{-8}	na	5.3×10^{-7}	na
Carcinogenic PAHs	2.5×10^{-8}	6.5×10^{-9}	2.9×10^{-7}	7.6×10^{-8}	7.3×10^{-8}	1.9×10^{-8}	8.5×10^{-7}	2.2×10^{-7}
Chromium[a]	5.7×10^{-6}	na	6.6×10^{-5}	na	5.2×10^{-6}	na	6.1×10^{-5}	na
Copper[a]	8.6×10^{-6}	na	1.0×10^{-4}	na	7.7×10^{-6}	na	9.0×10^{-5}	na
Total DDTs	6.1×10^{-10}	3.7×10^{-11}	7.2×10^{-9}	4.3×10^{-10}	1.9×10^{-9}	1.1×10^{-10}	2.2×10^{-8}	1.3×10^{-9}
Dieldrin	2.3×10^{-10}	4.5×10^{-11}	2.7×10^{-9}	5.3×10^{-10}	5.2×10^{-10}	1.0×10^{-10}	6.1×10^{-9}	1.2×10^{-9}
Dioxin/furan TEQ	3.9×10^{-13}	2.3×10^{-14}	4.5×10^{-12}	2.7×10^{-13}	nd	nd	nd	nd
Iron[a]	7.1×10^{-3}	na	8.2×10^{-2}	na	5.9×10^{-3}	na	6.9×10^{-2}	na
Manganese[a]	9.8×10^{-5}	na	1.1×10^{-3}	na	1.8×10^{-4}	na	2.1×10^{-3}	na
Mercury[a]	2.5×10^{-8}	na	2.9×10^{-7}	na	2.7×10^{-8}	na	3.2×10^{-7}	na
Molybdenum[a]	3.0×10^{-7}	na	3.5×10^{-6}	na	4.3×10^{-7}	na	5.0×10^{-6}	na
PCB TEQ	1.3×10^{-13}	3.6×10^{-14}	1.5×10^{-12}	4.2×10^{-13}	4.3×10^{-13}	1.2×10^{-13}	5.0×10^{-12}	1.4×10^{-12}
Total PCBs	5.2×10^{-8}	1.5×10^{-8}	6.1×10^{-7}	1.7×10^{-7}	5.2×10^{-8}	1.5×10^{-8}	6.1×10^{-7}	1.7×10^{-7}
Silver[a]	8.0×10^{-8}	na	9.3×10^{-7}	na	2.7×10^{-8}	na	3.2×10^{-7}	na
Thallium[a]	5.7×10^{-8}	na	6.6×10^{-7}	na	1.4×10^{-8}	na	1.6×10^{-7}	na

Chemical	Beach Play – Area 7				Beach Play – Area 8			
	Cancer		Non-Cancer		Cancer		Non-Cancer	
	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption	Incidental Ingestion	Dermal Absorption
Toxaphene	1.1×10^{-8}	2.2×10^{-9}	1.3×10^{-7}	2.6×10^{-8}	1.1×10^{-8}	2.2×10^{-9}	1.3×10^{-7}	2.6×10^{-8}
Vanadium[a]	1.5×10^{-5}	na	1.7×10^{-4}	na	1.3×10^{-5}	na	1.5×10^{-4}	na
Zinc[a]	1.9×10^{-5}	na	2.2×10^{-4}	na	2.5×10^{-5}	na	2.9×10^{-4}	na
Undetected chemicals								
4,6-Dinitro-o-cresol	6.8×10^{-8}	1.4×10^{-8}	8.0×10^{-7}	1.6×10^{-7}	6.8×10^{-8}	1.4×10^{-8}	8.0×10^{-7}	1.6×10^{-7}
Benzidine	nd	nd	nd	nd	nd	nd	nd	nd
Bis(2-chloroethyl)ether	6.8×10^{-9}	1.4×10^{-9}	8.0×10^{-8}	1.6×10^{-8}	6.8×10^{-9}	1.4×10^{-9}	8.0×10^{-8}	1.6×10^{-8}
N-Nitrosodimethylamine	3.9×10^{-9}	7.7×10^{-10}	4.5×10^{-8}	9.0×10^{-9}	3.9×10^{-9}	7.7×10^{-10}	4.5×10^{-8}	9.0×10^{-9}
N-Nitroso-di-n-propylamine	4.6×10^{-9}	9.1×10^{-10}	5.3×10^{-8}	1.1×10^{-8}	4.6×10^{-9}	9.1×10^{-10}	5.3×10^{-8}	1.1×10^{-8}

[a] No absorption factor is available for this chemical. Dermal exposure for this chemical will be discussed in the uncertainty analysis (Section B.6).

[b] An oral adjustment factor was applied to the RfD to determine the non-cancer risk from dermal absorption of this chemical because dermal absorption data are not available. This adjustment was necessary in order to account for differences between absorption across the skin versus the gastrointestinal tract.

na – not applicable

nd – no data were available for this chemical in this beach play area

ATTACHMENT 4. TOXICOLOGICAL PROFILES FOR CHEMICALS OF POTENTIAL CONCERN

Glossary **2**

Toxicological Profiles for Principal Risk Drivers **5**

1. 2,3,7,8-TCDD (DIOXIN) 5
2. ARSENIC 7
3. CARCINOGENIC POLYCYCLIC AROMATIC HYDROCARBONS (CPAHS) 8
4. POLYCHLORINATED BIPHENYLS (PCBS) 9

Toxicological Profiles for All Other Chemicals of Potential Concern **13**

1. 1,2-DIPHENYLHYDRAZINE 13
2. 1,3-DICHLOROBENZENE AND 1,4-DICHLOROBENZENE 14
3. 2,4,6-TRICHLOROPHENOL 15
4. 2,4-DICHLOROPHENOL 16
5. 2,4-DINITROPHENOL 17
6. 2,4-DINITROTOLUENE AND 2,6-DINITROTOLUENE 18
7. 2-CHLOROPHENOL 19
8. 3,3′-DICHLOROBENZIDINE 20
9. 3-NITROANILINE AND 4-NITROANILINE 21
10. 4,6-DINITRO-O-CRESOL 22
11. 4-CHLOROANILINE 23
12. 4-METHYLPHENOL OR P-CRESOL 24
13. ALDRIN 25
14. ALUMINUM 26
15. ANILINE 27
16. ANTIMONY 28
17. BARIUM 29
18. BENZIDINE 30
19. BIS(2-CHLOROETHYL)ETHER 31
20. BIS(2-CHLOROISOPROPYL)ETHER 32
21. BIS(2-ETHYLHEXYL)PHTHALATE 33
22. BUTYL BENZYL PHTHALATE 33
23. CADMIUM 34
24. CARBAZOLE 35

25. CHLORDANE 36
26. CHROMIUM 37
27. COPPER 38
28. DDT AND METABOLITES 39
29. DIELDRIN 40
30. ENDRIN AND ENDRIN ALDEHYDE 41
31. GAMMA-BHC (LINDANE) AND METABOLITES (ALPHA-BHC AND BETA-BHC) 42
32. HEPTACHLOR 44
33. HEPTACHLOR EPOXIDE 44
34. HEXACHLOROBENZENE 46
35. HEXACHLOROBUTADIENE 47
36. HEXACHLOROCYCLOPENTADIENE (HCCPD) 47
37. HEXACHLOROETHANE 49
38. IRON 50
39. LEAD 51
40. MANGANESE 52
41. MERCURY 53
42. MOLYBDENUM 54
43. NICKEL 55
44. NITROBENZENE 56
45. N-NITROSODIMETHYLAMINE 57
46. N-NITROSO-DI-N-PROPYLAMINE 58
47. N-NITROSODIPHENYLAMINE 58
48. PENTACHLOROPHENOL 59
49. SILVER 60
50. THALLIUM 61
51. TOXAPHENE 62
52. TRIBUTYLTIN 63
53. VANADIUM 64
54. ZINC 65

References 66

Attachment 4. Toxicological Profiles for Chemicals of Potential Concern

The following sections provide toxicological information for each of the chemicals that were identified as COPCs in this human health risk assessment (HHRA). The toxicity values used in this risk assessment (i.e., RfD or SF) are in bold type. Toxicity information was obtained primarily from:

- US Environmental Protection Agency (EPA's) Integrated Risk Information System (IRIS) (EPA 2006b)
- EPA's 1997 Health Effects Summary Tables (HEAST)(EPA 1997b)
- Toxicological profiles presented in *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories* (EPA 2000)
- EPA's Office of Ground Water and Drinking Water (OGWDW) (EPA 2006a)
- Agency for Toxic Substance and Disease Registry (ATSDR; 2006b)
- ToxFAQs (ATSDR 2006a)
- Hazardous Substance Data Bank (HSDB) (TOXNET 2006)

Since quantitative estimates of toxicity potential have been developed by EPA and other agencies, it is necessary to establish a hierarchy to determine what toxicity values should be used. EPA (2003b) has developed a hierarchical order of toxicity values for use in human health risk assessments:

- Tier 1 – EPA's IRIS
- Tier 2 – EPA's Provisional Peer Reviewed Toxicity Values (PPRTVs), Office of Research and Development/National Center for Environmental Assessment
- Tier 3 – Other toxicity values. Tier 3 includes additional EPA and non-EPA sources of toxicity information. Priority is given to those sources of information that are the most current, the basis for which is transparent and publicly available, and which have been peer reviewed. Sources include EPA Regional offices, EPA HEAST values, Cal/EPA, and ATSDR minimal risk levels (MRLs).

Online versions of IRIS, HSDB, and ToxFAQs are cited by acronym only in the sections below. These databases were accessed between February and April 2006. Other citations are presented in standard form. The carcinogenic evaluation of chemicals by the International Agency for Research on Cancer (IARC) and toxicity evaluations by the National Center for Environmental Assessment (NCEA, a division of EPA), as cited in other documents, are also referred to by their acronyms.

Glossary

Anorexia	An abnormal loss of appetite and desire to eat that results in severe weight loss.
Aplastic anemia	A condition whereby the capacity of bone marrow to generate red blood cells is defective.
Ataxia	The sudden onset of uncoordinated muscle movement.
Bronchiolitis	Inflammation of the small airways of the lung.
Chloracne	A rare acne-like skin condition caused by certain toxic chemicals.
Cirrhosis	A chronic disease of the liver that results in scarring of the liver and liver dysfunction.
Coma	A state of unconsciousness in which a person is unable to respond to stimuli.
Contact dermatitis	An inflammation of the skin caused by direct contact with an irritating or allergy-causing substance.
Conjunctivitis	An inflammation of the clear membrane that covers the white part of the eye and lines the inner surface of the eyelids, commonly known as pinkeye.
Cyanosis	A bluish discoloration of the skin or mucous membranes caused by lack of oxygen in the blood.
Defoliant	Any substance designed to destroy or remove foliage.
Dyspnea	Difficulty breathing or shortness of breath.
Edema	Swelling or enlargement of organs, skin, or other parts of the body caused by the excessive buildup of fluid in tissue.
Erythrocyte	Red blood cell.
Fungicide	Any substance used to kill fungus.
Gout	A disorder in which the body overproduces or cannot eliminate uric acid, which results in joint pain, especially in the feet and legs.

Hemoglobin	The red substance in blood that carries oxygen to cells throughout the body.
Hemolysis	The premature breakdown and destruction of red blood cells, which results in an inadequate number of red blood cells for the transport of oxygen.
Hemolytic anemia	A condition in which an inadequate number of circulating red blood cells (anemia) is caused by the premature destruction of red blood cells.
Herbicide	Any substance used to kill plants.
Jaundice	A condition, characterized by a yellow color in the skin, the mucous membranes, or the eyes, in which bilirubin, a byproduct of old red blood cells, is not adequately eliminated from the body.
Keratitis	Also known as dry-eye syndrome, a condition in which tear glands produce fewer tears.
Lacrimation	The production of tears; crying.
Lethargy	A feeling of fatigue, tiredness, or general lack of energy.
Leukocyte	White blood cell.
Leukemia	A type of cancer that targets bone marrow and causes an uncontrolled increase in the production of white blood cells.
LOAEL	Lowest-observed-adverse-effect level. The lowest dose at which an adverse reaction to a chemical or substance was observed.
LD50	Lethal dose at 50%. The amount of a chemical or other toxic substance that is sufficient to kill 50% of a population of test animals.
Lymphocyte	The nearly colorless cells formed in lymphatic tissue (lymph nodes, spleen, thymus, and tonsils) that constitute nearly a third of all white blood cells in the blood.
Lymphoma	Malignancy (cancer) of lymph tissue found in the lymph nodes, spleen, liver, and bone marrow.

Malaise	A general, non-specific feeling of discomfort, illness, or lack of well-being, often accompanied by exhaustion or low energy.
Methemoglobin anemia	See Methemoglobinemia.
Methemoglobinemia	A condition in which the iron in red blood cells is defective, which prevents it from transporting oxygen effectively.
Methemoglobinuria	The presence of methemoglobin in urine.
Necrosis	Death of cells or tissue caused by injury or disease, especially in a localized area of the body.
Necrotizing bronchitis	Bronchitis characterized by cell death in the airways or deeper tissues of the lungs.
NOAEL	No-observed-adverse-effect level. The highest dose at which no effect was observed from exposure to a certain chemical or toxic agent.
Pesticide	Any substance used to repel or kill pests.
Pulmonary edema	Fluid accumulation and swelling in the lungs.
Pulmonary fibrosis	The scarring or thickening of tissues deep in the lung without a known cause.
Proteinuria	The presence of protein in urine.
Rhinitis	A condition characterized by a constant runny nose, sneezing, and nasal congestion.
Siderosis	Chronic inflammation of the lungs caused by excessive inhalation of dust that contains iron. May also refer to the discoloration of organs or tissue as the result of excess iron in the blood.
Tachycardia	Irregular heartbeat or palpitations, often accompanied by sensations of heart pounding or racing.
Vertigo	A sudden sensation of spinning or dizziness, typically provoked by head movement.

Toxicological Profiles for Principal Risk Drivers

1. 2,3,7,8-TCDD (DIOXIN)

Tetrachlorodibenzo-*p*-dioxin (TCDD) is produced as an unwanted contaminant during the manufacture of chlorobenzenes, chlorophenols and their derivatives. TCDD is the most toxic of the 210 polychlorinated dioxin and furan congeners, although it should be noted that other congeners are also quite toxic. TCDD is released to the environment primarily through emissions from the incineration of municipal and chemical wastes, in exhaust from automobiles using leaded gasoline, and from the improper disposal of certain chlorinated chemical wastes. The major route of exposure to dioxins for the general population is through the ingestion of food, a result of the highly bioaccumulative nature of dioxins (Schecter and Gasiewicz 2001). Exposure can also occur through the inhalation of output from various incineration processes and exhausts from leaded gasoline engines. However, the main issue with these emissions is that they accumulate in virtually all food products, where they are ingested by humans (HSDB).

Pharmacokinetics

Dioxins are absorbed through the gastrointestinal tract, respiratory tract, and skin and distributed throughout the body. Absorption is congener-specific, with decreased absorption of hepta- and octa-congeners compared with dioxins with fewer chlorines. Because of their lipophilic nature, dioxins tend to accumulate in fat and the liver. Dioxins are slowly metabolized by oxidation or reductive dechlorination and conjugation, and the major routes of excretion are the bile and feces. Reported half lives in the body range from 5 to 15 years. Small amounts may be eliminated in the urine (EPA 2000).

Acute toxicity

The most commonly reported symptom related to TCDD exposure in humans is chloracne. The lesions of the skin may develop a few weeks after the exposure and may persist for over a year following the cessation of exposure. Other skin problems which have been reported include hyperpigmentation, hirsutism, increased skin fragility, and vesicular eruptions on exposed areas of the skin. Other less consistently reported non-carcinogenic effects from dioxin exposure in humans include asthenia, headaches, and pain in the extremities, peripheral neuropathy, ulcers, altered liver function, enzyme induction, altered lipid metabolism, and abnormal urinary porphyrin patterns. Immune system dysfunction and altered T-cell subsets have been reported by some investigators but have not been found by others (HSDB).

Chronic toxicity

In animal studies, numerous effects have been documented, including hepatic, gastrointestinal, hematological, dermal, body weight changes, endocrine, immunological, neurological, reproductive, and developmental effects. Most of the studies have involved oral exposure.

In humans, even low levels of dioxin exposure have been shown to have adverse health effects (EPA 2003a). The effect most commonly associated with exposure to dioxin-like agents is the skin disease chloracne, a particularly severe and prolonged acne-like skin disorder. Human studies have also shown that numerous developmental effects occurred after dioxin exposure (Mocarelli et al. 1991). Adverse human health effects were also noted following consumption of heated rice oil contaminated with PCBs and chlorinated dibenzofurans (CDFs). Some epidemiological studies have suggested that dioxins may cause immunosuppression, respiratory effects, cardiovascular effects, and liver effects in humans (EPA 2000). EPA has not developed an RfD for dioxins because the United States population is already exposed to unsafe levels of dioxins as a result of background concentrations of this chemical in most food items (EPA 2003a).

Carcinogenicity

There is limited evidence in humans for the carcinogenicity of TCDD, although there is sufficient evidence in experimental animals for the carcinogenicity of TCDD. The World Health Organization (IARC 1995) has concluded that TCDD is carcinogenic to humans (Group 1). In making the overall evaluation, the Working Group took into consideration the following supporting evidence: 1) TCDD is a multi-site carcinogen in experimental animals that has been shown by several lines of evidence to act through a mechanism involving the Ah receptor, 2) this receptor is highly conserved in an evolutionary sense and functions the same way in humans as in experimental animals, and 3) tissue concentrations are similar in both heavily exposed human populations in which an increased overall cancer risk was observed and in rats exposed to carcinogenic dosage regimens in bioassays.

To assess the risk associated with dioxins, it is important to understand that although 2,3,7,8-TCDD is the most toxic congener, many other congeners are also quite toxic and are important because of the additive nature of dioxin toxicity. For this reason, a toxic equivalency (TEQ) approach is used for dioxin and dibenzofuran congeners, where the toxic equivalency factor (TEF) for each congener is applied to determine the overall dioxin toxicity. The TEQ approach is discussed in greater detail in Sections B.2.2 and B.4 of the risk assessment. EPA has established 2,3,7,8-TCDD as a probable human carcinogen (category B2) and has established an **oral cancer slope factor of 150,000 per mg/kg-day** (IRIS). Since 2,3,7,8-TCDD is the most toxic congener, the cancer slope factor for this chemical is used to ensure a conservative estimate of dioxin toxicity.

2. ARSENIC

Arsenic is a naturally occurring element in the earth's crust that is usually found combined with other elements. Arsenic combined with elements such as oxygen, chlorine, and sulfur is referred to as inorganic arsenic; arsenic combined with carbon and hydrogen is referred to as organic arsenic. Arsenic in seafood is more commonly in the organic form (EPA 1997a). Most of the common organic forms, such as arsenobetaine and arsenocholine, are non-toxic, but other forms that may also occur to some extent, such as dimethylated and monomethylated arsenic acids, are more toxic (EPA 1997a). Some seafood may also contain arseno-sugars, which may be metabolized to dimethyl arsenic (Chew 1996).

Pharmacokinetics

Pharmacokinetic studies show that water-soluble arsenic compounds are well absorbed across the gastrointestinal tract. They appear to be transported throughout the body; analysis of tissues taken at autopsy from people who were exposed to arsenic found arsenic present in all tissues of the body. The arsenic levels in hair and nails were the highest, with somewhat lower levels in internal organs (ATSDR 2005a).

The metabolism of arsenic consists mainly of a reduction reaction, which converts pentavalent arsenic to trivalent arsenic, and methylation reactions, which convert arsenite to monomethylarsonic acid and dimethylarsenic acid (EPA 2000). Recent research suggests that trivalent forms of methylated arsenic generated during methylation may be more toxic than inorganic arsenic (Petrick et al. 2000; Petrick et al. 2001; Thomas et al. 2001). The primary excretion route for arsenic and metabolites is in the urine, with human studies showing that 45 to 85 percent is excreted in the urine within 1 to 3 days. Very little is excreted in the feces (ATSDR 2005a).

Acute toxicity

Arsenicals have been recognized as a human poison since ancient times, and large doses, approximately 600 µg/kg-day or higher, taken orally have resulted in death (EPA 2000). Oral exposure to lower levels of arsenic has resulted in effects on the gastrointestinal system (nausea, vomiting); central nervous system (headaches, weakness, delirium); cardiovascular system (hypotension, shock); and the liver, kidney, and blood (anemia, leucopoenia). Because significant information is available on the acute effects of arsenic poisoning in humans, few animal studies have been carried out. The limited available data have shown arsenic to have low to moderate acute toxicity to animals, based on LD50s between 50 and 5,000 mg/kg (ATSDR 2005a).

Chronic toxicity

The primary effects noted in humans from chronic exposure to arsenic are effects on the skin. Oral exposure has resulted in a pattern of skin changes that include the formations of warts or corns on the palms and soles, along with areas of darkened skin

on the face, neck, and back (EPA 2000). Blackfoot disease, a disease characterized by a progressive loss of circulation in the hands and feet, leading ultimately to necrosis and gangrene, is associated with arsenic (ATSDR 2005a). Other effects noted from chronic oral exposure include peripheral neuropathy, cardiovascular disorders, and liver and kidney disorders.

EPA's IRIS database provides an **RfD for inorganic arsenic of 0.0003 mg/kg-day**, based on a no observed adverse effects level (NOAEL) (adjusted to include arsenic exposure from food) of 0.0008 mg/kg-day. The RfD was based on two studies that showed that the prevalence of blackfoot disease increased with both age and dose for individuals exposed to high levels of arsenic in drinking water.

Carcinogenicity

There is clear evidence that chronic exposure of humans to inorganic arsenic increases the risk of cancer. Ingestion of arsenic has been associated with an increased risk of non-melanoma skin cancer, and bladder, liver, and lung cancer. In addition, studies have reported that inhalation of arsenic results in an increased risk of lung cancer (EPA 2000). Dimethyl arsenic may be a promoter of various forms of cancer in rats and mice (Kenyon and Hughes 2001). EPA has classified inorganic arsenic in Group A—Known Human Carcinogen, based on the increased incidence in humans of lung cancer through inhalation exposure and the increased risk of skin, bladder, liver, and lung cancer through drinking water exposure.

The **oral cancer slope factor for arsenic is 1.5 per mg/kg-day** (IRIS). EPA used data from Taiwan concerning skin cancer incidence, age, and level of exposure via drinking water. In 37 villages that had obtained drinking water for 45 years from artesian wells with various elevated levels of arsenic, 40,421 individuals were examined for hyperpigmentation, keratosis, skin cancer, and blackfoot disease. The local well waters were analyzed for arsenic, and the age-specific cancer prevalence rates were correlated with both local arsenic concentrations and duration of exposure.

3. Carcinogenic Polycyclic Aromatic Hydrocarbons (cPAHs)

PAHs are a group of organic chemicals that have a fused ring structure of two or more benzene rings, and are formed during the incomplete combustion of organic materials. Industrial activities which produce PAHs include: coal coking, production of carbon blacks, creosote, coal tar, petroleum refining, synfuel production from coal, and the use of Soderberg electrodes in aluminum smelters and ferrosilicum and iron works (EPA 2000). Domestic activities which produce PAHs include: cigarette smoke, burning of wood and fossil fuels, waste incineration, broiling and smoking foods, and the use of combustion engines. Benzo(a)pyrene is the PAH with the most available health effects data.

Pharmacokinetics

PAHs can be absorbed through the lungs, the stomach, or the skin. Oral absorption increases with more lipophilic PAHs or in the presence of oil in the gastrointestinal tract. Upon inhalation, oral or dermal exposure of animals, the highest levels of PAHs were found in highly perfused tissues, such as the lung, liver, gastrointestinal tract and kidneys. It has been demonstrated that PAHs metabolize to reactive intermediates by enzyme systems, which then covalently bind to cellular macromolecules leading to mutation and tumor development (EPA 2000).

Acute toxicity

There are little data describing acute toxicity of PAHs after inhalation, oral, or dermal exposure in humans or animals. However, benzo(a)pyrene is fatal to mice following ingestion, and the liver and the skin have been identified as target organs in animals after oral or dermal exposure, respectively (ATSDR 1995). The intraperitoneal LD50 values (injected dose which kills ½ of the animals being tested) in mice for pyrene, anthracene, and benzo(a)pyrene are 514, >430, and 232 mg/kg, respectively.

Chronic toxicity

PAHs have a high chronic exposure toxicity characterized by chronic dermatitis and hyperkeratosis (ATSDR 1995). Chronic studies in animals exposed to PAHs via ingestion, intratracheal installation, or skin-painting have not as yet identified adverse health effects other than cancer. RfDs have not been developed for any of the carcinogenic PAHs being evaluated in this Phase 1 HHRA.

Carcinogenicity

Occupational studies of workers exposed to mixtures containing PAHs have shown that mixtures of PAHs are carcinogenic to humans. Cancer associated with exposure to PAH containing mixtures in humans occurs mainly in the lung and skin following inhalation and dermal exposure.

The EPA and California EPA describe the cancer causing ability of individual cPAHs relative to the cancer causing ability of a reference compound, benzo(a)pyrene (EPA 1993; California EPA 1994). This approach is described in greater detail in Sections B.2.2 and B.4 of the risk assessment. The oral cancer slope factor developed by EPA for carcinogenicity of benzo(a)pyrene is **7.3 per mg/kg-day** (IRIS). EPA has classified benzo(a)pyrene as a probable human carcinogen (B2) based on observations of significant dose-related increases in multiple studies of rats and mice of both sexes (IRIS). The oral cancer potency factor was applied to the sum of cPAHs, using the TEFs described in Section B.2.2.

4. POLYCHLORINATED BIPHENYLS (PCBS)

Although the production and use of PCBs were banned in this country in 1979, this chemical group is extremely persistent in the environment and bioaccumulates

through the food chain (EPA 2000). There is evidence that some dioxin-like PCB congeners, which are assumed to be the most toxic, preferentially accumulate in organisms higher on the food chain, including humans. As a result, the composition of PCB mixtures in fish tissue may differ significantly from the environmental PCB source. Often the mixtures of interest are not those that have been used in studies of laboratory animals to determine toxicity (EPA 2000).

Pharmacokinetics

PCBs are absorbed through the gastrointestinal tract and distributed throughout the body, although the highest accumulation is typically in lipid-rich tissues. Human milk may contain relatively elevated PCB concentrations due to its high fat content (ATSDR 2000b).

The retention of PCBs in fatty tissues is linked to the degree of chlorination and also to the position of the chlorine atoms in the biphenyl ring. In general, more chlorinated congeners persist for longer periods of time. In occupationally exposed individuals, less chlorinated congeners had half-lives between 1 and 6 years, while more chlorinated congeners had half-lives ranging from 8 to 24 years (ATSDR 2000b). In subjects who consumed PCB-contaminated rice in Taiwan, the half-lives of several PCBs ranged from 3 to 24 months (EPA 2000).

Acute toxicity

Studies in animals have shown that exposure to very high doses of PCBs can cause death. However, doses of such magnitude are unlikely in environmental exposures and current industrial settings. There have been no reports of deaths in humans after exposure to PCBs even where exposures were much higher than those typically identified with environmental exposures (ATSDR 2000b).

Chronic toxicity

Numerous effects have been documented in animal studies including hepatic, gastrointestinal, hematological, dermal, body weight, endocrine, immunological, neurological, reproductive, developmental, and liver cancer (ATSDR 2000b). One of the most distinct effects associated with PCB exposure is the skin condition chloracne, which is generally associated with high levels of exposure (ATSDR 2000b). Evidence of other chronic effects in humans is not nearly as definitive. Several studies in humans have suggested that PCB exposure, particularly via in utero exposure through maternal fish consumption, may cause adverse effects in children and in developing fetuses (ATSDR 2000b). Neurobehavioral effects in such children with a range of PCB exposure levels have been documented by Fein et al. (1984), Jacobson and Jacobson (1996; 1997), and Schantz (1996). A review of exposure evaluation in 10 more recent studies associating neurodevelopmental effects with PCBs is also available (Longnecker et al. 2003). This will facilitate future comparisons across studies and future updates to neurodevelopmental toxicity metrics. PCBs have also been

associated with immunological effects in several epidemiological studies (Dallaire et al. 2006).

Over intermediate durations (i.e., less than 10% of an organism's lifetime), learning problems have been noted in monkeys fed PCB mixtures similar in composition to human breast milk (ATSDR 2000b). Some studies also indicate a possible connection between PCB exposure and cardiovascular effects; although this has been better demonstrated in assessments of dioxins, which share a similar chemical structure to PCBs (see structure activity relationships at the end of the PCB section).

EPA has derived an **RfD of 0.00002 mg/kg-day** for Aroclor 1254 (IRIS). The RfD was based on a LOAEL of 0.005 mg/kg-day for ocular and immunological effects in monkeys. This RfD is considered to be protective of developmental effects as well, and is used for total PCBs in this HHRA.

Carcinogenicity

PCBs are classified by EPA as Class B2, probable human carcinogens. This designation is based on studies that have found liver tumors in rats exposed to Aroclors 1260, 1254, 1242, and 1016. Occupational mortality data indicate that exposures to PCBs during capacitor manufacturing and repairing were associated with cancer of the liver, biliary tract and/or gall bladder, intestinal cancer, and skin melanoma (Brown and Jones 1981; Brown 1987); however, previous reviews of human epidemiological studies of PCBs have not yielded conclusive results (Silberhorn et al. 1990). Some more recent studies have indicated an increase in melanoma, brain, prostate, or liver cancer mortality in populations occupationally exposed to PCBs (Prince et al. 2006a; Prince et al. 2006b; Ruder et al. 2006). Elevated risk of non-Hodgkin lymphoma has been associated with detection of PCBs in carpet dust (Colt et al. 2005) and in elevated PCB concentrations in blood (De Roos et al. 2005).

EPA has developed a range of slope factors for PCBs (EPA 1996). Using information on environmental processes, they have provided guidance for choosing an appropriate slope factor based on the class of the mixture and the exposure pathway. Because bioaccumulated PCBs appear to be more toxic and more persistent in the body than commercial PCBs, **the upper bound slope factor associated with high risk and persistence (2.0 per mg/kg-day) was used in this HHRA** (IRIS).

When assessing PCB mixtures, it is important to recognize that both dioxin-like and non-dioxin-like modes of action contribute to overall PCB toxicity. It is possible that concentrations of dioxin-like congeners are increased in an environmental mixture. When congener concentrations are available, the mixture-based approach based on Aroclor analyses can be supplemented by analysis of dioxin TEQs to evaluate the PCB congeners with dioxin-like toxicity. In the TEQ approach, all PCB congeners with dioxin-like properties are analyzed in order to assess their impact on the overall risk from PCBs. For the analysis of dioxin-like PCB congeners, the **dioxin slope factor of 150,000 per mg/kg-day** (EPA 2005) is used with the estimated dioxin toxic equivalency

(Van den Berg et al. 2006). Details of the structure activity relationship are presented below. The TEFs for PCBs are presented in Section B.2.2.

Structure Activity Relationships

Some non- and mono-ortho substituted PCBs may adapt a planar conformation and activate the aryl hydrocarbon (Ah) receptor. These PCBs are thought to share a common mode of toxic action with dioxin (2,3,7,8-TCDD) and are sometimes referred to as dioxin-like PCBs (Van den Berg et al. 2006). Some polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are also capable of activating the Ah receptor and also have TEFs (see Section B.2.2). The mono-ortho substituted PCBs have higher TEFs because they are structurally better able to take on the planar conformation needed to activate the Ah receptor.

A recent study assessed cancer in rodents exposed to an equal TEQ quantity from either one PCB, one PCDF, a mixture (equal parts of one PCB, one PCDF, and TCDD), or TCDD (Walker et al. 2005). The results of this study supported the use of the TEF approach for the estimation of PCB cancer risk.

PCBs used in most laboratory studies and found in the environment are complex mixtures, and it is not known exactly what portion of observed effects is attributable to dioxin-like or non-dioxin-like PCBs. The EPA Science Advisory Board cited the van der Plas et al. (2000) study of rats exposed to Aroclor 1260, which suggested that most of the tumor promotion potential of PCB mixtures is attributable to the non-dioxin-like fraction (EPA 2001). Because this fraction is not included in the TEQ calculation, van der Plas et al. (2000) concluded that the tumor promotion potential of PCBs might be underestimated by the TEQ approach alone. This is also supported by estimates of TEQs for the different Aroclors. Although EPA's SF included consideration of several Aroclors, the SFs for 1260, followed by 1254, were the highest in the studies evaluated and were used for the development of the SF for total PCBs (EPA 1996). The TEQ potency for Aroclor 1260 on a mass basis is lower than the potencies for several other Aroclors (Rushneck et al. 2004; Van den Berg et al. 2006). This also suggests that some of its carcinogenic potency is not attributable to dioxin-like PCB congeners.

Another recent review also supports PCB carcinogenicity as acting through an indirect mechanism, such as tumor promotion (rather than initiation) (Knerr and Schrenk 2006). Across the carcinogenicity studies evaluated, the TEQ dose (but not the total PCB dose) was found to be primarily responsible for the development of neoplasms in rats. However, tumor promotion experiments in rodents have shown that both dioxin-like and non-dioxin-like PCB congeners may act as liver tumor promoters. In the case of the van der Plas (2000) study, Knerr and Schrenk (2006) asserted that the purity data provided in that study were not sufficient to exclude the potential contribution of some dioxin-like PCB congeners to the observed toxicity (Knerr and Schrenk 2006). Although the dioxin-like PCB congeners showed much greater potency, some weak carcinogenic potency of non-dioxin-like PCBs cannot be excluded (Knerr and Schrenk 2006). For this reason, some scientists have suggested that the carcinogenic evaluation

of PCBs based solely on PCB TEQ evaluation is not sufficient (Safe 1994). Research and debate is active on the most appropriate methods to evaluate carcinogenic potential of environmental PCB mixtures.

The contribution of dioxin-like and non-dioxin-like PCBs to non-cancer effects is also an area of active research and discussion. Unlike carcinogenicity, health risks from PCBs associated with these effects are not evaluated on a TEQ basis. For these endpoints, non-dioxin-like PCBs may play a greater role than they do in carcinogenicity. The mechanisms of action for PCB neurotoxicity are not thought to be mediated by the Ah receptor, suggesting that non-dioxin-like PCBs may be important. However, the specific PCB congeners involved have not been well-characterized. It is possible that the most potent congeners for these endpoints may be enhanced or diluted in environmental mixtures relative to mixtures used to develop toxicity metrics. For example, environmental mixtures of PCBs may have more- or less-potent immunological effects than do the Aroclor 1254 mixture used in the study that is the basis for the reference dose.

Toxicological Profiles for All Other Chemicals of Potential Concern

1. 1,2-DIPHENYLHYDRAZINE

1,2-Diphenylhydrazine is a white solid that is only slightly soluble. It adheres to soil and can be carried into the air along with windblown dust. Once in water or exposed to air it is transformed into other chemicals within minutes, including azobenzene and benzidine. 1,2-Diphenylhydrazine is used to make fabric dyes in other countries, and to make certain medicines. There are no other major anthropogenic or natural sources of 1,2-diphenylhydrazine (ATSDR 1990).

Pharmacokinetics

Pharmacokinetic studies of 1,2-diphenylhydrazine have not been conducted with humans. Soil particles contaminated by this compound may be inhaled or ingested, but it is likely that most of the chemical would be excreted via urine (ATSDR 1990).

Acute toxicity

The acute health effects of 1,2-diphenylhydrazine in humans have not been studied. Animals die if they swallow large amounts of 1,2-diphenylhydrazine (ATSDR 1990).

Chronic toxicity

Animals develop liver disease if they eat small amounts of 1,2-diphenylhydrazine for more than a year (ATSDR 1990). Chronic toxicity data for humans are not available.

Carcinogenicity

EPA has determined that 1,2-diphenylhydrazine is a probable carcinogen (B2) because it causes cancer in rats and mice that have eaten it in food for most of their lifetime. EPA has established an **oral cancer slope factor of 0.8 per mg/kg-day** (IRIS).

2. 1,3-DICHLOROBENZENE AND 1,4-DICHLOROBENZENE

1,3-Dichlorobenzene is a clear liquid used to make herbicides, insecticides, medicine, and dyes. 1,4-Dichlorobenzene is a clear to white solid with a strong, pungent odor. When exposed to air, it slowly evaporates from a solid to a vapor. Most people can smell 1,4-dichlorobenzene in the air at very low levels. 1,4-Dichlorobenzene is the more common of the two chemicals. Dichlorobenzenes do not dissolve easily in water, and the small amounts that enter water quickly evaporate into the air. Sometimes, dichlorobenzenes bind to soil and sediment. Dichlorobenzenes in soil usually are not easily broken down by soil organisms. Evidence suggests that plants and fish absorb dichlorobenzenes (ToxFAQs).

Pharmacokinetics

Very little is known about the health effects of 1,3-dichlorobenzene, especially in humans, but they are likely to be similar to those of 1,4-dichlorobenzene (ToxFAQs). Pharmacokinetic information for oral 1,4-diclorobenze exposure is very limited, although the liver and kidney have been identified as target organs (IRIS).

Acute toxicity

Dichlorobenzenes have low acute toxicity. Nausea and vomiting are common, while liver damage is a rare effect after large exposures. Ingestion may result in hemolytic anemia, jaundice, and methemoglobinemia. Irritation of nose and eyes may be caused by exposure to vapors. CNS depression has been observed when airborne concentrations become extremely objectionable to the eyes and nose. The solid material produces a sensation of warmth or burning when held in contact with the skin, but the resulting irritation is slight; warm fumes or strong solutions of dichlorobenzene may irritate the intact skin slightly on prolonged or repeated contact (HSDB).

Chronic toxicity

Dichlorobenzene may cause liver damage and, in severe cases, cirrhosis. Individuals who are exposed to higher concentrations of p-dichlorobenzene may show weakness, dizziness, headache, rhinitis, twitching of the facial muscles, weight loss, and acute hemolytic anemia with methemoglobinuria (HSDB). NCEA has developed **RfDs of 0.003 and 0.03 mg/kg-day for 1,3-dichlorobenzene and 1,4-dichlorobenzene, respectively** (EPA 2005).

Carcinogenicity

Although there is inadequate evidence in humans for the carcinogenicity of dichlorobenzenes, there is sufficient evidence in animal models to determine the carcinogenicity of 1,4-dichlorobenzene. 1,4-Dichlorobenzene was shown to cause a high incidence of liver tumors in male and female mice. Evidence that its mechanism of action of carcinogenesis in the liver may be relevant for humans supports the IARC ruling that 1,4-dichlorobenzene is possibly carcinogenic to humans (Group 2B). The carcinogenicity of 1,3-dichlorobenzene has not been assessed by IARC due to a lack of human and animal studies on the topic (ToxFAQs). An **oral cancer slope factor of 0.024 per mg/kg-day** was developed by NCEA for 1,4-dichlorobenzene (EPA 2005).

3. 2,4,6-TRICHLOROPHENOL

Chlorophenols are a group of chemicals that are created by adding chlorines to a phenol molecule. Phenol is an aromatic compound derived from benzene. There are 5 basic types of chlorophenols and 19 different chlorophenols. Most are solid at room temperature with a strong, medicinal taste and smell. Even very small amounts may be tasted in water.

Some chlorophenols are used as pesticides while others are used in antiseptics. Small amounts are produced as a byproduct of water disinfection with chlorine. Significant amounts may also be released to the environment if the water or wastewater undergoing chlorination contains phenol (HSDB). Chlorophenols are also produced by paper mills while bleaching wood pulp with chlorine to make paper. Chlorophenols enter the environment when they are created as byproducts of disinfection or bleaching or during manufacture and use of pesticides and herbicides such as Prochloraz and Chloranile. Chloranile is currently used as a bleaching agent, but was previously used as a defoliant, herbicide and fungicide. Chlorophenols are generally released into the environment via water, and are rarely released into the air. Airborne chlorophenols are broken down by sunlight and then washed out of the sir by rain. Chlorophenols stick to soil and sediments at the bottom of lakes, streams, and rivers. Micro-organisms break down and remove low levels of chlorophenols in water, soil, and sediment from the environment in a few days to weeks (ToxFAQs).

Pharmacokinetics

A Finnish study of sawmill workers with occupational exposure to 2,4,6-trichlorophenol showed that it is readily absorbed across the skin and through the mucous membranes of the lungs. The mechanism of action of 2,4,6-trichlorophenol is interference with metabolism, which prevent cells from producing energy (HSDB).

Acute toxicity

Redness and edema result from dermal contact. If contact is prolonged, moderate and severe chemical burns may occur. 2,4,6-Trichlorophenol is also a lung and eye irritant (HSDB).

Chronic toxicity

Long-term exposure to this chemical results in reduced lung function, and possibly pulmonary fibrosis (HSDB). NCEA has developed an **oral RfD of 0.0001 mg/kg-day** for 2,4,6-trichlorophenol (EPA 2004).

Carcinogenicity

Although there is no human data available, sufficient animal studies have been conducted to classify 2,4,6-trichlorophenol in group 2B, a probable human carcinogen (IARC). Increased incidences of lymphomas or leukemia were observed in male rats and tumors in the livers of male and female mice (HSDB). EPA established an **oral cancer slope factor of 0.011 per mg/kg-day** for 2,4,6-trichlorophenol (IRIS).

4. 2,4-DICHLOROPHENOL

Chlorophenols are a group of chemicals that are created by adding chlorines to a phenol molecule. Phenol is an aromatic compound derived from benzene. There are 5 basic types of chlorophenols and 19 different chlorophenols. Most are solid at room temperature with a strong, medicinal taste and smell. Even very small amounts may be tasted in water.

Some chlorophenols are used as pesticides while others are used in antiseptics. Small amounts are produced as a byproduct of water disinfection with chlorine. Significant amounts may also be released to the environment if the water or wastewater undergoing chlorination contains phenol (HSDB). Chlorophenols are also produced by paper mills while bleaching wood pulp with chlorine to make paper. Chlorophenols enter the environment when they are created as byproducts of disinfection or bleaching or during manufacture and use of pesticides and herbicides such as Prochloraz and Chloranile. Chloranile is currently used as a bleaching agent, but was previously used as a defoliant, herbicide and fungicide. Chlorophenols are generally released into the environment via water, and are rarely released into the air. Airborne chlorophenols are broken down by sunlight and then washed out of the sir by rain. Chlorophenols stick to soil and sediments at the bottom of lakes, streams, and rivers. Micro-organisms break down and remove low levels of chlorophenols in water, soil, and sediment from the environment in a few days to weeks (ToxFAQs).

Pharmacokinetics

2,4-Dichlorophenol (DCP) has been shown to disrupt metabolism in rats. It has a demonstrated half-life in these animal models of 4 to 30 minutes, meaning that it does not build up in the body before it is excreted in urine (HSDB).

Acute toxicity

Acute exposures may cause muscular weakness, gastroenteric disturbances, severe depression, and collapse. 2,4-DCP primarily affects the nervous system, but it may also cause edema of the lung and injury to the liver, pancreas and spleen. Contact

dermatitis may be seen as well. Additional chlorination of the chemical increases its toxicity (HSDB).

Chronic toxicity

Chronic exposure may result in chloracne (HSDB). EPA has developed an **RfD of 0.003 mg/kg-day for 2,4-dichlorophenol** (IRIS).

Carcinogenicity

IARC states that combined exposure to polychlorophenols or to their sodium salts is possibly carcinogenic to humans, and ranks them in Group 2B. There is evidence to suggest that people exposed to chlorophenols for a long time may have slightly higher incidences of cancer. However, the people studied were exposed to other chemicals as well, so no definite link can be made between exposure to chlorophenol and increased incidence of cancer (HSDB).

5. 2,4-DINITROPHENOL

Dinitrophenols are a group of manufactured chemicals that do not exist naturally in the environment. There are six different dinitrophenols. The most commonly found and most commercially important dinitrophenol, 2,4-dinitrophenol (DNP), is a yellow solid with no smell. It is used to make dyes, wood preservatives, explosives, insect control substances, and other chemicals. It is also used in the process of developing photographic film. Currently, exposure to dinitrophenols occurs mainly from breathing air, drinking water, or eating food that contains the chemicals. Before being banned in 1938, DNP was used in diet pills. The majority of the information on the health effects of dinitrophenols comes from studies of patients who were prescribed diet pills containing DNP before it was banned (ToxFAQs).

When DNP enters the environment, it dissolves only slightly in water, and will not easily evaporate to air. It can be broken down slowly in water and soil by small organisms or through reactions with other chemicals. DNP sticks to small particles in water, which will cause it to eventually settle to the bottom sediment. Because DNP also sticks to some types of soil particles, it does not usually migrate deep into the soil with rainwater (ToxFAQs).

Pharmacokinetics

DNP acts as a stimulant to increases metabolic rate which then causes the body to increase temperature, heart and breathing rates. DNP is metabolized by humans, dogs, and mice into various phenolic compounds which are excreted through urine (HSDB).

Acute Toxicity

The dose of dinitrophenols ingested that will cause harmful effects varies among people. Increased basal metabolic rate (the rate that you use energy at complete rest), increased sweating, extreme thirst, a feeling of warmth, increased heart rate, breathing

rate, and body temperature have all been observed in people who swallowed as little as 1 mg/kg-day or as much as 46 mg/kg-day for short or long periods of time. Ingestion may cause stimulation or excitement, followed by marked fatigue and depression (HSDB).

Chronic Toxicity

Ingesting 2-4 mg/kg-day DNP for short or long periods has caused cataracts in some people, while ingesting 1 to 4 mg/kg-day for short or long periods has caused skin rashes and decreases in white blood cells. A significant decrease in white blood cell count may prevent the immune system from functioning properly. Similar doses have also been observed to cause weight loss. Cyanosis and jaundice caused by liver damage have been observed both in individuals exposed for long periods of time and in those surviving the acute phase of poisoning (HSDB). EPA has developed an **RfD of 0.002 mg/kg-day** for DNP based on human exposures and studies conducted with animals (IRIS).

Carcinogenicity

Currently, there are no studies available in people or animals on the carcinogenic effects of DNP. Due to this lack of data, the Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the EPA have not classified DNP for carcinogenicity (IRIS, ToxFAQs).

6. 2,4-DINITROTOLUENE AND 2,6-DINITROTOLUENE

Both 2,4-dinitrotoluene (DNT) and 2,6-DNT are pale yellow solids with a slight odor. They are two of the six forms of DNT. DNT is not a natural substance, and it is created by mixing toluene with nitric acid. DNT is usually used to make flexible polyurethane foams used in the bedding and furniture industries. It is also used in the air bags of automobiles and in the production of explosives, ammunition, and dyes.

DNT is present in the environment in many media, including soil, surface and ground water, and air. It has been found at some hazardous waste sites that contain buried ammunition wastes; however, it is found mostly in the air outside of certain manufacturing plants.

DNT does not usually evaporate, and it does not stay in the environment because it is broken down by sunlight and by bacteria. In water, DNT tends to be more stable and less likely to break down. DNT can be transferred to plants by root uptake from contaminated water or soil (ToxFAQs).

Pharmacokinetics

The human health effects from DNT exposure that have been reported are from occupational exposure studies in which workers were exposed primarily by inhalation, but with some contribution assumed from dermal absorption and ingestion. Humans appear to metabolize DNT fairly quickly with rapid absorption

followed by urinary excretion of metabolites. A similar pattern of metabolism has been observed in animal models (HSDB).

Acute toxicity

The following symptoms have been reported as a result of varying doses of DNT: vertigo, fatigue, dizziness, weakness, nausea, vomiting, insomnia, tremor, paralysis, unconsciousness, chest pain, shortness of breath, palpitation, anorexia, and loss of weight. Following poisoning, the following symptoms have been observed: methemoglobinemia, anemia and liver necrosis. Liver injury may be more common than cyanosis (HSDB).

Chronic toxicity

Major effects from chronic exposure include methemoglobinemia, cyanosis; neurotoxicity; and possible excess mortality from ischemic heart disease and residual circulatory system effects. Neurotoxicity is characterized by vertigo, tremors, unconsciousness, and paralysis (HSDB). EPA has developed an **RfD of 0.002 mg/kg-day** for 2,4-DNT based on animal studies as well as human exposures (IRIS). For 2,6-DNT, a HEAST **RfD of 0.001 mg/kg-day** is available (EPA 2004).

Carcinogenicity

In animal studies, both 2,4-DNT and 2,6-DNT caused liver cancer in rats. There are no studies on the effects of 2,4-DNT and 2,6-DNT on people. The International Agency for Research on Cancer (IARC) has determined that 2,4-DNT and 2,6-DNT are possible human carcinogens (ToxFAQs). An **oral cancer slope factor of 0.68 mg/kg-day** has been developed for a mixture of 2,4-DNT and 2,6-DNT (IRIS).

7. 2-CHLOROPHENOL

Chlorophenols are a group of chemicals that are created by adding chlorines to a phenol molecule. Phenol is an aromatic compound derived from benzene. There are 5 basic types of chlorophenols and 19 different chlorophenols. Most are solid at room temperature with a strong, medicinal taste and smell. Even very small amounts may be tasted in water.

Some chlorophenols are used as pesticides while others are used in antiseptics. Small amounts are produced as a byproduct of water disinfection with chlorine. Significant amounts may also be released to the environment if the water or wastewater undergoing chlorination contains phenol (HSDB). Chlorophenols are also produced by paper mills while bleaching wood pulp with chlorine to make paper. Chlorophenols enter the environment when they are created as byproducts of disinfection or bleaching or during manufacture and use of pesticides and herbicides such as Prochloraz and Chloranile. Chloranile is currently used as a bleaching agent, but was previously used as a defoliant, herbicide and fungicide. Chlorophenols are generally released into the environment via water, and are rarely released into the air. Airborne

chlorophenols are broken down by sunlight and then washed out of the sir by rain. Chlorophenols stick to soil and sediments at the bottom of lakes, streams, and rivers. Micro-organisms break down and remove low levels of chlorophenols in water, soil, and sediment from the environment in a few days to weeks (ToxFAQs).

Pharmacokinetics

2-Chlorophenol disrupts metabolism, and may increase heart and breathing rates before it is excreted through bile and urine (HSDB).

Acute toxicity

Acute exposures may cause muscular weakness, gastroenteric disturbances, severe depression, and collapse. 2-Chlorophenol primarily affects the nervous system, but it may also cause edema of the lung and injury to the liver, pancreas and spleen. Contact dermatitis may be seen as well. Additional chlorination of the chemical increases its toxicity (HSDB).

Chronic toxicity

Chronic exposure may result in chloracne (HSDB). EPA has developed an **RfD of 0.005 mg/kg-day for 2-chlorophenol** (IRIS).

Carcinogenicity

Carcinogenicity of 2-chlorophenol is unknown, EPA has not completed its evaluation for carcinogenic potential of this chemical. There is evidence to suggest that people exposed to chlorophenols for a long time may have slightly higher incidences of cancer. However, the people studied were exposed to other chemicals as well (ToxFAQs).

8. 3,3'-DICHLOROBENZIDINE

3,3'-Dichlorobenzidine is a gray- to purple-colored crystalline solid. It changes from a solid to a gas very slowly. The salt of this compound is the major form in actual use. Neither 3,3'-dichlorobenzidine nor its salt are found naturally in the environment. They are manufactured for pigments for printing inks, textiles, plastics and enamels, paint, leather, and rubber. Human exposure may occur in industrial settings via inhalation or direct contact, or in residential settings through contact with contaminated dirt or water.

Pharmacokinetics

When 3,3'-dichlorobenzidine enters the body, very little of it leaves the body unchanged. Over 90% of the parent compound is transformed to metabolites which leave the body, mainly in urine and to a lesser extent in feces, within 72 hours after exposure (ToxFAQs).

Acute toxicity

The salt form of 3,3'-dichlorobenzidine may have caused sore throat, respiratory infections, stomach upset, headache, dizziness, caustic burns, and dermatitis in workers exposed to the chemical. Death has occurred in laboratory animals that ate very high levels of 3,3'-dichlorobenzidine mixed in their food for short periods of time (ToxFAQs).

Chronic toxicity

IRIS does not provide a discussion of chronic effects of exposure to 3,3′-dichlorobenzidine or an RfD. Laboratory animals exposed to moderate levels of 3,3'-dichlorobenzidine mixed with food for a long time suffered mild injury to the liver (ToxFAQs).

Carcinogenicity

Studies show that 3,3'-dichlorobenzidine caused cancer of the liver, skin, breast, bladder, and tissues that form blood and other organs in laboratory animals that ate it in their food (ToxFAQs). To date, evidence is inconclusive with regard to whether 3,3'-dichlorobenzidine has caused cancer in people who worked with it or who were exposed to it unknowingly or by accident for a short or long time. However, because of the many types of cancer that 3,3'-dichlorobenzidine has caused in different tissues of many types of laboratory animals, 3,3'-dichlorobenzidine has been classified as a probable human carcinogen (B2) with an **oral cancer slope factor of 0.45 per mg/kg-day** (IRIS).

9. 3-NITROANILINE AND 4-NITROANILINE

Nitroaniline may be released to the environment from process and waste emissions involved in its production or use as a chemical intermediate and through stack emissions from hazardous waste incineration. Once in the environment, it has a half life of approximately one day in the air, and four days in water. Nitroaniline persists for the longest time in soil, where it binds to particles and is prevented from mobilizing. Exposure to sunlight will break down the chemical (HSDB).

Pharmacokinetics

Nitroaniline is quickly absorbed across the skin and through the lining of the lungs. It has a biological half-life of approximately 1 hour, and is excreted largely through urine (HSDB).

Acute Toxicity

Nitroaniline may be harmful if absorbed through skin or inhaled. It is absorbed rapidly and induces methemoglobinemia. Symptoms of methemoglobinemia include cyanosis, headache, dizziness, weakness, lethargy, loss of coordination, dyspnea, coma, and death. Heart, liver, and kidney effects may be secondary to hemolysis.

Other symptoms of overexposure include irritability, vomiting, diarrhea, cyanosis, ataxia, tachycardia, convulsions, respiratory arrest, and anemia (HSDB).

Chronic Toxicity

Nitroaniline is a potent methemoglobin-inducing agent and given sufficiently high or prolonged exposures, hemolysis can occur. Liver damage is also a known effect of nitroaniline exposure, and prolonged exposure may also result in heart or kidney damage (HSDB). EPA has developed **provisional RfDs of 0.0003 mg/kg-day and 0.003 mg/kg-day for 3-nitroaniline and 4-nitroaniline, respectively** (EPA 2005).

Carcinogenicity

Nitroaniline was found to be not classifiable as to carcinogenicity by IARC (HSDB). EPA's PPRTV provides a **provisional oral cancer slope factor of 0.021 per mg/kg-day** for both 3-nitroaniline and 4-nitroaniline (EPA 2005).

10. 4,6-DINITRO-O-CRESOL

Dinitrocresols are manufactured chemicals that do not exist naturally in the environment. There are 18 different dinitrocresols of which 4,6-dinitro-o-cresol (DNOC) is the most commercially important. In its pure form, DNOC is a yellow solid with no smell that stains human skin on contact. Used primarily for insect control and crop protection, DNOC may be sold under several trade names including Antinonnin, Detal, and Dinitrol (ToxFAQs).

DNOC enters the environment during its manufacture and use, and is occasionally formed through reactions with other airborne chemicals. Another path for DNOC to enter the environment is through leaks at landfills, or accidental spills that occur during manufacture or transport.

Once in the environment, DNOC will only dissolve slightly in water, and it does not evaporate easily into air. DNOC will adhere to suspended particles in water, eventually settling to the bottom of lakes and streams. Because of its tendency to adhere to particles, DNOC does not usually migrate through soil to groundwater. Small organisms present in air, water and soil slowly break DNOC down (ToxFAQs).

Pharmacokinetics

DNOC is a yellow compound that. Absorption of DNOC by any route and subsequent distribution to tissues results in a characteristic yellow staining of visceral organs and tissues including the eyes, blood serum, skeletal tissues, and urine. The mechanism of acute toxicity is disruption of metabolism and diversion of energy into heat production which leads to an increase in body temperature (HSDB).

Blood levels of DNOC below 10 ppm are considered of trivial importance; levels of 11 to 20 ppm indicate appreciable absorption; and above these blood levels toxic

manifestations are likely. Levels greater than 50 ppm are critically dangerous (Doull et al. 1986).

Acute toxicity

Acute poisoning may result in increased basal metabolic rate, including increased temperature, heart and breathing rates. Other symptoms of exposure include nausea, gastric upset, restlessness, sensation of heat, flushed skin, sweating, rapid respiration, increased pulse rate, tachycardia, fever, cyanosis, and finally collapse and coma (ToxFAQs, HSDB).

Chronic toxicity

Chronic exposure to DNOC may cause increased basal metabolic rate, feelings of fatigue, restlessness, or anxiety, excessive sweating, unusual thirst, and loss of weight. A yellow staining of the whites of the eyes has been noted, and cataract formation is another possible result of chronic DNOC exposure (ToxFAQs, HSDB). EPA has established a **provisional RfD of 0.0001 mg/kg-day** for 4,6-dinitro-o-cresol (EPA 2004).

Carcinogenicity

DNOC is not rated by IARC, EPA or DHHS. Its carcinogenicity has not been classified, as studies have not been conducted to determine carcinogenic potential in humans or animals (ToxFAQs, IRIS).

11. 4-CHLOROANILINE

4-Chloroaniline or para-chloroaniline (PCA) is a colorless to amber-colored crystalline solid with a slight aromatic odor. The chemical is soluble in water and in common organic solvents. PCA is used as an intermediate in the production of a number of products, including agricultural chemicals, azo dyes and pigments, cosmetics, and pharmaceutical products (HSDB).

Pharmacokinetics

In humans, damage to hemoglobin molecules and a reduced ability to circulate oxygen throughout the body is detectable as early as 30 minutes after accidental exposure, with a maximum level at 3 hours. Due to common genetic differences in how humans metabolize chemical compounds, certain individuals may be at greater risk from exposure. Excretion in humans occurs primarily via the urine, with PCA and its conjugates appearing as early as 30 minutes after exposure. Excretion takes place mainly during the first 24 hours and is almost complete within 72 hours. Repeated exposure to PCA leads to cyanosis and methemoglobinemia, followed by effects in blood, liver, spleen, and kidneys (HSDB).

Acute Toxicity

Exposure via inhalation and/or simultaneous dermal absorption may result in severe headache, nausea, vomiting, dryness of throat, confusion, ataxia, vertigo, lethargy, drowsiness, and finally coma. Many of these symptoms can be attributed to increased levels of methemoglobin (HSDB).

Chronic Toxicity

Long term exposure to PCA may result in the development of anemia or cyanosis. A Heart, liver, and kidney effects may be secondary to hemolysis (HSDB). EPA has developed an **RfD of 0.004 mg/kg-day** based on studies conducted with rats (IRIS).

Carcinogenicity

IARC states that there is inadequate evidence in humans for the carcinogenicity of PCA. However, because there is sufficient evidence in experimental animals for the carcinogenicity of PCA, it has been designated as a Group 2B chemical, ranking it as possibly carcinogenic to humans (ToxFAQs). EPA has not fully evaluated this chemical for carcinogenic potential (IRIS).

12. 4-METHYLPHENOL OR P-CRESOL

Cresols are a widely occurring natural and manufactured group of chemicals. In their pure form, they are colorless solids and may be liquids if they are mixtures. Cresols generally smell like medicine or tar. There are three forms of cresols that are only slightly different in their chemical structure: ortho-cresol (o-cresol), meta-cresol (m-cresol), and para-cresol (p-cresol). These forms may occur separately or as a mixture. They are used as solvents to dissolve other chemicals, as disinfectants and deodorizers, and are ingredients in various pesticides.

Cresols are found in many foods as well as in wood and tobacco smoke, crude oil, and coal tar. Cresols are also a main component of brown mixtures such as creosote and cresylic acids, which are wood preservatives. Small organisms in soil and water produce cresols when they break down materials in the environment. Cresols enter the environment from natural sources, car exhaust, combustion, manufacturing use, and waste sites. Cresols are ubiquitous in our environment, but usually occur only at low levels because they quickly break down. In air, cresols quickly break down into other chemicals. Cresols do not evaporate quickly from water, but they can be removed by bacteria. Cresols may persist longer when it reaches deep groundwater or water that does not have bacteria.

In soil, approximately one half of the total amount of cresols present will break down in a week. Cresols do not appear to accumulate in fish or meat.

Pharmacokinetics

Cresols are corrosive to tissues and can cause serious burns. These chemicals are rapidly absorbed by all routes, cause systemic effects, and can be fatal by any route of exposure. Systemic effects include profound CNS depression, seizures, hemolysis, methemoglobinemia, pulmonary edema, and lung, liver, pancreas, spleen, heart, and kidney damage. Metabolic acidosis may occur. Cresol is a cellular poison and is toxic to all cells (HSDB).

Acute Toxicity

Skin contact is the main exposure route. Pain is followed by numbness; skin reddens, then blanches, blisters, and forms a scab. Skin contact can result in severe skin burns. Eye contact produces irritation, redness, corneal burns, keratitis, and possibly, in severe cases, blindness.

Inhalation produces coughing and labored, fast breathing; respiratory failure may result. Ingestion causes a burning pain in the mouth and throat, and abdominal pain, nausea, vomiting, bloody diarrhea and collapse. White necrotic lesions of the mouth, throat and stomach are also seen (HSDB).

Chronic Toxicity

Short-term and long-term studies with animals have shown similar effects from exposure to cresols. No human or animal studies have shown harmful effects from cresols on the ability to have children.

It is not known what the effects are from long-term ingestion or skin contact with low levels of cresols (ToxFAQs). Chronic exposure may produce allergic dermatitis, digestive disturbances, CNS effects, and liver and kidney damage. Effects of chronic exposure to p-cresol may include vomiting, difficulty swallowing, excessive salivation, diarrhea, loss of appetite, headache, fainting, dizziness, mental disturbances, skin rash, or death from severe damage to the liver or kidneys (HSDB). HEAST has developed an **RfD of 0.005 mg/kg-day** for 4-methylphenol (EPA 2005).

Carcinogenicity

4-methylphenol has received classification as a class C chemical (a possible human carcinogen) from IARC. This ruling was based on limited evidence from animal studies, as the data from human exposures is inadequate (HSDB).

13. ALDRIN

Aldrin is the common name for a popular insecticide that was used extensively until 1970, at which time the US Department of Agriculture cancelled all uses. In 1972, however, EPA approved aldrin for killing termites. Use of aldrin to control termites continued until 1987, at which time the manufacturer voluntarily canceled the registration for use in controlling termites. Pure aldrin is a white powder, but

technical-grade aldrin (>85% aldrin) is a tan powder. Aldrin slowly evaporates in the air.

Pharmacokinetics

Exposure of the general population to aldrin most likely occurs through eating contaminated food. Exposure of some infants occurs by drinking mother's milk containing aldrin. Studies in animals show that aldrin enters the body quickly after exposure. Once inside the body, aldrin quickly breaks down to dieldrin, where it is stored in lipid reserves.

Acute toxicity

Exposure to very high levels of aldrin for a short time causes convulsions or kidney damage. One very young child died from drinking a solution containing a very high level of dieldrin. Another very young child died after eating food contaminated with aldrin (ToxFAQs). Animal studies have shown that exposure to moderate levels of aldrin for a short time causes decreased ability to fight infections.

Chronic toxicity

Exposure to moderate levels of aldrin for a long time causes headaches, dizziness, irritability, vomiting, or uncontrollable muscle movements. Some sensitive people develop a condition in which aldrin or dieldrin causes the body to destroy its own blood cells (ToxFAQs).

EPA established an **RfD of 0.00003 mg/kg-day** based on observed liver toxicity in a chronic rat feeding study (IRIS).

Carcinogenicity

EPA has classified aldrin as a probable human carcinogen (B2) and has established an **oral cancer slope factor of 17 per mg/kg-day** based on observations of significant increases in tumor responses in three different strains of mice in both males and females following aldrin exposure (IRIS).

14. ALUMINUM

Aluminum is the most abundant metal and the third most abundant element, after oxygen and silicon, in the earth's crust. It is widely distributed and constitutes approximately 8 percent of the earth's surface layer. However, aluminum is a very reactive element and is never found as free metal in nature. It is found combined with other elements, most commonly with oxygen, silicon, and fluorine. High concentrations in the environment can be caused by the mining and processing of its ores and by the production of aluminum metal, alloys, and compounds. Small amounts of aluminum are released into the environment from coal-fired power plants and incinerators (ATSDR 1999b).

Pharmacokinetics

Since little aluminum is absorbed, it is excreted in the feces, much of it in the form of aluminum phosphate. There is no generally no increase in the amount of aluminum in tissues, except in bone, as demonstrated in animal experiments. Some aluminum may be absorbed by patients undergoing dialysis; the kidney is responsible for removing the majority of absorbed aluminum (HSDB). Aluminum in lung tissue appears to be derived from inhaled particulates rather than any affinity of lung tissue for aluminum.

Acute toxicity

Low-level exposure to aluminum from food, air, water, or contact with skin is not thought to harm your health (ATSDR 1999b). Aluminum, however, is not a necessary substance for our bodies and too much may be harmful. People who are exposed to high levels of aluminum in air may have respiratory problems including coughing and asthma from breathing dust.

Chronic toxicity

Some studies show that people with Alzheimer's disease have more aluminum than usual in their brains. Data are inconclusive on whether aluminum causes the disease or whether the buildup of aluminum happens to people who already have the disease. Aluminum is known to cause additional neurological problems such as memory loss and impaired motor skills. Infants and adults who received large doses of aluminum as a treatment for another problem developed bone diseases, which suggests that aluminum may cause skeletal problems. Some sensitive people develop skin rashes from using aluminum chlorohydrate deodorants (ATSDR 1999b). EPA's PPRTV provides an **RfD for aluminum of 1 mg/kg-day** (EPA 2004).

Carcinogenicity

EPA has not conducted a complete evaluation and determination of the carcinogenicity of aluminum (IRIS). Available data suggest that this element is not carcinogenic (ATSDR 1999b).

15. ANILINE

Aniline is a clear to slightly yellow liquid with a characteristic odor. It does not readily evaporate at room temperature, is slightly soluble in water, and mixes readily with most organic solvents. Aniline is used to make a wide variety of products such as polyurethane foam, agricultural chemicals, synthetic dyes, antioxidants, stabilizers for the rubber industry, herbicides, varnishes and explosives. Aniline in the air is broken down within a few days by other chemicals and by sunlight. In water, it can stick to sediment and particulate matter or evaporate to the air. Most of it will be broken down by bacteria and other micro-organisms. Aniline will only partially stick to the soil. Small amounts may evaporate into air or pass through the soil to groundwater. Most

of the aniline in soil will be broken down by bacteria and other micro-organisms. Aniline does not accumulate in the food chain (ToxFAQs).

Pharmacokinetics

Humans appear to be more sensitive than rats to aniline exposure (as indicated by formation of methemoglobin) Jenkins et al. (1972) noted that after oral administration of aniline to volunteers and rats, the dose that produced increased levels of methemoglobin was much lower for humans than for rats. The reason for this increased sensitivity in humans is not known and does not appear to be related to the half-life of methemoglobin in the serum, which is three times longer in rats than in humans. Human half-life of methemoglobin is less than one hour (IRIS).

Acute Toxicity

Aniline can be toxic if it is ingested, inhaled, or contacts the skin. Aniline damages hemoglobin, a protein that normally transports oxygen in the blood. The damaged hemoglobin cannot carry oxygen. This condition is known as methemoglobinemia and its severity depends on how much a person is exposed to and for how long. Methemoglobinemia is the most prominent symptom of aniline poisoning in humans, resulting in cyanosis (a purplish blue skin color) following acute high exposure to aniline. Dizziness, headaches, irregular heart beat, convulsions, coma, and death may also occur. Direct contact with aniline can also produce skin and eye irritation (ToxFAQs).

Chronic Toxicity

Long-term exposure to lower levels of aniline may cause symptoms similar to those experienced in acute high-level exposure. There is no reliable information on whether aniline has adverse reproductive effects in humans. Studies in animals have not demonstrated reproductive toxicity for aniline (ToxFAQs). A provisional EPA **RfD of 0.007 mg/kg-day** was used in this HHRA (EPA 2005).

Carcinogenicity

Evidence from human studies is inadequate to determine whether exposure to aniline can increase the risk of developing cancer in people. Rats that ate food contaminated with aniline for life developed cancer of the spleen. The International Agency for Research on Cancer (IARC) determined that aniline is not classifiable as to its carcinogenicity to humans. The EPA has determined that aniline is a probable human carcinogen (ToxFAQs). EPA has established an **oral cancer slope factor of 0.0057 mg/kg-day** (IRIS).

16. ANTIMONY

Antimony is naturally present in the earth's crust. The release of antimony into the environment occurs primarily through anthropogenic sources like non-ferrous metal

mining, smelting, refining, and production, the use and disposal of antimony alloys and compounds, coal combustion, and refuse and sludge combustion. Antimony exposure occurs through inhalation, ingestion of food containing antimony, and through dermal contact (IRIS).

Pharmacokinetics

Antimony is absorbed by erythrocytes and distributed to other tissues such as liver, adrenals, spleen, and thyroid. Much of the absorbed antimony is excreted via urine and feces. Of the antimony that is not excreted, the longest biological half-life is believed to occur in the lungs. The highest concentrations of antimony after acute or chronic exposure have been found in the thyroid, adrenals, liver, and kidney (HSDB).

Acute toxicity

Violent vomiting, diarrhea, lowered respiratory rate, myocardial edema, hyperemia, and capillary engorgement are major results of acute exposure to antimony. Seventy people became acutely ill after ingesting lemonade containing 0.013% antimony. Fifty-six of the victims were treated for burning stomach pains, colic, nausea, and vomiting. Most recovered after approximately three hours, while some required hospitalization for a few days (IRIS).

Chronic toxicity

Dyspnea, weight and hair loss, popular eruptions on the skin, jaundice, damage to the heart and liver, and spleen, kidney damage, abnormal increase in erythrocytes, and a decrease in leukocytes are reported from long-term exposure to antimony. Chronic inhalation results in damage to the lungs, liver and heart (HSDB). EPA developed an **RfD for antimony of 0.0004 mg/kg-day** based on a study in which rats were exposed to potassium antimony tartrate (IRIS).

Carcinogenicity

EPA has not conducted a complete evaluation and determination of the carcinogenicity of antimony (IRIS).

17. BARIUM

Barium metal does not occur in nature. The most common barium ores are sulfate, barite, carbonate, and witherite. The largest use of barium is in the removal of traces of gases from vacuum and television picture tubes. Barium is released into the environment through the disposal of drilling waste, copper smelting, manufacture of motor vehicle parts, combustion of coal and oil, and the mining, refining, and production of barium and barium-based chemicals (OGWDW).

Pharmacokinetics

The human body contains approximately 22 mg of barium, 66% of which is in the bones. Common routes of exposure are ingestion, inhalation of dust or fumes, and skin or eye contact (HSDB).

Acute toxicity

Exposure to large quantities of barium can cause gastrointestinal disturbances and muscular weakness. No Health Advisories have been established for short-term exposure to barium (OGWDW).

Chronic toxicity

Chronic exposure to barium can cause hypertension (OGWDW). Populations with pulmonary diseases are especially at risk. Barium is not considered an industrial health hazard (HSDB). EPA has established an **oral RfD for barium of 0.2 mg/kg-day** (IRIS).

Carcinogenicity

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of barium (IRIS). EPA has placed barium in weight-of-evidence group D, not classifiable as to human carcinogenicity.

18. BENZIDINE

Benzidine may be released as emissions and in wastewater during its production and use as an intermediate in the manufacture of direct azo dyes. Large-scale manufacturing of benzidine in the US has been suspended since 1976. It is now produced in the US for domestic consumption only with strict regulations that it be maintained in isolated or closed systems that would limit its release (HSDB). Exposure to benzidine is primarily occupational via dermal adsorption, inhalation, and ingestion in workers connected with its production and conversion into direct azo dyes. The respiratory route is of major importance under some manufacturing conditions.

Pharmacokinetics

Absorbed doses of benzidine are rapidly transferred to the excretory organs, liver, gastrointestinal tract, kidney, and bladder. Half-lives determined experimentally range from 65 hr in rat to 88 hr in dogs (HSDB).

Acute toxicity

Ingestion of benzidine may produce nausea, vomiting, liver, and kidney damage (HSDB).

Chronic toxicity

Long-term exposure to benzidine has been shown to produce a spectrum of lesions of the epithelium of the urinary bladder, which may precede appearance of malignancy. Presence of visible or occult of blood in urine or the development of pain or difficulty in urinating may signal appearance of such lesions (HSDB). EPA has established an **RfD of 0.003 mg/kg-day** based on a chronic oral mouse bioassay (IRIS).

Carcinogenicity

EPA has classified benzidine as a known (Class A) carcinogen based on observations of increased incidence of bladder cancer and bladder cancer-related deaths in exposed workers. EPA has established an **oral cancer slope factor of 230 per mg/kg-day** (IRIS).

19. BIS(2-CHLOROETHYL)ETHER

Bis(2-chloroethyl)ether (BCEE) is a colorless nonflammable liquid with a strong, unpleasant odor. It does not occur naturally, but is manufactured for use in the production of pesticides and other chemicals. Limited amounts of BCEE will dissolve in water, and it also will slowly evaporate into air. In the environment, BCEE is broken down by bacteria in soil and water and by chemical reactions in the air, so it does not tend to persist for long periods (ToxFAQs).

Pharmacokinetics

BCEE enters the body easily after being swallowed in food or water, or after being inhaled in air. It may also enter by crossing the skin when dermal contact occurs. Once inside the body, BCEE is broken down to a number of different chemicals, and these are eliminated in the urine or the breath. Most BCEE that enters the body is removed in this way within two to three days, so BCEE does not tend to bioaccumulate (ToxFAQs).

Acute toxicity

People exposed to BCEE vapors report that it is highly irritating to the eyes and the nose. Animal studies show that BCEE vapors can cause severe injury to the lungs, and may lead to death (ToxFAQs).

Chronic toxicity

The chronic effects of BCEE on other organs (besides the lung) and body functions have not been well studied. It is not known if BCEE impairs reproduction or the development of fetuses (ToxFAQs). EPA has not established an RfD for BCEE.

Carcinogenicity

Mice given repeated doses of BCEE through the mouth developed liver tumors. This suggests that BCEE might cause cancer in humans, although no cases of cancer due to BCEE have been reported in people and BCEE was also not found to induce excess

cancer after feeding to rats. EPA has classified BCEE as a probable human carcinogen (B2) and has established an **oral cancer slope factor of 1.1 per mg/kg-day** based on positive carcinogenicity results in two strains of mice and evidence of mutagenicity (IRIS).

20. BIS(2-CHLOROISOPROPYL)ETHER

Bis(2-chloroisopropyl)ether is used primarily as a solvent in the manufacture of fats, waxes, and greases; as an extractant; in paint and varnish removers; in spotting and cleaning solutions; and in textile processing (HSDB). There is no evidence of commercial production of this compound within the US.

If released to water or moist soil, bis(2-chloroisopropyl)ether will hydrolyze rapidly based on an estimated hydrolysis half-life of < 38.4 sec in water. Therefore, biodegradation, bioconcentration in aquatic organisms and adsorption to soil and sediment are not expected to be significant fate processes (HSDB).

Pharmacokinetics

After single oral doses, bis-chloroisopropylether appeared to be readily absorbed by both female rats and monkeys (HSDB). With respect to the percentage of the radiolabeled administered dose recovered in the tissues and excreta, higher amounts of radioactivity were found in the fat (1.98%), urine (63.36%), feces (5.87%), and expired air (15.96%) of the rat compared to the monkey. The corresponding figures in the monkey were 0.78%, 28.61%, 1.19%, and 0%.

Acute toxicity

The acute toxicity of bis(2-chloroisopropyl)ether is not well-studied. Studies with rats exposed to an atmosphere saturated with bis(2-chloroisopropyl) ether exhibited signs of immediate eye irritation and lack of coordination; the maximum exposure time causing no death was 1 hr. When rats were exposed to 700 ppm, deaths occurred after 6 hr of exposure. Autopsy revealed slight lung irritation and moderate to severe liver damage (HSDB).

Chronic toxicity

EPA has established an **RfD of 0.04 mg/kg-day** based a chronic oral study with mice that documented a decrease in hemoglobin and possible red blood cell destruction (IRIS).

Carcinogenicity

There is limited evidence in experimental animals for the carcinogenicity of bis(2-chloroisopropyl)ether. The IARC (1995) indicated the carcinogenicity of this chemical to humans is not classifiable (Category 3). HEAST provides an oral cancer slope factor of **0.07 per mg/kg-day for bis(2-chloroisopropyl)ether** (EPA 2005).

21. BIS(2-ETHYLHEXYL)PHTHALATE

Bis(2-ethylhexyl)phthalate (BEHP) is a man-made chemical that is commonly added to plastics to make them flexible. This compound is present in plastic products such as rainwear, footwear, upholstery materials, imitation leather, waterproof gloves, tablecloths, shower curtains, food packaging materials, floor tiles, and children's toys. It can be an ingredient in paints, flexible tubing, plastic bags, containers for blood, printing inks, pesticides, cosmetics, and vacuum pump oil and can be used for testing air filtration systems.

Pharmacokinetics

Small amounts of BEHP may enter your body by skin contact with plastics, but most evidence indicates that very little enters this way (ToxFAQs). Most BEHP that enters the body in food, water, or air is taken up into the blood from the intestines and lungs. After BEHP is absorbed into your body, most of it is rapidly broken down to mono(ethylhexyl)phthalate (MEHP) and 2-ethylhexanol. The toxicities of MEHP and 2-ethylhexanol are similar to the toxicity of BEHP. These compounds travel through the bloodstream to the liver, kidneys, and testes, and small amounts will become stored in fat or secreted in breast milk. Most of the BEHP, MEHP, and 2-ethylhexanol leave your body within 24 hours in the urine and feces.

Acute toxicity

BEHP appears to affect rats and mice more than it affects humans and some other animals. Short-term exposures to high levels of BEHP interfered with sperm formation in mice and rats. These effects were reversible, but sexual maturity was delayed when the animals were exposed before puberty. Short-term exposures appeared to have no effect on male fertility (ToxFAQs).

Chronic toxicity

Long-term exposure of rats to BEHP resulted in structural and functional changes in the kidney. The structural kidney changes seen in rats are similar to those in the kidneys of long-term dialysis patients (ToxFAQs).

EPA has established an **RfD of 0.02 mg/kg-day** based on a sub-chronic to chronic bioassay with guinea pig that documented increased relative liver weight (IRIS).

Carcinogenicity

EPA has classified BEHP as a probable human carcinogen (B2) and has established an **oral cancer slope factor of 0.014 per mg/kg-day** based on observations of significant dose-related increases in liver tumor responses in rats and mice of both sexes (IRIS).

22. BUTYL BENZYL PHTHALATE

Butyl benzyl phthalate (BBP) is a clear oily liquid that is used as a plasticizer mainly in the polyvinyl chloride for vinyl floor tile, vinyl foams and carpet backing and in

cellulose plastics and polyurethane (HSDB). It can exist in both the vapor and particulate phase when exposed to air, and will evaporate from water and moist soils. The half life of BBP is between four and thirteen days (HSDB).

Pharmacokinetics

No evidence was found describing the metabolism pathway of BBP in humans. In animal models it has been shown to absorb quickly across the walls of the intestine, and has a half life of 10 minutes in rats (Eigenberg et al. 1986). In general, phthalate esters are not absorbed through skin (Clayton and Clayton 1981) (HSDB).

Acute toxicity

No human data is available from controlled studies, however, observations of workers exposed in occupational settings has shown that the acute toxicity of phthalates is very slight and decreases generally with the increasing molecular weight (HSDB).

Chronic toxicity

Available data in humans are inadequate to serve a basis for assessment of effects of long term exposure to butyl benzyl phthalate in human populations (HSDB). EPA has established an **RfD of 0.02 mg/kg-day** for this chemical (IRIS) based on numerous animal studies. In studies with rats, effects included changes in the blood, kidney damage, and lesions on the testes (IRIS).

Carcinogenicity

EPA has classified butyl benzyl phthalate as a Class C chemical; a possible human carcinogen. There have been no studies done on humans to test for carcinogenicity, this rating is based solely on rat studies which showed a statistically significant increase in tumors in exposed rats (HSDB). IARC states that carcinogenicity of BBP in humans is not classifiable.

23. CADMIUM

Cadmium is a heavy metal that is released through a wide variety of industrial and agricultural activities. The accumulation of cadmium in human and other biological tissue has been evaluated in both epidemiological and toxicological studies. ATSDR (1999c) has determined that exposure conditions of most concern are long-term exposure to elevated levels in the diet.

Pharmacokinetics

Cadmium is not readily absorbed when exposure occurs via ingestion. Absorption may be much higher in iron-deficient individuals. Evaluations of the impact of cadmium complexation indicate that cadmium absorption from food is not dependent upon chemical complexation. Some populations with high dietary cadmium intakes

have elevated blood cadmium levels, which could be due to the particular forms of cadmium in their food (ATSDR 1999c).

Cadmium is not directly metabolized, but absorption appears to involve sequestering by metallothionein, and plasma cadmium is found primarily bound to this protein. This type of binding appears to protect the kidney. It is thought that kidney damage by cadmium occurs primarily due to unbound cadmium (ATSDR 1999c). Once cadmium is absorbed, it is eliminated slowly; the biological half-life has been estimated at 10 to 30 years (FDA 1993).

Acute toxicity

Effects of acute oral exposure to cadmium include gastrointestinal irritation, nausea, vomiting, abdominal pain, cramps, salivation, and diarrhea. Lethal doses in humans caused massive fluid loss, edema, and widespread organ destruction. The ingested doses were 25 and 1,500 mg/kg (ATSDR 1999c; FDA 1993).

Chronic toxicity

Kidney toxicity is the main concern with cadmium exposure, with the critical effect being significant proteinuria (an indicator of kidney toxicity). The **RfD for cadmium in food was calculated to be 0.001 mg/kg-day** (IRIS). The RfD was calculated using a toxicokinetic model to determine the highest level of cadmium in the human renal cortex not associated with significant proteinuria (EPA 2000).

Cadmium causes many other types of toxic effects in addition to kidney toxicity, such as reducing the gastrointestinal uptake of iron, bone disorders, and increased calcium excretion. Some human studies have shown cardiovascular toxicity and elevated blood pressure, but the results are conflicting (ATSDR 1999c). In addition, animal studies indicate that cadmium causes a wide variety of alterations in the function of the immune system.

Carcinogenicity

No animal or human oral exposure studies suggest that cadmium is carcinogenic via the oral exposure route, although cadmium is classified as a probable human carcinogen (B1) by EPA based on inhalation studies in humans (EPA 2000). ATSDR has concluded that there is minimal evidence of an association between cadmium exposure and increased cancer risk in humans but that the statistical power of the studies examined to detect an effect was not high. They determined that neither the human nor the animal studies provided enough evidence to agree on the carcinogenic status of cadmium by the oral route (ATSDR 1999c).

24. CARBAZOLE

In its pure form, carbazole exists as clear or white crystals. Human exposure to carbazole typically occurs through smoking tobacco and inhaling polluted air. Exposure may also occur through drinking water containing traces of carbazole and

eating charbroiled food. Carbazole is released to the atmosphere in emissions from waste incineration, tobacco smoke, aluminum manufacturing, and rubber, petroleum, coal, and wood combustion. Workers may be exposed to carbazole and other anthracene derivatives via inhalation of vapors and dust and through dermal contact. Carbazole is naturally contained in coal, petroleum, and peat; indicating that it will be released to the environment from the incomplete combustion of these materials. If released to the atmosphere, vapor-phase carbazole is rapidly degraded by photochemically produced hydroxyl radicals leading to an estimated half-life of 3 hours (HSDB).

Pharmacokinetics

No human studies have been conducted. In rats and rabbits, carbazole has been shown to break down into 3-hydroxycarbazole (HSDB).

Acute toxicity

Acute health affects of carbazole on humans have not been studied. Animal studies did not show any acute effects (HSDB).

Chronic toxicity

Chronic effects of carbazole have not been determined in humans, but various types of tumors were observed in rats fed carbazole (HSDB).

Carcinogenicity

No epidemiological data relevant to the carcinogenicity of carbazole to humans are readily available. There is some limited evidence in experimental animals supporting the carcinogenicity of carbazole. Overall, IRAC states that carbazole is not classifiable as to its carcinogenicity to humans and is therefore a Group 3 chemical (HSDB). A HEAST **oral cancer slope factor of 0.02 mg/kg-day** is available for carbazole in the EPA Region 9 PRG table (EPA 2004).

25. CHLORDANE

Chlordane is an organochlorine insecticide comprised of the sum of cis- and trans-chlordane and trans-nonachlor and oxychlordane for purposes of health advisory development. First introduced in 1947, it was used extensively on agricultural crops, livestock, lawns, and for termite control. Because of concern over cancer risk, human exposure, and effects on wildlife, most uses were banned in 1978, and all uses were banned by 1988. Due to its long half-life and ability to concentrate in biological materials, it is still widely distributed in fish in the United States (EPA 2000).

Pharmacokinetics

Chlordane is extremely lipid soluble, and lipid partitioning of chlordane and its metabolites has been documented in both humans and animals. Chlordane is

metabolized via oxidation, which results in a number of metabolites that are very persistent in body fat. Human studies have found chlordane in pesticide applicators, residents of homes treated for termites, and those with no known exposures other than background (EPA 2000).

Acute toxicity

Chlordane is moderately to highly toxic with an estimated lethal dose to humans of 6 to 60 g (IRIS). Effects reported in humans after acute exposure include headaches, irritability, excitability, confusion, loss of coordination, seizures, and convulsions. There is also some evidence that acute exposures to chlordane may be associated with impaired immune function and aplastic anemia in humans (EPA 2000).

Chronic toxicity

IRIS provides an **RfD of 0.0005 mg/kg-day** based on a NOAEL of 0.15 mg/kg-day for hepatic necrosis in a 2-yr feeding study in mice (IRIS). The LOAEL in the principal study was 0.75 mg/kg-day.

Carcinogenicity

Chlordane is classified as a probable human carcinogen (B2) by EPA based on oral studies in animals. An increased incidence of hepatocellular carcinoma was observed in both sexes in mice in two separate studies using different strains. Hepatocellular carcinomas were also observed in another study in male mice using a third strain. The **oral cancer slope factor of 0.35 per mg/kg-day** is the geometric mean of the cancer potencies calculated from five data sets (IRIS).

26. CHROMIUM

Trivalent chromium is a naturally occurring chemical with low toxicity. Hexavalent chromium, however, is released into the environment through industrial emissions and is highly toxic due to its strong oxidation characteristics and membrane permeability. Hexavalent chromium is used in chromate manufacturing, ferrochromium industries, and in metal alloys (HSDB).

Pharmacokinetics

Trivalent chromium is an essential ion required for lipid, protein, and fat metabolism and to maintain normal glucose metabolism. The most common routes of exposure to toxic levels of chromium are through inhalation and ingestion (ToxFAQs).

Acute toxicity

The acute toxic effects of hexavalent chromium were studied in 1965 when 155 people were exposed to 20 mg/L hexavalent chromium in their drinking water. The victims suffered from mouth sores, diarrhea, stomachaches, indigestion, vomiting, increased white blood cell counts, and a higher per capita cancer rate. Acute exposure to

hexavalent chromium may also affect fetal development. Dermal exposure to hexavalent chromium can cause skin irritation and allergic contact dermatitis (IRIS).

Chronic toxicity

Chronic exposure to chromium can cause damage to the liver, kidney, and circulatory system, as well as cause nerve tissue damage and dermatitis (OGWDW). **EPA has developed RfDs of 1.5 and 0.003 mg/kg-day for trivalent and hexavalent chromium, respectively** (IRIS). The RfD for hexavalent chromium will be applied to all chromium data in this HHRA since the proportion of trivalent chromium in the total chromium measurements is not known.

Carcinogenicity

EPA has classified trivalent chromium as Group D, not classifiable as to human carcinogenicity. Hexavalent chromium is a Group A known human carcinogen via the inhalation pathway (IRIS). EPA has not developed an oral cancer potency factor for hexavalent chromium.

27. COPPER

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (Faust 1992). Copper can be absorbed by the oral, inhalation, and dermal routes of exposure.

Pharmacokinetics

Copper is an essential nutrient that is normally present in a wide variety of human tissues (Faust 1992). Copper is incorporated into more than a dozen specific copper proteins. Copper is essential for hemoglobin formation, carbohydrate metabolism, catecholamine biosynthesis, and cross-linking of collagen, elastin, and hair keratin (EPA 1987).

Acute toxicity

In humans, ingestion of gram quantities of copper salts may cause gastrointestinal, hepatic, and renal effects with symptoms such as severe abdominal pain, vomiting, diarrhea, hemolysis, hepatic necrosis, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, and death (Faust 1992). Acute inhalation exposure to copper dust or fumes at concentrations of 0.075 to 0.12 mg Cu/m^3 may cause metal fume fever with symptoms such as cough, chills, and muscle ache (Faust 1992). Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and increase in liver size.

Chronic toxicity

Gastrointestinal disturbances and liver toxicity have resulted from long-term exposure to drinking water containing 2.2 to 7.8 mg Cu/L (Faust 1992). The chronic toxicity of copper has been characterized in patients with Wilson's disease, a genetic disorder causing copper accumulation in tissues. Vineyard workers chronically exposed to Bordeaux mixture (copper sulfate and lime) exhibit degenerative changes of the lungs and liver. Dermal exposure to copper may cause contact dermatitis in some individuals (ATSDR 2004). Additionally, high levels of copper are known to cause kidney and liver damage (ATSDR 2004).

EPA has not developed an oral RfD for elemental copper. EPA's HEAST proposed a provisional value of **0.04 mg/kg-day** (EPA 2005). Provisional RfDs have greater uncertainty than RfDs certified by EPA.

Carcinogenicity

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of copper (Faust 1992). EPA has placed copper in weight-of-evidence group D, not classifiable as to human carcinogenicity.

28. DDT AND METABOLITES

DDT is an organochlorine pesticide that has not been marketed in the United States since 1972 but is ubiquitous due to its widespread use in previous decades and its relatively long half-life. DDT's close structural analogs, DDE and DDD, are metabolites of DDT and have also been formulated as pesticides in the past (EPA 2000). DDT is very widely distributed; it has been found in wildlife all over the world and in many human samples as well.

Although some use of DDT continues throughout the tropics, it remains of human health concern in the United States primarily due to its presence in water, soil, and food. Because individuals are typically exposed to a mixture of DDE, DDT, and DDD and their degradation and metabolic products, the sum of the 4,4' and 2,4' isomers of DDT, DDE, and DDD will be evaluated together in this HHRA.

Pharmacokinetics

DDT and its analogs are stored in fat, liver, kidney, and brain tissue; trace amounts can be found in all tissues (EPA 2000). DDE is stored more readily than DDT. DDT is eliminated through first-order reduction to DDD and, to a lesser extent, to DDE. The DDD is converted to more water-soluble bis(p-chlorophenyl)acetic acid, with a biological half-life of 1 year. DDE is eliminated much more slowly, with a biological half-life of 8 years. Because elimination occurs slowly, ongoing exposure may lead to an increase in the body burden over time.

Acute toxicity

The low effect dose for severe effects (acute pulmonary edema) in infants has been reported to be 150 mg/kg. In adults, behavioral effects were noted at 5 to 6 mg/kg and seizures at 16 mg/kg (HSDB). Evidence from acute exposure studies of dogs indicates that DDT may sensitize the myocardium to epinephrine. This was observed for both injected epinephrine and epinephrine released by the adrenal glands during a seizure and resulted in ventricular fibrillation. DDT may concurrently act on the CNS, in a manner similar to that of other halogenated hydrocarbons, to increase the likelihood of fibrillation. Chronic exposure to 10 mg/kg-day did not produce increased incidence of arrhythmias in rats or rabbits (EPA 2000).

DDD is considered less toxic than DDT in animals. Symptoms develop more slowly and have a longer duration with DDD than with DDT exposure. Lethargy is more significant and convulsions are less common than with DDT exposure (HSDB).

Chronic toxicity

Extensive research has been conducted on chronic and sub-chronic exposure effects of DDT in animals and in humans working with DDT. These studies have primarily focused on carcinogenic effects, which are discussed in the following section. Studies have also identified liver damage, and there is limited evidence that DDT may cause an increase in the number of white blood cells and decreased hemoglobin level (EPA 2000). Immunological effects have been associated with exposure to DDT.

IRIS lists an **oral RfD of 0.0005 mg/kg-day** for DDT based on liver effects with a NOAEL of 0.05 mg/kg-day from a 27-week rat feeding study conducted in 1950 (IRIS).

Carcinogenicity

DDE, DDT, and DDD are all considered probable human carcinogens (category B2) based on animal studies, with **oral cancer slope factors of 0.24, 0.34, and 0.34 per mg/kg-day**, respectively (IRIS). Liver tumors were associated with each chemical. The occupational studies of workers exposed to DDT are of insufficient duration to assess carcinogenicity (IRIS). Elevated leukemia incidence, particularly chronic lymphocytic leukemia, was noted in two studies of workers. Lung cancer has also been implicated in one study. Bone marrow cells in experimental animals have also been affected by exposure, including an increase in chromosomal fragments in the cells (HSDB). The **oral cancer slope factor for DDT (0.34) is used for total DDTs in this HHRA**, in accordance with EPA (2000) recommendations.

29. DIELDRIN

Dieldrin is an organochlorine pesticide that was phased out between 1974 and 1987. It continues to be detected nationwide due to its relatively long half-life. Dieldrin is also a product of aldrin metabolism (ATSDR 1999a).

Pharmacokinetics

Dieldrin is absorbed from the gastrointestinal tract and transported through the hepatic portal vein and the lymphatic system. Soon after ingestion, it is found in the liver, blood, stomach, and duodenum. Dieldrin is lipophilic and ultimately stored primarily in fat and tissues with lipid components. A correlation between exposure and dieldrin levels in human breast milk has been established, and placental transfer of dieldrin has been observed in women (ATSDR 1999a).

Acute toxicity

The following symptoms are commonly associated with exposure to organochlorines: behavioral changes, sensory and equilibrium disturbances, involuntary muscle activity, depression of vital centers, myocardial irritability, convulsion, and unconsciousness (EPA 2000). Additional effects of dieldrin exposure include: possible hematological effects in humans (ATSDR 1999a). The estimated human lethal dose is 65 mg/kg-day (EPA 2000).

Chronic toxicity

Liver toxicity has been observed in multiple animal studies and in human acute exposure episodes. Neurotoxicity has been observed in humans with chronic inhalation and dermal exposures (ATSDR 1999a). Chronic exposures of pesticide applicators to dieldrin led to idiopathic epilepsy, which ceased when exposure was terminated (EPA 2000).

IRIS provides an **RfD of 0.00005 mg/kg-day for dieldrin** based on a NOEL of 0.005 mg/kg-day from a 1969 2-year rat feeding study that found liver lesions.

Carcinogenicity

Dieldrin is classified as a probable human carcinogen (Group B2) by EPA based on oral studies in animals. EPA has developed an **oral cancer slope factor of 16 per mg/kg-day for dieldrin**. ATSDR has concluded, based on studies that have been reviewed, that dieldrin is probably a tumor promoter. Varieties of tumor types have been observed in animal studies including pulmonary, lymphoid, thyroid, and adrenal (ATSDR 1999a). In addition, dieldrin has recently been observed to have estrogenic effects on human breast cancer estrogen-sensitive cells, and it may cause disruption of the endocrine system due to its estrogenic activity (Soto et al. 1994).

30. ENDRIN AND ENDRIN ALDEHYDE

Endrin is a solid, white, nearly odorless substance that was used in the past as a pesticide to control insects, rodents, and birds. Endrin has not been produced or sold for general use in the United States since 1986. Endrin aldehyde is a minor impurity of the pesticide endrin, which is no longer produced. The production and use of endrin may have resulted in endrin aldehyde's release to the environment through direct release or from various waste streams. Little is known about the properties of endrin

aldehyde (an impurity and breakdown product of endrin) or endrin ketone (a product of endrin when it is exposed to light) (ToxFAQs).

Pharmacokinetics

Endrin can be absorbed by any route. Endrin is metabolized relatively rapidly by the body. Following absorption, the primary target of action is the central nervous system (CNS). Both endrin and its water-soluble metabolites are excreted in urine and feces. Small amounts of the chemical may remain in the fatty tissues of the body (HSDB).

Acute Toxicity

Following exposure to endrin, CNS excitation and convulsions may occur. Respiratory depression may occur concurrently with convulsions, and respiratory failure is the most common cause of death from endrin poisoning. Symptoms in less-severe cases of endrin poisoning may include headache, dizziness, leg weakness, abdominal discomfort, nausea, vomiting, insomnia, agitation, and, occasionally, slight mental confusion. Buildup of fluid in the lungs and kidney damage has been reported following endrin ingestion. Symptoms including headache, dizziness, weakness, lethargy, and weight loss may persist for 2 to 4 weeks (HSDB).

Chronic Toxicity

Enlargement of the liver commonly occurs. Liver enzymes have been induced with occupational exposure. Anorexia, fatigue, and malaise have occurred with chronic exposure to organochlorine insecticides (HSDB). EPA has developed an **RfD of 0.0003 mg/kg-day for endrin** based on animal studies (IRIS).

Carcinogenicity

IARC has assigned a Classification D (not classifiable as to carcinogenicity in humans) for endrin. Animal studies failed to conclusively show carcinogenic effects, and inadequate human exposure data exists to determine carcinogenicity of this compound. Because so little is known about endrin aldehyde, it has been grouped with other organochlorine pesticides and may be assumed to be a class B2 carcinogen (IARC) and therefore a possible human carcinogen. However, there is limited animal evidence and no human studies to support this classification (HSDB and ToxFAQs).

31. GAMMA-BHC (LINDANE) AND METABOLITES (ALPHA-BHC AND BETA-BHC)

Lindane is an organochlorine pesticide that is comprised of isomers of hexachlorocyclohexane (BHC), with the gamma isomer constituting the major (> 99%) component. There appears to be some difference in toxicity of the various hexachlorocyclohexane isomers (EPA 2000). Lindane is used primarily for controlling wood-inhabiting beetles and as a seed treatment. Lindane is also used as a prescription pharmaceutical to control head lice and mites (scabies) in humans.

Pharmacokinetics

Lindane is readily absorbed by the gastrointestinal tract following oral exposure. Distribution is primarily to the adipose tissue but also to the brain, kidney, muscle, spleen, adrenal glands, heart, lungs, blood, and other organs. It is excreted primarily through urine as chlorophenols. The epoxide metabolite may be responsible for carcinogenic and mutagenic effects (EPA 2000). Male exposure to lindane through the environment results in accumulation in testes and semen in addition to the tissues listed above (ATSDR 2005c).

Acute toxicity

The estimated human lethal dose is 125 mg/kg (HSDB). Occupational and accidental exposures in humans have resulted in headaches, vertigo, abnormal EEG patterns, seizures, and convulsions. Death has occurred primarily in children.

Chronic toxicity

Liver damage has been observed in many animal studies and appears to be the most sensitive effect (EPA 2000). Immune system effects have been observed in humans exposed via inhalation and in orally dosed animals. A 5-week study in rabbits found immunosuppression at 1 mg/kg-day (ATSDR 2005c). IRIS provides an **RfD for lindane of 0.0003 mg/kg-day** based on a NOAEL of 0.33 mg/kg-day from a sub-chronic rat study that found liver and kidney toxicity at higher doses (IRIS).

NCEA provides RfDs for lindane's related isomers. The **RfD for alpha-BHC is 0.0005 mg/kg-day** and the **RfD for beta-BHC is 0.0002 mg/kg-day** (EPA 2004). Liver effects are a known result of exposure to both alpha-BHC and beta-BHC (ATSDR 2005c).

Most observed effects in humans exposed accidentally to lindane are neurological. Behavioral effects have also been noted in many studies on experimental animals, and at relatively high levels seizures were reported. More subtle behavioral effects were noted at an LOAEL of 2.5 mg/kg-day with 40 days of exposure in rats. No NOAEL was reported (ATSDR 2005c).

Carcinogenicity

Lindane has been classified as Group B2 (probable human carcinogen) (EPA 2000) and a HEAST **oral cancer slope factor of 1.3 per mg/kg-day** has been listed (EPA 2005). Lindane's related isomers, alpha and beta hexachlorocyclohexane, are classified as probable human carcinogens and have cancer potencies similar to that of lindane: **6.3 per mg/kg-day for alpha-BHC and 1.8 per mg/kg-day for beta-BHC** (IRIS). In addition to tumors identified in experimental animals, human study data indicate that this chemical may cause aplastic anemia (EPA 2000).

32. HEPTACHLOR

Heptachlor is a synthetic chemical that was used in the past for killing insects in homes, buildings, and on food crops. Heptachlor is both a breakdown product and a component of the pesticide chlordane (approximately 10% by weight). Pure heptachlor is a white powder. Technical-grade heptachlor is a tan powder. Heptachlor may be found in the soil or air of homes treated for termites, dissolved in surface water or groundwater, or in the air near hazardous waste sites. Heptachlor is still approved by EPA for killing fire ants in power transformers.

Pharmacokinetics

Approximately 20% of heptachlor is changed within hours into heptachlor epoxide in the environment and in your body. Heptachlor has been shown to bioaccumulate in fish and cattle. People store heptachlor epoxide in their fatty tissue. Some studies show that heptachlor epoxide can still be measured in fatty tissue 3 years after a person is exposed (ToxFAQs). Most of the heptachlor that is swallowed is absorbed into blood. Heptachlor can pass directly from a mother's blood to an unborn baby through the placenta.

Acute toxicity

Blood tests suggest that heptachlor may cause mild liver changes in humans. A few human cases show that breathing pesticide mixtures containing heptachlor may affect the nervous system causing dizziness, fainting, or convulsions (ToxFAQs). Studies of people who made or used pesticides that included heptachlor found no serious health effects. Acute toxicity studies with animals indicate that heptachlor can cause tremors, convulsions, and loss of kidney function at high doses.

Chronic toxicity

Sub-chronic dietary studies with mice resulted in liver and adrenal gland damage. Animals that ate food containing heptachlor before and/or during pregnancy had smaller litters (ToxFAQs). EPA has established an **RfD of 0.0005 mg/kg-day** for heptachlor based on a 2-yr rat feeding study that documented increased liver weight in males (IRIS).

Carcinogenicity

Animals fed heptachlor throughout their lifetime had more liver tumors than animals that ate food without heptachlor. EPA has classified heptachlor as a probable human carcinogen (B2) and established an **oral cancer slope factor of 4.5 per mg/kg-day** (IRIS).

33. HEPTACHLOR EPOXIDE

Heptachlor epoxide is a breakdown product of the organochlorine pesticides heptachlor and chlordane and is a contaminant of both products. It is more toxic than

either parent compound (ATSDR 2005b). Although most uses of heptachlor were suspended in 1978 and chlordane was removed from the market in 1988 (EPA 2000), heptachlor epoxide continues to be a widespread contaminant due to its relatively long half-life.

Pharmacokinetics

Based upon animal and limited human data, heptachlor epoxide is absorbed through the gastrointestinal tract and is found primarily in the liver, bone marrow, brain, and fat, although it is distributed widely to other tissues as well. It is stored primarily in fat. Fetal blood levels were approximately four times those measured in women.

Heptachlor epoxide has a very long half-life, particularly in adipose tissue. Human tissue levels have correlated well to age, with 97 percent of North Texas residents tested (ages 41 to 60) having measurable levels. Based on the Texas study, heptachlor epoxide tissue levels have not decreased appreciably since the 1960s (EPA 2000).

Acute toxicity

The LD50s for heptachlor epoxide range from 40 to 162 mg/kg in rodents (EPA 2000).

Chronic toxicity

IRIS provides an **RfD of 0.000013 mg/kg-day** based on an LOAEL of 0.0125 mg/kg-day from a 60-week dog feeding study reported in 1958. The critical effect was increased liver-to-body-weight ratios in both males and females at the lowest dose tested.

Animal studies have identified the following effects associated with heptachlor (and subsequently heptachlor epoxide via metabolism) or heptachlor epoxide directly: elevated bilirubin and white blood cell count, increased serum creatinine phosphokinase levels suggestive of muscle damage, muscle spasms secondary to CNS stimulation, adrenal gland pathology, and neurological disorders (EPA 2000).

Carcinogenicity

Heptachlor epoxide is classified as a probable human carcinogen (category B2) by EPA based on oral studies in animals. The **oral cancer slope factor is 9.1 per mg/kg-day** (IRIS). This value is based on the geometric mean of several studies that identified liver carcinomas.

Heptachlor (and consequently heptachlor epoxide) exposures have been associated with brain tumors in children exposed prenatally. Multiple chromosomal abnormalities were also identified in the tumor cells. It was not determined whether the effects were caused by environmental or familial factors (EPA 2000).

34. HEXACHLOROBENZENE

Hexachlorobenzene is a byproduct of manufacturing and in the past it has been used as a fungicide seed protectant. At ambient temperatures, it exists as a solid, and in aquatic environments, it is found in higher quantities in sediment than water due to its low solubility (ATSDR 2002).

Pharmacokinetics

Hexachlorobenzene is persistent in the body due to its lipophilic nature. It is found in human breast milk (ATSDR 2002), which may be a significant route of exposure for young children.

Acute toxicity

The following symptoms are commonly associated with exposure to organochlorines: behavioral changes, sensory and equilibrium disturbances, involuntary muscle activity, depression of vital centers, myocardial irritability, convulsion, and unconsciousness (EPA 2000). Acute exposure studies in animals have demonstrated a low acute toxicity for hexachlorobenzene with LD50s between 1,700 and 4,000 mg/kg. Based on animal studies, the following systems are negatively affected following acute exposure: liver, kidney, hematological, and dermal (ATSDR 2002).

Chronic toxicity

A large number of people in Turkey were exposed from 1955 to 1959 to grain contaminated with hexachlorobenzene. Precise exposure estimates are not available, but it was estimated that exposure levels of 0.7 to 2.9 mg/kg-day for a 70-kg individual occurred (ATSDR 2002). The following effects were associated with this exposure: shortening of the digits due to osteoporosis, painless arthritis, muscle weakness, rigidity and sensory shading, thyroid enlargement, and histopathological changes in the liver often accompanied by skin lesions (ATSDR 2002). These effects have also been observed in numerous animal studies.

Based on animal studies, the hepatic system appears to be the most sensitive systemic endpoint for hexachlorobenzene exposure. The results from these studies have been converted by EPA to an **RfD of 0.0008 mg/kg-day** in the IRIS database.

Carcinogenicity

Carcinogenic assays of hexachlorobenzene in animals have identified an increased incidence of multiple tumor types. Hexachlorobenzene is classified as a possible human carcinogen (B2) based on the results of animal studies (EPA 2000). EPA has established an **oral cancer slope factor of 1.6 per mg/kg-day** (IRIS). Follow-up studies of the exposure of hexachlorobenzene to the victims in Turkey have not identified cancers in the 25-year or 20- to 30-year exposure cohorts. However, ATSDR notes that the enlarged thyroids noted in members of these cohorts have not been adequately investigated (ATSDR 2002).

35. HEXACHLOROBUTADIENE

Hexachlorobutadiene, also known as HCBD, is formed during the processing of other chemicals such as tetrachloroethylene, trichloroethylene, and carbon tetrachloride. Hexachlorobutadiene is an intermediate in the manufacture of rubber compounds and lubricants. It is used as a fluid for gyroscopes, a heat transfer liquid, or a hydraulic fluid. Outside of the United States it is used to kill soil pests (ToxFAQs).

Pharmacokinetics

In animal studies, most of the hexachlorobutadiene is metabolized into more toxic compounds. It is not known how rapidly hexachlorobutadiene and its breakdown products are removed from your body through your urine and feces. Some is expected to remain in your body fat for long periods (ToxFAQs).

Acute toxicity

Ingestion of hexachlorobutadiene damaged the kidneys of rats and mice and, to a lesser extent, the liver of rats. These effects occurred after both short- and long-term exposures at very low dose levels. Young rats were affected more than adult rats. The kidneys of female rats appeared to be affected more than those of males. On the other hand, the liver of male rats was affected, but the liver of female rats was not. It is not clear if the differences between the sexes might be seen in humans. Kidney, brain, and liver damage were also seen in rabbits after contact of their skin with the compound for a short period.

Chronic toxicity

Hexachlorobutadiene was shown to affect the function of the liver in one study of workers at a solvent production plant who breathed hexachlorobutadiene for long periods. Hexachlorobutadiene decreased fetal body weight in rats, but did not affect fetal development or impair their ability to produce offspring. The lungs, heart, brain, blood, muscles, and skeleton in rats or mice were not damaged after long-term exposure. HEAST developed an **RfD of 0.0002 mg/kg-day for hexachlorobutadiene** (EPA 2005).

Carcinogenicity

Studies in rats indicate that hexachlorobutadiene may increase the risk of kidney cancer if exposures occur for long periods. EPA has classified hexachlorobutadiene as a possible human carcinogen (C) based on observations of renal neoplasms in male and female rats in one study. EPA established an **oral cancer slope factor of 0.078 per mg/kg-day** (IRIS).

36. HEXACHLOROCYCLOPENTADIENE (HCCPD)

HCCPD is a manufactured chemical that does not exist naturally in the environment. It is a light, lemon-yellow liquid with a sharp musty odor. It easily evaporates into the

air where the vapor looks like a blue haze. HCCPD is a component used in the manufacture of certain pesticides. In addition to the use as a pesticide, it is also used to make flame retardants, resins that won't burn, shock-proof plastics, esters, ketones, fluorocarbons, and dyes. HCCPD most commonly enters the environment during its production and disposal. When HCCPD becomes airborne, it is broken down quickly by sunlight and reactions with other chemicals. HCCPD doesn't dissolve readily in water, and will evaporate from the surface. About half the HCCPD in water will be changed to other chemicals by light in only 4 minutes. HCCPD that gets into soil binds to decaying plant and animal matter. If the soil is sandy, HCCPD can move through it to reach underground water. About half of the HCCPD in the soil will be changed to other chemicals by bacteria in 1 to 2 weeks. Small amounts of HCCPD can accumulate in fish (ToxFAQs).

Pharmacokinetics

HCCPD primarily targets the tissues that line the lungs, regardless of the exposure route. Cough, dyspnea, and chest discomfort have been reported in exposed humans. Experimental animals have developed pulmonary edema, pulmonary hemorrhages, and necrotizing bronchitis and bronchiolitis.

Acute Toxicity

HCCPD is highly irritating and corrosive to tissues. Exposure effects may include cough, dyspnea, chest discomfort, headache, dizziness and burns. Proteinuria and elevated levels of liver enzymes may occur. Pulmonary damage may range from bronchitis, chemical pneumonitis, bronchiolitis, and pulmonary edema to respiratory failure. Degenerative changes of the brain, heart, liver (elevations in liver enzymes), adrenals, and kidneys have been reported (HSDB).

Chronic Toxicity

Long term exposure has not been studied in humans. However, sub-chronic or pre-chronic exposure to HCCPD vapors caused lacrimation, salivation, gasping, and at high concentrations, tremors in laboratory animals. Diffuse degenerative changes were observed in the brain, heart, liver, adrenal glands, and kidneys. Severe pulmonary edema and acute necrotizing bronchitis and bronchiolitis demonstrated the severity of irritation with incidence and severity dependent on dose. During testing on animal models, adverse effects were observed at the lowest exposure concentration (0.15 ppm) (HSDB). Due to the lack of available human data, EPA has developed an **RfD of 0.006 mg/kg-day** for HCCPD based on animal studies (IRIS).

Carcinogenicity

IARC has given HCCPD a classification D status, meaning that it is not classifiable as to human carcinogenicity. This ruling is based on inadequate data in humans and animals concerning carcinogenicity of HCCPD (HSDB and ToxFAQs).

37. HEXACHLOROETHANE

Hexachloroethane is a colorless solid that gradually evaporates when it is exposed to air. It is also called HCE, perchloroethane, and carbon hexachloride. Its vapors smell like camphor. In the United States, about half of the hexachloroethane is used by the military for smoke-producing devices. It is also used to remove air bubbles in melted aluminum. Hexachloroethane may be present as an ingredient in some fungicides, insecticides, lubricants, and plastics. Hexachloroethane does not occur naturally in the environment. It is no longer made in the United States, but it is formed as a byproduct in the production of some chemicals. Some hexachloroethane can be formed by incinerators when materials containing chlorinated hydrocarbons are burned. Hexachloroethane itself does not catch fire easily. Some hexachloroethane can also be formed when chlorine reacts with carbon compounds in drinking water. Hexachloroethane can be released to the environment during its production, use, transport, or disposal. In air, hexachloroethane does not break down to other compounds. Some hexachloroethane that is in lakes or streams and surface soils will evaporate into the air. Microscopic organisms can break it down more easily in an anaerobic environment (without oxygen) than with oxygen. Hexachloroethane does not appear to build up in plants or animals used for food (ToxFAQs).

Pharmacokinetics

Following absorption, hexacloroethane is distributed systemically, but tends to accumulate in fat. Metabolism of hexachloroethane in animals has been shown to be rather extensive. Multiple chlorine atoms are removed by the cytochrome P450 complex, and the resulting metabolites, primarily trichloroethanol or trichloroacetic acid, have been shown to be excreted in urine. The half life in mice is thought to be 2.5 days (HSDB).

Acute Toxicity

Exposure has caused eye irritation, lacrimation and redness, but no lasting damage. Hexachloroethane is also irritating to the skin and may act as a central nervous system depressant (HSDB).

Chronic Toxicity

Hexachloroethane is not a very toxic substance. Significant exposure over a long period of time may cause damage to the liver and kidneys. Animal studies have not shown hexachloroethane to cause birth defects or to affect reproduction (ToxFAQs). EPA has developed an **RfD of 0.001 mg/kg-day** for HCCPD based on animal studies (IRIS).

Carcinogenicity

There is inadequate evidence in humans for the carcinogenicity of hexachloroethane. Because there is sufficient evidence in experimental animals for the carcinogenicity of

hexachloroethane, IARC has given it a classification of group 2B, and considers it to be a possible carcinogen to humans (HSDB). EPA developed an **oral cancer slope factor of 0.014 per mg/kg-day** for hexachloroethane, and ranks the chemical in group C, a possible human carcinogen because cancer was observed in one strain of mice following exposure to hexachloroethane (IRIS).

38. IRON

Iron is the second most abundant metal in the earth's crust. The most common iron ores include hematite, magnetite, limonite, and siderite (HSDB). Iron salts are used as fertilizer micronutrients, herbicides, electrolytes in dry cell batteries, animal feed additives, galvanizers, and as emulsion breakers. The major route of exposure to iron is through the mining and handling of iron ores (HSDB).

Pharmacokinetics

Iron is found naturally in the body as an important component of hemoglobin. In overdoses (>20 mg/kg-day), iron may be absorbed into the body may be extremely fast, where it is incorporated into structural proteins (Spanierman 2001). Excretion may be extremely slow in these cases.

Acute toxicity

Acute iron toxicity is the main cause of pediatric poisoning death in the United States. The hallmark feature of iron overdose is gastrointestinal bleeding. Iron is an extremely corrosive substance in the gastrointestinal tract. The absorption of excessive quantities of ingested iron will result in systemic iron toxicity. Severe overdose causes impaired mitochondrial dysfunction, which can result in cellular death. One of the most affected organs is the liver, but other organs, such as the heart, kidneys, lungs and the hematologic systems may be impaired (Spanierman 2001).

Chronic toxicity

Chronic exposure to iron oxide fume or dust can cause the appearance of a pulmonary condition called siderosis. This is considered a benign condition and does not ordinarily cause significant physiologic impairments (HSDB). Iron is also suspected to be a cardiovascular or blood toxicant, gastrointestinal or liver toxicant, neurotoxicant, and respiratory toxicant (HSDB). EPA's NCEA has developed a **provisional RfD for iron of 0.3 mg/kg-day** (EPA 2005). Provisional RfDs have greater uncertainty than RfDs published by EPA in the IRIS database.

Carcinogenicity

At this time, there is no information regarding the carcinogenicity of iron to humans or animals.

39. LEAD

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. Lead's most important industrial use is in the production of some types of batteries. It is also used in the production of ammunition, in some kinds of metal products (such as sheet lead, solder, some brass and bronze products, and pipes), and in ceramic glazes. Human activities (such as the former use of "leaded" gasoline) have spread lead and substances that contain lead to all parts of the environment. Before the use of leaded gasoline was banned, most of the lead released into the US environment came from car exhaust. Other sources of lead released to the air include burning fuel, such as coal or oil, industrial processes, and burning solid waste.

Sources of lead in dust and soil include lead that falls to the ground from the air, and weathering and chipping of lead-based paint from buildings and other structures. Lead in dust may also come from windblown soil. Disposal of lead in municipal and hazardous waste dump sites may also add lead to soil. Mining wastes that have been used for sandlots, driveways, and roadbeds can also be sources of lead (ATSDR 1999d).

People living near hazardous waste sites may be exposed to lead and chemicals that contain lead by breathing air, drinking water, eating foods, or swallowing or touching dust or dirt that contains lead. For people who do not live near hazardous waste sites, exposure to lead may occur in several ways: 1) by eating foods or drinking water that contain lead, 2) by spending time in areas where leaded paints have been used and are deteriorating, 3) by working in jobs where lead is used, 4) by using health-care products or folk remedies that contain lead, and 5) by having hobbies in which lead may be used such as sculpturing (lead solder) and staining glass.

Pharmacokinetics

Absorbed lead is distributed in various tissue compartments.

Acute toxicity

Lead can affect almost every organ and system in your body. The most sensitive is the central nervous system, particularly in children. Studies have shown that children exposed to low levels of lead have lower IQs, reduced motor skills, developmental problems, hyperactivity, and increased aggression (Canfield et al. 2003; Pattee and Pain 2003).

Lead also damages kidneys and the reproductive system. The toxic effects of lead are the same regardless of the route of entry into the body, and they are correlated with internal exposure as blood lead level.

Chronic toxicity

At high levels over long periods of time, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect the memory. Lead may

cause anemia, a disorder of the blood. It can also damage the male reproductive system. Even low levels of exposure to lead may have significant effects.

Since most of the toxicity data for lead is based on an internal dose, a reference dose, which is based on an external dose (i.e., mg/kg-day) has not been developed. Data on external exposure (i.e., mg/kg-day) are available from animal studies, but these data are generally not used to assess human health impacts because of the large database available using blood levels. Risks from lead exposure were evaluated using the IEUBK model for young children and the adult lead model for risks to fetal development, as described in Section B.3.4.4. EPA and the Centers for Disease Control and Prevention have determined that child or fetal blood lead concentrations at or above 10 µg/dL present risks to children's health.

Carcinogenicity

The Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably be anticipated to be carcinogens based on studies in animals. There is inadequate information to clearly determine lead's carcinogenicity in people (ToxFAQs).

40. MANGANESE

Manganese is an element considered essential to human health. However, divalent manganese is about 2.5 to 3 times more toxic than trivalent manganese, and the anions of manganese salts influence the overall manganese toxicity. Industrial activities which use manganese include steel manufacturing, nonferrous alloys, purifying and scavenging agent in metal production, manufacturing of aluminum, ceramics, matches, glass, and welding rods (HSDB).

Pharmacokinetics

Humans ingest manganese from three main sources: diet, drinking water, and inhaled particles. Manganese that is inhaled is mostly brought up from the respiratory tract by ciliary action and swallowed, eventually being absorbed in the gastrointestinal tract (Clayton and Clayton 1981). After oral exposure, absorbed manganese is quickly eliminated from blood and distributed mainly to the liver, kidneys, and endocrine glands. Minor amounts go to the brain and bone as shown in studies using mice, rats and monkeys.

Acute toxicity

Acute manganese poisoning has effects similar to other heavy metals if dust or fumes are inhaled in sufficient quantity. The minimum dose that produces effects on the central nervous system is not known and, with few exceptions, such effects have been observed only in occupationally exposed individuals. Sixteen cases of manganese poisoning have been described for a small Japanese community, three of which were fatal (including one suicide). The manganese content of the water was about 14 mg/L

and concentrations of about 8 and 11 mg/L were found in two other wells. The subjects exhibited psychological and neurological disorders associated with manganese poisoning and high manganese and zinc levels were found in organs at autopsy (WHO 1981).

Chronic toxicity

The usual form of chronic manganese poisoning primarily involves the central nervous system. Early symptoms include, languor, sleepiness, and weakness in legs, emotional disturbances such as uncontrollable laughter and a tendency to fall while walking (ACGIH 1986). Long-term exposure to manganese is known to cause a condition with symptoms that are similar to Parkinson's disease and are debilitating and permanent. Exposure to this metal has also been linked to reproductive problems and reduced red and white blood cell counts (ATSDR 2000a).

Experimental studies have suggested that populations at greatest risk of adverse effects due to manganese exposure are the very young and those with an iron deficiency, and workers exposed to manganese at or near the recommended threshold limit value. EPA has established an **RfD of 0.14 mg/kg-day** for a 70 kg adult (IRIS).

Carcinogenicity

Manganese is not classified as a carcinogen to humans (a class D chemical) because existing studies are inadequate to assess the carcinogenicity of manganese to humans and animals (ToxFAQs).

41. MERCURY

Mercury is widely distributed in the environment due to both natural and anthropogenic processes. It is released generally as elemental mercury (Hg^0) or divalent mercury (Hg^{2+}). It can be converted between these forms and may form mercury compounds by chemical processes in air, water, and soil. Biological processes in other media, primarily soil and sediment, can convert inorganic mercury into organic mercury, primarily methylmercury. In fish tissue, the majority of mercury is in the form of methylmercury (EPA 2000).

Pharmacokinetics

Methylmercury is rapidly and nearly completely absorbed; estimates of absorption efficiency are 90 percent or greater (ATSDR 1999e; EPA 1997c; WHO 1990). Methylmercury is readily distributed to all tissues following absorption from the gastrointestinal tract. Methylmercury in the body is considered to be relatively stable and is only slowly demethylated to form mercuric mercury. Estimates for the half-life of methylmercury in the body range from 44 to 80 days (EPA 1997c).

Methylmercury binds readily to protein and can be found throughout fish tissue. A substantial portion of the mercury in fish can be found in trimmed filets, making it difficult to reduce exposure by trimming fat and skin prior to cooking (EPA 2000).

Acute toxicity

Acute high-level exposures to methylmercury may result in kidney damage and failure, gastrointestinal damage, cardiovascular collapse, shock, and death. The estimated lethal dose is 10 to 60 mg/kg-day (ATSDR 1999e).

Chronic toxicity

Neurotoxicity is the chronic effect of greatest concern, both to the developing embryo or fetus and to adults and children (EPA 2000). Neurotoxicological effects include tremors, decreased IQ, and decreased motor function. In addition, damage to the liver and kidney can occur with chronic exposure (ATSDR 1999e). Effects to humans from consumption of contaminated food have been documented in Japan and Iraq.

The current IRIS **RfD for methylmercury of 0.0001 mg/kg-day** was originally based on data on neurological changes in 81 Iraqi children who had been exposed *in utero*. This value was subsequently updated using data from a population in the Faroe Islands who were exposed to methylmercury and PCBs through consumption of fish and pilot whale. In deriving the RfD, EPA used a benchmark dose (BMD) approach to quantify a dose-effect relationship between methylmercury in cord blood and a neurological endpoint. A BMD limit of 58 µg/L cord blood was estimated based on findings from the Boston Naming Test, a neuropsychological evaluation. A methylmercury intake level associated with a blood level of 58 µg/L was then calculated to be 1.0 µg/kg-day. The current RfD of 0.1 µg/kg-day (i.e., 0.0001 mg/kg-day) derived from the Faroe Islands data, is thus unchanged from the previous RfD derived from the Iraqi data. The RfD for methylmercury is used for mercury in this HHRA.

Carcinogenicity

Methylmercury is currently a Class C chemical, a possible carcinogen based on inadequate data in humans and limited evidence in animals. Dietary exposure of mice to methylmercury resulted in significant increases in the incidences of kidney tumors in males but not in females (EPA 1997c). Evidence points to a mode of action for methylmercury carcinogenicity that operates at high doses certain to produce other types of toxicity in humans. Given the relatively low levels of exposure, even among consumers of highly contaminated fish, methylmercury is not likely to present a carcinogenic risk to the US population (EPA 2000). An oral slope factor is currently not available for methylmercury.

42. MOLYBDENUM

Molybdenum occurs as a dark-gray or black powder with metallic luster or as a mass of silver-white color. In nature, molybdenum occurs in small amounts in the earth's crust (HSDB). Common industrial activities in which exposure to soluble molybdenum compounds may occur include molybdenum steel processing, welding operations, petroleum refining, manufacture of corrosion inhibitors, application of agricultural chemicals, and electroplating processes (HSDB).

Pharmacokinetics

While the mechanism of molybdenum toxicity is not yet fully understood, it is assumed that the primary factor is the formation of a copper-containing complex in the gastrointestinal tract which reduces the body's ability to utilize copper. Molybdenum has been found to accumulate in large amounts in the liver, and another study found a high accumulation in the lungs, spleen, and heart of rats exposed to chronic inhalation. In the blood stream, molybdenum binds to red blood cells and plasma proteins. Molybdenum is excreted rapidly as a molybdate, and excesses may also be excreted by the bile (HSDB).

Acute toxicity

Most molybdenum toxicity studies have been conducted over an extended time period, and thus little acute toxicity is available. Due to the relatively lower toxicity of molybdenum as compared to most metals, a massive dose would be required to cause harmful effects in humans (IMOA 2006). Exposure to molybdenum has been reported to cause diarrhea in animals such as cows (HSDB).

Chronic toxicity

Chronic exposure of humans to molybdenum may result in increased uric acid levels, gout-like symptoms, and anemia. Animal studies also reported damage to the kidney and liver, as well as impaired reproductive functions and sterility (HSDB). EPA's IRIS has developed an **RfD of 0.005 mg/kg-day** for molybdenum based on elevated uric acid levels in humans who were exposed to the chemical.

Carcinogenicity

No information is available regarding the carcinogenicity of molybdenum to humans or animals (HSDB, IRIS).

43. NICKEL

Nickel is used in a wide variety of industries. Occupational exposure is the predominant cause of harmful exposure to nickel.

Pharmacokinetics

Nickel is toxic to the liver in animals and is shown to affect kidney function in humans. It binds to tissues within the kidney and reduces their ability to function. Divalent nickel ions can penetrate the skin through openings at sweat ducts and hair follicles. The ions then bind with keratin and cause contact dermatitis. Nickel has a biological half-life of 20 to 34 hours in plasma and 17 to 39 hours in urine (HSDB).

Acute toxicity

Dermal contact with nickel causes contact dermatitis. Nickel poisoning occurred in 23 dialyzed patients when nickel leached in dialysate from a nickel-plated stainless steel

water heater. The victims experienced nausea, vomiting, weakness, headache, and palpitation (HSDB).

Chronic toxicity

Nasal and lung cancer have resulted from chronic inhalation of nickel particles (IRIS). Damage to the nasal mucosa, asthma, pneumoconiosis, and conjunctivitis have also been observed after long term exposure (HSDB). EPA has developed an **RfD of 0.02 mg/kg-day for nickel** based on decreased body and organ weights in a long-term rat feeding study (IRIS).

Carcinogenicity

EPA has classified nickel refinery dust as a known (Class A) carcinogen, but the soluble salts of nickel on which the oral RfD is based are not classified as carcinogenic. This classification was based on a study of sulfide nickel matte refinery workers who developed lung and nasal tumors after being exposed to nickel refinery dust, and also on data collected from nickel carcinogenicity studies with rats (IRIS).

44. NITROBENZENE

Nitrobenzene is an industrial chemical. It is an oily yellow liquid with an almond-like odor. It is produced in large quantities for use in industry. Most of the nitrobenzene produced in the United States is used to manufacture a chemical called aniline. Nitrobenzene is also used to produce lubricating oils such as those used in motors and machinery. A small amount of nitrobenzene is used in the manufacture of dyes, drugs, pesticides, and synthetic rubber. Very small amounts of nitrobenzene may be found in the air, and although nitrobenzene dissolves only slightly in water it will evaporate to air. It may be present in water from industrial releases. In water, nitrobenzene will be broken down by sunlight. Nitrobenzene in soil can move into the groundwater, be taken up by plants, evaporate to the air, and be broken down by bacteria. It does not appear to concentrate in fish or other aquatic animals (ToxFAQs).

Pharmacokinetics

Nitrobenzene is metabolized via two main pathways that yield aniline and nitrophenols. All metabolites were observed to be excreted through urine in lab animals with a half life of approximately 2 days (HSDB).

Acute toxicity

Nitrobenzene may be toxic by all routes of exposure, depending on dose; usual routes of exposure are inhalation of vapor and skin contact with vapor or liquid. The mean adult lethal oral dose is estimated to be about 1 to 5 grams. Alcohol ingestion may worsen the effects. Symptoms of exposure may include eye and skin irritation and methemoglobinemia, associated with headache, cyanosis, weakness, dizziness, confusion, rapid heart rate, labored breathing, chest pain, nausea and vomiting, and

coma. These are usually delayed in onset for up to 1 to 4 hours. A bitter almond odor may be present in urine or vomit, which suggests cyanide poisoning, but cyanide produces symptoms much more rapidly than nitrobenzene (HSDB).

Chronic toxicity

Chronic exposure may produce liver toxicity (HSDB). EPA has developed an **RfD of 0.0005 mg/kg-day** for Nitrobenzene based on animal studies (IRIS).

Carcinogenicity

No studies are available on whether nitrobenzene causes cancer in people. In animals, breathing nitrobenzene resulted in an increase in liver, thyroid, and kidney tumors. IARC has determined that nitrobenzene is a group 2B chemical, and is possibly carcinogenic to humans. However, EPA's IRIS states that nitrobenzene is not classifiable as to human carcinogenicity, and is a group D chemical. Both sources state that insufficient evidence exists to fully evaluate the carcinogenicity of this compound (ToxFAQs, IRIS).

45. N-NITROSODIMETHYLAMINE

N-nitrosodimethylamine is not currently used in industrial processes, except for research purposes where it may be released to the environment with laboratory waste. It was once used as an antioxidant, additive for lubricant, as a softener of copolymers, and in the production and use of rocket fuels (HSDB).

Pharmacokinetics

N-nitrosodimethylamine is absorbed from gastrointestinal tract and lung; skin absorption is slow. When administered to rats, mice, and rabbits, it is distributed uniformly in tissue and has a half-life of approximately 4 hr. Although the liver is the main organ concerned with its metabolism and is a site of selective toxicity, N-nitrosodimethylamine does not concentrate there (HSDB).

Acute toxicity

Systemic effects are characterized by onset in a few hours of nausea and vomiting, abdominal cramps and diarrhea. Headache, fever, and weakness may also occur. Ultimately liver disease may result (HSDB).

Chronic toxicity

Chronic toxic effects other than liver disease and cancer have not been well-documented (ToxFAQs). EPA has established a **provisional RfD of 0.000008 mg/kg-day** for N-nitrosodimethylamine (EPA 2004).

Carcinogenicity

EPA has classified N-nitrosodimethylamine as a probable human carcinogen (B2) based on the induction of tumors at multiple sites in both rodents and non-rodent mammals exposed by various routes (IRIS). EPA has established an **oral cancer slope factor of 51 per mg/kg-day** (IRIS).

46. N-NITROSO-DI-N-PROPYLAMINE

N-Nitroso-di-n-propylamine is produced primarily as a research chemical and not for commercial purposes. However it has been identified as a contaminant in the substituted dinitrotrifluralin herbicides, and thus may be released to the environment when these herbicides are used and from spills, as well as from some industrial effluents. The general population may be exposed to N-nitroso-di-n-propylamine in spray drifts from fields where trifluralin is used. N-Nitroso-di-n-propylamine is rarely found in food (HSDB).

Pharmacokinetics

N-Nitroso-di-n-propylamine is distributed evenly throughout the body. When administered to pregnant animals, the compound crosses the placental barrier and can be found in fetal tissue. It has been measured in milk and blood one hour after oral administration (HSDB).

Acute toxicity

Acute toxic effects from N-nitroso-di-n-propylamine are not well-documented.

Chronic toxicity

Chronic toxic effects other than teratogenicity and carcinogenicity have not been well-documented. EPA has not established an RfD for N-nitroso-di-n-propylamine (IRIS).

Carcinogenicity

EPA has classified N-nitroso-di-n-propylamine as a probable human carcinogen (B2) based on the increased tumor incidence at multiple sites in two rodent species and in monkeys administered the compound by various routes. EPA has established an **oral cancer slope factor of 7 per mg/kg-day** (IRIS).

47. N-NITROSODIPHENYLAMINE

N-Nitrosodiphenylamine (NNDP) is an industrial compound. It is an orange-brown or yellow solid that has been produced since 1945 to make rubber products such as tires or to make other chemicals. In the early 1980s, most US rubber manufacturers replaced it with more efficient chemicals, and now, only one manufacturer in the United States produces this chemical. Whether or not it occurs naturally in the environment remains unknown, but there is some evidence that micro-organisms make it (ToxFAQs).

Pharmacokinetics

Evidence in animals has found that NNDP is metabolized to 1,1-diphenylhydrazine in the liver. Additionally, increased levels of nitrate and diphenylamine were detected in the urine of rats given NNDP (HSDB).

Acute toxicity

No acute toxicity information is available for humans. Mortality was observed in two studies in which NNDP was orally administered at high levels to mice and rats (HSDB).

Chronic toxicity

Overall little information regarding the health effects of exposure to NNDP. However, animal studies have identified levels and exposures that can cause death. Animals given high levels of NNDP in their diets for long periods of time developed liver damage, swelling, cancer of the bladder, and changes in body weight (ToxFAQs). A provisional **RfD of 0.02 mg/kg-day** was developed by EPA (2004) for NNDP.

Carcinogenicity

While there is no evidence of carcinogenicity in humans, EPA has characterized NNDP as a probable human carcinogen based on the limited available animal studies. In a long-term study done, rats showed an increase in bladder cancer after being exposed to high levels of NNDP (HSDB). EPA has developed an **oral cancer slope factor of 0.0049 per mg/kg-day** for NNDP (IRIS).

48. PENTACHLOROPHENOL

Pentachlorophenol is a man-made substance that does not occur naturally in the environment. At one time, it was one of the most widely used biocides in the United States. Now the purchase and use of pentachlorophenol are restricted to certified applicators. It is no longer available to the general public. Application of pentachlorophenol in the home as an herbicide and pesticide accounted for only 3% of its consumption. Before use restrictions, pentachlorophenol was widely used as a wood preservative. It is now used industrially as a wood preservative for power line poles, cross arms, and fence posts (ToxFAQs).

Pharmacokinetics

The most common exposure routes for pentachlorophenol are inhalation and dermal contact. Human studies have estimated half lives of less than 33 hours. Bioaccumulation appears to be minor; most absorbed pentachlorophenol does not break down, but instead leaves in urine. Much smaller amounts leave in feces (ToxFAQs).

Acute toxicity

Many, but not all, the harmful effects associated with exposure to pentachlorophenol may be due to impurities present in commercial pentachlorophenol. Short exposures to large amounts of pentachlorophenol in the workplace or through the misuse of products that contain it can cause harmful effects on the liver, kidneys, blood, lungs, nervous system, immune system, and gastrointestinal tract. Contact with pentachlorophenol (particularly in the form of a hot vapor) can irritate the skin, eyes, and mouth. If large enough amounts enter the body, heat is produced causing an increase in body temperature. The body temperature can increase to dangerous levels, causing injury to various organs and tissues and even death (ToxFAQs).

Chronic toxicity

Long-term exposure to low levels such as those that occur in the workplace can cause damage to the liver, kidneys, blood, and nervous system. The major organs or systems affected by long-term exposure to low levels in animals are the liver, kidney, nervous system, and immune system. All these effects get worse as the level of exposure increases (ToxFAQs).

EPA has established an **RfD for pentachlorophenol of 0.03 mg/kg-day** based on a rat chronic oral study that documented liver and kidney pathology (IRIS).

Carcinogenicity

EPA's IRIS database classifies pentachlorophenol as a probable human carcinogen (B2) and provides an **oral cancer slope factor of 0.12 per mg/kg-day** based on statistically significant increases in the incidences of multiple biologically significant tumor types in mice. In addition, a high incidence of two uncommon tumors was also observed.

49. SILVER

Silver is toxic by all routes of exposure. Exposure is predominantly occupational via industries associated with electroplating, photographic materials, brazing, welding, and the manufacturing of jewelry, mirrors, coinage, pigments, and antiseptics (HSDB).

Pharmacokinetics

Silver is retained in all body tissues. It is primarily deposited in the skin, adrenals, lung, muscle, pancreas, kidney, heart, and spleen. Excretion of silver from the body is mainly via the gastrointestinal tract. Silver has a biological half-life of 1.7 to 2.5 days, as determined in studies using dogs, rats, monkeys, and mice (HSDB).

Acute toxicity

Acute exposure to silver can result in skin and eye irritation, mild bronchitis, metal fume fever and hepatic damage, stomach pain, and lung and throat irritation (HSDB).

Chronic toxicity

The most common result of chronic exposure to silver is generalized argyria, a blue-gray discoloration of the skin, mucous membranes, and eyes (HSDB). Workers involved in manufacturing precious metal powder experienced elevated urine and blood silver concentrations and respiratory irritation. EPA has calculated the **RfD for silver at 0.005 mg/kg-day** based on a long-term study of argyria in humans (IRIS).

Carcinogenicity

EPA has placed silver in Class D, not classifiable as to human carcinogenicity (IRIS).

50. THALLIUM

Thallium occurs in nature as a trace compound and is mainly associated with potassium and rubidium. Anthropogenic sources of thallium are gaseous emissions from cement factories, coal burning power plants, metal sewers, and leaching from ore processing operations (OGWDW).

Pharmacokinetics

Thallium has been shown to inhibit enzymatic action in the body. In acute thallium poisoning, human brain areas densely populated with neurons were found to accumulate thallium more than other areas. Thallium excretion is a very slow process mainly occurring via the kidney, gut, and salivary glands (HSDB).

Acute toxicity

Acute exposure to thallium can cause sever paroxysmal abdominal pain, vomiting, and diarrhea. Thallium poisoning often causes an increase in heart rate and blood pressure. Some victims have experienced a loss of vision in industrial exposures. In very severe cases, tremors, delirium, convulsions, hypotension, bradycardia, paralysis, coma, and death can occur (HSDB).

Chronic toxicity

Long-term exposure to thallium causes the relaxation of vascular smooth muscle, increased sympathetic tone, vagus nerve damage, fatty infiltration and necrosis of the liver, nephritis, gastroenteritis, pulmonary edema, degenerative changes in the adrenals, degeneration of peripheral and central nervous system, alopecia, and in some cases death (HSDB). EPA provides RfDs for several forms of thallium, with an **RfD for thallium chloride of 0.00008 mg/kg-day**, which is based on a subchronic study with rats. Because no data are available regarding the forms of thallium most commonly present in the LDW, the conversion factors provided in IRIS were used to convert the RfD for thallium chloride to an RfD for thallium. Thus, the **RfD used in this risk assessment for thallium is 0.00007 mg/kg-day**.

Carcinogenicity

EPA has placed thallium in Class D, not classifiable as to human carcinogenicity.

51. TOXAPHENE

Toxaphene is an organochlorine pesticide that is comprised of a mixture of at least 670 chlorinated camphenes. Toxaphene was probably the most heavily used pesticide in the United States during the 1970s after DDT was banned. It was banned for most uses in 1982; all uses were banned in 1990. However, due to its relatively long half-life, it persists in the environment. The soil half-life is approximately 1 to 14 years (HSDB).

Pharmacokinetics

The components of toxaphene are metabolized in mammals via dechlorination, dehydrodechlorination, and oxidation, primarily through the action of the mixed function oxidase system and other hepatic microsomal enzymes. Conjugation may occur but is not a major route of metabolism. Each component of toxaphene has its own rate of biotransformation, making the characterization of toxaphene pharmacokinetics complex. Some components of toxaphene are highly lipophilic and poorly metabolized; these components may accumulate in body fat (ATSDR 1996).

Acute toxicity

Acute high-level exposures to toxaphene and toxaphene-contaminated food have resulted in death in adults and children with an estimated minimum lethal dose of 2 to 7 g/day, which is equivalent to 29 to 100 mg/kg-day for an adult male. LD50 values in rats were 80 mg/kg-day for females and 90 mg/kg-day for males. Transient liver and kidney effects, and periods of memory loss have been observed in humans after single large oral exposures. In animals, the most sensitive organ is the liver. Toxicity to the central nervous system, kidney, and adrenal glands have also been observed (ATSDR 1996).

Chronic toxicity

IRIS does not provide a discussion of chronic effects of exposure to toxaphene or an RfD. Chronic exposure to toxaphene may result in damage to the following organ systems: liver, kidney, adrenal, immunological, and neurological (ATSDR 1996). Chronic exposure to toxaphene may cause hormonal alterations. A study on chronic exposures found increased levels of hepatic metabolism of the hormones estradiol and estrone and a decrease in their uterotropic action. Some adverse effects of toxaphene that do not occur with a single exposure may result from repeated exposures. Exposures at 0.06 mg/kg-day over 5 weeks caused adrenal hormone reductions, whereas a single dose of 16 mg/kg-day did not cause effects.

Carcinogenicity

Toxaphene is classified as a probable human carcinogen (B2) by EPA based on oral studies in animals (IRIS). However, no conclusive human epidemiological studies are available for toxaphene (ATSDR 1996). Oral administration of toxaphene resulted in an increased incidence of hepatocellular carcinomas and neoplastic nodules in mice, and thyroid tumors in rats (IRIS). The **oral cancer slope factor for toxaphene is 1.1 per mg/kg-day**, based on liver tumors in experimental animals (IRIS).

Toxaphene has recently been observed to have estrogenic effects on human breast cancer estrogen-sensitive cells (Soto et al. 1994). Xenoestrogens have been hypothesized to have a role in human breast cancer. In addition to potential carcinogenic effects, toxaphene may also cause disruption of the endocrine system due to its estrogenic activity (Soto et al. 1994).

52. TRIBUTYLTIN

TBT is one of several organotin compounds that have been used as biocides, disinfectants, and antifoulants. This overview focuses primarily on bis(tri-n-butyltin) oxide (TBTO) because this is the only TBT compound for which the EPA has established an RfD for assessing chronic toxicity to humans and because more toxicological information is available for this compound than for other organotin compounds.

Pharmacokinetics

No studies are available regarding the distribution of tin in human tissues following oral exposure (ATSDR 2005d). Laboratory studies with mammals have shown that organotin compounds are absorbed; studies with rats detected tin compounds in the gastrointestinal tract, kidney, and liver. Rats that orally ingested tin compounds showed the highest concentrations in the liver and kidneys; concentrations in the brain and adipose tissue were 10 to 20 percent of those found in the kidneys and liver (Krajnc et al. 1984). Studies involving trialkyltin compounds show that absorbed compounds are metabolized, with the data suggesting that the liver is the active site and dealkylation the principle metabolic pathway (ATSDR 2005d).

Acute toxicity

There are no controlled studies on the effects of TBTO in humans. The available data demonstrate that TBT is toxic to animals, with LD50 values ranging from 122 to 194 mg/kg-day in rats.

Chronic toxicity

There are no studies on the effects of TBTO in humans. Animal studies have shown effects on the blood and liver, and immunological effects, including thymus atrophy and depletion of T-lymphocytes in the spleen and lymph nodes (ATSDR 2005d). In addition, numerous studies have documented the endocrine-disrupting properties of

TBT in invertebrates, and much of this data can also be applied to humans (DeFur et al. 1999).

EPA's IRIS database provides an **RfD for TBTO of 0.0003 mg/kg-day**, based on a NOAEL of 0.025 mg/kg-day. This was based on a chronic feeding study of rats in which immunologic function analyses for specific and nonspecific resistance were performed after 4 to 6 or 15 to 17 months of exposure to test doses of TBTO ranging from 0.025 to 2.5 mg/kg-day (Vos et al. 1990). The RfD for TBTO can be converted to TBT ion units by multiplying it by the ratio (0.49) of the molecular weights for the two substances. **The resulting RfD for the TBT ion is 0.00015 mg/kg-day**.

Carcinogenicity

TBTO is currently Class D, which is defined as a chemical not classifiable with respect to human carcinogenicity. There are no data documenting the development of cancer in humans following exposure to TBTO. A large number of studies show that TBTO is not genotoxic, and there are no structure-activity relationships suggesting that TBTO might be a carcinogen.

53. VANADIUM

Vanadium compounds are widely distributed in the earth's crust. Elemental vanadium does not occur in nature, but its compounds exist in over 50 different mineral ores and in association with fossil fuels (HSDB). The route of entry of vanadium compounds most commonly seen in industrial exposures is through the respiratory system. Exposures are usually limited to areas where vanadium pentoxide is produced, in steel mills where vanadium pentoxide is used, and in cleaning boilers fired by oil containing vanadium (HSDB).

Pharmacokinetics

Vanadium compounds and metallic vanadium, when absorbed, are rapidly excreted and exhibit low degrees of toxicity, as indicated by minor irritation and lack of systemic effects. Absorbed vanadium is widely distributed in the body. In animals, the highest values are found in bone, kidney, liver, spleen and lung. Bone maintains essentially unchanged levels for several weeks. The lowest values are found in the brain, but in human autopsy material, brain concentrations of vanadium are more or less the same as those found in other organs (HSDB).

Acute toxicity

Vanadium and its compounds are principally eye and respiratory tract irritants that result in conjunctivitis, coughing, wheezing, difficulty in breathing, and industrial bronchitis. A metallic taste and throat irritation may occur. Greenish discoloration of the fingers, scrotum, and upper legs may also be present. A greenish black discoloration of the tongue indicates heavy exposure (HSDB).

Chronic toxicity

Some studies suggest exposure to vanadium may impair the lung resistance to respiratory infection, although the available data on chronic respiratory effects of vanadium are still inconclusive. NCEA provides an **RfD of 0.001 mg/kg-day** for vanadium (EPA 2004).

Carcinogenicity

At this time, there is no information regarding the carcinogenicity of vanadium to humans or animals.

54. ZINC

Zinc is an essential trace element that plays a necessary role in enzymatic functions, protein synthesis, and carbohydrate metabolism. Small doses of zinc are necessary for normal growth and development in birds and mammals. Zinc also has many industrial uses. It is used as a galvanizing agent, component in brass, bronze alloys, light metal alloys, and in wet batteries (HSDB). The most common route of high-level exposure to zinc is through consumption of liquid contained in galvanized metal containers or by water contaminated with industrial zinc waste (ToxFAQs).

Pharmacokinetics

Absorption of zinc occurs in the intestine when ingested or through the lung when zinc dust or fumes are inhaled. Zinc is mainly stored in skeletal muscle, but significant concentrations can also occur in the pancreas, prostate, liver, and retina. Zinc has a biological half-life of 162 to 500 days (HSDB).

Acute toxicity

In humans, ingestion of gram quantities of zinc may cause pancreatic derangement, light-headedness, and mild derangement of cerebellar function. Acute exposure to zinc can also cause dizziness, nausea, tightness in the throat, diarrhea, and vomiting. Metal fume fever has been observed after inhalation of zinc oxide fumes (HSDB).

Chronic toxicity

Prolonged exposure to drinking water that contained 40 mg/L of zinc triggered symptoms such as irritability, muscular stiffness and pain, loss of appetite, and nausea (HSDB). EPA has established an **RfD of 0.3 mg/kg-day for zinc** based on a human diet supplement study in which adult females experienced a 47% decline in erythrocyte superoxide dismutase (ESOD) after 10 weeks of exposure (IRIS).

Carcinogenicity

EPA has placed zinc in Class D, not classifiable as to human carcinogenicity (IRIS).

References

ACGIH. 1986. Documentation of the threshold limit values and biological exposure indices. Fifth edition. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.

ATSDR. 1990. Toxicological profile for 1,2-diphenylhydrazine. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1995. Toxicological profile for polycyclic aromatic hydrocarbons (PAHs). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1996. Toxicological profile for toxaphene. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1999a. Toxicological profile for aldrin/dieldrin. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1999b. Toxicological profile for aluminum. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1999c. Toxicological profile for cadmium. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1999d. Toxicological profile for lead (update). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1999e. Toxicological profile for mercury. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2000a. Toxicological profile for manganese [online]. Agency for Toxic Substances and Disease Registry, Atlanta, GA. [Cited 10/9/06]. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp151.html

ATSDR. 2000b. Toxicological profile for polychlorinated biphenyls (PCBs). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2002. Toxicological profile for hexachlorobenzene. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2004. Toxicological profile for copper. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2005a. Toxicological profile for arsenic (update). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2005b. Toxicological profile for heptachlor and heptachlor epoxide. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2005c. Toxicological profile for hexachlorocyclohexane (lindane). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2005d. Toxicological profile for tin. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 2006a. ToxFAQs™: Frequently Asked Questions about contaminants found at hazardous waste sites Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services, Atlanta, GA.

ATSDR. 2006b. Toxological profiles [online]. Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services, Atlanta, GA. Available from: http://www.atsdr.cdc.gov/.

Brown DP. 1987. Mortality of workers exposed to polychlorinated biphenyls--an update. Arch Environ Health 42(6):333-339.

Brown DP, Jones M. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Arch Environ Health 36(3):120-129.

California EPA. 1994. Health effects of benzo(a)pyrene. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Berkeley, CA.

Canfield RL, Henderson CRJ, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. 2003. Intellectual impairment in children with blood lead concentrations below 10 ug per deciliter. N Engl J Med 348(16):1517-1526.

Chew C. 1996. Toxicity and exposure concerns related to arsenic in seafoods: an arsenic literature review for risk assessments. Prepared for US Environmental Protection Agency, Region 10. ICF Kaiser, Inc., Bellevue, WA.

Clayton GD, Clayton FE. 1981. Patty's industrial hygiene and toxicology: Volumes 2A, 2B, 2C: Toxicology. 3rd ed. John Wiley and Sons, New York, NY.

Colt JS, Severson RK, Lubin JH, Rothman N, Camann D, Davis S, Cerhan JR, Cozen W, Hartge P. 2005. Organochlorines in carpet dust and non-Hodgkin lymphoma. Epidemiology 16:516-525.

Dallaire F, Dewailly E, Vézina C, Muckle G, Weber J-P, Bruneau S, Ayotte P. 2006. Effect of prenatal exposure to polychlorinated biphenyls on incidence of acute respiratory infections in preschool Inuit children. Environ Health Persp 114(8):1301-1305.

De Roos AJ, Hartge P, Lubin JH, Colt JS, Davis S, Cerhan JR, Severson RK, Cozen W, Patterson DG, Jr, Needham LL, Rothman N. 2005. Persistent organochlorine chemicals in plasma and risk of non-Hodgkin's lymphoma. Cancer Res 2005 65(23):11214-11226.

DeFur PL, Crane M, Ingersoll C, Tattersfield L. 1999. Endocrine disruption in invertebrates: endocrinology, testing, and assessment. SETAC Press, Pensacola, FL.

Doull J, Klassen CD, Amdur MD, eds. 1986. Casarett and Doull's Toxicology. 3rd ed. Macmillan Co., Inc., New York, NY.

Eigenberg DA, Bozigian HP, Carter DE, Sipes IG. 1986. Distribution, excretion, and metabolism of butylbenzyl phthalate in the rat. J Toxicol Environ Health 17(4):445-456.

EPA. 1987. Summary review of the health effects associated with copper. Health issue assessment. EPA/600/8-87/001. Office of Health and Environmental Assessment, US Environmental Protection Agency, Washington, DC.

EPA. 1993. Provisional guidance for quantitative risk assessment of polycyclic aromatic hydrocarbons. EPA-600/R-93/089. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, US Environmental Protection Agency, Cincinnati, OH.

EPA. 1996. PCBs: Cancer dose-response assessment and application to environmental mixtures. EPA/600/P-96/001F. Office of Pollution Prevention and Toxics, US Environmental Protection Agency, Washington, DC.

EPA. 1997a. Arsenic and fish consumption. EPA-822-R-97-003. Health and Ecological Criteria Division, US Environmental Protection Agency, Washington DC.

EPA. 1997b. Health Effects Assessment summary tables. FY 1997 update. EPA-540-R-97-036. Office of Research and Development and Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1997c. Mercury study report to Congress. EPA/452/R-97-003. Health and Ecological Criteria Division, Office of Research and Development and Office of Air Quality Planning and Standards, US Environmental Protection Agency, Washington, DC.

EPA. 2000. Guidance for assessing chemical contaminant data for use in fish advisories. Volume 2: Risk assessment and fish consumption limits. Third ed. EPA 823-B-00-008. US Environmental Protection Agency, Washington, DC.

EPA. 2001. Dioxin reassessment -- an SAB review of the Office of Research and Development's reassessment of dioxin. Review of the revised sections (dose response modeling, integrated summary, risk characterization, and toxicity equivalency factors) of the EPA's reassessment of dioxin by the dioxin reassessment review subcommittee of the EPA Science Advisory Board (SAB). EPA-SAB-EC-01-006. US Environmental Protection Agency, Washington, DC.

EPA. 2003a. Exposure and human health reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 1: estimating exposure to dioxin-like compounds. Vol 2: Properties, environmental levels, and background exposures. NAS review draft. National Center for Environmental Assessment, US Environmental Protection Agency, Washington, DC.

EPA. 2003b. Human health toxicity values in Superfund risk assessments. OSWER Directive 9285.7-53. Office of Solid Waste and Emergency Response, US Environmental Protection Agency, Washington, DC.

EPA. 2004. EPA Region IX preliminary remediation goals: 2004 PRG table [online]. US Environmental Protection Agency Region IX, San Francisco, CA. Updated October 2004. Available from: http://www.epa.gov/region09/waste/sfund/prg/index.html.

EPA. 2005. EPA Region III risk-based concentration table [online]. US Environmental Protection Agency Region III, Philadelphia, PA. Updated October 2005. Available from: http://www.epa.gov/reg3hwmd/risk/human/index.htm.

EPA. 2006a. Ground water and drinking water: Drinking water contaminants [online]. Office of Ground Water and Drinking Water (OGWDW), US Environmental Protection Agency, Washington, DC. Updated 2/28/2006. Available from: http://www.epa.gov/OGWDW/hfacts.html.

EPA. 2006b. Integrated Risk Information System (IRIS) database [online]. US Environmental Protection Agency, Washington, DC. [Cited 1/2006]. Available from: http://www.epa.gov/iris/.

Faust RA. 1992. Toxicity summary for copper. Health and Research Safety Division, Biomedical and Environmental Information Analysis Section, Chemical Hazard Evaluation and Communication Group, Oak Ridge National Laboratory, Oak Ridge, TN.

FDA. 1993. Guidance document for cadmium in shellfish. Center for Food Safety and Applied Nutrition, US Food and Drug Administration, Washington, DC.

Fein GG, Jacobson JL, Jacobson SW. 1984. Prenatal exposure to polychlorinated biphenyls: effects on birth size and gestational age. J Pediatr 105:315-320.

IARC. 1995. Monographs on the evaluation of the carcinogenic risk of chemicals to man. World Health Organization, International Agency for Research on Cancer, Geneva, Switzerland.

IMOA. 2006. IMOA Health, Safety & Environment website: Environmental data overview [online]. International Molybdenum Association, Brussels, Belgium. [Cited 4/3/2006]. Available from: http://hse.imoa.info/Default.asp?page=80.

Jacobson JL, Jacobson SW. 1996. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N Engl J Med 335(11):783-789.

Jacobson JL, Jacobson SW. 1997. Evidence for PCBs as neurodevelopmental toxicants in humans. Neurotoxicol 8(2):415-424.

Jenkins FP, Robinson JA, Gellatly JBM, Salmond GWA. 1972. The no-effect dose of aniline in human subjects and a comparison of aniline toxicity in man and the rat. :. Cosmet Toxicol 10:671-679.

Kenyon EM, Hughes MF. 2001. A concise review of the toxicity and carcinogenicity of dimethylarsinic acid. Toxicology 160(1-3):227-236.

Knerr S, Schrenk D. 2006. Carcinogenicity of "non-dioxinlike" polychlorinated biphenyls. Crit Rev Toxicol 36(9):663-694.

Krajnc E, Webster PW, Loeber JG. 1984. Toxicity of bis(tri-*n*-butyltin) oxide in the rat. I: Short-term effects of general parameters and on the endocrine and lymphoid systems. Toxicol Appl Pharmacol 75(363-386).

Longnecker MP, Wolff MS, Gladen BC, Brock JWG, P, Jacobson JL, Korrick SA, Rogan WJ, Weisglas-Kuperus N, Hertz-Picciotto I, Ayotte P, Stewart P, Winneke G, Charles MJ, Jacobson SW, Dewailly E, Boersma ER, Altshul LM, Heinzow B, Pagano JJ, Jensen AA. 2003. Comparison of polychlorinated biphenyl levels across studies of human neurodevelopment. Environ Health Persp 111(1):65-70.

Mocarelli P, Marocchi A, Brambilla P, Gerthoux PM, Colombo L, Mondonico A, Meazza L. 1991. Effects of dioxin exposure in humans at Seveso, Italy. In: Gallo MA, Scheuplein RJ, Van der Heijden KA, eds, Banbury Report 35: Biological basis for risk assessment of dioxins and related compounds. Cold Spring Harbor Laboratory Press, Plainview NY, pp 95-110.

Pattee OH, Pain DJ. 2003. Lead in the environment. In: Hoffman DJ, Rattner BA, Burton GA, Jr, Cairns J, Jr, eds, Handbook of ecotoxicology. Second ed. Lewis Publishers, Boca Raton, FL, pp 373-408.

Petrick JS, Ayala-Fierro A, Cullen WR, Carter DE, Aposhian HV. 2000. Monomethylarsonous acid (MMAIII) is more toxic than arsenite in Chang human hepatocytes. Toxicol Appl Pharmacol 163:203-207.

Petrick JS, Jagadish J, Mash EA, Aposhian HV. 2001. Monomethylarsonous acid (MMAIII) and arsenite: LD50 in hamsters and in vitro inhibition of pyruvate dehydrogenase. Chem Res Toxicol 14:651-656.

Prince MM, Ruder AM, Hein MJ, Waters MA, Whelan EA, Nilsen N, Ward EM, Schnorr TM, Laber PA, Davis-King KE. 2006a. Mortality and exposure response among 14,458 electrical capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). Environ Health Persp 114(10):1508-1514.

Prince MM, Hein MJ, Ruder AM, Waters MA, Laber PA, Whelan EA. 2006b. Update: cohort mortality study of workers highly exposed to polychlorinated biphenyls (PCBs) during the manufacture of electrical capacitors, 1940-1998. Environ Health Glob Access 5(13):10 pp.

Ruder AM, Hein MJ, Nilsen N, Waters MA, Laber PA, Davis-King K, Prince MM, Whelan EA. 2006. Mortality among workers exposed to polychlorinated biphenyls (PCBs) in an electrical capacitor manufacturing plant in Indiana: an update. Environ Health Persp 114(1):18-23.

Rushneck DR, Beliveau A, Fowler B, Hamilton C, Hoover D, Kaye K, Berg M, Smith T, Telliard WA, Roman H, Ruder E, Ryan L. 2004. Concentrations of dioxin-like PCB congeners in unweathered Aroclors by HRGC/HRMS using EPA method 1668A. Chemosphere 54:79-87.

Safe SH. 1994. Polychlorinated biphenyls (PCBs): environmental impact, biochemical and toxic responses, and implications for risk assessment. Crit Rev Toxicol 24(2):87–149.

Schantz SL. 1996. Developmental neurotoxicity in humans: what do we know and where do we go from here. Neurotoxicol Teratol 18(3):217-227.

Schecter A, Gasiewicz TA. 2001. Dioxins and health: second edition. John Wiley and Sons Inc., Hoboken, NJ.

Silberhorn EM, Glauert HP, Robertson LW. 1990. Carcinogenicity of polyhalogenated biphenyls: PCBs and PBBs. Crit Rev Toxicol 20(6):397-426.

Soto AM, Chung KL, Sonnenschein C. 1994. The pesticides endosulfan, toxaphene, and dieldrin have estrogenic effects on human estrogen-sensitive cells. Environ Health Persp 102(4):380-383.

Spanierman C. 2001. Toxicity, iron. eMedicine Clinical Knowledge Base [online]. eMedicine, Omaha, NE. [Cited May 17, 2001]. Available from: http://www.emedicine.com/EMERG/topic285.htm.

Thomas DJ, Styblo M, Lin S. 2001. The cellular metabolism and systemic toxicity of arsenic. Toxicol Appl Pharmacol 176:127-144.

TOXNET. 2006. Hazardous substances data bank (HSDB) [online]. TOXNET Toxicology Data Network, US National Library of Medicine, Bethesda, MD. Available from: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB.

Van den Berg M, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, Fiedler H, Hakansson H, Hanberg A, Haws L, Rose M, Safe S, Schrenk D, Tohyama C, Tritscher A, Tuomisto J, Tysklind M, Walker N, Peterson RE. 2006. The 2005 World Health Organization reevaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Tox Sci 93(2):223-241.

van der Plas SA, Sundberg H, van den Bert H, Scheu G, Wester P, Jensen S, Berbman A, de Boer J, Koeman JH, Brouwer A. 2000. Contribution of planar (0-1 *ortho*) and nonplanar (2-4 *ortho*) fractions of Aroclor 1260 to the induction of altered hepatic foci in female Sprague-Dawley rats. Toxicol Appl Pharmacol 169:255-268.

Vos J, Deklerk A, Krajnc E, Van Loveren L, Rozing J. 1990. Immunotoxicity of bis(tri-*n*-butyltin) oxide in the rat: effects on thymus-dependent immunity and on

nonspecific resistance following long-term exposure in young versus aged rats. Toxicol Appl Pharmacol 105:144-155.

Walker NJ, Crockett PW, Nyska A, Brix AE, Jokinen MP, Sells DM, Hailey JR, Easterling M, Haseman JK, Yin MW, M E, Bucher JR, Portier CJ. 2005. Dose-additive carcinogenicity of a defined mixture of "dioxin-like compounds". Environ Health Persp 113(1):43-48.

WHO. 1981. Environmental health criteria 17: Manganese [online]. World Health Organization, Geneva, Switzerland. Available from: http://www.inchem.org/documents/ehc/ehc/ehc017.htm.

WHO. 1990. Environmental health criteria 101: Methylmercury [online]. World Health Organization, Geneva, Switzerland. Available from: http://www.inchem.org/documents/ehc/ehc/ehc101.htm.

Lower Duwamish Waterway Group

Port of Seattle / City of Seattle / King County / The Boeing Company

Lower Duwamish Waterway Remedial Investigation

REMEDIAL INVESTIGATION REPORT

APPENDIX B, BASELINE HUMAN HEALTH RISK ASSESSMENT

ERRATA: ADJUSTMENT TO TULALIP TRIBES SEAFOOD CONSUMPTION RATES AND THE IMPACT ON RISK ESTIMATES

For submittal to:

The US Environmental Protection Agency
Region 10
Seattle, WA

The Washington State Department of Ecology
Northwest Regional Office
Bellevue, WA

July 17, 2009

Prepared by: Windward environmental LLC

200 West Mercer Street, Suite 401 • Seattle, Washington • 98119

Human Health Risk Assessment Errata: Adjustment to Tulalip Tribes' Seafood Consumption Rates and the Impact on Risk Estimates

The final baseline human health risk assessment (HHRA) for the Lower Duwamish Waterway (LDW) (Windward 2007) was based, in part, on seafood consumption rate data supplied by the US Environmental Protection Agency (EPA) (EPA 2007). Some of the seafood consumption rates in the Framework were developed by EPA from data collected from a survey of the Tulalip Tribes' consumption of Puget Sound seafood. EPA recently discovered that some of the consumption rates it had calculated were incorrect and thus issued corrected rate information in comments on the draft final Remedial Investigation report for the LDW site (EPA 2009). Specifically, the apportionment of shellfish consumption (but not the total consumption rates) for scenarios developed from the Tulalip Tribes seafood consumption study were corrected. These errata present the revised shellfish consumption rates and the associated changes in the risk estimates for the Tulalip survey-based scenarios.

UPDATED SEAFOOD CONSUMPTION RATES

Table 1 compares the seafood consumption rates used in the final HHRA (Windward 2007) for the Tulalip Tribe scenarios to the revised consumption rates provided by EPA (2009). Essentially, the corrected apportionment of total shellfish consumption for crab and other shellfish categories is reversed from what was initially presented in the final HHRA. In the final HHRA, the percentage of shellfish consumption attributed to crabs was set at 53% while the percentage of shellfish associated with clams was set at 46%; the corrections provided by EPA reverse these percentages. The revised consumption rates were developed from these corrected percentages, following the process described in the final HHRA.

Table 1. HHRA consumption rates

SCENARIO NAME	CONSUMPTION CATEGORY	FINAL HHRA CONSUMPTION RATE (g/day)	REVISED CONSUMPTION RATE (g/day)
Adult Tribal RME (Tulalip data)	Pelagic fish – fillet	8.1	8.1
	Benthic fish – fillet	7.5	7.5
	Benthic fish – whole body	0	0
	Crab – edible meat	33	**28.7**
	Crab – whole body	10.4	**9.0**
	Mussels	0.8	0.8
	Clams	37.7	**43.4**

Scenario Name	Consumption Category	Final HHRA Consumption Rate (g/day)	Revised Consumption Rate (g/day)
Adult Tribal CT (Tulalip data)	Pelagic fish – fillet	1.3	1.3
	Benthic fish – fillet	1.2	1.2
	Benthic fish – whole body	0	0
	Crab – edible meat	5.0	**4.4**
	Crab – whole body	1.6	**1.4**
	Mussels	0.1	0.1
	Clams	5.8	**6.6**
Child Tribal RME (Tulalip data)	Pelagic fish – fillet	3.2	3.2
	Benthic fish – fillet	3.0	3.0
	Benthic fish – whole body	0	0
	Crab – edible meat	13.2	**11.5**
	Crab – whole body	4.2	**3.6**
	Mussels	0.33	0.33
	Clams	15.1	**17.4**
Child Tribal CT (Tulalip data)	Pelagic fish – fillet	0.52	0.52
	Benthic fish – fillet	0.48	0.48
	Benthic fish – whole body	0	0
	Crab – edible meat	2.0	**1.8**
	Crab – whole body	0.64	**0.6**
	Mussels	0.04	0.04
	Clams	2.3	**2.6**

Note: Underlined and bolded entries indicate that the rate has changed.
CT – central tendency
HHRA – human health risk assessment
RME – reasonable maximum exposure

As shown in Table 1, based on the EPA correction, the crab consumption rate decreased and the clam consumption rate increased. EPA did not make any changes in consumption rates for pelagic fish, benthic fish, or mussels.

Updated Risk Estimates

Risk estimates for the Tulalip survey-based seafood consumption scenarios were updated using the corrected consumption rates. Tables 2 through 5 present a comparison of the cancer chronic daily intakes (CDIs) based on the consumption rates from the final HHRA and EPA's revised rates, the percent change in the CDIs, and the excess cancer risks calculated using the consumption rates from the final HHRA and EPA's revised rates. Consistent with the final HHRA, the CDIs for the central tendency (CT) scenarios were developed using mean concentrations as the exposure point

concentrations (EPCs), whereas the CDIs for the reasonable maximum exposure (RME) scenarios were developed using the 95% upper confidence limit on the mean (UCL). Hence, the percent differences in the CDIs for the same chemicals may differ between the CT and RME scenarios.

Table 2. Comparison of excess cancer risk estimates for the adult tribal RME seafood consumption scenario based on Tulalip data

Chemical	Cancer CDI (mg/kg-day)		Percent Change in CDI	Excess Cancer Risk	
	Final HHRA	Revised		Final HHRA	Revised
Arsenic[a, b]	9.7×10^{-4}	1.1×10^{-3}	+13%	1×10^{-3}	2×10^{-3}
Bis(2-ethylhexyl) phthalate	4.5×10^{-4}	4.5×10^{-4}	0%	6×10^{-6}	6×10^{-6}
cPAHs[a, c]	9.8×10^{-6}	1.1×10^{-5}	+12%	7×10^{-5}	8×10^{-5}
PCB TEQ[a]	8.1×10^{-9}	8.1×10^{-9}	0%	1×10^{-3}	1×10^{-3}
Total PCBs	8.0×10^{-4}	8.1×10^{-4}	+1%	2×10^{-3}	2×10^{-3}
Pentachlorophenol	7.3×10^{-4}	7.2×10^{-4}	-1%	9×10^{-5}	9×10^{-5}
Subtotal excluding PCB TEQ				**3×10^{-3}**	**4×10^{-3}**
Subtotal excluding total PCBs				**2×10^{-3}**	**3×10^{-3}**
Tentatively identified chemicals (JN-qualified)					
Aldrin	2.8×10^{-6}	2.7×10^{-6}	-4%	5×10^{-5}	5×10^{-5}
alpha-BHC	2.6×10^{-6}	2.4×10^{-6}	-8%	2×10^{-5}	2×10^{-5}
beta-BHC	3.5×10^{-6}	3.4×10^{-6}	-3%	6×10^{-6}	6×10^{-6}
Carbazole	2.3×10^{-3}	2.2×10^{-3}	-4%	5×10^{-5}	4×10^{-5}
Total chlordane	1.7×10^{-5}	1.6×10^{-5}	-6%	6×10^{-6}	6×10^{-6}
Total DDTs	6.2×10^{-5}	6.0×10^{-5}	-3%	2×10^{-5}	2×10^{-5}
Dieldrin	8.2×10^{-6}	8.8×10^{-6}	+7%	1×10^{-4}	1×10^{-4}
gamma-BHC	4.2×10^{-6}	4.1×10^{-6}	-2%	6×10^{-6}	5×10^{-6}
Heptachlor	3.2×10^{-6}	3.0×10^{-6}	-6%	1×10^{-5}	1×10^{-5}
Heptachlor epoxide	3.4×10^{-6}	3.3×10^{-6}	-3%	3×10^{-5}	3×10^{-5}
Hexachlorobenzene	6.5×10^{-6}	6.0×10^{-6}	-8%	1×10^{-5}	1×10^{-5}
Subtotal				**3×10^{-4}**	**3×10^{-4}**
Total excess cancer risk excluding PCB TEQ				**3×10^{-3}**	**4×10^{-3}**
Total excess cancer risk excluding total PCBs				**2×10^{-3}**	**3×10^{-3}**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for cPAHs are from only 2004; historical data were not used because of high reporting limits. Risk related to 2004 and historical data for cPAHs were analyzed in the uncertainty analysis of the HHRA (Windward 2007).

BHC – benzene hexachloride
CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
DDT – dichlorodiphenyltrichloroethane
HHRA – human health risk assessment
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table 3. Comparison of excess cancer risk estimates for the adult tribal CT seafood consumption scenario based on Tulalip data

Chemical	Cancer CDI (mg/kg-day)		Percent Change	Excess Cancer Risk	
	Final HHRA	Revised		Final HHRA	Revised
Arsenic[a, b]	4.1×10^{-5}	4.5×10^{-5}	+10%	6×10^{-5}	7×10^{-5}
Bis(2-ethylhexyl) phthalate	1.2×10^{-5}	1.2×10^{-5}	0%	2×10^{-7}	2×10^{-7}
cPAHs[a, c]	5.1×10^{-7}	5.5×10^{-7}	+8%	4×10^{-6}	4×10^{-6}
PCB TEQ[a]	3.8×10^{-10}	3.5×10^{-10}	-8%	6×10^{-5}	5×10^{-5}
Total PCBs	3.2×10^{-5}	3.1×10^{-5}	-3%	6×10^{-5}	6×10^{-5}
Pentachlorophenol	1.4×10^{-5}	1.4×10^{-5}	0%	2×10^{-6}	2×10^{-6}
Subtotal excluding PCB TEQ				**1×10^{-4}**	**1×10^{-4}**
Subtotal excluding total PCBs				**1×10^{-4}**	**1×10^{-4}**
Tentatively identified chemicals (JN-qualified)					
Aldrin	8.7×10^{-8}	8.3×10^{-8}	-5%	1×10^{-6}	1×10^{-6}
alpha-BHC	8.6×10^{-8}	8.2×10^{-8}	-5%	5×10^{-7}	5×10^{-7}
beta-BHC	1.4×10^{-7}	1.4×10^{-7}	-0%	3×10^{-7}	2×10^{-7}
Carbazole	5.0×10^{-5}	4.6×10^{-5}	-8%	1×10^{-6}	9×10^{-7}
Total chlordane	5.7×10^{-7}	5.5×10^{-7}	-4%	2×10^{-7}	2×10^{-7}
Total DDTs	3.1×10^{-6}	3.0×10^{-6}	-3%	1×10^{-6}	1×10^{-6}
Dieldrin	1.6×10^{-7}	1.6×10^{-7}	0%	3×10^{-6}	3×10^{-6}
gamma-BHC	9.8×10^{-8}	9.4×10^{-8}	-4%	1×10^{-7}	1×10^{-7}
Heptachlor	8.9×10^{-8}	8.5×10^{-8}	-4%	4×10^{-7}	4×10^{-7}
Heptachlor epoxide	1.3×10^{-7}	1.2×10^{-7}	-8%	1×10^{-6}	1×10^{-6}
Hexachlorobenzene	1.5×10^{-7}	1.5×10^{-7}	0%	2×10^{-7}	2×10^{-7}
Subtotal				**9×10^{-6}**	**9×10^{-6}**
Total risk across all exposure routes/pathways excluding PCB TEQ				**1×10^{-4}**	**1×10^{-4}**
Total risk across all exposure routes/pathways excluding total PCBs				**1×10^{-4}**	**1×10^{-4}**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for carcinogenic PAHs are from only 2004; historical data were not used because of high reporting limits. Risk related to 2004 and historical data for cPAHs were analyzed in the uncertainty analysis of the HHRA (Windward 2007).

BHC – benzene hexachloride
CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
DDT – dichlorodiphenyltrichloroethane
HHRA – human health risk assessment
PCB – polychlorinated biphenyl
TEQ – toxic equivalent

Table 4. Comparison of excess cancer risk estimates for the child tribal RME seafood consumption scenario based on Tulalip data

Chemical	Cancer CDI (mg/kg-day)		Percent Change	Excess Cancer Risk	
	Final HHRA	Revised		Final HHRA	Revised
Arsenic[a, b]	1.8×10^{-4}	2.0×10^{-4}	+11%	3×10^{-4}	3×10^{-4}
Bis(2-ethylhexyl) phthalate	8.3×10^{-5}	8.3×10^{-5}	0%	1×10^{-6}	1×10^{-6}
cPAHs[a, c]	1.8×10^{-6}	2.1×10^{-6}	+17%	7×10^{-5}	8×10^{-5}
PCB TEQ[a]	1.5×10^{-9}	1.5×10^{-9}	0%	2×10^{-4}	2×10^{-4}
Total PCBs	1.5×10^{-4}	1.5×10^{-4}	0%	3×10^{-4}	3×10^{-4}
Pentachlorophenol	1.3×10^{-4}	1.3×10^{-4}	0%	2×10^{-5}	2×10^{-5}
Subtotal excluding PCB TEQ				**7×10^{-4}**	**7×10^{-4}**
Subtotal excluding total PCBs				**6×10^{-4}**	**6×10^{-4}**
Tentatively identified chemicals (JN-qualified)					
Aldrin	5.2×10^{-7}	5.0×10^{-7}	-4%	9×10^{-6}	8×10^{-6}
alpha-BHC	4.8×10^{-7}	4.5×10^{-7}	-6%	3×10^{-6}	3×10^{-6}
beta-BHC	6.5×10^{-7}	6.2×10^{-7}	-5%	1×10^{-6}	1×10^{-6}
Carbazole	4.2×10^{-4}	4.0×10^{-4}	-5%	8×10^{-6}	8×10^{-6}
Total chlordane	3.1×10^{-6}	3.0×10^{-6}	-3%	1×10^{-6}	1×10^{-6}
Total DDTs	1.1×10^{-5}	1.1×10^{-5}	0%	4×10^{-6}	4×10^{-6}
Dieldrin	1.5×10^{-6}	1.6×10^{-6}	+7%	2×10^{-5}	3×10^{-5}
gamma-BHC	7.8×10^{-7}	7.5×10^{-7}	-4%	1×10^{-6}	1×10^{-6}
Heptachlor	5.9×10^{-7}	5.6×10^{-7}	-5%	3×10^{-6}	3×10^{-6}
Heptachlor epoxide	6.2×10^{-7}	6.1×10^{-7}	-2%	6×10^{-6}	6×10^{-6}
Hexachlorobenzene	1.2×10^{-6}	1.1×10^{-6}	-8%	2×10^{-6}	2×10^{-6}
Subtotal				**6×10^{-5}**	**7×10^{-5}**
Total risk across all exposure routes/pathways excluding PCB TEQ				**8×10^{-4}**	**8×10^{-4}**
Total risk across all exposure routes/pathways excluding total PCBs				**7×10^{-4}**	**7×10^{-4}**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for carcinogenic PAHs are from only 2004; historical data were not used because of high reporting limits. Risk related to 2004 and historical data for cPAHs were analyzed in the uncertainty analysis of the HHRA (Windward 2007).

BHC – benzene hexachloride
CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
DDT – dichlorodiphenyltrichloroethane
HHRA – human health risk assessment
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table 5. Comparison of excess cancer risk estimates for the child tribal CT seafood consumption scenario based on Tulalip data

Chemical	Cancer CDI (mg/kg-day)		Percent Change	Excess Cancer Risk	
	Final HHRA	Revised		Final HHRA	Revised
Arsenic[a, b]	1.8×10^{-5}	1.9×10^{-5}	+6%	3×10^{-5}	3×10^{-5}
Bis(2-ethylhexyl) phthalate	5.2×10^{-6}	5.3×10^{-6}	+2%	7×10^{-8}	7×10^{-8}
cPAHs[a, c]	2.1×10^{-7}	2.3×10^{-7}	+10%	8×10^{-6}	9×10^{-6}
PCB TEQ[a]	1.5×10^{-10}	1.5×10^{-10}	0%	2×10^{-5}	2×10^{-5}
Total PCBs	1.4×10^{-5}	1.3×10^{-5}	-7%	3×10^{-5}	3×10^{-5}
Pentachlorophenol	5.9×10^{-6}	6.1×10^{-6}	+3%	7×10^{-7}	7×10^{-7}
Subtotal excluding PCB TEQ				**7×10^{-5}**	**7×10^{-5}**
Subtotal excluding total PCBs				**6×10^{-5}**	**6×10^{-5}**
Tentatively identified chemicals (JN-qualified)					
Aldrin	3.7×10^{-8}	3.6×10^{-8}	-3%	6×10^{-7}	6×10^{-7}
alpha-BHC	3.7×10^{-8}	3.5×10^{-8}	-5%	2×10^{-7}	2×10^{-7}
beta-BHC	6.0×10^{-8}	5.9×10^{-8}	-2%	1×10^{-7}	1×10^{-7}
Carbazole	2.2×10^{-5}	2.0×10^{-5}	-9%	4×10^{-7}	4×10^{-7}
Total chlordane	2.5×10^{-7}	2.3×10^{-7}	-8%	9×10^{-8}	8×10^{-8}
Total DDTs	1.3×10^{-6}	1.2×10^{-6}	-8%	4×10^{-7}	4×10^{-7}
Dieldrin	6.9×10^{-8}	6.9×10^{-8}	0%	1×10^{-6}	1×10^{-6}
gamma-BHC	4.2×10^{-8}	4.0×10^{-8}	-5%	5×10^{-8}	5×10^{-8}
Heptachlor	3.8×10^{-8}	3.7×10^{-8}	-3%	2×10^{-7}	2×10^{-7}
Heptachlor epoxide	5.6×10^{-8}	5.3×10^{-8}	-5%	5×10^{-7}	5×10^{-7}
Hexachlorobenzene	6.6×10^{-8}	6.3×10^{-8}	-5%	1×10^{-7}	1×10^{-7}
Subtotal				**4×10^{-6}**	**4×10^{-6}**
Total risk across all exposure routes/pathways excluding PCB TEQ				**7×10^{-5}**	**7×10^{-5}**
Total risk across all exposure routes/pathways excluding total PCBs				**6×10^{-5}**	**6×10^{-5}**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] cPAH concentrations are presented as benzo(a)pyrene equivalents. Tissue data for carcinogenic PAHs are from only 2004; historical data were not used because of high reporting limits. Risk related to 2004 and historical data for cPAHs were analyzed in the uncertainty analysis of the HHRA (Windward 2007).

BHC – benzene hexachloride
CDI – chronic daily intake
cPAH – carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
DDT – dichlorodiphenyltrichloroethane
HHRA – human health risk assessment
PCB – polychlorinated biphenyl
TEQ – toxic equivalent

As shown in Tables 2 through 5, changes to the consumption rates resulted in increased CDIs and chemical-specific risk estimates in some cases and decreased CDIs and chemical-specific risk estimates in other cases. The largest increase in the cancer CDI (17% change) using the revised consumption rates was for carcinogenic polycyclic aromatic hydrocarbons (cPAHs) for the child tribal RME seafood consumption scenario based on the Tulalip data. The largest decrease in the cancer CDI (-9% change) using the revised consumption rates was for carbazole for the child tribal CT seafood consumption scenario based on the Tulalip data. Overall, the total excess cancer risk estimates across chemicals stayed the same or increased a relatively small amount.

Revised non-cancer CDIs and non-cancer hazard quotients for the four tribal seafood consumption scenarios based on Tulalip data were also calculated (Tables 6 through 9). Changes to the consumption rates resulted in increased non-cancer CDIs and hazard quotients in some cases and decreased non-cancer CDIs and hazard quotients in other cases. The largest increase in the non-cancer CDI (14% change) using the revised consumption rates was for tributyltin (TBT) for the adult tribal RME seafood consumption scenario based on the Tulalip data and also for arsenic for the child tribal RME seafood consumption scenario based on the Tulalip data. The largest decrease in the non-cancer CDI (-17% change) using the revised consumption rates was for butyl benzyl phthalate for the child tribal CT seafood consumption scenario based on the Tulalip data. In almost all cases, hazard indices by endpoint stayed the same or increased slightly.

Table 6. Comparison of non-cancer hazard estimates for the adult tribal RME seafood consumption scenario based on Tulalip data

Chemical	Non-Cancer CDI (mg/kg-day)		Percent Change	Hazard Quotient	
	Final HHRA	Revised		Final HHRA	Revised
4-Methylphenol	5.0×10^{-4}	4.6×10^{-4}	-8%	0.1	0.09
Antimony	4.7×10^{-5}	5.3×10^{-5}	+13%	0.1	0.1
Arsenic[a, b]	9.7×10^{-4}	1.1×10^{-3}	+13%	3	4
Bis(2-ethylhexyl) phthalate	4.5×10^{-4}	4.5×10^{-4}	0%	0.02	0.02
Butyl benzyl phthalate	5.4×10^{-4}	4.9×10^{-4}	-9%	0.003	0.002
Cadmium	9.3×10^{-5}	9.6×10^{-5}	+3%	0.09	0.1
Chromium	4.7×10^{-4}	5.1×10^{-4}	+9%	0.2	0.2
Copper	8.6×10^{-3}	8.3×10^{-3}	-3%	0.2	0.2
Mercury	5.1×10^{-5}	4.8×10^{-5}	-6%	0.5	0.5
Nickel	4.0×10^{-4}	4.5×10^{-4}	+13%	0.02	0.02
Total PCBs	8.0×10^{-4}	8.1×10^{-4}	+1%	40	40
Pentachlorophenol	7.3×10^{-4}	7.2×10^{-4}	-1%	0.02	0.02
TBT (as ion)	2.1×10^{-4}	2.4×10^{-4}	+14%	1	2
Vanadium	8.3×10^{-4}	9.3×10^{-4}	+12%	0.8	0.9
Zinc	3.4×10^{-2}	3.3×10^{-2}	-3%	0.1	0.1
Tentatively identified chemicals (JN-qualified)					
Aldrin	2.8×10^{-6}	2.7×10^{-6}	-4%	0.09	0.09
alpha-BHC	2.6×10^{-6}	2.4×10^{-6}	-8%	0.005	0.005
beta-BHC	3.5×10^{-6}	3.4×10^{-6}	-3%	0.02	0.02
Total chlordane	1.7×10^{-5}	1.6×10^{-5}	-6%	0.03	0.03
Total DDTs	6.2×10^{-5}	6.0×10^{-5}	-3%	0.1	0.1
Dieldrin	8.2×10^{-6}	8.8×10^{-6}	+7%	0.2	0.2
Endrin	3.0×10^{-6}	2.8×10^{-6}	-7%	0.01	0.009
Endrin aldehyde	1.1×10^{-5}	1.1×10^{-5}	0%	0.04	0.04
gamma-BHC	4.2×10^{-6}	4.1×10^{-6}	-2%	0.01	0.01
Heptachlor	3.2×10^{-6}	3.0×10^{-6}	-6%	0.006	0.006
Heptachlor epoxide	3.4×10^{-6}	3.3×10^{-6}	-3%	0.3	0.3
Hexachlorobenzene	6.5×10^{-6}	6.0×10^{-6}	-8%	0.008	0.008
Hazard index for cardiovascular endpoint[c]				**4**	**5**
Hazard index for developmental endpoint[d]				**41**	**41**
Hazard index for hematologic endpoint[e]				**0.2**	**0.2**
Hazard index for immunological endpoint[f]				**41**	**42**
Hazard index for kidney endpoint[g]				**0.4**	**0.4**
Hazard index for liver endpoint[h]				**1**	**1**
Hazard index for neurological endpoint[i]				**41**	**41**
Hazard index for dermal endpoint[j]				**3**	**4**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] Cardiovascular endpoint is for arsenic and vanadium.

[d] Developmental endpoint is for PCBs and mercury.

[e] Hematologic endpoint is for antimony and zinc.

[f] Immunological endpoint is for PCBs and TBT.

[g] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[h] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[i] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[j] Dermal endpoint is for 4-methylphenol and arsenic.

BHC – benzene hexachloride

CDI – chronic daily intake

DDT – dichlorodiphenyltrichloroethane

HHRA – human health risk assessment

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

TBT – tributyltin

Table 7. Comparison of non-cancer hazard estimates for the adult tribal CT seafood consumption scenario based on Tulalip data

Chemical	Non-Cancer CDI (mg/kg-day)		Percent Change	Hazard Quotient	
	Final HHRA	Revised		Final HHRA	Revised
4-Methylphenol	4.5×10^{-5}	4.1×10^{-5}	-9%	0.009	0.008
Antimony	4.0×10^{-6}	4.4×10^{-6}	+10%	0.01	0.01
Arsenic[a, b]	9.7×10^{-5}	1.0×10^{-4}	+3%	0.3	0.4
Bis(2-ethylhexyl) phthalate	2.8×10^{-5}	2.9×10^{-5}	+4%	0.001	0.002
Butyl benzyl phthalate	5.4×10^{-5}	4.9×10^{-5}	-9%	0.0003	0.0002
Cadmium	1.2×10^{-5}	1.3×10^{-5}	+8%	0.01	0.01
Chromium	5.5×10^{-5}	6.2×10^{-5}	+13%	0.02	0.02
Copper	1.1×10^{-3}	1.1×10^{-3}	0%	0.03	0.03
Mercury	6.9×10^{-6}	6.6×10^{-6}	-4%	0.07	0.07
Nickel	5.3×10^{-5}	5.8×10^{-5}	+9%	0.003	0.003
Total PCBs	7.5×10^{-5}	7.3×10^{-5}	-3%	4	4
Pentachlorophenol	3.2×10^{-5}	3.4×10^{-5}	+6%	0.001	0.001
TBT (as ion)	2.4×10^{-5}	2.7×10^{-5}	+13%	0.2	0.2
Vanadium	1.1×10^{-4}	1.2×10^{-4}	+9%	0.1	0.1
Zinc	4.8×10^{-3}	4.7×10^{-3}	-2%	0.02	0.02
Tentatively identified chemicals (JN-qualified)					
Aldrin	2.0×10^{-7}	1.9×10^{-7}	-5%	0.007	0.007
alpha-BHC	2.0×10^{-7}	1.9×10^{-7}	-5%	0.0004	0.0004
beta-BHC	3.3×10^{-7}	3.2×10^{-7}	-3%	0.002	0.002
Total chlordane	1.3×10^{-6}	1.3×10^{-6}	0%	0.003	0.003
Total DDTs	7.1×10^{-6}	6.9×10^{-6}	-3%	0.01	0.01
Dieldrin	3.7×10^{-7}	3.8×10^{-7}	+3%	0.007	0.008
Endrin	2.6×10^{-7}	2.5×10^{-7}	-4%	0.0009	0.0008
Endrin aldehyde	3.0×10^{-7}	2.9×10^{-7}	-3%	0.001	0.001
gamma-BHC	2.3×10^{-7}	2.2×10^{-7}	-4%	0.0008	0.0007
Heptachlor	2.1×10^{-7}	2.0×10^{-7}	-5%	0.0004	0.0004
Heptachlor epoxide	3.0×10^{-7}	2.9×10^{-7}	-3%	0.02	0.02
Hexachlorobenzene	3.6×10^{-7}	3.4×10^{-7}	-6%	0.0004	0.0004
Hazard index for cardiovascular endpoint[c]				**0.4**	**0.5**
Hazard index for developmental endpoint[d]				**4**	**4**
Hazard index for hematologic endpoint[e]				**0.03**	**0.03**
Hazard index for immunological endpoint[f]				**4**	**4**
Hazard index for kidney endpoint[g]				**0.05**	**0.05**
Hazard index for liver endpoint[h]				**0.1**	**0.1**
Hazard index for neurological endpoint[i]				**4**	**4**
Hazard index for dermal endpoint[j]				**0.3**	**0.4**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] Cardiovascular endpoint is for arsenic and vanadium.

[d] Developmental endpoint is for PCBs and mercury.

[e] Hematologic endpoint is for antimony and zinc.

[f] Immunological endpoint is for PCBs and TBT.

[g] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[h] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[i] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[j] Dermal endpoint is for 4-methylphenol and arsenic.

BHC – benzene hexachloride

CDI – chronic daily intake

CT – central tendency

DDT – dichlorodiphenyltrichloroethane

HHRA – human health risk assessment

J – estimated concentration

N – tentative identification

PCB – polychlorinated biphenyl

TBT – tributyltin

Table 8. Comparison of non-cancer hazard estimates for the child tribal RME seafood consumption scenario based on Tulalip data

Chemical	Non-Cancer CDI (mg/kg-day)		Percent Change	Hazard Quotient	
	Final HHRA	Revised		Final HHRA	Revised
4-Methylphenol	1.1×10^{-3}	9.9×10^{-4}	-10%	0.2	0.2
Antimony	1.0×10^{-4}	1.1×10^{-4}	+10%	0.3	0.3
Arsenic[a, b]	2.1×10^{-3}	2.4×10^{-3}	+14%	7	8
Bis(2-ethylhexyl) phthalate	9.7×10^{-4}	9.7×10^{-4}	0%	0.05	0.05
Butyl benzyl phthalate	1.2×10^{-3}	1.1×10^{-3}	-8%	0.006	0.005
Cadmium	2.0×10^{-4}	2.1×10^{-4}	+5%	0.2	0.2
Chromium	1.0×10^{-3}	1.1×10^{-3}	+10%	0.3	0.4
Copper	1.8×10^{-2}	1.8×10^{-2}	0%	0.5	0.4
Mercury	1.1×10^{-4}	1.0×10^{-4}	-9%	1	1
Nickel	8.7×10^{-4}	9.7×10^{-4}	+11%	0.04	0.05
PCBs (total calc'd)	1.7×10^{-3}	1.7×10^{-3}	0%	86	87
Pentachlorophenol	1.6×10^{-3}	1.5×10^{-3}	-6%	0.05	0.05
TBT (as ion)	4.5×10^{-4}	5.1×10^{-4}	+13%	3	3
Vanadium	1.8×10^{-3}	2.0×10^{-3}	+11%	2	2
Zinc	7.3×10^{-2}	7.2×10^{-2}	-1%	0.2	0.2
Tentatively identified chemicals (JN-qualified)					
Aldrin	6.1×10^{-6}	5.8×10^{-6}	-5%	0.2	0.2
alpha-BHC	5.6×10^{-6}	5.2×10^{-6}	-7%	0.01	0.01
beta-BHC	7.6×10^{-6}	7.3×10^{-6}	-4%	0.04	0.04
Chlordane (total calc'd)	3.6×10^{-5}	3.5×10^{-5}	-3%	0.07	0.07
DDTs (total calc'd)	1.3×10^{-4}	1.3×10^{-4}	0%	0.3	0.3
Dieldrin	1.8×10^{-5}	1.9×10^{-5}	+6%	0.4	0.4
Endrin	6.4×10^{-6}	6.0×10^{-6}	-6%	0.02	0.02
Endrin aldehyde	2.4×10^{-5}	2.4×10^{-5}	0%	0.08	0.08
gamma-BHC	9.1×10^{-6}	8.8×10^{-6}	-3%	0.03	0.03
Heptachlor	6.9×10^{-6}	6.5×10^{-6}	-6%	0.01	0.01
Heptachlor epoxide	7.3×10^{-6}	7.1×10^{-6}	-3%	0.6	0.5
Hexachlorobenzene	1.4×10^{-5}	1.3×10^{-5}	-7%	0.02	0.02
Hazard index for cardiovascular endpoint[c]				**9**	**10**
Hazard index for developmental endpoint[d]				**87**	**88**
Hazard index for hematologic endpoint[e]				**0.5**	**0.5**
Hazard index for immunological endpoint[f]				**89**	**90**
Hazard index for kidney endpoint[g]				**1**	**0.9**
Hazard index for liver endpoint[h]				**3**	**2**
Hazard index for neurological endpoint[i]				**87**	**88**
Hazard index for dermal endpoint[j]				**7**	**8**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] Cardiovascular endpoint is for arsenic and vanadium.

[d] Developmental endpoint is for PCBs and mercury.

[e] Hematologic endpoint is for antimony and zinc.

[f] Immunological endpoint is for PCBs and TBT.

[g] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[h] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[i] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[j] Dermal endpoint is for 4-methylphenol and arsenic.

BHC – benzene hexachloride

CDI – chronic daily intake

DDT – dichlorodiphenyltrichloroethane

HHRA – human health risk assessment

J – estimated concentration

N – tentative identification

PCB – polychlorinated biphenyl

RME – reasonable maximum exposure

TBT – tributyltin

Table 9. Comparison of non-cancer hazard estimates for the child tribal CT seafood consumption scenario based on Tulalip data

Chemical	Non-Cancer CDI (mg/kg-day)		Percent Change	Hazard Quotient	
	Final HHRA	Revised		Final HHRA	Revised
4-Methylphenol	9.7×10^{-5}	8.9×10^{-5}	-8%	0.02	0.02
Antimony	8.5×10^{-6}	9.4×10^{-6}	+11%	0.02	0.02
Arsenic[a, b]	2.1×10^{-4}	2.2×10^{-4}	+5%	0.7	0.7
Bis(2-ethylhexyl) phthalate	6.1×10^{-5}	6.2×10^{-5}	+2%	0.003	0.003
Butyl benzyl phthalate	1.2×10^{-4}	1.0×10^{-4}	-17%	0.0006	0.0005
Cadmium	2.7×10^{-5}	2.7×10^{-5}	0%	0.03	0.03
Chromium	1.2×10^{-4}	1.3×10^{-4}	+8%	0.04	0.04
Copper	2.5×10^{-3}	2.3×10^{-3}	-8%	0.06	0.06
Mercury	1.5×10^{-5}	1.4×10^{-5}	-7%	0.1	0.1
Nickel	1.1×10^{-4}	1.2×10^{-4}	+9%	0.006	0.006
Total PCBs	1.6×10^{-4}	1.5×10^{-4}	-6%	8	8
Pentachlorophenol	6.9×10^{-5}	7.2×10^{-5}	+4%	0.002	0.002
TBT (as ion)	5.2×10^{-5}	5.8×10^{-5}	+12%	0.3	0.4
Vanadium	2.3×10^{-4}	2.5×10^{-4}	+9%	0.2	0.3
Zinc	1.0×10^{-2}	1.0×10^{-2}	0%	0.03	0.03
Tentatively identified chemicals (JN-qualified)					
Aldrin	4.4×10^{-7}	4.2×10^{-7}	-5%	0.01	0.01
alpha-BHC	4.3×10^{-7}	4.1×10^{-7}	-5%	0.0009	0.0008
beta-BHC	7.0×10^{-7}	6.8×10^{-7}	-3%	0.004	0.003
Total chlordane	2.9×10^{-6}	2.7×10^{-6}	-7%	0.006	0.005
Total DDTs	1.5×10^{-5}	1.4×10^{-5}	-7%	0.03	0.03
Dieldrin	8.0×10^{-7}	8.0×10^{-7}	0%	0.02	0.02
Endrin	5.6×10^{-7}	5.3×10^{-7}	-5%	0.002	0.002
Endrin aldehyde	6.5×10^{-7}	6.2×10^{-7}	-5%	0.002	0.002
gamma-BHC	4.9×10^{-7}	4.7×10^{-7}	-4%	0.002	0.002
Heptachlor	4.5×10^{-7}	4.3×10^{-7}	-4%	0.0009	0.0009
Heptachlor epoxide	6.5×10^{-7}	6.1×10^{-7}	-6%	0.05	0.05
Hexachlorobenzene	7.7×10^{-7}	7.3×10^{-7}	-5%	0.001	0.0009
Hazard index for cardiovascular endpoint[c]				**0.9**	**1**
Hazard index for developmental endpoint[d]				**8**	**8**
Hazard index for hematologic endpoint[e]				**0.05**	**0.05**
Hazard index for immunological endpoint[f]				**8**	**8**
Hazard index for kidney endpoint[g]				**0.1**	**0.1**
Hazard index for liver endpoint[h]				**0.3**	**0.3**
Hazard index for neurological endpoint[i]				**8**	**8**
Hazard index for dermal endpoint[j]				**0.7**	**0.7**

[a] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[b] Arsenic risk estimates are based on inorganic arsenic.

[c] Cardiovascular endpoint is for arsenic and vanadium.

[d] Developmental endpoint is for PCBs and mercury.

[e] Hematologic endpoint is for antimony and zinc.

[f] Immunological endpoint is for PCBs and TBT.

[g] Kidney endpoint is for 4-methylphenol, cadmium, copper, gamma-BHC, and pentachlorophenol.

[h] Liver endpoint is for 4-methylphenol, aldrin, alpha-BHC, beta-BHC, bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, chlordane, copper, total DDTs, dieldrin, endrin, endrin aldehyde, gamma-BHC, heptachlor, heptachlor epoxide, hexachlorobenzene, and pentachlorophenol.

[i] Neurological endpoint is for 4-methylphenol, mercury, and total PCBs.

[j] Dermal endpoint is for 4-methylphenol and arsenic.

BHC – benzene hexachloride

CDI – chronic daily intake

CT – central tendency

DDT – dichlorodiphenyltrichloroethane

HHRA – human health risk assessment

J – estimated concentration

N – tentative identification

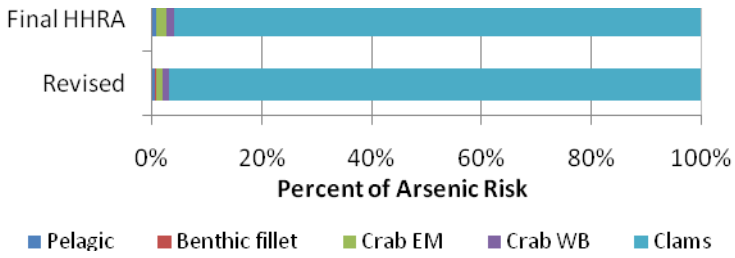
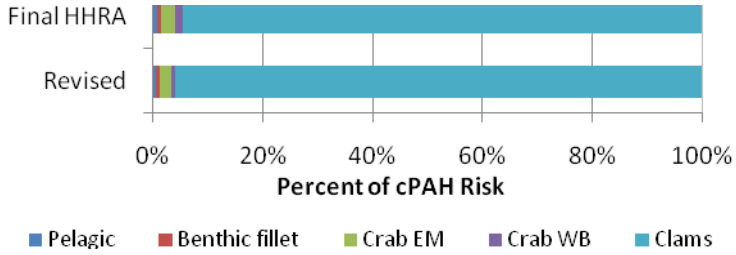
PCB – polychlorinated biphenyl

TBT – tributyltin

UPDATED RISK ESTIMATES BY SEAFOOD CATEGORY

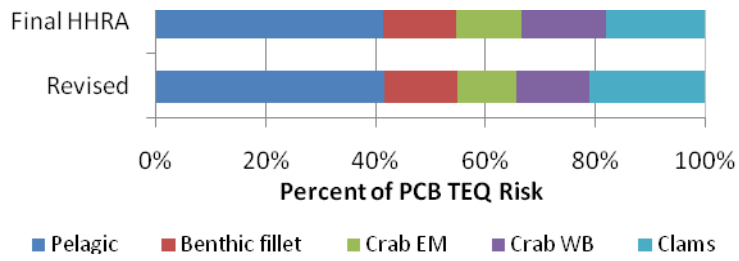
This section summarizes the contribution of different seafood consumption categories to the total risk estimates by chemical (for arsenic, cPAHs, polychlorinated biphenyl [PCB] toxic equivalent [TEQ], and total PCB TEQ) based on the consumption rates used in the final HHRA and the revised consumption rates. Comparisons of the percent contribution to the risk estimates for each seafood consumption category are presented in Tables 10 and 11 for the adult/child tribal RME and the adult/child tribal CT seafood consumption scenarios, respectively, based on Tulalip data. For all four scenarios, the estimates using the revised consumption rates, which assumed a higher consumption of clams and a lower consumption of crabs, resulted in a slightly higher percentage of the risk attributable to clams and a lower percentage of risk attributable to crabs.

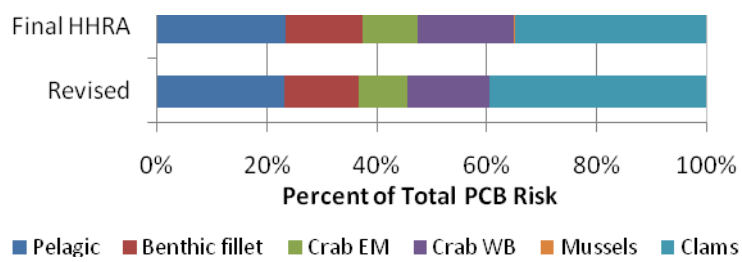
Table 10. Comparison of the percent contribution of seafood consumption categories to the excess cancer risks and non-cancer hazards for selected chemicals in the adult tribal RME and child tribal RME seafood consumption scenarios based on Tulalip data

Consumption Category	Adult Consumption Rate (g/day)		Child Consumption Rate (g/day)		Percent of Chemical's Risk		Adult or Child Tribal RME (Tulalip data) Seafood Consumption Risk Expressed as Percentage of the Total Risk[a]
	Final HHRA	Revised	Final HHRA	Revised	Final HHRA	Revised	
Arsenic[b]							
Pelagic	8.2	8.2	3.3	3.2	0.9%	0.8%	
Benthic – fillet	7.6	7.6	3.0	3.0	0.06%	0.05%	
Crab – edible meat	33	29	13	12	1.8%	1.3%	
Crab – whole body	10	9.1	4.2	3.6	1.5%	1.1%	
Clams	38	44	15	18	95.8%	96.7%	
cPAHs (2004 data only)[b, c]							
Pelagic	8.2	8.2	3.3	3.2	1.0%	0.8%	
Benthic – fillet	7.6	7.6	3.0	3.0	0.6%	0.5%	
Crab – edible meat	33	29	13	12	2.7%	2.1%	
Crab – whole body	10	9.1	4.2	3.6	1.2%	0.9%	
Clams	38	44	15	18	94.5%	95.7%	

Consumption Category	Adult Consumption Rate (g/day)		Child Consumption Rate (g/day)		Percent of Chemical's Risk	
	Final HHRA	Revised	Final HHRA	Revised	Final HHRA	Revised
PCB TEQ[b]						
Pelagic	8.2	8.2	3.3	3.2	41.4%	41.7%
Benthic – fillet	7.6	7.6	3.0	3.0	13.3%	13.4%
Crab – edible meat	33	29	13	12	12.0%	10.6%
Crab – whole body	10	9.1	4.2	3.6	15.3%	13.3%
Clams	38	44	15	18	18.0%	21.0%
Total PCBs[d]						
Pelagic	8.1	8.1	3.2	3.2	23.6%	23.3%
Benthic – fillet	7.5	7.5	3.0	3.0	13.8%	13.6%
Crab – edible meat	33	29	13	12	10.1%	8.7%
Crab – whole body	10	9.0	4.2	3.6	17.6%	15.0%
Mussels	0.82	0.80	0.33	0.30	0.05%	0.05%
Clams	38	43	15	17	34.8%	39.4%

Adult or Child Tribal RME (Tulalip data) Seafood Consumption Risk Expressed as Percentage of the Total Risk[a]





[a] Figures represent both cancer and non-cancer risks. Risk percentages are based on EPC and consumption rates, meaning that the percentage of risk from each consumption category is the same for cancer and non-cancer risks.

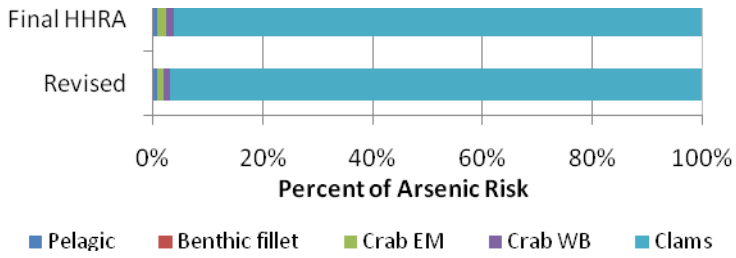
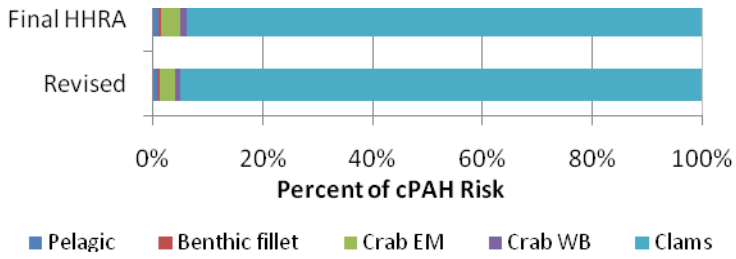
[b] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (HHRA, Section B.6).

[d] Although total PCBs data for mussels were included in the risk estimates, mussels are not shown in the bar charts because their percentage of total risk was so small.

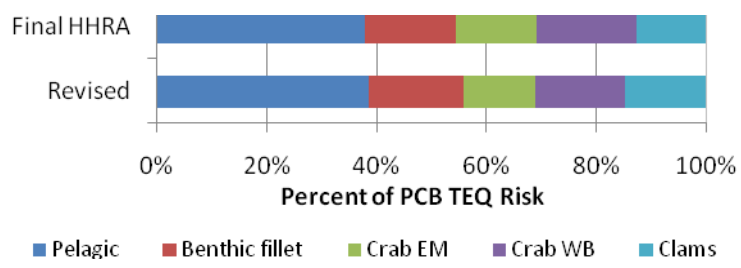
cPAH – carcinogenic polycyclic aromatic hydrocarbon
EM – edible meat
EPC – exposure point concentration
HHRA – human health risk assessment
na – not applicable
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent
WB – whole body

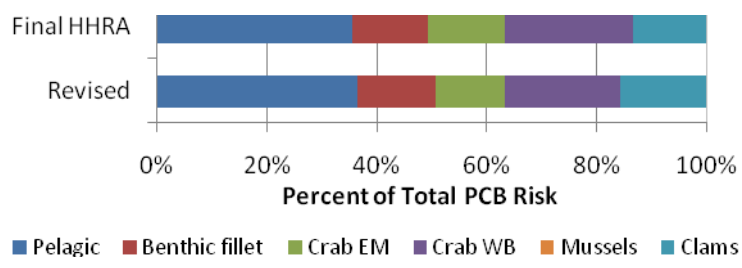
Table 11. Comparison of the percent contribution of seafood consumption categories to the excess cancer risks and non-cancer hazards for selected chemicals in the adult tribal CT and child tribal CT seafood consumption scenarios based on Tulalip data

Consumption Category	Adult Consumption Rate (g/day)		Child Consumption Rate (g/day)		Percent of Chemical's Risk		Adult or Child Tribal RME (Tulalip data) Seafood Consumption Risk Expressed as Percentage of the Total Risk[a]
	Final HHRA	Revised	Final HHRA	Revised	Final HHRA	Revised	
Arsenic[b]							
Pelagic	1.4	1.3	0.55	0.52	0.9%	0.9%	
Benthic – fillet	1.3	1.2	0.51	0.48	0.06%	0.06%	
Crab – edible meat	5.3	4.4	2.1	1.8	1.5%	1.2%	
Crab – whole body	1.7	1.4	0.68	0.60	1.6%	1.2%	
Clams	6.1	6.6	2.4	2.6	95.8%	96.6%	
cPAHs (2004 data only)[b, c]							
Pelagic	1.4	1.3	0.55	0.52	1.1%	1.0%	
Benthic fillet	1.3	1.2	0.51	0.48	0.6%	0.5%	
Crab –edible meat	5.3	4.4	2.1	1.8	3.4%	2.7%	
Crab – whole body	1.7	1.4	0.68	0.60	1.3%	1.0%	
Clams	6.1	6.6	2.4	2.6	93.6%	94.8%	

Consumption Category	Adult Consumption Rate (g/day)		Child Consumption Rate (g/day)		Percent of Chemical's Risk	
	Final HHRA	Revised	Final HHRA	Revised	Final HHRA	Revised
PCB TEQ[b]						
Pelagic	1.4	1.3	0.55	0.52	37.9%	38.8%
Benthic – fillet	1.3	1.2	0.51	0.48	16.7%	17.1%
Crab – edible meat	5.3	4.4	2.1	1.8	14.7%	13.2%
Crab – whole body	1.7	1.4	0.68	0.60	18.1%	16.2%
Clams	6.1	6.6	2.4	2.6	12.6%	14.7%
Total PCBs[d]						
Pelagic	1.3	1.3	0.52	0.52	35.6%	36.6%
Benthic – fillet	1.2	1.2	0.48	0.48	13.8%	14.1%
Crab – edible meat	5.0	4.4	2.0	1.8	13.9%	12.6%
Crab – whole body	1.6	1.4	0.64	0.60	23.3%	21.0%
Mussels	0.10	0.10	0.40	0.04	0.06%	0.06%
Clams	5.8	6.6	2.3	2.6	13.3%	15.6%

Adult or Child Tribal RME (Tulalip data) Seafood Consumption Risk Expressed as Percentage of the Total Risk[a]





[a] Figures represent both cancer and non-cancer risks. Risk percentages are based on EPC and consumption rates, meaning that the percentage of risk from each consumption category is the same for cancer and non-cancer risks.

[b] No mussel data were available for this chemical. When calculating the CDI and risk estimates, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[c] cPAH concentrations are given in terms of benzo(a)pyrene equivalents. Data used in the risk characterization portion of this document are from only 2004 because of high reporting limits in historical data. All cPAH data are analyzed in the uncertainty analysis (HHRA, Section B.6).

[d] Although total PCBs data for mussels were included in the risk estimates, mussels are not shown in the bar charts because their percentage of total risk was so small.

cPAH –carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
EM – edible meat
EPC – exposure point concentration
HHRA – human health risk assessment
na – not applicable
PCB – polychlorinated biphenyl
TEQ – toxic equivalent
WB – whole body

CONCLUSIONS

As presented in these errata, the revised consumption rates for the child and adult tribal seafood consumption scenarios based on Tulalip data do not result in substantial changes to the risk estimates presented in the final LDW HHRA (Windward 2007). Risk estimates increased for some chemicals and decreased for other chemicals. In addition, although the revised consumption rates resulted in a slightly higher percentage of the risk attributable to clams, this change was minimal. The changes in excess cancer risk estimates or hazard quotients with the revised shellfish consumption rates do not change the chemicals of concern or risk drivers for any of the Tulalip survey-based seafood consumption scenarios that were identified in the final HHRA.

References

EPA. 2007. Framework for selecting and using tribal fish and shellfish consumption rates for risk-based decision making at CERCLA and RCRA cleanup sites in Puget Sound and the Strait of Georgia. U.S. Environmental Protection Agency, Region 10, Seattle, WA.

EPA. 2009. EPA and Ecology comments on Revised Draft Remedial Investigation report, dated October 30, 2008; comments dated February 23, 2009. US Environmental Protection Agency Region 10, Seattle, WA.

Windward. 2007. Lower Duwamish Waterway remedial investigation. Baseline human health risk assessment. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle, WA.