



HANFORD NATURAL RESOURCE DAMAGE ASSESSMENT INJURY ASSESSMENT PLAN



Hanford Natural Resource Trustees

U.S. Department of Energy

U.S. Department of the Interior

U.S. Department of Commerce

State of Washington

State of Oregon

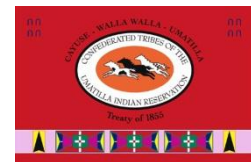
Yakama Nation

Confederated Tribes of the Umatilla Indian Reservation

Nez Perce Tribe

FINAL

January 31, 2013



LEGAL DISCLAIMER

This report was prepared as an account of work sponsored by an agency of the United States Government. Neither the United States Government nor any agency thereof, nor any of their employees, makes any warranty, express or implied, or assumes any legal liability or responsibility for the accuracy, completeness, or any third party's use or the results of such use of any information, apparatus, product, or process disclosed, or represents that its use would not infringe privately owned rights. Reference herein to any specific commercial product, process, or service by trade name, trademark, manufacturer, or otherwise, does not necessarily constitute or imply its endorsement, recommendation, or favoring by the United States Government or any agency thereof or its contractors or subcontractors. The views and opinions of authors expressed herein do not necessarily state or reflect those of the United States Government or any agency thereof.

TABLE OF CONTENTS

EXECUTIVE SUMMARY

CHAPTER 1 INTRODUCTION

- 1.1 Trusteeship 1-4
- 1.2 The Natural Resource Damage Assessment Process 1-5
- 1.3 Assessment Activities at Hanford 1-10
 - Summary 1-10
 - Use of Available Data 1-10
 - Coordination with Site Remedial Activities 1-12
 - Cooperation with the Responsible Party 1-12
 - Geographic Scope 1-12
 - Temporal Scope 1-12
- 1.4 Public Participation 1-13
 - Public Review and Comment 1-13
- 1.5 Schedule for Injury Assessment 1-14
- 1.6 Plan Organization 1-14

CHAPTER 2 BACKGROUND INFORMATION

- 2.1 Site History 2-1
 - Major Landscape Features and Setting 2-1
 - Tribal Presence 2-3
 - Other Land Use/Development 2-6
 - Federal Government Operations 2-7
- 2.2 Overview of Releases of Hazardous Substances 2-9
 - 300 Area 2-9
 - 100 Area 2-10
 - 200 Area 2-11
 - Other Releases 2-13
 - Hanford Site Hazardous Substances 2-14

CHAPTER 3 HABITATS, NATURAL RESOURCES, AND ASSOCIATED ECOLOGICAL SERVICES

- 3.1 Aquatic Habitats 3-1
 - Columbia River 3-1
 - Springs and Streams 3-3
 - Ponds and Ditches 3-4
- 3.2 Terrestrial Habitats 3-4
 - Shrub-Steppe 3-4
 - Sand Dunes 3-5
 - White Bluffs 3-6

- Columbia River Islands 3-6
- Basalt Outcrops, Scarps, and Screens 3-7
- Abandoned Fields/Disturbed Areas 3-7
- 3.3 Natural Resources 3-8
 - Surface Water Resources 3-8
 - Geological Resources 3-9
 - Groundwater Resources 3-11
 - Biological Resources 3-13
 - Air 3-19
- 3.4 Ecological Services 3-19
- 3.5 Preliminary Determination of Recovery Period 3-20

CHAPTER 4 NATURAL RESOURCE HUMAN USE SERVICES

- 4.1 Natural Resource Services Provided to Tribal Communities 4-1
- 4.2 Non-Tribal Human Use Services 4-9
 - Recreation 4-9
 - Agriculture 4-14

CHAPTER 5 CONFIRMATION OF EXPOSURE AND INJURY ASSESSMENT PROCESS

- 5.1 Confirmation of Exposure 5-1
 - Surface Water 5-1
 - Sediment 5-2
 - Geological (Soil) 5-2
 - Groundwater 5-2
 - Biological 5-3
- 5.2 Injury Determination 5-4
 - Pathway 5-4
 - Determining Injury 5-5
- 5.3 Injury Quantification 5-6
 - Baseline 5-7
 - Ecological Injury Quantification 5-8
 - Groundwater Injury Quantification 5-8
 - Lost Human Use Services Quantification 5-10
 - Remediation-Related Impacts 5-14

CHAPTER 6 DEFINITION OF INJURY

- 6.1 Surface Water 6-1
- 6.2 Groundwater 6-2
- 6.3 Geological 6-3
- 6.4 Biological 6-3
- 6.5 Air 6-6
- 6.6 Linking Injury Studies to DOI Regulations 6-6

CHAPTER 7 INJURY ASSESSMENT STUDIES

- 7.1 Introduction 7-1
 - Efforts to Date 7-2
 - Injury Studies 7-2
- 7.2 Aquatic Resources 7-10
 - Overview of Existing Site Aquatic Resource Data 7-10
 - Implications of Existing Data for Injury Assessment 7-16
 - Surface Water: Comparison to Injury Thresholds 7-16
 - Sediment: Comparison with Effects Thresholds 7-17
 - Aquatic Biota: Comparison to Effects Thresholds – Tissues 7-18
 - Aquatic Biota: Review of Hanford Sediment and Pore Water Toxicity Studies 7-20
 - Benthic Invertebrates: Sediment Toxicity Testing 7-20
 - Mussels: Distribution, Abundance, and Histopathology 7-22
 - Mussels: Toxicity Testing 7-23
 - Mussels: Caged (*In-Situ*) Study 7-24
 - Fish: Chinook Salmon Spawning Habitat Evaluation 7-24
 - Fish: Chinook Salmon Artificial Redd Evaluation 7-26
 - Fish: Prickly Sculpin Habitat Use 7-26
 - Fish: Early Life Stage Sculpin, White Sturgeon, and Rainbow Trout Toxicity Testing 7-28
 - Aquatic Resources: Quantification of Lost Aquatic Ecological Services 7-29
- 7.3 Terrestrial Resources 7-29
 - Overview of Existing Site Terrestrial Resource Data 7-29
 - Implications of Existing Data for Injury Assessment 7-32
 - Soils: Comparison with Effects Thresholds 7-33
 - Soils: Geospatial Evaluation 7-33
 - Terrestrial Biota: Comparison with Injury Thresholds-Tissues 7-35
 - Terrestrial Biota: Review of Hanford Soil Toxicity Studies 7-36
 - Plants: Native Plant Toxicity Testing 7-37
 - Plants: Assessment of Plant Community Health 7-39
 - Invertebrates: Nematode Toxicity Testing 7-40
 - Invertebrates: Assessment of Terrestrial Invertebrate Abundance 7-41
 - Birds: Assessment of Avian Abundance and Diversity 7-42
 - Birds: Evaluation of Exposure to Hanford Site Avian Species 7-42
 - Mammals: Small Mammal Population Assessment 7-43
 - Mammals: Great Basin Pocket Mouse - Carbon Tetrachloride and Histopathology 7-44
 - Terrestrial Resources: Impacts of Remedial Activities 7-45
 - Terrestrial Resources: Quantification of Lost Terrestrial Ecological Services 7-46
- 7.4 Vadose/Geological Resources 7-46
 - Overview of Existing Site Vadose Zone Data 7-47
 - Implications of Existing Data for Injury Assessment 7-48

Characterizing Vadose Zone (Geological Resource) Contamination and the Potential for Long-term Injury to Groundwater and Surface Water due to Contaminants that have been Released to the Vadose Zone 7-48

Evaluation of Existing Vadose Zone Models 7-49

7.5 Groundwater 7-50

 Overview of Existing Site Groundwater Resource Data 7-50

 Implications of Existing Data for Injury Assessment 7-52

 Developing a Comprehensive Database and Comparison to Injury Thresholds 7-53

 Review of Contaminant Plume Mapping 7-54

 Define the Legal, Political, and Economic Environment for Baseline Services Provided by Groundwater 7-55

 Verify Validity and Limitations to Hanford Groundwater Models 7-55

 Groundwater Upwellings 7-56

 Synoptic Sampling of River Corridor Wells 7-57

 Vertical Distribution of Contaminant Plumes 7-57

 Geology of Columbia River Bed 7-58

 Quantify Injured Groundwater Volume and Time Dimensions 7-58

7.6 Tribal Use 7-59

 Ethnographic Study to Identify Traditional Cultural Properties at Hanford 7-59

 Assess Tribal Service Losses 7-61

 Current Resource Characterization to Allow for Restoration of Lost Tribal Services 7-62

7.7 Other Human Uses 7-65

 Inventory of Institutional Controls Related to the Release of Hazardous Substances, and Description of Associated Limits on Human Use of the Site 7-65

7.8 All Resources 7-66

 Treatment of Non-Detects in Studies Analyzing Existing Data 7-66

CHAPTER 8 QUALITY ASSURANCE MANAGEMENT

8.1 Project Management 8-2

8.2 Quality System Description 8-3

8.3 Data Generation and Acquisition 8-5

 New Data Generation Activities 8-5

 Historical Data Acquisition and Use 8-5

8.4 Assessment and Oversight 8-6

8.5 Data Validation and Usability 8-6

REFERENCES R-1

APPENDICES

APPENDIX A THE FOUR HANFORD NPL SITES

APPENDIX B ECOTOXICITY SUMMARIES FOR SELECTED CONTAMINANTS

APPENDIX C SPECIES DOCUMENTED ON THE HANFORD SITE

LIST OF EXHIBITS

| | |
|--------------|---|
| Exhibit ES-1 | Summary of Injury Assessment Studies <i>ES-2</i> |
| Exhibit ES-2 | Phases of the Natural Resource Damage Assessment Process <i>ES-8</i> |
| Exhibit ES-3 | Preliminary List of Contaminants of Potential Concern <i>ES-10</i> |
| Exhibit 1-1 | Hanford Site <i>1-2</i> |
| Exhibit 1-2 | Assessment Phase Components <i>1-9</i> |
| Exhibit 1-3 | Summary of the Natural Resource Damage Assessment Process at Hanford <i>1-11</i> |
| Exhibit 2-1 | Central Hanford and the Hanford Reach National Monument <i>2-2</i> |
| Exhibit 2-2 | Preliminary Assessment-Focused List of Hazardous Substances <i>2-14</i> |
| Exhibit 3-1 | Surface Water Features on Hanford Site <i>3-10</i> |
| Exhibit 4-1 | Hanford Tribal Services Matrix <i>4-5</i> |
| Exhibit 6-1 | Linking Injury Assessment Plan Studies to DOI Natural Resource Damage Assessment Regulations <i>6-7</i> |
| Exhibit 7-1 | Overview of Injury Assessment Studies <i>7-6</i> |
| Exhibit 8-1 | Quality Assurance Management Organization for the Hanford Natural Resource Damage Assessment <i>8-2</i> |
| Exhibit 8-2 | Components of the Quality System <i>8-4</i> |

LIST OF ACRONYMS

| | |
|--------|---|
| ALE | Arid Lands Ecology |
| ANN | Artificial Neural Network |
| ASTM | American Society for Testing and Materials |
| ATSDR | Agency for Toxic Substances and Disease Registry |
| CERCLA | Comprehensive Environmental Response, Compensation, and Liability Act |
| COPC | Contaminant of Potential Concern |
| CTUIR | Confederated Tribes of the Umatilla Indian Reservation |
| CWA | Clean Water Act |
| DAP | Data Acquisition Plan |
| DMP | Data Management Plan |
| DMS | Data Management System |
| DOE | U.S. Department of Energy |
| DOI | Department of the Interior |
| EPA | U.S. Environmental Protection Agency |
| ERDF | Environmental Restoration Disposal Facility |
| ESA | Endangered Species Act |
| FEMA | Federal Emergency Management Agency |
| FS | Feasibility Study |
| HEIS | Hanford Environmental Information System |
| HH&E | Human Health and Environment |
| HLAN | Hanford Local Area Network |
| HNRTC | Hanford Natural Resource Trustee Council |
| IAP | Injury Assessment Plan |
| MC | Markov chain |
| MOA | Memorandum of Agreement |
| NEPA | National Environmental Policy Act |
| NOAA | National Oceanic and Atmospheric Administration |
| NPL | National Priorities List |
| NPT | Nez Perce Tribe |
| NRD | Natural Resource Damage |
| NRDA | Natural Resource Damage Assessment |
| OU | Operable Unit |
| PAS | Pre-assessment Screen |
| PNNL | Pacific Northwest National Laboratory |
| QA | Quality Assurance |
| QAP | Quality Assurance Plan |

| | |
|--------|---|
| QAPP | Quality Assurance Project Plan |
| QA/QC | Quality Assurance/Quality Control |
| QC | Quality Control |
| QMP | Quality Management Plan |
| RCDP | Restoration and Compensation Determination Plan |
| RCBRA | River Corridor Baseline Risk Assessment |
| RI | Remedial Investigation |
| RI/FS | Remedial Investigation/Feasibility Study |
| RM | River Mile |
| SDWA | Surface Drinking Water Act |
| SOPs | Standard Operating Procedures |
| TCP | Traditional Cultural Property |
| TEDF | Treated Effluent Disposal Facility |
| TNC | The Nature Conservancy |
| TP | Transition Probability |
| TWG | Technical Working Group |
| UCWSRI | Upper Columbia White Sturgeon Recovery Initiative |
| US FWS | U.S. Fish and Wildlife Service |
| USGS | U.S. Geological Survey |
| VOC | Volatile Organic Compound |
| WCH | Washington Closure Hanford |
| WDFW | Washington Department of Fish and Wildlife |

EXECUTIVE SUMMARY

This Injury Assessment Plan is intended to describe the Hanford Trustees' current understanding of the studies necessary to determine and quantify contaminant-related injury to Hanford Site natural resources and to assess associated service losses.

The identified studies, which are summarized in Exhibit ES-1, include efforts to carefully evaluate existing information as well as efforts designed to generate new information relevant to natural resource injury determination and quantification.

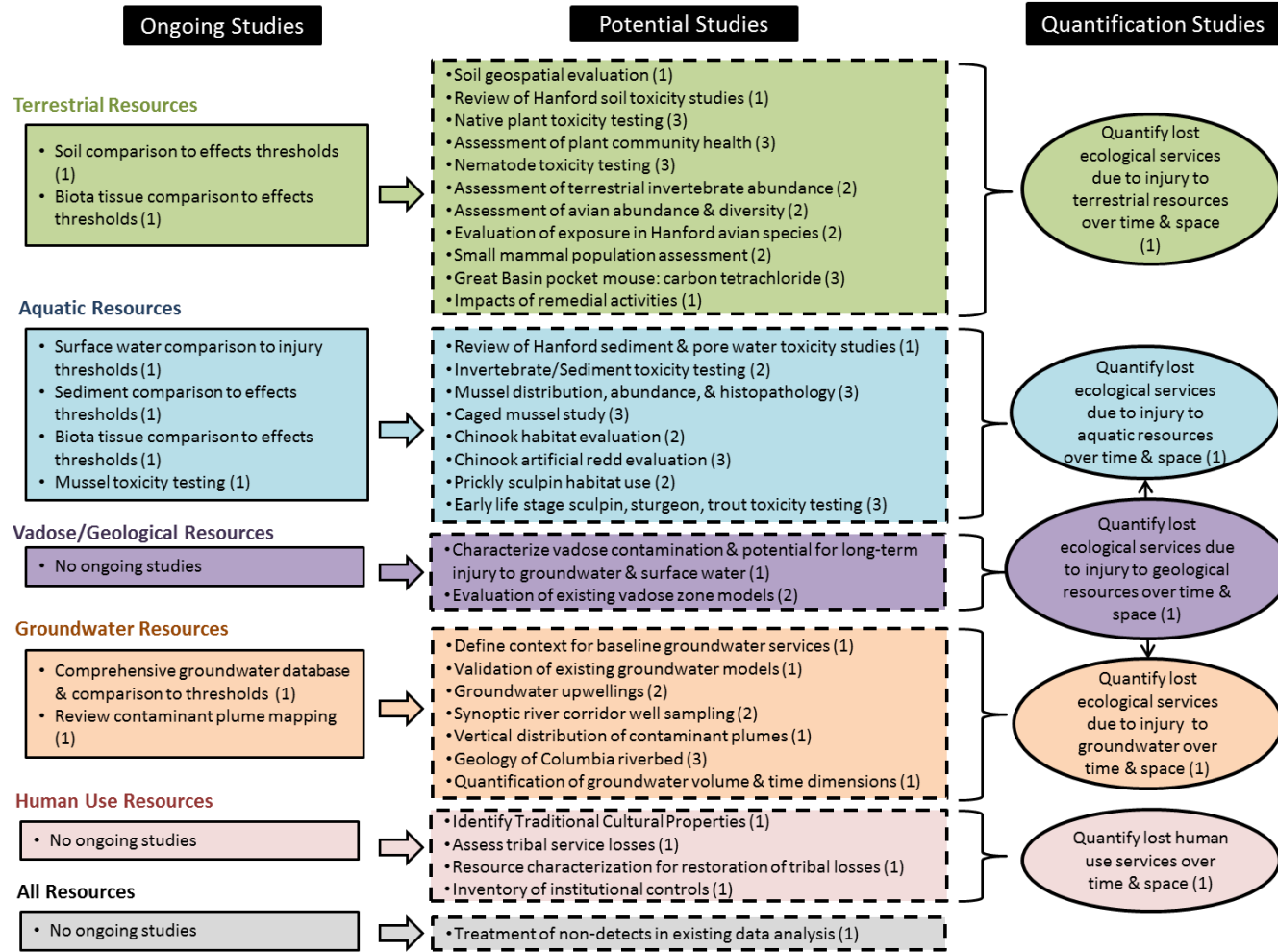
The Trustees have selected these studies and produced this document as part of their duties in connection with the ongoing Hanford natural resource damage assessment (NRDA). The following paragraphs describe the purpose and need for a NRDA, identify opportunities for public involvement, describe the identity and role of the Trustees (who work on behalf of the public), provide more information about NRDA, and briefly summarize the events and general processes undertaken by the Trustees that resulted in the selection of the indicated studies.

PURPOSE AND NEED Public lands, waters, air, and living resources are held in trust for the benefit of all people and future generations. Since the 1970s, the U.S. Congress has enacted a number of statutes to protect and manage the natural resources that belong to all Americans. Certain of these statutes designate natural resource Trustees. These Trustees serve as stewards of natural resources on behalf of the public, identifying potential natural resource injuries and restoring resources when they are threatened or harmed by releases of hazardous substances. In the case of Hanford, designated Trustees include several Federal agencies as well as states and tribes.

Since 1943, activities on the Hanford Site in south-central Washington have resulted in the widespread release of a large volume of radiological and other hazardous contaminants into the environment. Cleanup of the Site began around 1989 and will continue for several more decades. While cleanup efforts continue, the Hanford Natural Resource Trustees are conducting a natural resource damage assessment.

EXHIBIT ES-1

SUMMARY OF INJURY ASSESSMENT STUDIES



Notes: The studies for quantifying lost services due to injury to geological and groundwater resources, and the study for quantifying lost human use services are listed here for illustrative purposes and are not unique studies described in Chapter 7; these studies will be completed using the results of the other resource-specific studies. The numbers in parentheses indicates the priority group of each study, as described in the text. Additional studies may be added to this list.

The goal of the NRDA is to restore, replace, or acquire the equivalent of natural resources that have been injured as a result of the release of hazardous substances.

Trustees undertake natural resource damage assessments on behalf of the public. The purpose of these assessments is to define the scope and scale of natural resource restoration required to make the public whole for natural resource injuries and associated service losses.

As defined by the Department of the Interior's (DOI) regulations implementing the damage assessment provisions of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), the purpose of this Plan is to outline the approach the Hanford Trustees will take to assess injuries to natural resources stemming from releases of Site-related hazardous substances. The development of a Plan is intended to ensure that the natural resource damage assessment is conducted in a planned and systematic manner and at a reasonable cost (43 CFR § 11.30(b)). This Injury Assessment Plan describes ongoing and anticipated studies designed to evaluate past, current, and future natural resource injury and associated losses of resource services. Ultimately, the information collected through implementation of this Plan will inform the scope and scale of restoration activities needed to make the public whole for natural resource injuries and associated service losses.

This Plan describes the Trustees' current understanding of the studies necessary to determine and quantify injury to Site resources and resource services. The studies have been initially grouped into three general prioritization categories (nearer-term, middle-term, and longer-term). The exact timing of studies has not been determined and will depend on a number of considerations including but not limited to available funding. The DOI regulations also provide that an assessment plan may be modified as new information becomes available (43 CFR § Section 11.33(e)). Implementation of initial studies may result in the addition of studies to the current list, and may deprioritize others.

**PUBLIC
INVOLVEMENT**

The DOI regulations provide that an assessment plan, as well as any significant subsequent revisions that may be made to it, be made available for review and comment by potentially responsible parties, other natural resource trustees, other affected Federal, state, or tribal agencies, and any other interested members of the public for a period of at least 30 calendar days, with reasonable extensions granted as appropriate (43 CFR § 11.32(c) and (e)).

The Trustees are interested in receiving feedback on this Injury Assessment Plan. To facilitate this process, the Trustees are asking the public to review the Assessment Plan and provide feedback on the proposed approach and studies. Comments should be

submitted by December 31, 2012. These comments will help the Trustees plan and conduct an assessment that is scientifically valid, cost effective, and that incorporates a broad array of perspectives. To that end, the Trustees request that you carefully consider this Plan and provide any comments you may have.

Modifications to the Assessment Plan documents may occur at any time during the Assessment phase as new and additional information becomes available. Such modifications may result in additional need for public notification and opportunities for comment. Minor modifications could result in public notification, but need not result in delay of the implementation of those modifications pending public comment (43 CFR § 11.32(e)).

Commenters are encouraged to submit electronic comments to Larry.Goldstein@ecy.wa.gov. Comments can also be sent via U.S. mail to:

Mr. Larry Goldstein
Hanford Natural Resource Trustee Council Chair
Washington State Department of Ecology
Nuclear Waste Program
PO Box 47600
Olympia, WA 47600

For more information, please visit www.hanfordnrda.org.

**THE HANFORD
TRUSTEES**

Designated Federal, state, and tribal entities are authorized to act as Trustees of natural resources on behalf of the public.¹ In this role, Trustees may assess and recover damages for natural resource injuries resulting from the release of hazardous substances to the environment to ensure that the services that would have been provided by the injured resources but for Hanford Site-related contamination are restored, and the public made whole. Natural Resource Trustees for the Hanford Assessment Area include:

- U.S. Department of Energy (DOE);
- U.S. Department of the Interior (DOI) through U.S. Fish and Wildlife Service (US FWS);
- U.S. Department of Commerce through the National Oceanic and Atmospheric Administration (NOAA);
- State of Washington, through the Washington Department of Ecology, in consultation with the Washington Department of Fish and Wildlife (WDFW);

¹ More specifically, CERCLA as amended (42 U.S.C. 9601 *et. seq.*), the Oil Pollution Act of 1990 (OPA) (33 U.S.C. 2701 *et. seq.*) and the Federal Water Pollution Control Act (the "Clean Water Act" (CWA)), as amended (33 U.S.C. 1251 *et. seq.*), authorize the Federal government, states, and Indian tribes to recover, on behalf of the public, damages for injuries to, destruction of, or loss of natural resources belonging to, managed by, appertaining to, or otherwise controlled by them (42 CFR § 9607(f)(1); 9601(16)). Under the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), when there is injury to, destruction of, loss of, or threat to the supporting ecosystems of natural resources, the Trustees are also authorized to act (40 CFR Subpart G § 300.600).

- State of Oregon, through the Oregon Department of Energy;
- Confederated Tribes and Bands of the Yakama Nation (Yakama Nation);
- Confederated Tribes of the Umatilla Indian Reservation (CTUIR); and
- The Nez Perce Tribe.

The Trustees have formed the Hanford Natural Resources Trustee Council (HNRTC), a collaborative working group chartered to address natural resources injured by Hanford Site releases of hazardous substances. The Trustees have established several Technical Working Groups (TWGs) that provide technical expertise and guidance to the Council.

The party responsible for discharges and releases of oil or hazardous substances at this site (i.e., the “responsible party”) is DOE. DOE is also responsible for site remediation; in addition, as noted above, DOE is a Trustee. The Trustees have agreed to follow a cooperative assessment process, as recommended by the DOI Natural Resource Damage Assessment regulations, meaning that DOE and the other Trustees are jointly and collaboratively conducting the assessment, including development of this Plan.

**NATURAL
RESOURCE DAMAGE
ASSESSMENT VS.
REMEDATION**

Following the release of a hazardous substance that resulted in injury to a natural resource or resources, CERCLA provides an avenue by which the affected sites and resources can be remediated and restored. “Remediation” and “restoration” represent two related, but distinct processes under CERCLA.

Remediation and/or cleanup activities are risk-based. They are designed to reduce current and future risks to public health and the environment to acceptable levels. At Hanford, remediation activities are overseen by the U.S. Environmental Protection Agency (EPA) and the Washington State Department of Ecology.

“Remediation” and “restoration”
represent two related, but distinct
processes under CERCLA.

In contrast, restoration – the focus of the natural resource damage assessment process – is designed to restore injured natural resources to their “baseline” condition, defined as the conditions that would have existed in the assessment area (over time) absent the release of the hazardous contaminants in question. Achieving a risk-based cleanup goal (remediation) does not necessarily return injured natural resources to their baseline condition. However, Trustees are directed in the DOI regulations to take cleanup activities and outcomes into account – and whenever possible coordinate with the remedial process – in order to enhance the cost-effectiveness of proposed restoration activities.

THE NRDA PROCESS Section 301(c) of CERCLA provides the statutory authority for natural resource Trustees to assess and recover damages resulting from the “injury to, destruction of, or loss of natural resources resulting from the release of oil or hazardous substances.” Injury assessment planning represents one step within the multi-phased framework of natural resource damage assessments. As noted above, the ultimate goal of the assessment is to restore (or replace) injured natural resources and services lost due to the release of hazardous substances. To achieve this goal, Trustees must complete a number of interim steps which are outlined within the DOI regulations, and can be divided into three sequential phases. These phases are presented graphically in Exhibit ES-2, and are described below.

In the **Pre-Assessment Phase**, a review of readily available information is conducted that allows the authorized official to make an early decision on whether a natural resource damage assessment can and should be performed. During this phase, the Trustees determine whether an injury has occurred and if a pathway of exposure exists.² The pre-assessment phase is a pre-requisite to conducting a formal assessment. The Hanford Trustees have completed this process and confirmed that a formal assessment of injuries to resources on the Site is warranted.

Development of the present injury assessment plan, indicated by a red outline in Exhibit ES-2, is the first step within the **Assessment Phase** of a natural resource damage assessment.³ There are two primary components of the Assessment Phase: planning and implementation. First, the Trustees must write a plan, or series of plans, to ensure that the assessment is performed in a systematic manner, and that the methodologies selected can be conducted at a reasonable cost.⁴ Second, the Plan is implemented.

This report represents the Trustees’ current plan for injury assessment. It focuses on studies to be undertaken as part of the injury determination and injury quantification phases of the assessment. It does not include studies associated with the damage determination phase—i.e., it does not include efforts aimed at identifying the appropriate amount of compensation, expressed either in dollars or in terms of actions to be taken to restore natural resources and the services they provide, associated with any potential injuries. The Trustees will develop one or more additional planning documents when appropriate to describe efforts to be undertaken as part of damage determination. In addition, the Trustees may make modifications to this Plan over time to reflect new

² “Injury” is generally defined in the regulations as a measurable adverse change, either long- or short-term, in the chemical or physical quality or the viability of a natural resource resulting either directly or indirectly from exposure to a discharge of oil or release of a hazardous substance, or exposure to a product of reactions resulting from the discharge of oil or release of a hazardous substance” (43 CFR § 11.14(v)).

³ In addition to the assessment documents and steps listed in Exhibit ES-2, the Trustees have commissioned a Preliminary Estimate of Damages, which uses existing information to estimate the scale and scope of injury and damages at the Hanford Site. This document and the process of its development informed this injury assessment plan.

⁴ The U.S. Department of the Interior NRDA regulations at 43 CFR § 11 require that the Trustees perform either a Type A or Type B assessment. Type A assessments are assessments performed using the CERCLA Type A Natural Resource Damage Assessment Model for Coastal and Marine Environments. Type B assessments employ alternative methodologies for damages determination. In the case of Hanford, the Trustees are conducting a Type B assessment.

information and/or analyses as they become available. Future assessment planning documents will be developed that provide more technical details for particular studies (e.g., detailed sampling and analysis plans, statistical approaches). The implementation of studies generally described in this Injury Assessment Plan, and to be described in more detail in study-specific work plans, ultimately will result in the identification and quantification of injury to natural resources resulting from hazardous contaminant releases from the Site.

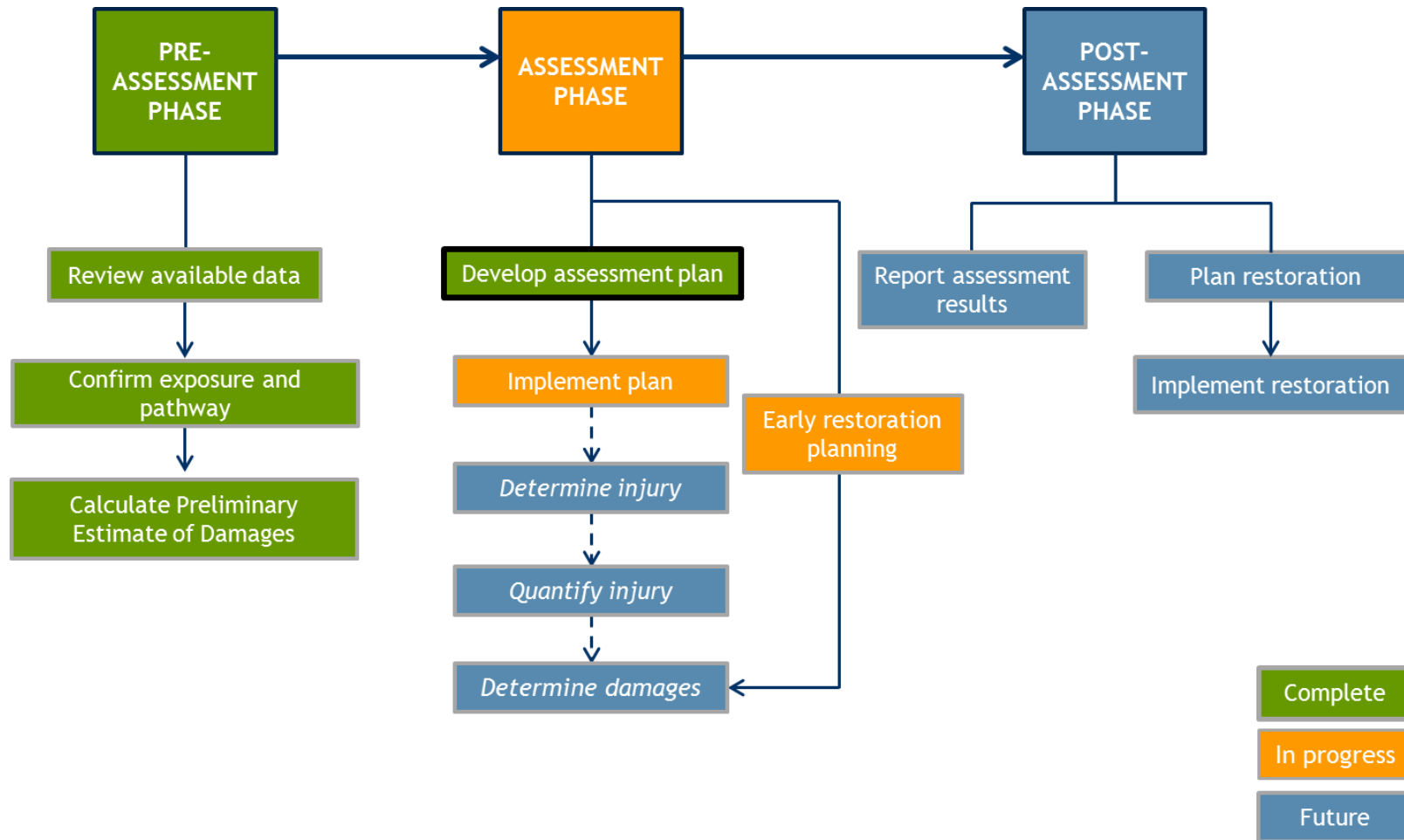
The DOI NRDA regulations state that a Restoration Compensation and Determination Plan (RCDP) shall be part of the Assessment Plan (43 CFR § 11.81(d)(1)). The RCDP is a document that lists a reasonable number of possible alternatives for restoration, rehabilitation, replacement, and/or acquisition of equivalent resources and their related services, selects one of the alternatives, and provides a rationale for the alternative (43 CFR § 11.81(a)). The DOI NRDA regulations, however, allow Trustees to defer development and public release of a RCDP after completion of injury determination or quantification phases if existing data are not sufficient to develop a RCDP at the time that the overall assessment plan is released (43 CFR § 11.81(d)(1)). The Hanford Trustees believe there is insufficient information to complete a RCDP at this time, and have chosen to develop a RCDP later in the assessment process.

After completing injury determination and quantification (including pathway determination), the damage determination planning and implementation will follow. Subsequent to damage determination, the Trustees enter the **Post-Assessment Phase**. As part of this phase, the Trustees will prepare: 1) a Report of Assessment detailing the results of the Assessment Phase; and 2) a Restoration Plan that is based upon the RCDP and describes how natural resources and the services they provide will be restored.

The Trustees note that although the various phases and steps of a natural resource damage assessment are set forth as a sequential process within the DOI NRDA regulations, in practice evaluations for different natural resources may occur at different rates: for some categories of injury the Trustees may choose to proceed through the steps in a sequential order; in others the availability of existing information or the ability to establish reasonable assumptions may allow the Trustees to take an alternative, but still sound approach to establish the scale and scope of required restoration.

In addition, the Trustees may from time to time identify early restoration opportunities—i.e., chances to commence with a restoration project before the assessment has proceeded completely through earlier phases. Because these opportunities may be short-lived in duration, the Trustees may agree to pursue them and to estimate restoration credits for such projects that could eventually be used to offset the final tally of environmental liabilities.

EXHIBIT ES-2 PHASES OF THE NATURAL RESOURCE DAMAGE ASSESSMENT PROCESS



**SITE HISTORY,
NATURAL RESOURCES,
AND INJURY
ASSESSMENT PLAN
DEVELOPMENT**

ACTIVITIES LEADING TO CONTAMINANT RELEASE

In 1943, the United States established the 586 square mile Hanford Site as the Hanford Nuclear Reservation to produce nuclear materials for national defense.⁵ In addition to producing the materials needed for nuclear weapons, Site activities produced significant quantities of waste containing hazardous chemicals and/or radioactive materials.⁶ The Federal government managed these wastes by storing them on land and by releasing them into ponds and ditches.⁷ Over time, many of these production facilities have leaked contaminants onto the land and into the air and water, including into the Columbia River.⁸ The production facilities, which included nine nuclear reactors and associated processing facilities (Poston *et al.* 2010), are now considered “closed” (not operational) and are being decommissioned and cleaned up by DOE, which is currently the Federal agency responsible for overall management of the Site.⁹

CONTAMINANTS OF POTENTIAL CONCERN

At Hanford, the list of contaminants known to have been used and released from the Site is extensive. The Hanford Trustees have identified a suite of contaminants on which to focus the assessment. The Trustees have reviewed a number of information sources in assembling this list, including but not limited to site risk assessments (e.g., CRCIA 1998, DOE 2011a, b), contaminant data for onsite underground tanks (e.g., Gephart 2003b) and major groundwater plumes (e.g., DOE 2011c), reports on historic and current releases (e.g., Hall 1991), and Site contaminant databases. Although the Trustees’ work in this area is ongoing and subject to further refinement, the preliminary list of contaminants of potential concern (COPCs) is presented in Exhibit ES-3.

⁵ Tri-Party Agreement, Article VI, Part 23 (A).

⁶ Tri-Party Agreement, Article VI, Part 23 (D) and <http://www.hanford.gov/page.cfm/HanfordsPresentMission>

⁷ <http://www.hanford.gov/page.cfm/HanfordsPresentMission>

⁸ Statement added by Trustees, in part supported by <http://www.hanford.gov/page.cfm/HanfordsPresentMission> and the Cleanup Progress at Hanford Factsheet.

⁹ Tri-Party Agreement, Article VI, Part 24 (E).

EXHIBIT ES-3 PRELIMINARY LIST OF CONTAMINANTS OF POTENTIAL CONCERN

| RADIOISOTOPES | ORGANICS | INORGANICS |
|-------------------------|-------------------------|--------------------------|
| Americium-241 | 1-2 Dichloroethane | Antimony |
| Carbon-14 | 1,4 Dioxane | Arsenic |
| Cesium-137 | 2,4,6 Trichlorophenol | Barium |
| Cobalt-60 | Acetonitrile | Boron |
| Europium-152 | Benzo(a)pyrene | Cadmium |
| Gadolinium-152 | Carbon tetrachloride | Chromium (includes Cr6+) |
| Iodine-129 | Chlorodane | Cobalt |
| Neptunium-237 | Chloroform | Copper |
| Plutonium-239/240 | Cyanide | Fluoride |
| Potassium-40 | DDT/DDE | Lead |
| Radium-226, Ra-228 | Dichloromethane | Manganese |
| Strontium-90 | Glyphosate | Mercury |
| Technetium-99 | Hydrazine | Molybdenum |
| Thorium-232 | Hexone | Nitrate |
| Tritium | PCBs | Nickel |
| Uranium-233/34/35/38 | Tributyl Phosphate | Phosphate |
| Zirconium-93 | Trichloroethylene (TCE) | Selenium |
| Total radiological dose | Total Petroleum | Silver |
| | Hydrocarbons/PAHs | Strontium |
| | Vinyl chloride | Uranium |
| | | Vanadium |
| | | Zinc |

RESOURCES OF CONCERN

Natural resources of concern include all Trust resources within the assessment area, including groundwater, surface water, sediment, soil, plants, insects and other invertebrates, fish, amphibians, reptiles, birds, and mammals.¹⁰ The Hanford Site has unique terrestrial and aquatic ecosystems that are home to 40 species of mammals (Fitzner and Gray 1991), over 200 species of birds (TNC 1999), and a large variety of amphibians, reptiles, and invertebrates (Fitzner and Gray 1991). Rare plant surveys conducted by The Nature Conservancy confirm the Site is a critical area for the conservation of rare shrub-steppe, riparian, and aquatic plants. At least 725 individual plant species have been identified on the Site (Sackschewsky and Downs 2001), 13 of which are listed by Washington State as threatened or endangered (Poston *et al.* 2010). The adjacent Columbia River also supports a number of economically and culturally important fish species including the Chinook salmon, coho salmon, steelhead, white sturgeon and Pacific lamprey.

The Chinook salmon species is managed as a federally protected species by population according to spawning location and timing of spawning. There are seventeen populations of Chinook that are considered to be “substantially reproductively isolated” and that are managed in divisions known as Evolutionarily Significant Units (ESUs). Two Chinook

¹⁰ Available information does not indicate that the air resource itself has been subject to injury due to releases from the Hanford Site. For the purposes of this Plan, air is considered as a pathway for contamination.

ESUs currently occur within the Hanford Reach (the portion of the Columbia River adjacent to the Hanford Site): (1) the Upper Columbia River (UCR) summer-/fall-run Chinook, and (2) the UCR spring-run Chinook. The fall-run Chinook naturally spawn in the Hanford Reach, as do fall-run steelhead trout (federally threatened) (Duncan *et al.* 2007). Spring-run Chinook, which pass through the Hanford Reach to their spawning grounds, are listed as federally endangered.

HUMAN USES OF NATURAL RESOURCES

Historically, the lands making up the Hanford Site were home to several mid-Columbia Indian Tribes and bands, including ancestors of the present-day Wanapum Band of Priest Rapids, Confederated Tribes and Bands of the Yakama Nation, Nez Perce Tribe, Cayuse

“The most appropriate way to understand our cultural values is to view our cultural practices conducted today on our landscape. They reflect a complex tradition showing high regard for the land. There isn't a daily activity of a traditional lifestyle that doesn't have oral traditions telling how the activity is part of the land and plays a role in taking care of the land” (Nez Perce 2010).

Tribe, Confederated Tribes of the Umatilla Indian Reservation, and Walla Walla people. The Site continues to have tremendous cultural and religious significance for local tribes. Non-tribal historical and present uses of the Site include recreation (e.g., fishing, hunting, birding) and agriculture. The Wanapum and Yakama Tribes continue to fish in the Columbia River, including spring and fall

fishing near Horn Rapids Dam and from Vernita Bridge to Wanapum Dam (Leah Aleck, personal communication, 2012).

The release of hazardous contaminants from Hanford Site operations has impacted people’s use of natural resources, and the well-being they derive from such uses. Changes in human use due to the presence of contaminants may result in the need for specific restoration actions to restore the scale and quality of human uses of natural resources of particular concern to the Trustees or losses in tribal services as a result of injury to natural resources.

ABOUT THE PROPOSED STUDIES

Purpose of Studies

It is well-established that natural resources have been injured as a result of release of hazardous contaminants from Hanford, as described in Chapter 5. Thus, the Trustees’ intent in designing this Injury Assessment Plan, and in selecting the studies identified therein, is to lay out a path by which the scope and scale of injury to natural resources can be understood and restoration may be planned and scaled appropriately.

The Plan as currently written represents the Trustees’ best understanding of the studies that may be necessary to robustly identify and quantify injury to Site natural resources and their services. Inclusion of a study within this Plan does not guarantee that it will be undertaken, and studies not included within the Plan may be deemed necessary at a later date. **The Plan does not limit in any way the extent and nature of studies that may be undertaken in the course of the Assessment.** Rather, it provides a starting point

from which the Trustees will begin to prioritize study efforts and implement the Injury Assessment process.

In developing this Plan, the Trustees have considered available information on the nature and extent of hazardous contaminants in the environment resulting from releases from Hanford operations. The Trustees have also considered information that can be used to establish the level of past, current, and likely future natural resource injuries and service losses resulting from these releases. There is, however, a great deal of uncertainty as to the potential for long-term future natural resource injuries and service losses that could result from sources of contamination at the Site that currently may not be well-characterized. There is also a great deal of uncertainty regarding the likely nature and effectiveness of future remedial actions in addressing these sources of contamination. For example, there are several existing sources of hazardous contaminants in the vadose zone (or deep soils above the groundwater) in the Central Plateau of the Hanford Site (Chronister 2011, DOE 2010a). These sources may not be remediated as part of the ongoing Site cleanup. As such, additional injuries and lost services associated with these contaminants may occur in the future that may not be foreseen or reliably quantified in the context of this Plan. DOE notes that ecological risk assessments, additional site characterization, and remedial investigation/feasibility studies will be performed and are intended to assure remedial actions are protective of human health and the environment.

Study Selection

A number of Trustee efforts have led to the selection of the particular studies included in this Plan. The Trustees have been meeting since 1993, and more recently on a monthly basis, to discuss Hanford assessment activities. There are six technical working groups (TWGs) that focus on more technical analyses including the aquatic, terrestrial, groundwater, human use, restoration, and source and pathway TWGs. Specifically, the Hanford TWGs have conducted preliminary analyses of geocoded (i.e., have associated location information) sediment and fish contaminant data to determine resources at risk, developed a number of species profiles, which summarize and evaluate historical contaminant data on a Hanford species of concern, conducted research on contaminant sources and resource use of several ponds and ditches on Hanford, evaluated groundwater contaminant plume maps, and began developing the Hanford Natural Resource Restoration Plan which addresses early restoration and restoration project evaluation criteria.

The Trustees held a number of workshops and expert panels to explore different methods for injury assessment as well as key questions on the effects of contamination at Hanford. Workshop and panel topics included data management, data quality assessment, ecosystem service valuation, human use services and service flows in natural resource damage assessments, compiling toxicity thresholds, injury to aquatic biota in the Hanford Reach, groundwater contaminant upwellings, the integration of groundwater and vadose zone analyses, and the effects of radionuclides on biota at Hanford.

With contractor support, the Trustees have completed a number of large technical analyses including a compilation and evaluation of natural resource information and

historical contaminant concentrations from the Hanford Site, an analysis and summary of key data gaps, and a preliminary estimate of injury at the Site. Together, these analyses have helped the Trustees to evaluate existing information and identify injury studies that will fill data gaps and allow the Trustees to determine and quantify injury at the Hanford Site.

Nature of Studies

This Injury Assessment Plan presents an array of potential studies to identify the scope and scale of injury and service losses to natural resources. Ultimately, these studies are intended to help the Trustees select the appropriate scope and scale of restoration projects that will restore site natural resources to their baseline condition – i.e., a condition in which the injured natural resource provides all of the services that would have been provided absent natural resource injury – and compensate the public for any lost services that occurred while natural resources were in an injured state. The identified studies fall generally within four categories:

1. Use of existing data to identify potential injury to site resources.

Since the Hanford Reservation was established in 1943, a tremendous volume of environmental data has been collected both at the Site and from adjacent lands and waterways. These data present a valuable source of information on the past and recent condition of site resources, and they will be used, to the extent possible, to help evaluate occurrence and magnitude of potential injury to site resources. Studies that may be undertaken in this regard include the comparison of existing data measuring concentrations of contaminants in various media to selected injury thresholds, and compilation of the results of toxicity testing that has been conducted on-site for non-assessment purposes.¹¹

2. Collection of new data to determine injury to site resources, including changes in natural resource services.

Preliminary analysis of existing site data indicates that those data alone will not be sufficient to fully characterize contamination and injury to site resources. For example, sampling of soil has largely been limited to specific geographic areas immediately around facilities and operational areas, and most data have been collected for specific purposes, potentially limiting its utility for natural resource damage assessment. In addition, comparison of existing data to published thresholds may not, in itself, be enough to demonstrate injury under the law.¹² Collection of new data to fill existing gaps, or to answer questions raised through the analysis of existing data, will represent a significant proportion of studies conducted under the Injury Assessment.

¹¹An “injury threshold” is a concentration of a contaminant found in a given media type or resource which has been demonstrated (e.g., in the peer-reviewed scientific literature) to cause a “...measurable adverse change, either long- or short-term, in the chemical or physical quality or the viability of a natural resource” (43 CFR § 11.14(v)).

¹² An exception may be in the case where the published threshold is based on a site-specific study.

3. Use of existing or newly collected data to identify the pathways of exposure of site resources to hazardous releases.

Responsible parties are only liable for injury due to contamination that can be positively linked to their own hazardous releases. However, for some contaminants, upstream or otherwise off-site sources may be contributing to the contamination identified in site resources. Studies of this nature are primarily focused on demonstrating a direct link between on-site activities and observed contamination, or identifying that portion of identified injury for which the responsible party can be held accountable.

4. Use of existing or newly collected data to quantify injury to site resources, including changes in natural resource services.

Determination that injury has occurred does not provide sufficient information to allow for the selection and scaling of restoration projects needed to restore that resource's services to their baseline condition. Once injury is identified, the Trustees must evaluate the scope and scale of that injury and the degree of natural resource services loss. These studies will evaluate the type of injury that has occurred, and quantify that injury, providing information so that restoration may be selected and scaled appropriately.

Study Timing / Relative Prioritization

To help guide future assessment efforts, the Trustees have grouped the proposed studies into three informal categories. The assignment of a study to a particular category (and, therefore, the expected relative prioritization of the study) is based on Trustee judgments about a variety of factors including but not necessarily limited to: cost effectiveness; technical study sequencing requirements; studies that, in the Trustees' view, may be more likely to demonstrate injury; studies most likely to contribute to the selection and scaling of restoration alternatives; and/or studies anticipated to address principal concerns of the public. Based on these types of considerations, the Trustees have grouped the studies in this Plan into three categories:

1. Nearer-term priorities,
2. Middle-term priorities, and
3. Longer-term priorities.

The first category, nearer-term priorities, includes studies that are presently ongoing, and studies the completion of which are prerequisites for subsequent work or that are expected to generate information of significant use in refining future study designs. The second category of studies is expected to include studies that may be more likely to identify injuries, studies anticipated to address principal concerns of the public, and/or studies that are expected to contribute the most towards informing the selection and scaling of restoration alternatives. The third category includes studies that depend on the prior completion of other efforts, and those that are presently expected to be subject to more difficult technical issues. Exhibit ES-1 lists the studies identified in this Plan and

indicates their current relative priority group (i.e., 1, 2, or 3) in parentheses after each study.

CHAPTER 1 | INTRODUCTION

The Hanford Site is located in south-central Washington State near the City of Richland – approximately 150 miles southwest of Spokane and 200 miles southeast of Seattle. The Site covers 586 square miles (375,000 acres) and includes an area now designated as the Hanford Reach National Monument (Exhibit 1-1).

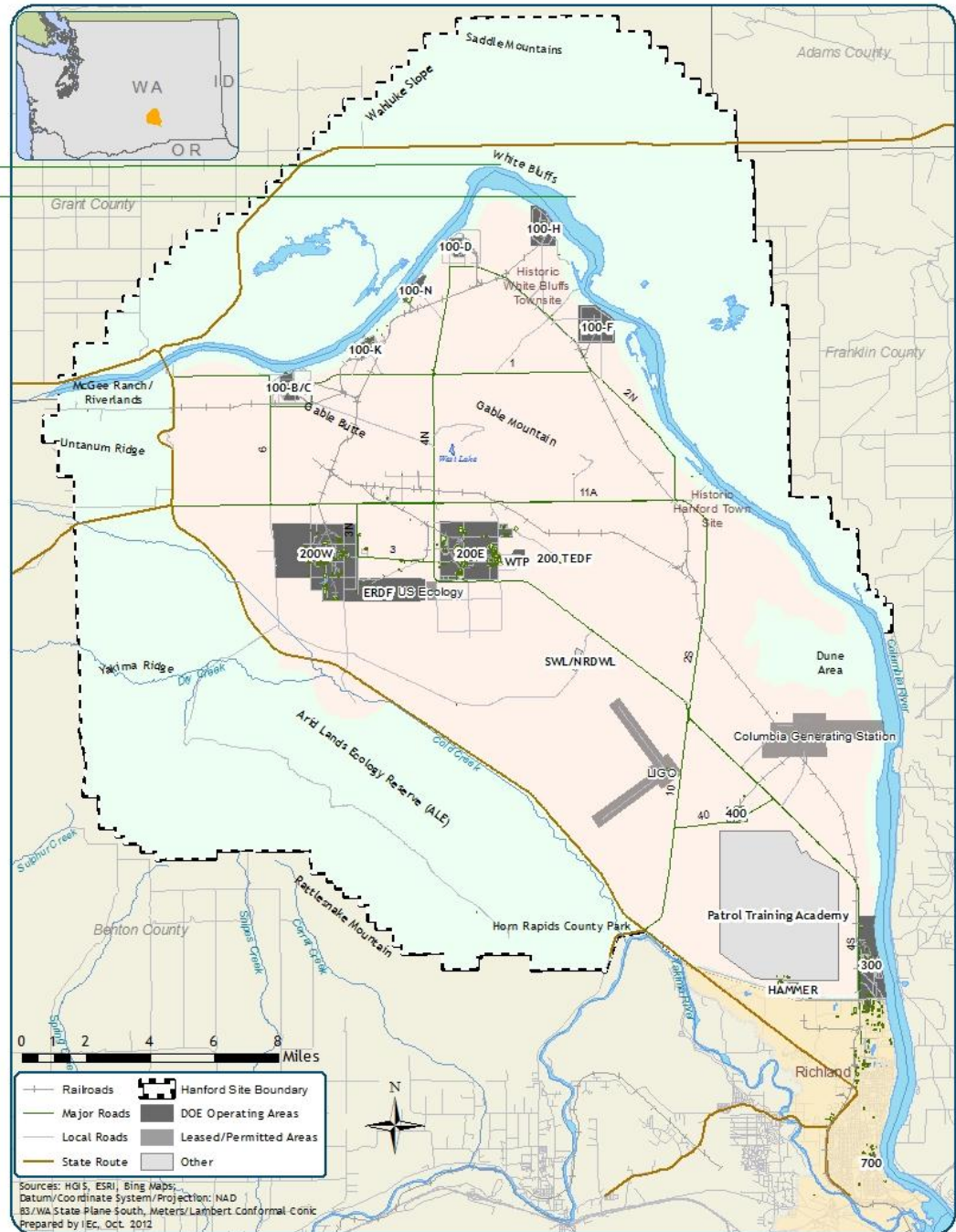
The Site has had restricted public access since 1943, “providing a buffer for areas currently used for storage of nuclear materials, waste treatment, and waste storage and/or disposal” (Duncan 2007). This restricted access has allowed the area to serve as a refuge for native plants and animals that were once far more common in the region (US FWS 2008). At present, the Site is surrounded primarily by agricultural lands. The U.S. Department of Energy (DOE) and the U.S. Fish and Wildlife Service (US FWS) each manage portions of the Site.

The Hanford Site is home to nine decommissioned nuclear reactors and associated processing facilities. From 1944 until 1987 these reactors produced plutonium for use in the United States’ atomic weapons program. The processes required to transform raw uranium into plutonium generated billions of gallons of liquid waste and millions of tons of solid waste. Radioactive wastes were piped to underground tanks, contaminated liquids and cooling water were pumped to ditches and ponds, and contaminated water discharged from the reactors was released to nearby soils and the Columbia River (Gephart 2003b). Major contaminants released to soil and groundwater include metals (e.g., chromium), organics (e.g., carbon tetrachloride), and radionuclides (e.g., cesium, tritium, strontium-90, technetium-99, uranium, and plutonium) (Hartman *et al.* 2001). Most radionuclides released to the Columbia River were short-lived; however, some longer-lived radionuclides such as cobalt-60, strontium-90, cesium-137, uranium-238, and plutonium-238, -239, and -240 were also released to the River (Gephart 2003b).

In May 1989, DOE, the U.S. Environmental Protection Agency (EPA), and Washington State signed the Hanford Federal Facilities Agreement and Consent Order (also known as the Tri-Party Agreement), and in November 1989, the Hanford Site was listed on the National Priorities List (NPL).¹³ Remedial actions have been ongoing since the early 1990s. Cleanup actions are conducted by DOE, with support and oversight from EPA and the Washington Department of Ecology.

¹³ “The *National Priorities List* (NPL) is the list of national priorities among the known releases or threatened releases of hazardous substances, pollutants, or contaminants throughout the United States and its territories. The NPL is intended primarily to guide the EPA in determining which sites warrant further investigation” (EPA 2012c).

EXHIBIT 1-1 HANFORD SITE



Radionuclides, metals, and organics released to on-site ditches, ponds, and soil have leached into groundwater beneath the Site. Along with contaminants discharged directly to the Columbia River, these hazardous substances (also generally referred to as contaminants in this Plan) have been transported downstream via surface water, sediments, and floodplain soils. Since the 1950s, Site natural resources have been monitored as part of various risk assessments and monthly and annual environmental

reporting requirements. Thousands of soil and sediment samples, as well as millions of groundwater samples are documented in the Hanford Environmental Information Systems database, confirming exposure of sediments, soils, groundwater, and biota to contaminants such as chromium, mercury, strontium-90, and technetium-99. In addition, EPA conducted fish surveys in the Columbia River from 1996-1998, and documented elevated levels of metals and organic contaminants in Hanford Reach fish compared to other areas of the Columbia River basin (EPA 2002a).

Releases of hazardous substances to the environment may cause injury to natural resources. Injury is generally defined in the Department of the Interior (DOI) regulations for Damage Assessment under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) as:

“a measurable adverse change, either long- or short-term, in the chemical or physical quality or the viability of a natural resource resulting either directly or indirectly from exposure to a discharge of oil or release of a hazardous substance, or exposure to a product of reactions resulting from the discharge of oil or release of a hazardous substance. As used in this part, injury encompasses the phrases “injury”, “destruction” and “loss”. Injury definitions applicable to specific resources are provided in Sec. 11.62 of this part.” (43 CFR § 11.14(v))

Natural resources or resources are defined in the DOI regulations under CERCLA as:

“land, fish, wildlife, biota, air, water, ground water, drinking water supplies, and other such resources belonging to, managed by, held in trust by, appertaining to, or otherwise controlled by the United States...any State or local government, any foreign government, any Indian tribe, or if such resources are subject to a trust restriction or alienation, any member of an Indian tribe. These natural resources have been categorized into the following five groups: Surface water resources, ground water resources, air resources, geologic resources, and biological resources.” (43 CFR § 11.14(z))

When injury to natural resources is suspected, Federal law authorizes government officials, acting as natural resource trustees, to enter into a Natural Resource Damage Assessment (NRDA) process. CERCLA¹⁴ and the Federal Water Pollution Control Act, also known as the Clean Water Act (CWA),¹⁵ authorize the Federal government, states, and Indian tribes to recover, on behalf of the public, damages for injuries to natural resources belonging to, managed by, appertaining to, or otherwise controlled by them. Under the authority of CERCLA and the CWA, DOI issued regulations to guide trustees in the assessment of natural resource injuries and damages and to plan and implement actions to restore, replace, or rehabilitate natural resources injured or lost as a result of the release of a hazardous substance, and/or to acquire the equivalent resources

¹⁴ As amended, 42 U.S.C. ● 9601, et seq.

¹⁵ As amended, 33 U.S.C. ● 1251, et seq.

(collectively referred to as “restoration”; 42 U.S.C. § 9601 *et seq.* (CERCLA); 43 CFR Part 11).

The DOI regulations under CERCLA define restoration or rehabilitation as:

“actions undertaken to return an injured resource to its baseline condition, as measured in terms of the injured resource’s physical, chemical, or biological properties or the services it previously provided, when such actions are in addition to response actions completed or anticipated, and when such actions exceed the level of response actions determined appropriate to the site pursuant to the NCP.” (43 CFR § 11.14(l))

The remainder of this Chapter describes the following:

- Trusteeship: the Hanford Trustees and their role and coordination;
- Overview of the natural resource damage assessment process;
- Assessment activities at Hanford;
- Public participation;
- Schedule for injury assessment; and,
- Plan organization.

1.1 TRUSTEESHIP The natural resource trustees for the Hanford Site (together, Trustees) include:

- The U.S. Department of Energy;
- The U.S. Department of the Interior through the U.S. Fish and Wildlife Service (US FWS);
- The U.S. Department of Commerce through the National Oceanic and Atmospheric Administration (NOAA);
- The State of Washington through the Washington Department of Ecology in consultation with the Washington Department of Fish and Wildlife (WDFW);
- The State of Oregon through the Oregon Department of Energy;
- The Confederated Tribes and Bands of the Yakama Nation (Yakama Nation);
- The Confederated Tribes of the Umatilla Indian Reservation (CTUIR); and
- The Nez Perce Tribe.

In 1993, DOE, DOI, the State of Washington, the State of Oregon, the Yakama Nation, CTUIR, and the Nez Perce Tribe formed the Hanford Natural Resource Trustee Council (HNRTC), a collaborative working group chartered to address natural resources affected by Hanford Site releases of contaminants. In 1996, these Trustees signed a Memorandum of Agreement (MOA) “intended to help coordinate decisions and actions made by the trustees pursuant to their legal authority to address natural resources impacted by Hanford Site releases of contaminants.” NOAA began participating in the HNRTC in 1997.

The Hanford Trustees have adopted a statement of guiding principles for protection of natural resources on the Hanford Site. These principles state, in broad terms, the Trustees' expectations for cleanup and future uses of the Hanford Site as they relate to natural resource restoration, and also Trustee goals for restoration of injured natural resources. Three broad goals are articulated in the principles document (Guiding principles for protection of natural resources Draft 4, March 11, 2011):

- 1) Achieve a cleanup of the Site sufficient to avoid or minimize residual injuries to natural resources and the services they provide to people and ecosystems.
- 2) Achieve cost-effective restoration of the Site. One way to achieve this will be to coordinate assessment restoration with post-cleanup revegetation and mitigation activities where practicable.
- 3) Post-cleanup land use decisions should not constrain, or preclude, effective natural resource damage assessment restoration. (HNRTC 2011)

1.2 THE NATURAL RESOURCE DAMAGE ASSESSMENT PROCESS

The ultimate goal of the natural resource damage assessment process is to restore, replace, or acquire the equivalent of natural resources injured due to the release of hazardous substances, and to compensate the public for any loss of services that occurs while natural resources are in an injured state (43 CFR § 11.80(b)).¹⁶ The Trustees must determine the scope and magnitude of damages, that is, the cost for restoration of injured natural resources and/or compensation for lost services.¹⁷

The DOI regulations under CERCLA define services as:

“the physical and biological functions performed by the resource including the human uses of those functions. These services are the result of the physical, chemical, or biological quality of the resource.” (43 CFR § 11.14 (nn))

The DOI regulations can be divided into three sequential phases in the assessment of damages: pre-assessment, assessment, and post-assessment.

Pre-Assessment Phase

In the pre-assessment phase, a review of readily available information is conducted that allows the authorized official to make an early decision on whether a natural resource damage assessment can and should be performed. During this phase, the Trustees determine whether an injury has occurred and a pathway of exposure exists. The pre-assessment phase is a prerequisite to conducting a formal assessment.

¹⁶ The regulations are not mandatory. However, they “must be followed by Federal or State natural resource trustees in order to obtain the rebuttable presumption contained in section 107(f)(2)(C) of CERCLA” (50 CFR Part 11). A rebuttable presumption is an assumption accepted by a court until disproved. The regulations state that the results of an assessment performed by a Federal or state natural resource trustee according to the NRDA regulation shall be accorded the evidentiary status of a rebuttable presumption under CERCLA.

¹⁷ Note that the responsible party may also choose to undertake restoration activities directly.

The Hanford Trustees completed the pre-assessment phase of the assessment in 2009 with the release of the Pre-assessment Screen (PAS) for the Site, in accordance with 43 CFR § 11.23-11.25. Yakama Nation and the Confederated Tribes of the Umatilla Indian Reservation each released a PAS in 2006 and 2007 respectively. The PAS determined there was a reasonable probability of making a successful claim for damages for injuries to natural resources. Specifically, the PAS concluded:

- Releases of hazardous substances have occurred;
- Natural resources for which the Trustees may assert trusteeship under CERCLA and/or the CWA may have been adversely affected by the discharge or release of hazardous substances;
- The quantity and concentration of the released hazardous substances are sufficient to potentially cause injury to natural resources;
- Data sufficient to pursue an assessment are readily available or likely to be obtained at a reasonable cost; and
- Response actions may not sufficiently restore, replace, or provide compensation for injured natural resources without further restoration action.

Therefore, the Trustees determined that further investigation and assessment is warranted.

Keep in mind that this Plan includes only injury assessment studies (i.e., those associated with injury determination and quantification), and does not address potential activities associated with the damage determination phase.

Assessment Phase

This is the current phase of the Hanford assessment. This Injury Assessment Plan describes studies to determine and quantify injury (components 1 and 2 below).

There are three main components of the Assessment Phase (Exhibit 1-2):

- 1) **Injury Determination:** Determine “whether an injury to one or more of the natural resources has occurred; and that the injury resulted from the discharge of oil or release of a hazardous substance based upon the exposure pathway and the nature of the injury” (43 CFR § 11.61(a)(1)).
- 2) **Injury Quantification:** “quantify for each resource determined to be injured and for which damages will be sought, the effect of the discharge or release in terms of the reduction from the baseline¹⁸ condition in the quantity and quality of services...provided by the injured resource” (43 CFR § 11.70(a)(1)).
- 3) **Damage Determination:** Estimate “the monetary damages resulting from the discharge of oil or release of a hazardous substance” (43 CFR § 11.80(a)(1)),

¹⁸ According to the DOI regulations, baseline is “... the condition or conditions that would have existed at the assessment area had the discharge of oil or release of hazardous substance under investigation not occurred.” (43 CFR § 11.14(e))

typically presented in a Restoration and Compensation Determination Plan (RCDP) (43 CFR § 11.80(c)).

For each of these components, the Trustees undertake a planning effort, then a subsequent implementation effort. First the Trustees must write a plan, or series of plans, to ensure that the assessment is performed in a systematic manner and that the methodologies selected can be conducted at a reasonable cost (43 CFR § 11.30(b)). This Injury Assessment Plan (“Plan”) describes the Trustees’ current approach to preparing for and implementing the injury assessment phase of the NRDA (i.e., injury determination and quantification). After injury quantification is completed, the Trustees will establish the amount of money (or damages) required to compensate for the quantity of injuries to natural resources resulting from the discharge of hazardous substances (i.e., the amount of monies needed to restore, replace, or acquire the equivalent of lost services). Note that damage determination activities are not addressed in the Plan, and will be described in a subsequent RCDP, as mentioned above.¹⁹

This Plan is intended to summarize ongoing and proposed studies that have been or will be used to evaluate Site-related contamination and corresponding effects of contamination on natural resources and resource services (Exhibit 1-2). The Trustees may make modifications to this Plan over time to reflect new information and/or analyses as they become available (43 CFR § 11.32(e)). In addition, future injury assessment planning documents will be developed that provide more technical details for particular studies (e.g., detailed sampling and analysis plans, statistical approaches). Consistent with the DOI NRDA regulations, Plan documents will be made available for public review and comment (43 CFR § 11.32(c)); see Public Participation section below).

As part of the assessment planning process, the Trustees must also decide to conduct either a simplified assessment (“Type A”) or a comprehensive assessment (“Type B”). The Type A procedures, which use minimal field observations in conjunction with computer models to generate a damage claim, are limited by the regulations to the assessment of relatively minor, short duration discharges or releases in coastal or marine environments or in the Great Lakes. Alternatively, Type B procedures allow for a range of scientific and economic methodologies to be used for Injury Determination, Quantification and Damage Determination. For this site, the Trustees concluded that the use of Type B procedures is appropriate based on the following determinations: (1) the release did not occur in a coastal, marine, or Great Lakes habitat, (2) the nature of the release and resource exposure to contaminants is long-term and spatially and temporally complex, (3) substantial site-specific data already exist to support the assessment, and (4) additional site-specific data can be collected at reasonable cost. As such, in accordance with the natural resource damage assessment regulations the Trustees have confirmed

¹⁹ The RCDP typically includes a number of possible alternatives for restoration, rehabilitation, replacement, and/or acquisition of equivalent resources. This Plan may also include the criteria used to select the Trustees preferred alternative and the methodologies selected for estimating cost or valuation of natural resource injuries to calculate damages. After public review and finalization of the RCDP is complete, the Plan is implemented.

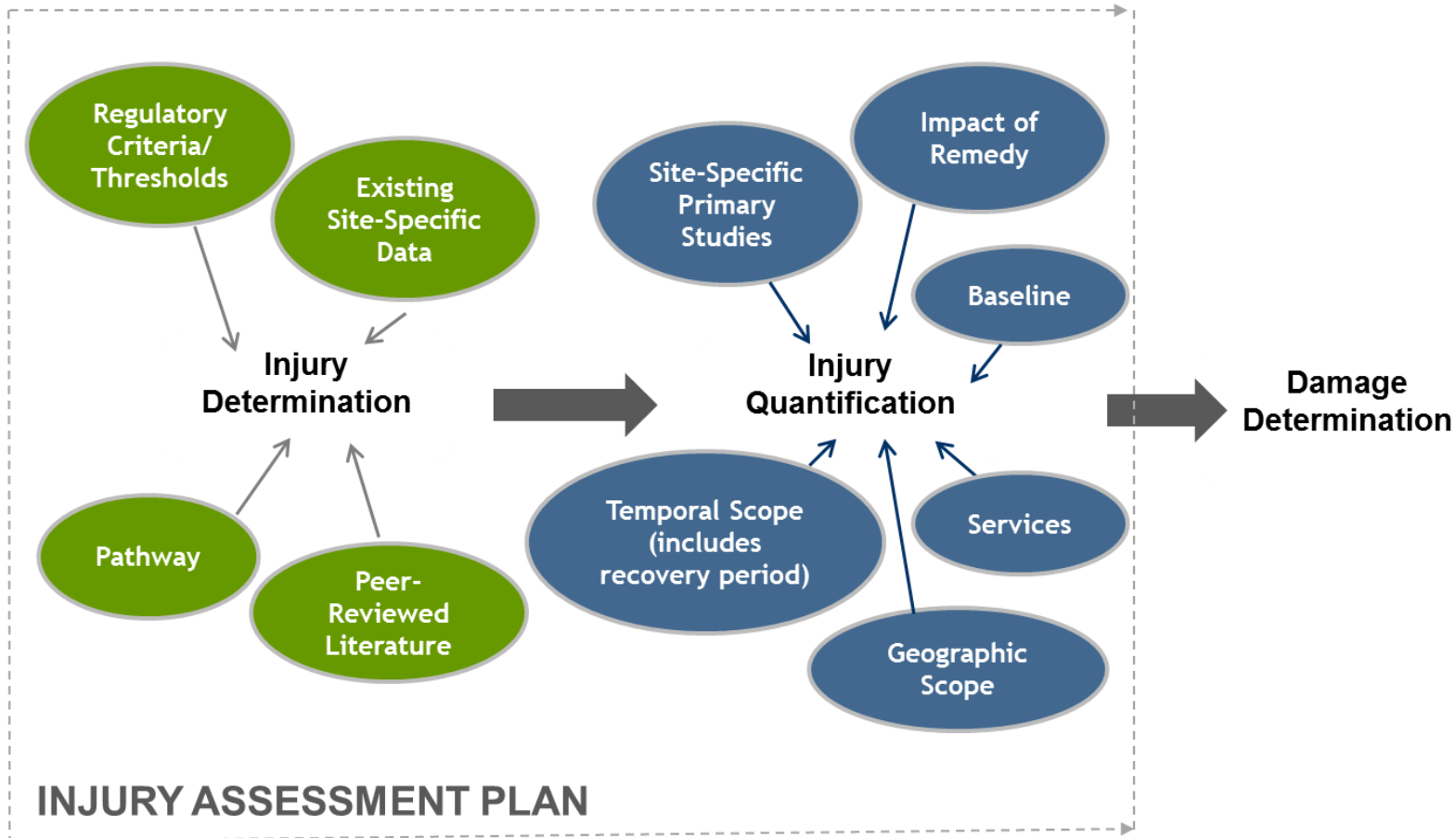
that at least one of the natural resources identified as potentially injured has in fact been exposed to the released hazardous substances (43 CFR § 11.33-11.35) (See Chapter 5).

Study implementation will take place in a phased manner, reflecting factors including, but not necessarily limited to, the availability of funding and prioritization (e.g., collection of ephemeral data before the opportunity to collect it is lost, priority implementation of studies that may generate information relevant to the design of other studies, efficiencies gained by integrating studies into other ongoing data collection activities, etc.).

Post-Assessment Phase

As part of this phase, the Trustees prepare: 1) a Report of Assessment detailing the results of the Assessment phase (i.e., the results of injury studies described in this Plan as well as the results of any subsequent damage determination studies); and 2) a Restoration Plan, based upon the RCDP created as part of the damage determination phase described above, which describes how awarded monies will be used.

EXHIBIT 1-2 ASSESSMENT PHASE COMPONENTS



1.3 ASSESSMENT SUMMARY
ACTIVITIES AT
HANFORD

Under the MOA described above, in the early years of the HNRTC, the Trustees focused much of their effort on the review of and providing technical assistance on ecological risk assessments and other cleanup activities being conducted on the Hanford Site, such as those associated with the Central Plateau cleanup, the River Corridor Closure Project and the Groundwater Project. In addition, during the pre-assessment phase, various Trustees developed their own PASs, including two for the 1100 Area (HNRTC 2000, Nez Perce 2000), as well as Site-wide PAS reports (CTUIR 2007, Ridolfi 2006). In 2007, the Trustees decided to proceed with a phased assessment approach and begin the assessment phase in parallel with ecological risk assessments.

In 2008, a contractor was hired to begin the injury assessment planning process including development of a list of potentially injured natural/cultural resources and defining the focus and scope of the injury assessment. This initial planning was completed in 2009. Since that time, assessment planning activities have continued, including development of this Injury Assessment Plan. The current status of the assessment process at Hanford is outlined in Exhibit 1-3.

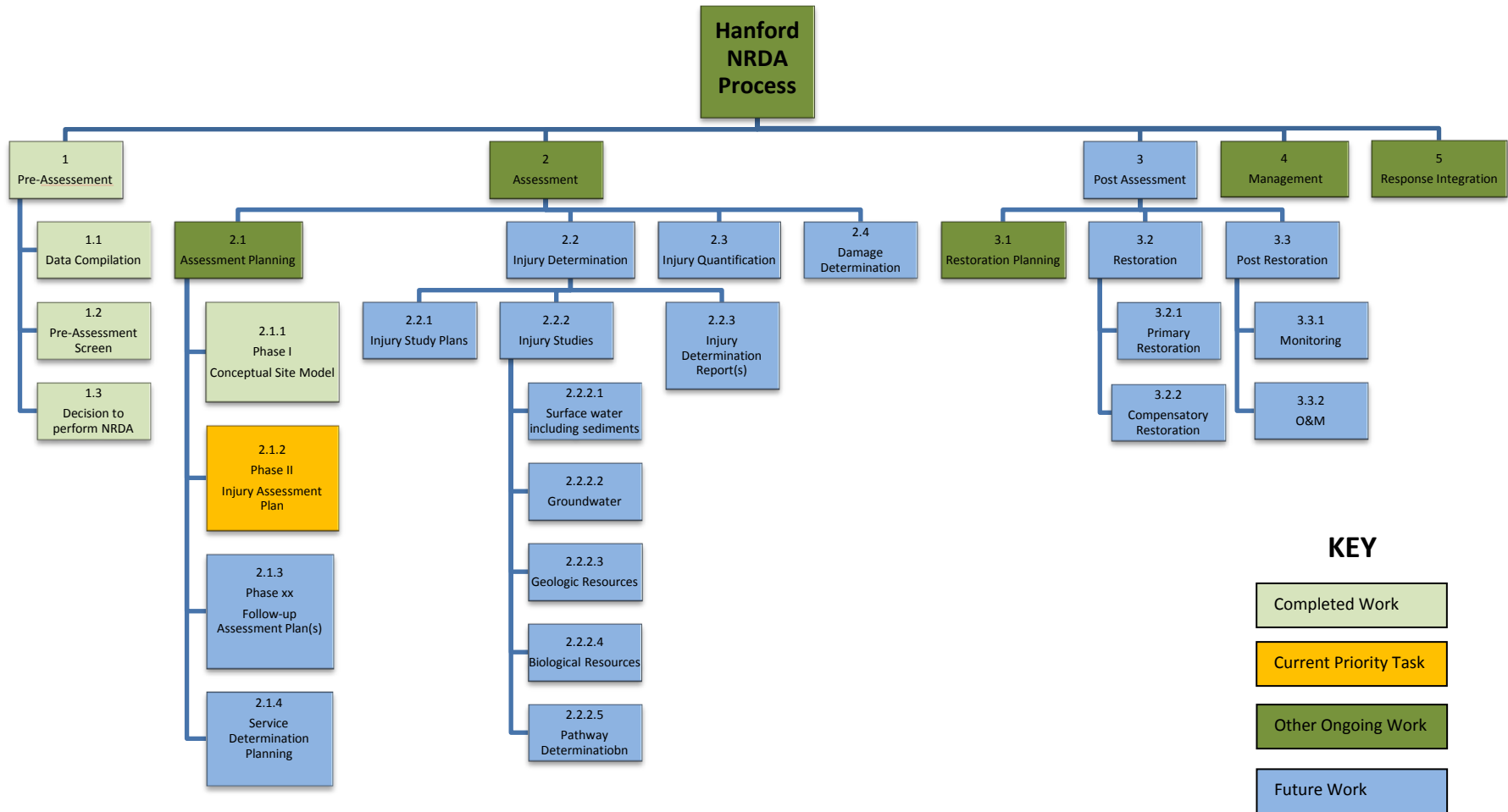
When available, updated information about assessment activities at the Hanford Site is posted at:

- <http://www.hanford.gov/page.cfm/HNRTCHistory>
- <http://www.hanfordnrda.org/>

USE OF AVAILABLE DATA

Analysis of existing data by Hanford Trustees is already underway, including preliminary pathway determination and injury determination efforts. To the extent possible, the Hanford Trustees anticipate using existing information to inform the assessment process. Such information includes data and information collected as part of site investigation and remediation. Going forward, the Hanford Trustees anticipate evaluating existing information and data prior to undertaking additional data collection as part of the assessment process, to better understand where additional information would assist in determining and quantifying injury and, ultimately, determining damages and required restoration. Such efforts are likely to inform the need for and extent of any additional primary research or study(ies) to support the assessment.

EXHIBIT 1-3 SUMMARY OF THE NATURAL RESOURCE DAMAGE ASSESSMENT PROCESS AT HANFORD



COORDINATION WITH SITE REMEDIAL ACTIVITIES

It is important to understand that remediation (i.e., cleanup) and NRDA are separate but related programs. *Remediation and/or response* activities, usually overseen by EPA or state environmental agencies, are intended to reduce present and future risks to public health and the environment. In contrast, *natural resource damage* claims compensate the public for past, present, and future injuries to natural resources and the services they provide.

The Trustees recognize the importance of coordinating efforts to meet assessment and remedial objectives as effectively and efficiently as possible. As noted above, the Trustees have focused, and continue to focus, significant effort in providing comments on and recommendations relating to the ecological risk assessments and other cleanup activities being conducted on the Hanford Site.

COOPERATION WITH THE RESPONSIBLE PARTY

Under CERCLA, the parties responsible for releases of hazardous substances may be invited to participate cooperatively in the assessment and restoration planning process (43 CFR § 11.32(a)(2)). Cooperative assessments can act to reduce duplication of effort, expedite the assessment, and accomplish resource restoration earlier than might otherwise be the case. For this Site, the primary party responsible for discharges and releases of oil or hazardous substances is the Federal Government, represented by DOE, which, as noted above, is also a Trustee and member of the Hanford Council along with other Federal Trustees. The Council has agreed to follow a cooperative assessment process.

GEOGRAPHIC SCOPE

The assessment area is defined in the DOI regulations as:

“the area or areas within which natural resources have been affected directly or indirectly by the discharge of oil or release of a hazardous substance and that serves as the geographic basis for the injury assessment.” (43 CFR § 11.14(c))

Existing data indicate that the exposure and potential impacts from contaminants of potential concern (COPCs) released from the Site may be affecting natural resources in the aquatic and terrestrial habitats of the Hanford Site (including the National Monument), and the adjacent portion of the Columbia River and associated floodplain (the Hanford Reach). Although the evaluation of natural resource damage (NRD) injuries is not limited to a specific geographic area, it is reasonable to develop an understanding of the nature, spatial extent and severity of injuries on the Hanford Site before determining whether the geographic scope of the assessment should be expanded to other, off-site areas.

TEMPORAL SCOPE

The date at which quantification of injuries will begin will depend on the type of natural resource injury. For instance, some natural resource injuries and subsequent damages may be assessed in a manner that allows for separation of damages pre- and post-

December 11, 1980 (in accordance with the passage of CERCLA). In those cases, the Trustees will focus their efforts on estimating damages for the post-December 11, 1980 period. In other cases, injuries and damages may be less clearly divisible over time. In these cases, the Trustees may choose to assess damages for the entire time period of injury. For example, cultural losses may be assessed beginning when tribal members began noticing changes in their environment, and may continue indefinitely. In either case, information available from pre-1980 may be used by the Trustees in understanding baseline conditions as well as injuries and damages post-1980.

Injuries will be quantified, and damages calculated, through the expected date of resource recovery to baseline (note that some injuries may be considered permanent if baseline conditions are not expected to be reestablished). The rate of recovery will be determined based on information related to remedial and restoration activities, natural attenuation, and resource recoverability.

1.4 PUBLIC PARTICIPATION

The Trustees intend to work with the general and tribal publics during this assessment and restoration process and encourage active public participation. Public participation is a required component of the Plan's development process. Specifically:

“The authorized official must make the Assessment Plan available for review by any identified potentially responsible parties, other natural resource trustees, other affected Federal or State agencies or Indian tribes, and any other interested member of the public for a period of at least 30 calendar days, with reasonable extensions granted as appropriate. The authorized official may not perform any type B procedures described in the Assessment Plan until after this review period.” (43 CFR § 11.32(c)(1))

The Hanford Natural Resource Damage Assessment website, available at <http://www.hanfordnrda.org>, provides updated information to the public regarding the status of the assessment and restoration process and opportunities for public involvement. Interested individuals may also sign up for the Hanford natural resource damage assessment Listserve, through which they will be notified about the release of key documents and of milestones within the assessment.

PUBLIC REVIEW AND COMMENT

During the assessment process, the Trustees have and will continue to produce and release for public comment several key documents. The public will be notified of opportunities for public comment through the Hanford Listserve, media releases, and mailings that will be distributed to key stakeholders.

This Plan, as well as any significant subsequent revisions which may be made to it, will be available for review and comment by interested members of the public for a period of at least 30 calendar days, with reasonable extensions granted as appropriate (43 CFR § 11.32(c) and (e)).

Commenters are encouraged to submit electronic comments to Larry.Goldstein@ecy.wa.gov. Comments can also be sent via U.S. mail to:

Larry Goldstein
Hanford Natural Resource Trustee Council Chair
Washington State Department of Ecology
Nuclear Waste Program
PO Box 47600
Olympia, WA 47600

Comments on this Plan must be submitted in writing to the Hanford Trustee contact listed above within 45 days of the publication of the Federal Register Notice of Availability.

As mentioned above, modifications to Assessment Plan documents may occur at any time during the Assessment Phase as new and additional information becomes available (43 CFR § 11.32(e)). Such modifications may result in additional need for public notification and opportunities for comment. Significant modifications (e.g., resource-specific study plan amendments) or additions to this Plan will also be made available for review by any interested members of the public for a period of at least 30 calendar days, with reasonable extensions granted as appropriate, and will be appended to this Plan. Non-significant modifications may also be made available for review, but implementation of such modifications need not be delayed as a result of the review. For more information regarding completed, ongoing, planned, and proposed Site-specific studies see Chapter 7.

**1.5 SCHEDULE
FOR INJURY
ASSESSMENT**

The Trustees do not yet have a firm schedule for the completion of the injury assessment phase of this natural resource damage assessment. Some efforts have been completed (e.g., compilation of biota contaminant concentration data) and others are ongoing (e.g. mussel toxicity testing and review of groundwater contaminant plume maps). As mentioned above, study implementation will take place in a phased manner, reflecting factors such as availability of funding, prioritization of studies, and the nature and timing of remedial alternatives. Other variables that may affect the schedule of the injury assessment phase include environmental conditions (e.g., weather) that could restrict study plan(s) implementation.

**1.6 PLAN
ORGANIZATION**

This Plan provides relevant background information and describes the Trustees' approach to the first two major steps in the assessment process: 1) injury determination, and 2) injury quantification. The third major step, damage determination, including restoration alternatives selection and scaling, will be assessed in a separate plan at a later date.

The remainder of this document contains the following chapters:

- **Chapter 2 - Background Information:** This chapter provides an overview of the history of the Hanford Site including natural history, tribal presence at Hanford, land use and development, and Federal government operations, and Hanford

operations and sources of contaminants, hazardous substance releases, and COPCs.

- **Chapter 3 – Natural Resources:** This chapter includes a description of the Hanford Site natural resources, a discussion of potential ecological service losses associated with contaminant releases from Hanford Site operations, and a discussion of the rate of recovery of services.
- **Chapter 4 – Human Uses:** This chapter provides a description of Hanford tribal and non-tribal human use services and associated potential service losses.
- **Chapter 5 – Confirmation of Exposure and Injury Assessment Process:** This chapter provides a description of data confirming exposure of Hanford resources to contaminants; a description of the injury determination process including a discussion of primary pathways and fate and transport of contaminants; and a description of the injury quantification process including a discussion of baseline and the quantification of ecological, groundwater, human use, and remediation-related impacts.
- **Chapter 6 – Injury Assessment Regulatory Definitions:** This chapter includes relevant DOI regulatory definitions for injury determination, pathway determination, and injury quantification.
- **Chapter 7 – Injury Assessment Studies:** This chapter includes descriptions of injury assessment studies that are currently proposed to support assessment of ecological injuries, groundwater injuries, and human use service losses.
- **Chapter 8 – Quality Assurance Management:** This chapter provides a discussion of the Quality Assurance Plan including project management, a description of the quality system, data generation and acquisition, assessment and oversight, and data validation and usability.

CHAPTER 2 | BACKGROUND INFORMATION

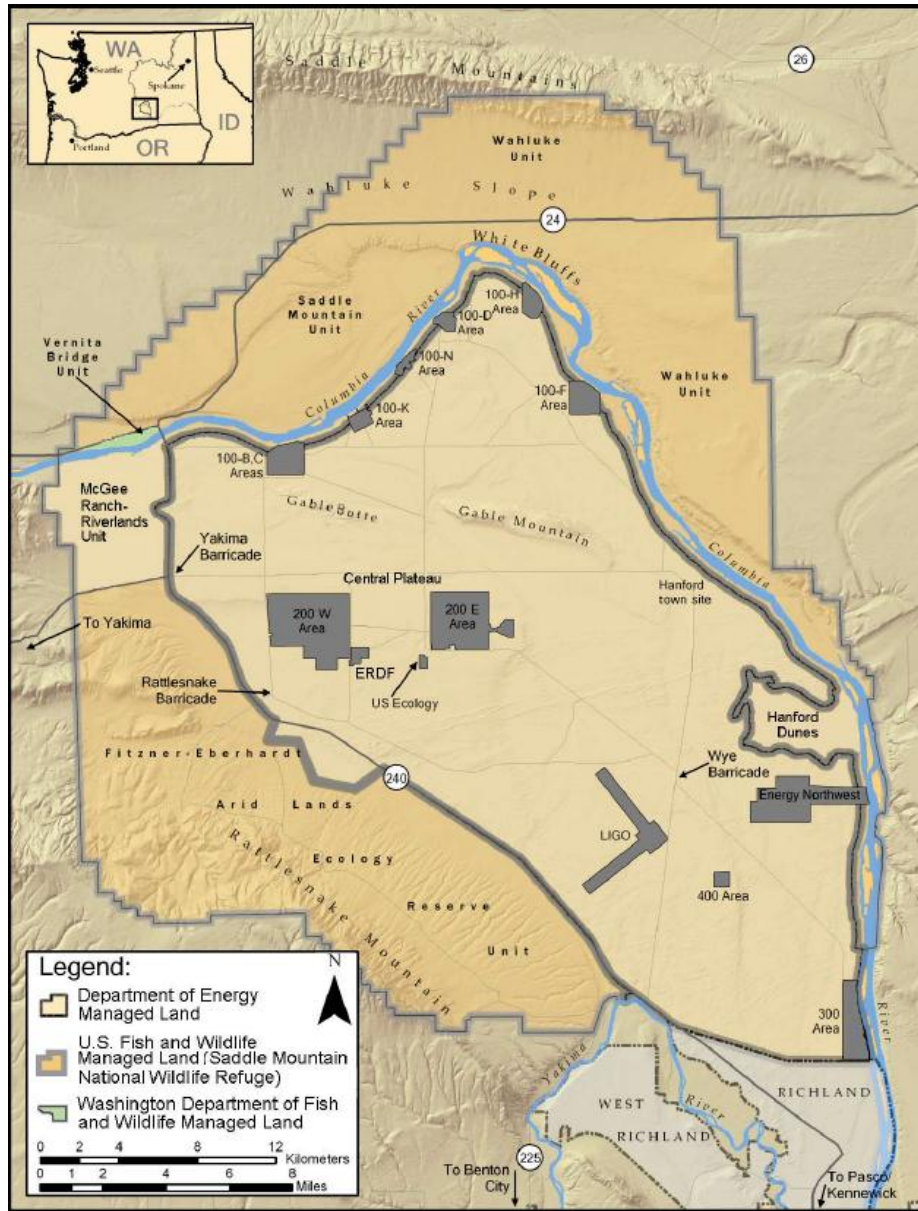
This chapter provides an overview of the Hanford Site's history, including key components of the Site's natural and cultural landscapes. Topics include the Site's major natural features, tribal presence, land use/development, and Federal government operations, including an overview of releases of hazardous substances. Subsequent chapters provide more detail on certain topics: Chapter 3 provides information on the Site's natural resources, while Chapter 4 describes human uses of these natural resources.

2.1 SITE HISTORY MAJOR LANDSCAPE FEATURES AND SETTING

The Hanford Site consists of Central Hanford (Central Plateau and Columbia River Corridor) and the Hanford Reach National Monument (Exhibit 2-1). The Columbia River flows east through the northern part of the Site and then turns south towards Richland. The Yakima River meets the Columbia River at Richland. Rattlesnake Mountain, Yakima Ridge, and Umtanum Ridge are major landforms on the Site's southwestern and western sides, while Saddle Mountain is to the north. Adjoining lands to the west, north, and east are principally range and agricultural land. The cities of Kennewick, Pasco, and Richland (the Tri-Cities), West Richland, and Benton City are the nearest population centers and are located south-southeast of the Hanford Site.

The Hanford Site includes a number of significant natural features, such as the Hanford Dunes—the only active dunefield within the State of Washington—along with Gable Mountain and Gable Butte in Central Hanford (TNC 2003). The Fitzner-Eberhardt Arid Lands Ecology (ALE) Reserve, officially recognized as a valuable site for scientific study in 1967 due to its rich and relatively undisturbed native shrub-steppe habitat, is on the southwest boundary of Hanford. Additionally, the McGee Ranch-Riverlands Unit, managed by DOE, contains the biologically diverse Umtanum Ridge area and some intact shrublands (TNC 2003).

EXHIBIT 2-1 CENTRAL HANFORD AND THE HANFORD REACH NATIONAL MONUMENT



Source: Poston *et al.* 2010

TRIBAL PRESENCE

For thousands of years before the Hanford Site was established, indigenous peoples used the natural resources of the area for hunting, fishing, gathering plants, and conducting religious ceremonies (Yakama 2010; NPT 2010; CTUIR 2012; DOE 2007a). Ancestors of the present day Nez Perce, Cayuse, Umatilla, Walla Walla, Yakama, Wanapum, and Colville fish for salmon; hunt deer, elk, sheep and rabbit; and collect and gather roots, seeds and berries. Natural resources are gathered primarily during spring to fall for foods, medicines, and materials for shelters and tools. Temporary camps are located at fishing sites along the River or in upland areas where resources are available.

Traditionally, the Yakama, Umatilla, and Nez Perce follow a seasonal round of subsistence where hunting, fishing, and gathering harvest is based on seasonal availability of these resources. Many families spent much of their time in the mountains during the summer and in the valley during the winter. The seasonal round is best described as a return to a specific area for the purpose of gathering resources: food, medicinal, or otherwise (NPT 2010). Rather than following a resource wherever it occurs, a seasonal round is “a return to an area to gather resources based on prior knowledge or experience” (NPT 2010). Thus, the ritual of returning to a site daily, seasonally, or annually, was critical to the culture, and the ability to sustain the culture, of these peoples. The three tribes documented this cultural knowledge of subsistence resource use through their Tribal Narratives (NPT 2010; CTUIR 2012; Yakama 2010), which are available in the Administrative Record.

Each Tribal Narrative describes the Columbia River as being culturally and economically central to the culture of these tribes. The CTUIR characterizes the regional importance of the Columbia River Plateau as follows:

“The Columbia River flows through what was a cultural and economic center for the Plateau communities. The indigenous communities were part of the land and its cycles, and the land was part of them. The land and its many entities and services provided for all their needs: hunting and fishing, food gathering, and endless acres of grass on which to graze their horses, commerce and economy, art, education, health care, and social systems. All of these services flowed among the elements of the natural resources, including humans, in continuous interlocking cycles. These elements and relationships form the basis for the unwritten laws or *Tamanwit* that were taught by those who came before, and are passed on through generations by oral tradition in order to protect those yet to arrive. The ancient responsibility to respect and uphold these teachings is directly connected to the culture, the religion, and the landscape of the Columbia Plateau. The cultural identity, survival, and sovereignty of the native nations along the Columbia River and its tributaries are still maintained by adhering to, respecting, and obeying these ancient unwritten laws here in this place along the *Nch’i-Wana*, or Big River” (CTUIR 2012).

In its “Perspective at Hanford,” the Nez Perce describes the historical use of the Hanford Site and surrounding areas as follows:

“Use of the Hanford site and surrounding areas by tribes was primarily tied to the robust Columbia River fishery. Tribal families and bands lived along the Columbia either year round or seasonally for catching, drying and smoking salmon. Past associated activities included gatherings for such events like marriages, trading, ceremonial feasts, harvesting, fishing, and mineral collection” (NPT 2010).

The Yakama Nation also emphasizes the Columbia River’s importance:

“Native Americans of the Columbia River Basin, including members of the Yakama Nation, depend on the Columbia River, known as *Nch’i-wa’na* (‘Big River’) for their livelihood. The spring Chinook salmon is considered a ‘first food,’ celebrated with a feast each spring to recognize the availability and abundance of food at the start of each growing season (ERWM personal communication, 2006-2007; Relander, 1986). In addition to dependence on fish as a major part of their diet for both nutritional and cultural health, the Yakama also depend on hunting local wild animals and birds for food and materials. They are also extremely dependent on the rich abundance and variety of wild plants, from above and below ground, which are used for food and medicine and some of which are also celebrated as ‘first foods’” (Yakama Nation 2010).

The Treaties of 1855

The Confederated Tribes of the Umatilla Indian Reservation (CTUIR)³² observe that “when Lewis and Clark and subsequent traders arrived in the Hanford area during the early 1800s, Native Americans were living in numerous villages along the Columbia River, including from the mouth of the Yakima River to Priest Rapids” (CTUIR 2012). Less than 50 years later, under separate treaties signed in 1855, the Confederated Tribes and Bands of the Yakama Indian Nation,³³ the CTUIR, and the Nez Perce Tribes, as well as numerous other tribes in the Columbia River Basin, ceded control of millions of acres of land to the United States in exchange for establishment of reservations set up for the exclusive use and benefit of those tribes. The Yakama and CTUIR treaties included ceding control of the area occupied by the present Hanford Site, but reserving rights to hunt, gather, fish, and other activities upon open and unclaimed land. These treaties all include similar language recognizing tribal rights to natural resources as follows:

“the exclusive right of taking fish in the streams running through and bordering said reservation is hereby secured to said Indians, and at all other usual and accustomed stations in common with citizens of the United States, and of erecting suitable buildings for curing the same; the privilege of hunting, gathering roots and berries and pasturing their stock on unclaimed lands in common with

³² Cayuse, Umatilla, and Walla Walla Tribes.

³³ The Yakama, Palouse, Pisuouse, Wenatshapam, Klikatat, Klinquit, Kow-was-say-ee, Li-ay-was, Skin-pah, Wish-ham, Shyiks, Oche-chotes, Kah-milt-pah, and Se-ap-cat tribes and bands were joined by their treaty agreement under the name “Yakama” (Treaty with the Yakama, 1855).

citizens, is also secured to them” (Treaty with the Walla Walla, Cayuse, and Umatilla Tribes in 1855).

Thus, the Yakama Nation, the CTUIR, and the Nez Perce Tribe all retain rights to fish, hunt, gather, pasture livestock, and erect structures in the usual and accustomed areas currently occupied by the Hanford Site. We note that the Wanapum People did not sign a treaty with the United States and are not a Federally-recognized Tribe; however, the Wanapum People were historical residents of what would become the Hanford Site and their interests in the area have been acknowledged by the State of Washington (DOE *et al.* 1999).

The Tribes note that in establishing these treaties, the U.S. Government and the Treaty did not "give" the indigenous people the rights to fish, hunt, and gather foods and medicines. Rather, the Treaty of 1855 recognized pre-existing indigenous rights that these peoples have held and exercised since time immemorial (CTUIR 2012). In the Treaty, “ancestors reserved those rights in order to ensure that the Tribes’ future generations would be able to maintain and exercise their traditions and customs, obtain foods and medicines, and retain that part of their identity that is associated with the specific lands and resources at Hanford. Because cultural identity is tied to specific lands and landscapes, every acre has its own unique importance and cannot necessarily be interchanged with another acre if the first acre is lost or injured” (CTUIR 2012).

Federal Trust Responsibility

The Tribes note that, in addition to rights they maintain under existing treaties, the U.S. government also has a responsibility to manage lands held in trust, as well as resources held in trust, for the benefit of tribes. As stated by CTUIR:

“Though often difficult to define, the federal Indian trust doctrine is considered a “cornerstone” of federal Indian law.³⁴ Federal courts have clarified that certain kinds of assets can be held by the United States in trust for Indian tribes and, generally, the United States must properly manage and protect those resources held in trust for tribes.³⁵ Regardless of the difficulty in defining the trust responsibility, it is clear that the United States has charged itself with moral obligations of the highest order in its conduct towards Indian tribes.”³⁶

³⁴ See *Dep’t of the Interior v. Klamath Water Users Protective Ass’n*, 532 U.S. 1, 11 (2001) (“The fiduciary relationship has been described as ‘one of the primary cornerstones of Indian law,’ and has been compared to one existing under a common law trust, with the United States as trustee, the Indian tribes or individuals as beneficiaries, and the property and natural resources managed by the United States as the trust corpus.”) See also Cohen, Felix S., *Handbook of Federal Indian Law* at 220 (Michie Bobbs-Merrill 1982) (trust relationship as one of the primary “cornerstones” of Indian law).

³⁵ Morisset, Mason D., *Recent Developments in Defining the Federal Trust Responsibility* (April 1999) (<http://www.msaj.com/papers/43099.htm>) (accessed July 5, 2012).

³⁶ *Seminole Nation v. United States*, 316 U.S. 286, 296-297 (1942) (stating that Federal government is “more than mere contracting partner” with tribes and has “charged itself with moral obligations of the highest responsibility and trust”); *Pyramid Lake Paiute Tribe of Indians v. Morton*, 354 F. Supp. 252, 256 (indicating that the Federal government’s conduct toward tribes should “be judged by the most exacting fiduciary standards”).

“In such cases where the federal government has a trust responsibility for a specific tribal resource, the government must assume the obligations of a trustee as in a typical, non-Indian fiduciary relationship. These principles include: 1) preserving and protecting the trust property; 2) informing the beneficiary about the condition of the trust resource; and 3) acting fairly, justly and honestly in the utmost good faith and with sound judgment and prudence.³⁷ *United States v. White Mountain Apache Tribe* recognizes that the fundamental common law duty of a trustee is to maintain trust assets and applies that principle in the context of the Indian trust doctrine.³⁸ In a typical fiduciary relationship the trustee must always act in the interests of the beneficiary and the Indian trust doctrine is no different.³⁹ The federal government can and should act on behalf of an Indian tribe if it is within its legal authority to do so” (CTUIR 2012).

OTHER LAND USE/DEVELOPMENT

Lewis and Clark were the first Euro-Americans to visit the Columbia Basin in 1805 (DOE 2007a; Gard 1992). By 1840, the area around Hanford had been mapped by the Army Corps of Topographical Engineers, laying the groundwork for settlers and development (Gard 1992). In 1856, cattle ranchers began making their way to the Columbia River Valley (Gard 1992). By the early 1880’s, settlers were abundant, much of the natural bunchgrasses in the region had been overgrazed, and much of the livestock lost due to lack of available feed (Gard 1992). In response, ranchers began to build small dams and irrigation systems in order to grow alfalfa as food for cattle (Gard 1992). Just after the turn of the century, new irrigation and water companies were developed, new canals and ditches were constructed, and desirable land adjacent to the canals were procured for farming (Gard 1992). Soon, the area was growing strawberries, root crops, fruit trees, onions, and barley in addition to alfalfa (Gard 1992).

Archaeological resources from thousands of years of indigenous occupation as well as the early settlement period are scattered over the Hanford Site, and include gold mining features along riverbanks, homestead remains, agricultural equipment and fields, ranches, and irrigation features (DOE 2007a). Identified traditional cultural places associated with early settlement and farming include home sites and townsites, orchards, fields, and places of former community activities (e.g., swimming hole and town square).

In 1943, the Federal government acquired the Hanford Site for the Manhattan Project. At this time, Native Americans were still living at Hanford in accordance with traditional beliefs and practices, and were among those evicted when the U.S. government took control of the area (CTUIR 2012). Livestock grazing has “presumably been prohibited on the unit since about 1950, although active enforcement was apparently sporadic until the

³⁷ See *Assiniboine and Sioux Tribes v. Board of Oil and Gas Conservation*, 792 F.2d 782, 794 (9th Cir. 1986); *Trust*, 89 C.J.S. §§ 246-62; Morisset, *Recent Developments in Defining the Federal Trust Responsibility*, *supra* note 3.

³⁸ 537 U.S. 465, 475 (2003)

³⁹ *Covelo Indian Community v. FERC*, 895 F.2d 581, 586 (9th Cir. 1990).

1970s” (TNC 2003). Incidences of trespass grazing by sheep continue to be reported occasionally along the western edge of the Site (TNC 2003).

“In May 2000, 175,000 acres of the Hanford Site surrounding Central Hanford was designated as the Hanford Reach National Monument by proclamation of President William J. Clinton. DOE continues to have administrative jurisdiction over Monument lands, is the primary manager for some portions of the Monument, and cooperates with US FWS in comanagement of other Monument Lands. Five management units of the Hanford Reach National Monument—the Fitzner-Eberhardt Arid Lands Ecology Reserve, the McGee Ranch– Riverlands Unit, the Saddle Mountain Unit, the Wahluke Unit, and the River Corridor Unit—encircle Central Hanford, which remains under DOE management” (TNC 2003).

FEDERAL GOVERNMENT OPERATIONS

Site Operational History

The Hanford Site was the world’s first nuclear production facility. The site location was originally selected due to its remoteness, available electrical power from the Grand Coulee Dam, a functional railroad, a cool, flowing water source (the Columbia River), and the availability of sand and gravel for construction (Poston 2010). Construction of nuclear facilities at the Site began in 1943 as part of the Manhattan Project, a secretive World War II government program with the goal of manufacturing an atomic bomb. Extraordinary measures were taken throughout the World War II era to ensure that progress continued on an accelerated schedule, often resulting in unprecedented scientific risks being taken and unorthodox means to acquire land and resources (DOE 2002).

In the over 40 years of nuclear operations, a total of nine reactors were constructed for the production of plutonium for national defense purposes. In 1943, DOE constructed the Site’s first three reactors (reactors B, D, and F). Of these, B Reactor was the world’s first industrial scale plutonium production reactor, and manufactured the plutonium used in the Trinity Test and Nagasaki atomic bombs.

After World War II, Hanford’s objective was shifted to nuclear production for the Cold War, and the Site underwent an extensive expansion phase including the construction of the DR and H complexes in the late 1940s. Construction of the C Reactor began in 1950, less than a mile from B Reactor, so that the two could share utilities, services, and facilities. The two reactors in the 100-K area were larger than all of their predecessors, and construction of these reactors began in 1953. The last reactor, N Reactor, was completed in 1963. All nine reactors were decommissioned by the late 1980s, although additional testing facilities (in the 400 Area, specifically) remained active until the early 1990s.

DOE operational and research areas on the Hanford Site include the 100, 200, 300, 400, and 1100 Areas (has since been transferred to Port of Benton), described below and shown in Exhibit 2-1. The 600 Area designation encompasses all areas not included within the 100, 200, 300, 400 or 1100 Areas.

- The **100 Areas**, consisting of six operable units, are where the nine plutonium-producing reactors were located;
- The **200 Area**, split into the East and West portions, includes facilities for chemical separation and extraction, and plutonium finishing. It also houses dozens of underground storage tanks (known as “tank farms”) that store highly contaminated radioactive waste, byproducts of the plutonium extraction process;
- The **300 Area**, where nuclear fuel fabrication and development were performed;
- The **400 Area**, located just north of the 300 Area, houses the Fast Flux Test Facility, a reactor that was designed to test and research various types of nuclear fuel.
- The **1100 Area** included an area just north of Richland and a non-adjacent area on the Arid Lands Ecology Reserve. The portion near Richland contained offices associated with administration, maintenance, transportation, materials procurement and distribution, waste sites, French drains, underground tanks, and a sand pit. The portion on the Arid Lands Reserve is a former missile base and control center. Remedial actions selected for the 1100 Area have been completed and the site was delisted from the NPL in 1996 (DOE 2011d).⁴⁰

The process areas were designed to have structural redundancy so that each could function as an independent unit. Each contained its own facilities for operations, support, administration, security, health, communication, utilities, and waste disposal, the ultimate goal being the uninterrupted production of weapons-grade plutonium (DOE 2002).

Presently, the DOE Richland Operations Office, the Office of River Protection, and the DOE Office of Science and their contractors jointly manage cleanup, treatment, disposal, and research in the central portion of the Hanford Site in what has become the world’s largest environmental remediation project (Poston 2010).

The buffer zone of the Site was established as a national monument in 2000 in order to protect rare resources, specifically, unimpounded portions of the Columbia River and areas of shrub-steppe ecosystem (Poston 2010). Units of the Hanford Reach National Monument are managed by DOE, US FWS, and WDFW.

National Priorities List (NPL) Designation

Nuclear fuel production activities, disposal practices, and releases at Hanford resulted in the Site qualifying for inclusion on the EPA’s NPL. In anticipation of Hanford’s inclusion on the NPL, in May 1989, DOE, EPA, and Washington State Department of Ecology signed the Hanford Federal Facility Agreement and Consent Order or Tri-Party

⁴⁰ The 1100 Area land and facilities have been transferred to the Port of Benton. However, DOE maintains institutional controls, as required by DOE 1996, *Superfund Final Closeout Report, U.S. Department of Energy 1100 Area*, and EPA/ROD/R10-93/063, *Record of Decision for the USDOE Hanford 1100 Area Final Remedial Action*.

Agreement, which established a legal framework and schedule for cleanup, and designated a lead regulatory agency (either EPA or Washington State Department of Ecology) for each operable unit.

On November 3, 1989, Hanford was added to the NPL as four separate sites: the 100 Area, 200 Area, 300 Area, and 1100 Area.⁴¹ In order to coordinate response actions, each of these sites is further subdivided into operable units (OUs), based on geographic area, common waste sources, and natural resource type (soil and groundwater contamination are addressed in separate OUs). Additionally, waste management units have been identified throughout the Hanford Site; these units, based on waste disposal practices, are much smaller than operable units and are grouped among the four NPL sites (DOE 2006a).

Ongoing and planned cleanup work at Hanford is expected to address, but will not be limited to, more than 50 million gallons of highly contaminated liquid waste in 177 underground storage tanks, 2,300 tons of spent nuclear fuel, 12 tons of plutonium in various forms, approximately 25 million cubic feet of buried or stored solid waste, and approximately 270 billion gallons of groundwater contaminated above drinking water standards (and occurring over an area of approximately 80 square miles), more than 1,700 waste sites, and approximately 500 contaminated facilities.

Additional summary information describing the four Hanford NPL sites and the current status of remediation efforts is provided in Appendix A. More detailed information can be found at <http://www.hanford.gov/>.

**2.2 OVERVIEW OF
RELEASES OF
HAZARDOUS
SUBSTANCES**

Between fuel fabrication in the 300 Area, fuel irradiation in the 100 Area, and fuel processing and plutonium recovery in the 200 Area, operations at Hanford resulted in the release of many hazardous substances, including radionuclides as well as other inorganic and organic contaminants (Ballinger and Hall 1991).

300 AREA

The 300 Area supported the first step of the plutonium production process, fuel fabrication, as well as research and development activities. Construction of fuel fabrication facilities began in 1943 and fuel fabrication operations began in 1944 (Ballinger and Hall 1991). Fuel fabrication consists of molding and encapsulating uranium in metallic alloy cladding so that it can be used as nuclear fuel in reactors (DOE 2008). Once the fuel was fabricated, it was transported to the 100 Areas for irradiation in the nuclear reactors (DOE 2011b).

Operations in the 300 Area generated both solid and liquid waste. While there is some evidence of air emissions associated with fuel fabrication and research activities in the 300 Area, these air emissions were relatively minor (Stratus 2009). Before 1973,

⁴¹ Remedial actions selected for the 1100 Area have been completed and the site was delisted from the NPL in 1996 (DOE 2011d).

operators at the Hanford Site stored solid waste and debris generated by 300 Area operations in solid waste burial grounds in the 300 Area. After 1973, these burial grounds were no longer used as waste was transported to other Hanford Site burial grounds (DOE 2008).

Contaminated liquid wastewater generated in the 300 Area was deposited in surface impoundments such as unlined ponds and trenches located in the 300 Area. These liquid wastes were primarily contaminated with uranium from the fuel fabrication process, and the ponds and trenches are now “suspected to be the primary source of uranium in the groundwater beneath the 300 Area” (DOE 2008). Evidence suggests that these underground storage tanks leaked hazardous substances to the subsurface, possibly further contributing to soil and groundwater contamination (Stratus 2009).

The 300 Area fuel fabrication operations ended in 1988 after the final nuclear reactor shut down (Ballinger and Hall 1991). Today, the “300 Area contains solid waste disposal sites, burn pits, ash pits, catch tanks, cribs, drains fields, dumping areas, foundations, French drains, injection wells, laboratories, process sewers, ponds, process facilities, radioactive process sewers, storage areas, storage tanks, surface impoundments, trenches, and unplanned releases” (DOE 2011b). Remediation operations in the 300 Area are ongoing.

100 AREA

Once nuclear fuel was fabricated in the 300 Area, it was transported to the 100 Area for irradiation in the nuclear reactors. From 1943 to 1963, over the course of three post-World War II production capability expansions and the peak years of plutonium production, nine nuclear reactors were built in the 100 Area (Gerber 2001). Eight of these nuclear reactors (the B, C, KW, KE, D, DR, H, and F Reactors) were single-pass reactors that relied upon water withdrawn from the Columbia River to cool the reactors before returning the water to the River. The ninth reactor (the N Reactor) “recirculated purified water through the reactor core in a closed-loop cooling system” (DOE 2008). These nuclear reactors used fabricated fuel to produce weapons grade plutonium via nuclear reactions. The closed-loop N Reactor, unlike the other reactors, acted as a dual-purpose reactor that also produced electrical power (Ballinger and Hall 1991).

Operations in the 100 Area produced contamination in the form of air emissions, solid wastes, and liquid wastes. Sources of air emissions in the 100 Area included stacks related to the nuclear reactors, as well as incinerators and open burn pits. Airborne emissions from the stacks primarily occurred in the 1940s and 1950s before the introduction of filtration systems in the 1960s (although ongoing radionuclide air emissions are still released at low levels from some Hanford operational sites, the emissions are permitted and regulated by Washington State and inventoried annually (DOE 2010b)). Radioactive waste generated in the 100 Area was divided into “soft waste (combustibles) and hard waste (greater than 99% metallic)” (DOE 2011b). Soft wastes with less potent radioactive contamination were buried in the 100-F Area, burned in open pits, or incinerated in the 100-K Area (DOE 2011b). For soft wastes that were burned, the open burn pits and incinerator operations resulted in the airborne release of

“radionuclides, organics, metals, and other hazardous substances” (Stratus 2009). Hard wastes generated in the 100 Area were disposed of in burial grounds or, for highly contaminated radioactive wastes, transported to the 200 Area for burial. In addition, irradiated fuel from N reactor was stored in canisters in the K basins located in the 100 Area after N reactor was closed (Stratus 2009).

Liquid wastes generated in the 100 Area were primarily related to the waters used to cool the nuclear reactors. For the closed-loop N Reactor, the highly contaminated liquid effluent resulting from cooling operations “was discharged to trenches and cribs near the river” (DOE 2008). For the eight single-pass reactors, water was withdrawn from the Columbia River, sent to treatment facilities for purification, passed through the reactors, and then sent to retention basins to cool and “allow for decay of short-lived radionuclides” (DOE 2008). From there, most of the water was returned to the Columbia River, while portions of highly radioactive water were diverted to surface impoundments, including trenches, cribs, and French drains (DOE 2008). The effluent water sent to the Columbia River was often discharged at high temperatures, with traces of hazardous substances such as radionuclides, chromium, and other hazardous substances (Stratus 2009). Although a change in the water treatment process in 1961 reduced radioactive contamination in the discharge water, this pathway of contamination continued until the last single-pass reactor was shut down in 1971 (Ballard and Hall 1991).

An additional issue linked to the single-pass reactors was sodium dichromate contamination of groundwater resources; sodium dichromate was used as a corrosion inhibitor and it likely migrated to groundwater via unplanned releases of reactor coolant water (DOE 2011b). Significant amounts of chromium contamination also resulted from inadvertent discharges of sodium dichromate spilled in the handling process, when granular dichromate was mixed in batches to create solutions for mixture into cooling waters. The solutions were delivered to treatment plants via pipeline, rail car, truck, and other methods, the process of which may have resulted in additional spills. There is likely an ongoing source of chromium contamination from a dichromate transfer station in the 100-D Area (Qafoku *et al.* 2011).

More highly contaminated water was diverted to surface impoundments such as trenches, cribs, and French drains. This water was frequently contaminated with radioactive isotopes such as cesium, strontium, and iodine, which led to contamination of the soil and the underlying groundwater (DOE 2011b).

The eight single-pass reactors were shut down between 1964 and 1971 and the closed-loop N Reactor was shut down in 1988 (Ballinger and Hall 1991). Following the cessation of reactor operations, remediation activities for the burial grounds, retention basins, groundwater resources, and other contamination sites commenced and are ongoing (DOE 2008).

200 AREA

Following irradiation in the 100 Area, fuel elements were transported to the 200 Area for processing and separation of the irradiated fuel. These processing operations were

designed to extract plutonium from the irradiated fuel by dissolving irradiated “fuel elements with acids and then chemically [separating] the plutonium isotopes from the liquefied materials” (DOE 2008). Five separation plants (T, B, U, REDOX, and PUREX) were constructed in the 200 Area between 1944 and 1952 (Ballinger and Hall 1991). This final processing step in the plutonium production process produced significant amounts of contamination, primarily in the form of air emissions and liquid wastes.

When nuclear operations first began in 1944, 200 Area stacks for the chemical separation plants generated large quantities of airborne emissions, including radioactive and non-radioactive hazardous substances (i.e., iodine-131, volatile organic compounds (VOCs), nitrate compound particulates, and gaseous ammonia) (Stratus 2009). Although these emissions were reduced in the late 1940s and early 1950s through a series of iterative improvements to the filtration devices on these stacks (Ballinger and Hall 1991), some significant releases continued into the 50s including large releases of ruthenium from 1952 to 1954 (Selby and Soldat 1958). In fact, several retired facilities continue to produce minor emissions, which are regulated and permitted by the State of Washington. As of 2009, the 200 Areas released nine different radionuclides, totaling 2.14 E-03 Ci (DOE 2010b).

In addition to airborne releases, the chemical processing of irradiated fuel in the 200 Area produced significant quantities of liquid waste. Less contaminated liquid wastes were primarily disposed of in “liquid waste receiving sites (i.e., ponds, cribs, trenches, reverse wells, ditches, and cribs)” (DOE 2008). These wastes percolated into the soil column and eventually migrated to groundwater resources, resulting in contamination of the vadose zone and groundwater (DOE 2008). More highly contaminated wastes were neutralized and directed to underground storage tanks in the 200 Area via underground pipes. Initially, the underground storage tanks were arranged in twelve groups, or tank farms, that collectively included 149 single-shell tanks (Ballinger and Hall, 1991). In the tanks, heavier components settled out of solution, forming sludge. Because tank space was limited, though, Hanford operators would discharge the remaining liquid effluent to the soil column via the waste receiving sites, making room for additional highly contaminated waste (Stratus 2009). Over time, environmental monitoring efforts discovered that the single-shell tanks were leaking. This prompted the construction of 28 double-shell tanks in the 200 Area, and drainable liquid wastes were pumped from the single-shell tanks to the double-shell tanks to prevent further leakage and contamination. Many of the 149 single-shell tanks, however, still contain highly contaminated non-drainable wastes, and remain a risk of future releases (DOE 2009). It is now believed that 67 out of 149 single-shell tanks leaked (Gephart 2003b). Between the storage tank leaks and the liquid waste discharges to the soil column, the 200 Area released significant quantities of radionuclides (e.g., cesium-137, iodine-129, strontium-90, uranium, and tritium), as well as inorganic and organic chemicals (e.g., nitrate, sodium, phosphate, sulfate, ammonia, carbon tetrachloride, and sodium dichromate), which have contaminated the underlying groundwater (DOE 2008).

OTHER RELEASES

In addition to hazardous substances generated and released in the course of managed Site operations, there have been “numerous episodic events at the Site, such as overland flow, spills, leaks, explosions and wildfires that may have resulted in the release of hazardous substances into the environment” (Stratus 2009). Examples of these releases include the following.⁴²

- 1948: In October of this year, a large liquid waste pond in the 300 Area failed, resulting in “the release of 14.5 million gallons of uranium-contaminated water” into the Columbia River (Stratus 2009). It is estimated that “12 to 16 pounds of elemental uranium entered the Columbia River” (*ibid*).
- 1949: To test the usefulness of atmospheric sampling for radioisotopes indicative of fuel processing, Hanford operators bypassed stack filters on the chemical separation plants and released radioactive gases, including 11,000 curies of iodine-131 and xenon-133. This experiment was known as the “Green Run” (Gephart 2003b).
- 1953: “An unintentional chemical reaction resulted in the violent ejection of metal waste spray from a vault in one of the tank farms in the 200 Area...The volume released was unspecified but should not have exceeded the 15,000 gallon storage capacity of the vault. The contamination spread to the southeast, and covered the eastern half of the tank farm” (Stratus 2009).
- 1956: “500 gallons of metal waste overflowed the 241-UR-151 diversion box at the northeast corner of the U tank farm. In the same year, tank U-104 leaked an estimated 55,000 gallons of metal waste” (Stratus 2009).
- 1966: In the 100 Area, a spill “released 140,000 pounds of sodium dichromate, much of which reached the Columbia River, as a result of a storage tank transfer pump malfunction at the 183-C Building” (Stratus 2009).
- 1969: In the 200 West Area, “approximately 2,600 gallons of cesium-137 recovery process feed solution leaked... It is estimated that 11,300 curies of cesium-137, 18.3 kilograms of uranium, and 5.01 curies of technetium-99 were released to the subsurface” (Stratus 2009).
- 1997: “Leachate tanks at the ERDF leaked approximately 190 liters (50 gallons) of contaminated leachate” (Stratus 2009).
- 2003: “Approximately 757 liters (200 gallons) of diesel fuel leaked from a 242-S Facility tank on January 22, 2003. Contaminated soil was excavated and moved to a remediation area” (Stratus 2009).

⁴² The official listing of all unplanned releases at the Hanford Site is available in the Hanford Site Waste Management Units Report (last updated February 2012), available at http://www.hanford.gov/files.cfm/DOERL-88-30_R21.pdf.

- 2007: “Approximately 322 liters (85 gallons) of radioactive waste spilled from Tank 241-S-102 at the S Tank Farm on July 27, 2007” (Stratus 2009).

HANFORD SITE HAZARDOUS SUBSTANCES

Hanford Site operations have resulted in releases of hundreds of different hazardous substances. The Hanford Trustees have been engaging in an effort to identify those contaminants likely to be of greatest concern in the context of this natural resource damage assessment. Towards that end, the Trustees have examined a number of sources of information, including but not limited to information in Site risk assessments (e.g., CRCIA 1998, DOE 2011a, b), information on chemicals in the underground tanks (e.g., Gephart 2003b), in major groundwater plumes (e.g., DOE 2011c), data on releases (e.g., Hall 1991), and chemical measurements in Site databases. The Trustees’ work in this area is ongoing, and their preliminary focused list of hazardous substances (Exhibit 2-2) is subject to refinement in the future.

EXHIBIT 2-2 PRELIMINARY ASSESSMENT-FOCUSED LIST OF HAZARDOUS SUBSTANCES

| RADIOISOTOPES | ORGANICS | INORGANICS |
|-------------------------|-------------------------|--------------------------|
| Americium-241 | 1-2 Dichloroethane | Antimony |
| Carbon-14 | 1,4 Dioxane | Arsenic |
| Cesium-137 | 2,4,6 Trichlorophenol | Barium |
| Cobalt-60 | Acetonitrile | Boron |
| Europium-152 | Benzo(a)pyrene | Cadmium |
| Gadolinium-152 | Carbon tetrachloride | Chromium (includes Cr6+) |
| Iodine-129 | Chlordane | Cobalt |
| Neptunium-237 | Chloroform | Copper |
| Plutonium-239/240 | Cyanide | Fluoride |
| Potassium-40 | DDT/DDE | Lead |
| Radium-226, Ra-228 | Dichloromethane | Manganese |
| Strontium-90 | Glyphosate | Mercury |
| Technetium-99 | Hydrazine | Molybdenum |
| Thorium-232 | Hexone | Nitrate |
| Tritium | PCBs | Nickel |
| Uranium-233/34/35/38 | Tributyl Phosphate | Phosphate |
| Zirconium-93 | Trichloroethylene (TCE) | Selenium |
| Total radiological dose | Total Petroleum | Silver |
| | Hydrocarbons/PAHs | Strontium |
| | Vinyl chloride | Uranium |
| | | Vanadium |
| | | Zinc |

Different hazardous substances have the potential for different types of adverse effects to natural resources. Effects on biota may include (but are not limited to) genotoxicity, carcinogenicity, reproductive impairment, behavioral impairment, immunotoxicity, endocrine disruption, disruption of other physiological functions, and/or lethality, depending on the degree of exposure and the sensitivity of the exposed organism.

Different life stages of a given species may experience differential degrees of exposure and may also be differentially sensitive to a given exposure. In addition, exceedances of certain standards (e.g., promulgated water quality standards and criteria) can constitute an injury under DOI's NRDA regulations. Natural resources (e.g., surface waters, sediments, soils, groundwater, air, biota) can also be considered as injured if exposure to hazardous substances in those natural resources results in injury to other natural resources.

Providing a detailed description of the potential effects of the full suite of hazardous substances under consideration is beyond the scope of this assessment plan; however, Appendix B contains a series of ecotoxicity summaries for a subset of these including uranium, plutonium, cesium-137, iodine-129, strontium-90, technetium-99, tritium, PCBs, mercury, chromium (including hexavalent chromium), and carbon tetrachloride.

CHAPTER 3 | HABITATS, NATURAL RESOURCES AND ASSOCIATED ECOLOGICAL SERVICES

The Hanford Site lies in the semi-arid Pasco Basin of the Columbia Plateau in southeastern Washington State (US FWS 2008). The Site is situated along the banks of the longest continually flowing stretch of the Columbia River (the Hanford Reach), and is home to one of the largest areas of native shrub-steppe habitat remaining in the state. The Hanford Site’s unique terrestrial and aquatic ecosystems are home to forty species of mammals, over two hundred species of birds, and a large variety of amphibians, reptiles, and invertebrates. Furthermore, rare plant surveys conducted by The Nature Conservancy confirm the Site is a critical area for the conservation of rare shrub-steppe, riparian and aquatic plants (TNC 2003). The Hanford Reach of the Columbia River also supports a number of economically and/or culturally important fish and mollusk species such as the Chinook salmon (including the endangered Upper Columbia spring-run Chinook), coho salmon, sockeye salmon, steelhead (a Federally-listed threatened species), Pacific lamprey (a Federal Species of Concern), bull trout (threatened), white sturgeon, land snail, freshwater snail, Columbia pebblesnail, freshwater Limpet shortface lanx, and the California floater.

This chapter provides information characterizing Site habitats, describes the Site natural resources (as defined by the DOI natural resource damage assessment regulations) found within those habitats, and summarizes the ecological services these resources typically provide, as well as a preliminary determination of the time required for injured resources to once again provide these services (i.e., the “recovery period”) (human use services provided by these resources are described in Chapter 4). A number of earlier reports describe the Hanford Site’s natural resources in more detail.⁴³ This Assessment Plan does not attempt to re-create or supplant those efforts, but rather summarizes key subjects useful in placing the proposed assessment studies into a historic and ecological context.

3.1 AQUATIC HABITATS COLUMBIA RIVER

The Columbia River is the fourth largest river in the contiguous United States as measured in terms of flow, and is the dominant surface water resource at the Hanford Site (Burk *et al.* 2007). The river forms the northern and eastern boundary of the Site, flowing east and then turning south. The Hanford Reach, the portion of the river most closely associated with the Hanford Site, is approximately 51 miles long, extending from

⁴³ See, for example, Downs *et al.* 1993, Burk *et al.* 2007, and Fitzner and Gray 1991.

Priest Rapids Dam (River Mile (RM) 397) to McNary Pool (RM 346; US FWS 2008). The Hanford Reach “is the last non-impounded, non-tidal segment of the Columbia River in the United States” (Burk *et al.* 2007) and “contains significant riparian habitat that is otherwise rare within the Columbia River system” (National Park Service 1994 as cited in US FWS 2008).

Hydrology

Burk *et al.* (2007) provides the following description of the river’s hydrology.

“Flows through the Hanford Reach fluctuate significantly and are controlled primarily by releases from three upstream storage dams: Grand Coulee in the United States, and Mica and Keenleyside in Canada. Flows in the Hanford Reach are directly affected by releases from Priest Rapids Dam; however, Priest Rapids operates as a run-of-the-river dam rather than a storage dam. Flows are controlled for purposes of power generation and to promote salmon egg and embryo survival.⁴⁴ ...

Columbia River flows typically peak from April through June during spring runoff from snowmelt and are lowest from September through October. As a result of daily discharge fluctuations from upstream dams [i.e., Priest Rapids dam], the depth of the river varies over a short time period. River stage changes of up to 3 m (10 ft) during a 24-hr period may occur along the Hanford Reach (Poston *et al.* 2006). The width of the river varies from approximately 300 m (1,000 ft) to 1,000 m (3,300 ft) within the Hanford Reach. The width also varies with the flow rate, which causes repeated wetting and drying of an area along the shoreline.”⁴⁵

Burk *et al.* (2007) states “Large Columbia River floods have occurred in the past (DOE 1987), but the likelihood of recurrence of large-scale flooding has been reduced by the construction of several flood control/water-storage dams upstream of the Hanford Site.” There are no Federal Emergency Management Agency (FEMA) floodplain maps for the Hanford Reach because FEMA maps developing areas, while lands adjacent to the Hanford Reach are primarily under Federal control (*ibid.*). However, assessments of the Reach’s flood potential, including a scenario of potential dam failures, have been made and are summarized by Burk *et al.* (2007).

Columbia River Habitat Types

The Columbia River includes a variety of riparian habitats, including riffles-pools (areas with graded geomorphic attributes of riffles and pools), gravel bars, backwater sloughs,

⁴⁴ The Vernita Bar Agreement (signed in 1988 and expanded in 2004, by the U.S. DOE, Federal and state agencies, tribal governments, and public utility districts in Grant, Chelan, and Douglas counties) was created to prevent redds (salmon nests) from being left high and dry when river flows fluctuate to meet peak power demands.

⁴⁵ The flow rate varies from year to year, which affects the development and extent of vegetation in nearshore areas.

and shorelines.⁴⁶ Pacific Northwest National Laboratory (PNNL) has collected spatial information on substrate type (including sediments, sand, gravel, and large boulders), and in 2002, PNNL developed more detailed spatial information about nearshore substrates from Vernita Bridge to the 300 Area (Downs et al. 2004).

The Hanford Reach includes several slack water areas, including the White Bluffs slough (between 100-H and 100-F Areas), the F Area slough (about 1 mile downstream of the 100-F Area), and the Hanford slough at the old Hanford townsite (Weiss and Mitchell 1992). These areas are generally depositional, and typically include more vegetation than erosional areas. A number of fish species also use slack water areas as nursery habitat.

Some contaminants adhere to sediment and tend to be transported along with sediments; consequently, sediment depositional areas can serve as sinks for certain types of contaminants. Biota that live on or in these sediments, or that derive part of their food from sediment-associated food webs, may receive increased exposures to these contaminants.

SPRINGS AND STREAMS

Downs (2007) states:

“Springs are found on the slopes of the Rattlesnake Hills along the western edge of the Hanford Site (DOE 1988). There is also an alkaline spring at the east end of Umtanum Ridge (Hall 1998). Rattlesnake and Snively springs form small surface streams. Water discharged from Rattlesnake Springs flows in Dry Creek for about 3 km (1.6 mi) before disappearing into the ground (Figure 4.4-1). Cold Creek and its tributary, Dry Creek are ephemeral streams within the Yakima River drainage system in the southwestern portion of the Hanford Site. These streams drain areas to the west of the Hanford Site and cross the southwestern part of the Site toward the Yakima River. When surface flow occurs, it infiltrates rapidly and disappears into the surface sediments in the western part of the Site. The quality of water in these springs and streams varies depending on the source. However, they are up-gradient of Hanford waste sites and groundwater contamination plumes.”

Jamison (1982) provides additional information about Rattlesnake Springs, noting that it begins “from ground seepage and is subsequently fed by small ground springs along its course, flows for approximately 3 km... before disappearing into the ground.” Biota present at the spring include algae, cattails and sedges, and watercress (*ibid.*).

⁴⁶ A riffle is a section of a streambed characterized by shallow, steep slopes and fast moving water broken by the presence of rocks and boulders, and are typically at cross over locations. A pool is a reach of a stream characterized by deep, low velocity water and a smooth surface, and typically has a greater depth of flow and slope of the bed than that of riffles, often located at the outside of meander bends. (<http://www.streamnet.org/glossarystream.html>). A backwater slough is an inlet off of another waterway; as defined in Alaska Statute AS 41.17.950, it “(A) has sluggish flow, is warm in summer, and is typically only connected to the main stem or a side channel at one end of the water body; (B) carries river current only under high water conditions; and (C) may have only a seasonal connection to the main stem or side channel.”

Invertebrates are also present, although “the number of species present is highly dependent on the size of the winter floods” (*ibid.*).

PONDS AND DITCHES

There are a number of ponds on the Hanford Site, some of which were created as a result of water releases through trenches from processing facilities. Some of the major ponds include Gable Mountain pond, U-pond, B-pond, S-pond, T-pond, Westlake, and associated ditches. Contaminated ponds have been decommissioned, filled, and covered with soil.

Gable Mountain pond was one of “the most significant and extensively studied” ponds onsite (Jamison 1982). Gable Mountain pond was much larger than many other Hanford ponds and supported an abundance of plant life which provided food and shelter for wildlife. Vegetation, primarily cattails and rushes, were the predominant biota type associated with these ponds (*ibid.*). Table 7-4 in Jamison (1982) lists major taxa identified at these ponds. Decommissioning of Gable Mountain pond was completed in 1988, and the water table beneath the pond declined more than three feet between 1979 and 1989 as wastewater discharges ceased (Newcomer 1990).

The B-pond system included a series of ponds used for disposal of liquid effluent from past Hanford production facilities starting in 1945 (Barnett *et al.* 2000). In 1994, some of the ponds were closed, leaving only the main pond and a portion of one of the ditches as the currently regulated facility (*ibid.*). Minor contamination in groundwater and soil has been detected at the site, and levels of gross alpha and gross beta radiation and specific conductance are monitored semi-annually (*ibid.*).

West Lake is a natural feature recharged from groundwater, the B-pond system, and two 300 Area Treated Effluent Disposal Facility (TEDF) ponds – Gable Mountain and U-pond (Burk *et al.* 2007).⁴⁷ There are also several natural vernal ponds near Gable Mountain and Gable Butte (Hall 1998 as cited in Burk *et al.* 2007). West Lake “has not received direct effluent discharges from Site facilities; rather, its existence is caused by the intersection of the elevated water table with the land surface in the topographically low area. Water levels of West Lake fluctuated with water table elevation, which were influenced by wastewater discharge in the 200 Areas. The water level and size of the lake has been decreasing over the past several years because of reduced wastewater discharge” (*ibid.*).

3.2 TERRESTRIAL SHRUB-STEPPE

HABITATS

The upland terrestrial habitat on the Hanford Site consists primarily of shrub-steppe, considered to be some of the highest quality of this habitat type remaining in the State of Washington (Burk *et al.* 2007).

⁴⁷ Although the waste disposal ponds were used by wildlife, they are mentioned for completeness and are not themselves considered to be a natural resource.

A variety of specific shrub-steppe habitats exist on-site, each defined by the dominant shrub and grass species at a given location (Downs *et al.* 1993). For example, the healthy, intact shrub-steppe habitat at Hanford is characterized by an overstory dominated by big sagebrush (*Artemisia tridentata*) and an understory of bunchgrasses and forbs. On the Columbia River Plain, habitat is usually dominated by big sagebrush and non-native cheatgrass (*Bromus tectorum*), or a mixture of cheatgrass and native bunchgrasses (e.g., Sandberg's bluegrass (*Poa secunda*), Indian ricegrass (*Achnatherum hymenoides*)) (Downs *et al.* 1993).

Microbiotic crusts, which are formed primarily by algae, lichens, and mosses, serve a number of important ecological functions as a component of the shrub-steppe ecosystem. These functions include soil stability, erosion protection, nitrogen fixation, and nutrient contribution, as well as increasing water infiltration, seedling germination, and plant growth (Burk *et al.* 2007).

The shrub-steppe habitat of Hanford provides a variety of important functions for the biota described later in this report including foraging, nesting, burrowing, and hunting habitat, as well as cover (Burk *et al.* 2007).

Remaining shrub-steppe habitat in Washington is threatened by a number of factors including soil disturbance (e.g., due to overgrazing), development, invasive species, and wildfires (Washington Native Plant Society 2008). Because of its importance to a number of wildlife species, and the scarcity of the habitat type, the State of Washington considers shrub-steppe habitat to be a priority habitat, and DOI identifies the native shrub and grassland steppe in Washington and Oregon as an endangered ecosystem (US FWS 2011c).

SAND DUNES

The sand dune habitat found at Hanford is distinctive due to its atypical association with a shrub-steppe habitat. Dune habitat is dynamic, ranging from 2.5 acres to several hundred acres in size (U.S. Department of the Army 1990 as cited in Burk *et al.* 2007). Areas of sand dunes are found in several locations on the Hanford Site including along the shoreline in the area north of the Energy Northwest complex, near the 100-F area and westward to the area north of Gable Mountain, and along the eastern border of the Site. Fire has also resulted in the formation of temporary dunes along State Route 240 (Burk *et al.* 2007).

Predominant vegetation in the dune areas includes shrubs such as bitterbrush (*Purshia tridentata*) and gray (*Ericameria nauseosa*) and yellow rabbitbrush (*Chrysothamnus viscidiflorus*), with an understory of forbs and grasses including Indian ricegrass, scurfpea (*Psoralidium lanceolatum*), needle-and-thread grass (*Hesperostipa comata*), and thickspike wheatgrass (*Elymus lanceolatus*) (Burk *et al.* 2007). Dunes are known to support several plant species of concern, and thus are considered to be a sensitive habitat. The gray cryptantha (*Cryptantha leucophaea*), an ESA Species of Concern and Washington State sensitive plant species, grows in sandy soils in a variety of locations

across the Columbia River Plain, and is likely distributed across the dune habitat at Hanford (Downs *et al.* 1993).

Dunes also provide habitat for burrowing owls (*Athene cunicularia*), coyotes (*Canis latrans*), and mule deer (*Odocoileus hemionus*) (Burk *et al.* 2007). In addition, the bitterbrush that grows in sandy soils is considered to be an important forage resource for mule deer (Downs *et al.* 1993). A 2003 study of biodiversity by The Nature Conservancy found that several of the invertebrate fauna found in sand dunes at Hanford are extremely limited outside of the Hanford Site (TNC 2003).

The Hanford Dunes are reported to be the only active non-coastal dunefield in the State of Washington although other dune areas exist (TNC 2003).

WHITE BLUFFS

The White Bluffs are located on the northern shoreline of the Columbia River from RM 376 to RM 356 (Burk *et al.* 2007). The tops of the bluffs are dominated by Indian ricegrass, while the slopes are dominated by shrubs including greasewood (*Sarcobatus vermiculatus*) and spiny hopsage (*Grayia spinosa*) (Burk *et al.* 2007). The bluffs are home to at least two species of sensitive plants – Geyer’s milkvetch (*Astragalus geyeri* Gray), recognized as a sensitive species by the State of Washington and White Bluffs bladderpod (*Lesquerella tuplashensis*), recognized respectively as sensitive and threatened by the State of Washington (Sackschewsky and Downs 2001). The White Bluffs bladderpod is additionally a Candidate for listing under the ESA.

The bluffs provide perching, nesting and escape habitat for a number of bird species existing on the Hanford Site, including the prairie falcon (*Falco mexicanus*), red-tailed hawk (*Buteo regalis*), cliff swallow (*Hirundo pyrrhonota*), bank swallow (*Riparia riparia*), rough-winged swallow (*Stelgidopteryx serripennis*), Canada goose (*Branta canadensis*), and bald eagle (*Haliaeetus leucocephalus*). The bluffs are known to provide habitat for at least one Federal Species of Concern, the peregrine falcon (*Falco peregrinus*) (Burk *et al.* 2007).

COLUMBIA RIVER ISLANDS

The total area of island habitat within the Hanford Reservation is 4.74 square kilometers (Hanson and Browning 1959). Islands within the main channel of the Hanford Reach, including Locke Island, Wooded Island, and others, provide important habitat for a variety of plant, mammalian and avian species. The shoreline of the island is dominated by willow (*Salix spp.*), poplar (*Populus spp.*), Russian olive (*Eleagnus angustifolia*), and mulberry (*Morus alba*) (Burk *et al.* 2007). Plants species populating the interior of the islands include buckwheat, lupine (*Lupinus spp.*), mugwort (*Artemisia lindleyana*), thickspike wheatgrass, giant wildrye (*Leymus cinereus*), yarrow (*Achillea millefolium*), and cheatgrass (Warren 1980). The islands are used for resting, nesting, and escape by a variety of waterfowl and shorebirds, including the Canada goose, American white pelican (*Pelecanus erythrorhynchos*), California gull (*Larus californicus*), ring-billed gull (*Larus*

delawarensis), and Forster's tern (*Sterna forsteri*). There has also been documented use of the islands by mule deer for birthing, and by coyote for hunting (Burk *et al.* 2007).

Slumping of the White Bluffs has caused accelerated erosion of Locke Island, which is of great concern due to the cultural significance of the island and potential losses of cultural resources (Bjornstad 2006). Eroding sediments may also be sources of contamination and may be reducing the suitability of important salmon habitat in the Columbia River (Mueller and Geist 1999).

BASALT OUTCROPS, SCARPS, AND SCREES

A number of features on the Hanford Site support lithosol habitats or stony soils.⁴⁸ The tops and slopes of Rattlesnake Mountain, Umtanum Ridge, Yakima Ridge, Saddle Mountains, Gable Butte, and Gable Mountain are all characterized by basalt outcrops, scarps (cliffs), screes (loose rock at the base of cliffs or on slopes), and thin, rocky soils. Diverse plant communities can establish on these stony soils, typically dominated by short shrubs and grasses (Sackschewsky and Downs 2001).

Outcrops support some plants, including thyme buckwheat (*Eriogonum thymoides*), and Sandberg's bluegrass (Burk *et al.* 2007). Areas with higher elevation, including habitat on Rattlesnake Mountain, typically support greater plant diversity than lower elevations (Downs *et al.* 1993). Most of the scarps and screes occur on Umtanum Ridge, and are nearly devoid of vascular plants (Downs *et al.* 1993). However, on north-facing slopes some small islands of stabilized substrate develop, and can support squaw currant (*Ribes cereum*), bluebunch wheatgrass (*Pseudoroegneria spicata*), Sandberg's bluegrass, and forbs in early spring when moisture is available (Downs *et al.* 1993). Hoover's desert parsley (*Lomatium tuberosum*), an ESA Species of Concern, is confined to steep scree slopes. Additionally, the shrub steppe immediately adjacent to the basalt outcrops of Umtanum Ridge and Juniper Springs are known to support other plant species of concern (Downs *et al.* 1993).

The unique geomorphology of basalt outcrops, scarps, and screes provide habitat for rattlesnakes (*Crotalus viridis*), woodrats (*Neotoma cinerea*), and other burrowing animals (Burk *et al.* 2007). Scarps on Umtanum Ridge, Rattlesnake Mountain, and Gable Butte provide nesting sites for prairie falcons and, historically, for ferruginous hawks (*Buteo regalis*); rock wrens (*Salpinctes obsoletus*), chukars (*Alectoris chukar*), and poorwills (*Phalaenoptilus nuttallii*) also nest on scarps and scree habitats (Downs *et al.* 1993).

ABANDONED FIELDS/DISTURBED AREAS

Past agricultural development, livestock grazing, and wildfires have created extensive areas of disturbed habitat that are dominated largely by non-native species. Additionally, contaminant releases and associated remedial activities have created disturbed areas.

⁴⁸ Lithosol is defined by Sackschewsky and Downs 2001 as gravelly, rocky, talus soils associated with basalt outcrops and cliffs.

These disturbed areas are concentrated around operational areas, and impact the distribution, movement, and extent of natural resources on the Hanford Site.

Dominant species in abandoned fields and disturbed areas include cheatgrass, tumble mustard (*Sisymbrium altissimum*), jagged chickweed (*Holosteum umbellatum*), and Russian thistle (*Salsola kali*) at low elevations. At higher elevations, such as the Snively Ranch in the Rattlesnake Hills, native black rye (*Secale cereale*) is still dominant (Downs *et al.* 1993). Similar species are found in areas that have been disturbed by grazing and wildfire.

3.3 NATURAL RESOURCES Pursuant to 43 CFR § 11.14(z), natural resources are defined as:

land, fish, wildlife, biota, air, water, ground water, drinking water supplies, and other such resources belonging to, managed by, held in trust by, appertaining to, or otherwise controlled by the United States...any State or local government...These natural resources have been categorized into the following five groups: surface water resources, groundwater resources, air resources, geologic resources, and biological resources.

This Plan focuses on abiotic and biological resources in the aquatic, riparian, and terrestrial habitats described above, and the ecological and human uses of those resources. Air, soil, and groundwater are exposed to Site-related contaminants and transport those contaminants to other resources (e.g., surface water, sediment, the hyporheic zone where groundwater and surface water mix, and biota). Within the aquatic habitat, surface water and sediment are the base of the aquatic ecosystem. The invertebrate community (e.g., mussels, crayfish, stoneflies) supports multiple species of fish, including special status fish, which vary depending on the microhabitat (e.g., riffle or pool). Other organisms that rely on aquatic invertebrates as prey include amphibians and reptiles, migratory and non-migratory birds, and multiple small mammals, such as several species of bats including the Hoary bat (*Lasiurus cinereus*) and Pallid bat (*Antrozous pallidus*). The invertivorous fish community is in turn preyed upon by piscivores such as the smallmouth bass (*Micropterus dolomieu*), bald eagle, great blue heron (*Ardea herodias*), and river otter (*Lutra canadensis*). The terrestrial habitat supports a wide array of species as well, including an invertebrate community (e.g., spiders, beetles, moths, and grasshoppers) reliant on soil for protection, food, etc.; several species of breeding songbirds; and several species of small mammals. Additionally, larger fauna such as mule deer, Rocky Mountain elk (*Cervus elaphus*), coyote, badger (*Taxidea taxus*), and black-tailed jackrabbit (*Lepus californicus*) utilize the shrub-steppe and grassland habitat.

SURFACE WATER RESOURCES

Surface water resources are defined as:

The waters of the United States, including the sediments suspended in water or lying on the bank, bed, or shoreline and sediments in or transported through coastal and marine areas (43 CFR § 11.14(pp)).

At the Hanford Site, surface water and sediment resources are found in all of the aquatic habitats described above (see section 3.1 on Aquatic Habitats). Surface water and sediment sources at the Site include:

- the Columbia River;
- springs on Columbia River riverbanks and Rattlesnake Springs;
- ponds and lakes, including West Lake;
- streams, including Cold Creek and Dry Creek; and,
- the Yakima River abutting the southernmost extent of the site.

The Columbia River is the predominant surface water resource at Hanford. While the Columbia defines the northern and eastern boundaries of the Site, the Yakima River also abuts the southern extent, and Cold Creek flows along the Site's southwestern edge.⁴⁹ Also prevalent, are a series of waste water ponds north of the Columbia within the National Monument. A map of significant surface water features at Hanford is provided below (Exhibit 3-1).

GEOLOGICAL RESOURCES

Geological resources are defined as:

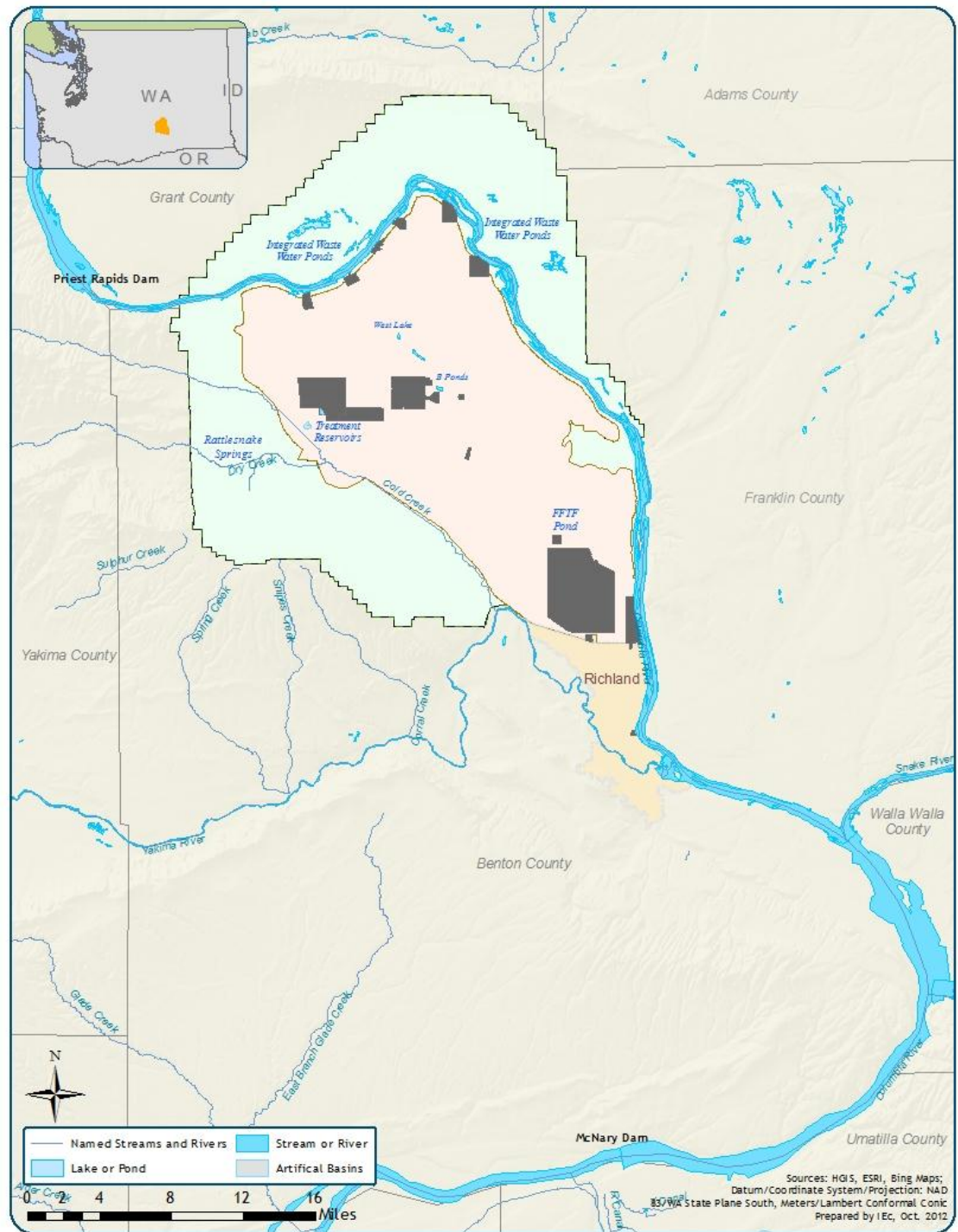
Those elements of the earth's crust such as soils, sediment, rocks, and minerals...that are not included in the definitions...of surface water resources (43 CFR Section 11.14 (s)).

The Hanford Site lies within the Pasco Basin, part of the larger Columbia Basin or Columbia Plateau. Relatively low-relief due to river and stream sedimentation filling in synclinal valleys and basins between the anticlinal ridges of the Yakima Fold Belt within the past several million years, the surface topography has been modified by Pleistocene cataclysmic flooding, Holocene eolian activity, and landsliding (Burk *et al.* 2007). Cataclysmic floods during the Pleistocene eroded sediments and scoured basalt bedrock, creating branching flood channels, giant current ripples, ice rafted erratics, and giant flood bars, which can all be found on the Hanford Site (Burk *et al.* 2007).

The Site consists of a layered depositional model, with basalt bedrock in the deepest (oldest) layer, overlain by Ringold formation sediments, Cold Creek sediments, and with Hanford formation sediments as the top (youngest) layer. However, these layers have been complicated by the method of deposition, and later by the removal of some of the sedimentary units (DOE 2011c). A description of each of the stratigraphic layers, from oldest to youngest, and additional information on some of these complications is provided in DOE 2011c.

⁴⁹ For additional detail regarding surface water resources, see section 3.1 on Aquatic Habitats, above.

EXHIBIT 3-1 SURFACE WATER FEATURES ON HANFORD SITE



Surface Soil

Of particular concern for this natural resource damage assessment are the soils in the suite of terrestrial habitats described above (i.e., shrub-steppe, sand dunes, white bluffs, Columbia River islands, basalt outcrops, scarps, scree, and, in particular locations, agricultural or disturbed habitat). Soils have been directly exposed to contaminants, and also act as a pathway of contaminants to terrestrial biota (see Chapter 5 for more details).

Vadose Zone Soil

In addition to the soils described above, Trustees are investigating injury to soils within the vadose zone at the Site – that is, the geological resources that extend from the surface of the ground to the water table. The Hanford Site vadose zone ranges in thickness from less than one meter near the Columbia River to over 50 meters on the Central Plateau (DOE 2011c).

Moisture consistently moves through the vadose zone to groundwater. Prior to the mid-1990s, the major source of moisture was liquid discharges from the Site; currently, the major moisture sources include precipitation and water used for dust suppression during remediation. The rate of deep drainage from the vadose zone into the groundwater (i.e., the migration path and time required for a contaminant to pass through the vadose zone) depends on hydraulic, physical, and chemical conditions in the soil, total soil moisture content, the total amount of water available, recharge rates, depth to the water table, and the presence of vegetation (Burk et al. 2007, Freeman et al. 2001). However, since precipitation is fairly low at Hanford, annual infiltration is limited but over time can be an important driving force for transport of near-surface contaminants.

GROUNDWATER RESOURCES

Groundwater resources are defined as:

Water in a saturated zone or stratum beneath the surface of land or water and the rocks or sediments through which groundwater moves. It includes groundwater resources that meet the definition of drinking water supplies (43 CFR Section 11.14 (t)). Drinking water supply means any raw or finished water source that is or may be used by a public water system, as defined in the SDWA [Safe Drinking Water Act], or as drinking water by one or more individuals (43 CFR Section 11.14 (o)).

As described in Burk *et al.* (2007), “groundwater at the Hanford Site originated as either recharge from rain and snowmelt, or from excess irrigation, canal seepage, and wastewater disposal.” Additionally, seasonal bank recharge from the Columbia River is an important source of groundwater on Site. Most of the Hanford groundwater eventually discharges into the Columbia River, although some may be brought to the surface through wells or evaporation and transpiration in areas where the water table is shallow (Burk *et al.* 2007).

The groundwater on the Hanford Site is found in both an upper unconfined sedimentary aquifer system and a deeper basalt confined or semi-confined aquifer system (DOE

2011c, Burk *et al.* 2007). Although parts of the unconfined aquifer are semiconfined or confined, the entire suprabasalt aquifer system is interconnected site-wide (DOE 2011c, Burk *et al.* 2007). Unconfined aquifer groundwater typically flows from recharge zones near the western part of the Site towards the Columbia River on the eastern and northern boundaries of the Site. The Yakima River near the southwest boundary of the Hanford Site is a source of recharge (DOE 2011c). Recharge rates vary across the Site, due to changes in vegetation and soil type, and range from 1.5 millimeters per year in natural shrub-steppe areas to 52 centimeters per year in un-vegetated areas (DOE 2011c). Recharge rates can also be artificially supplemented from Hanford wastewater disposal operations. To-date an estimated 1.68×10^{12} liters of wastewater have been discharged to disposal ponds, trenches, and cribs, increasing the water table elevation during operating years (DOE 2011c). Discharges in the Central Plateau caused groundwater mounding as high as 20 meters during peak operations (Stratus 2009). However, Hanford Site wastewater discharges have declined steadily in volume over the years, from approximately 14 billion liters in 1990 to 0.33 billion liters in 2010. Subsequently, the water table has been declining in most areas since 1980; Central Plateau levels have decreased to approximately 11 meters (Stratus 2009). Groundwater levels have also declined across the Site since non-permitted discharges to unlined ponds ceased in 1996 (DOE 2011c).

The confined/semi-confined aquifer system is located within the Columbia River Basalt Group. Most of the water in basaltic aquifers comes from precipitation and stream flow, and the groundwater generally flows toward the Columbia River; in some places, groundwater flows toward semi-confined areas where groundwater flows upward from the basalt into the overlying unconfined sedimentary aquifer system. This upward flow occurs in areas where the basalt is not completely confined and where there is an upward hydraulic gradient between the basalt and the overlying unconfined sedimentary aquifer system. Such upward gradients have been detected at several areas of the Site, due in part to significant declines in the unconfined water table as wastewater disposals ceased over the past 20 years.

Interactions between Groundwater and the Columbia River

The groundwater system at Hanford is highly influenced by the Columbia River flow system, and there is a dynamic zone of interaction where groundwater mixes with river water (DOE 2011c). This situation occurs in the 100 and 300 Areas, where during the high river stage, river water moves into the riverbank, overlaying the groundwater and mixing with it (Peterson and Johnson 1992). As the river water levels fall, the water flows back towards the river. Additionally, groundwater enters the Columbia River through a number of upwellings. Although the nature and extent of groundwater upwelling is unclear, upwelling locations have been identified within the 100 and 300 areas within the Hanford Reach (Hulstrom and Tiller 2010; Hulstrom 2010). A number of contaminants in these upwellings have been documented at levels exceeding water quality standards, including hexavalent chromium, nitrate, and uranium (Hulstrom 2011).

These interactions between groundwater and surface water can affect contaminant concentrations and cause varying hydraulic gradients by river-stage fluctuations. The effects of groundwater and surface water interaction on contaminant concentrations depends on a number of key variables such as flow patterns in the zone of interaction, the location of groundwater discharge, and the degree and timing of dilution prior to discharge into the riverbed substrate and the free stream (Peterson and Connelly 2001).⁵⁰

BIOLOGICAL RESOURCES

Biological resources are defined as:

Those natural resources referred to in section 101(16) of CERCLA as fish and wildlife and other biota. Fish and wildlife include marine and freshwater aquatic and terrestrial species; game, nongame, and commercial species; and threatened, endangered, and State sensitive species. Other biota encompass shellfish, terrestrial and aquatic plants, and other living organisms not otherwise listed in this definition (43 CFR Section 11.14 (f)).

The biological resources potentially exposed to releases from the Site include, but are not limited to, aquatic and terrestrial plants, aquatic and terrestrial invertebrates, reptiles and amphibians, fish, birds, and mammals that utilize the aquatic and terrestrial habitats described above. The following sections provide a brief description and inventory of the biological resources present on the Hanford Site. Additional information on the specific species documented on the Hanford Site is available in Appendix C and in other sources (e.g., Gray and Dauble 1977, Fitzner and Gray 1991, Downs *et al.* 2004, CRICIA 1998, TNC 1999, TNC 2003, Burk *et al.* 2007, US FWS 2008, and information from the Hanford Site Environmental Monitoring and Compliance Project presented in Downs *et al.* 1993 and the annual Hanford Site Environmental Monitoring Reports).⁵¹

Aquatic and Riparian Plants

Hundreds of plant species have been documented on the Hanford Site (Sackschewsky and Downs 2001). Aquatic plants are typically found in the narrow riparian areas along the Columbia River, which consist “of a number of forbs, grasses, sedges, reeds, rushes, cattails, and deciduous trees and shrubs. Much of the riparian zone has also been successfully invaded by exotic plant species” (Sackschewsky and Downs 2001). Dominant vegetation includes mulberry (*Morus alba*), willow, Siberian elm (*Ulmus pumila*), Northern wormwood (*Artemisia campestris*), sweet clover (*Melilotus spp.*), and reed canarygrass (*Phalaris arundinacea*) (DOE 2007a). Burk *et al.* (2007) provides a list

⁵⁰ Additional information on the interaction of groundwater and the Columbia River can be found in FLUOR 2008, Lee *et al.* 1997, Peterson and Johnson 1992, and Peterson and Connelly 2001.

⁵¹ These lists show many, but not all, species present at the Hanford Site. Note that inclusion of a species in this assessment plan does not imply an obligation on the part of the Trustees to evaluate it, nor does omission of a species preclude the Trustees from evaluating potential injury to that species.

(see Table B1) of riparian vegetative species in the area, based on Sackschewsky and Downs (2001). TNC (1999) identified rare riparian community plants in riverine emergent wetlands.

In the Hanford Reach, phytoplankton consists predominantly of diatoms (Weiss and Mitchell 1992), but green algae, blue-green algae, red algae, and dinoflagellates have also been found (Burk *et al.* 2007). Populations are heavily influenced by Priest Rapids dam and the changing water levels (Burk *et al.* 2007). Many of the free-floating algae species in the Hanford Reach are derived from the periphyton. The phytoplankton and periphyton community make up the base of the aquatic food web and are an important food source for many herbivores such as immature insects.

Macrophytes are “sparse in the Columbia River because of strong currents, rocky bottom, and frequently fluctuating water levels” (Burk *et al.* 2007), and are most prevalent in the slack water areas (Weiss and Mitchell 1992). Where present, macrophytes provide food, shelter, and breeding areas for fish. Weiss and Mitchel (1992) provide a list of macrophyte species present in the Hanford Reach.

Terrestrial Plants

The terrestrial vegetative communities on the Hanford Site are dominated by shrubs and steppe grasses. The shrub-steppe communities that once covered over 200,000 square miles of the American West have been largely eliminated or fragmented as a result of agricultural development and urbanization. The Hanford Site, with hundreds of documented plant species, represents one of the last relatively undisturbed tracts of this plant community remaining (Sackschewsky and Downs 2001).

Terrestrial plant community type (i.e., dominant shrub and grasses) is determined by climatic conditions, topographic conditions, soil type and depth, and land disturbance history. Big sagebrush is the dominant shrub in the majority of shrub-steppe plant communities found at Hanford. Other common species include grey rabbitbrush (*Chrysothamnus nauseosus*), green rabbitbrush (*Chrysothamnus viscidiflorus*), antelope bitterbrush (*Purshia tridentata*), spiny hopsage (*Grayia spinosa*), and black greasewood (*Sarcobatus vermiculatus*). Communities in which shrub species dominate are typically associated with an understory of grasses and forbs. Common grass species include Sandberg’s bluegrass (*Poa secunda*), cheatgrass (*Bromus tectorum*), needle-and-thread grass (*Hesperostipa comata*), bluebunch wheatgrass (*Pseudoroegneria spicata*), Indian ricegrass (*Oryzopsis (=Achnatherum) hymenoides*), saltgrass (*Distichlis stricta*), and Idaho fescue (*Festuca idahoensis*) (Sackschewsky and Downs 2001). At higher elevations, Sandberg’s bluegrass is replaced by bluebunch wheatgrass, Cusick’s bluegrass (*Poa cusickii*), hawk’s beard (*Crepis atrabarba*), and Idaho fescue become more abundant, and three-tip sagebrush (*Artemisia tripartita*) is found at the highest elevations (Downs *et al.* 1993).

Of the 725 plant species documented at Hanford, approximately 20 percent are non-native. A number of noxious weeds have successfully established and displaced native forbs, including rush skeletonweed (*Chondrilla juncea*) and several species of

knapweeds. Areas that have been disturbed by activities such as cultivation, fire, grazing, or construction activities are typically dominated by exotic annual species such as cheatgrass, tumble mustard (*Sisymbrium altissimum*), and Russian thistle. Past fires, such as the major fire in the year 2000 which consumed much of the shrub-steppe habitat in the ALE Reserve, have greatly contributed to altering the plant community – allowing non-native species to invade and significantly influence the Hanford habitat (Burk *et al.* 2007). The introduction of cheatgrass in particular has also resulted in significant alterations to distribution and abundance of native plants (Sackschewsky and Downs 2001).

A number of plant species whose populations are considered to be of concern by the Federal government and the State of Washington occur on the Hanford Site, such as Columbia milkvetch (*Astragalus columbianus*), Columbia yellowcress (*Rarippa columbiae*), and Hoover's desert parsley (*Lomatium tuberosum*). Although no plant species is currently listed as threatened or endangered under the ESA, two plant species are currently candidates for listing, Umtanum Desert buckwheat (*Eriogonum codium*) and White Bluffs bladderpod.

Terrestrial Invertebrates

Between 1994 and 1999, The Nature Conservancy conducted an insect inventory at Hanford that resulted in at least 1,536 species-level terrestrial invertebrate identifications, including identification of 43 previously unknown taxa. At the time of publication, researchers anticipated that after identification of all remaining samples the inventory would result in a total of over 2,000 species identified. Of those species identified during the survey, 142 were identified in the State of Washington for the first time, making Hanford the only known location for these species in Washington State (TNC 1999). The authors of this study attribute the high diversity of insect species on the Hanford Site to the size, complexity, and relatively undisturbed quality of the shrub-steppe habitat.

Biomass estimates indicate that the major taxonomic groupings at the Hanford Site are *Coleoptera* (beetles), *Hymenoptera* (ants, bees and wasps), and *Lepidoptera* (moths and butterflies) (Downs *et al.* 1993).

Two species, the Columbia River tiger beetle (*Cicindela columbica*) and the silver-bordered fritillary butterfly (*Boloria selene atrocotalis*) are listed as Candidate species by the State of Washington (Appendix C).

Aquatic Invertebrates

TNC (1999) conducted a limited reconnaissance survey and identified 52 taxa of aquatic invertebrates, including 21 not previously documented in the Hanford Reach. This discovery brought the total number of identified aquatic invertebrate taxa to 145 (*ibid.*). The study also investigated aquatic invertebrates in Hanford Reach tributaries and spring streams.

TNC (2003) continued the work of TNC (1999), surveying and compiling existing records of aquatic invertebrates in the Hanford Reach and other area locations. The

authors conclude that over the past 50 years a variety of changes have occurred in the Hanford Reach: “*Ephemeroptera* (mayfly) diversity has increased; *Plecoptera* (stoneflies) have disappeared; *Trichoptera* (caddisfly) diversity and abundance remain high; *Odonata* (dragonflies and damselflies), *Hemiptera* (true bugs), *Lepidoptera* (butterflies and moths) and *Coleoptera* (beetles) are rare; and *Diptera* (fly) diversity remains relatively constant.” The Pacific crayfish (*Pacifasticus leniusculus*) population “appears to be robust” and the introduced Asiatic clam (*Corbicula fluminea*) “appears to be extremely abundant” (*ibid.*).

Mueller *et al.* (2011) evaluated the species, distribution, and densities of native freshwater mussels in the Hanford Reach. Four species of native mussels were identified, of which the western and Oregon floaters (*Anodonta kennerlyi* and *Anodonta oregonensis*) were most abundant. The California floater (*Anodonta californiensis*), though it is listed as a Federal Species of Concern and State Candidate species, was the next most abundant, while the formerly-abundant western pearlshell (*Margaritafera falcata*) appears to have been extirpated.

In addition to the California floater, two additional species, the Giant Columbia River spire snail (*Fluminicola* (also known as *Lithoglyphus*) *columbiana*), and the shortfaced lanx (*Fisherola nuttalli*), are of special conservation concern (Appendix C).

Reptiles and Amphibians

A variety of reptiles and amphibians are found in and around the Hanford Site. However, Fitzner and Gray (1991) note that distribution and abundance of these species is poorly understood. Nine unique species of reptiles have been identified at Hanford (Fitzner and Gray 1991). The most common reptile species is the side-blotched lizard (*Uta stansburiana*) (Downs *et al.* 1993). The short-horned lizard (*Phrynosoma douglassi*), sagebrush lizard (*Sceloporous graciosus*) (an ESA Species of Concern and Candidate for State listing), striped whipsnake (*Masticophis taeniatus*) (a Washington State Candidate for listing) and desert nightsnake (*Hypsiglena torquata*) are also documented, though infrequently and the painted turtle was once commonly found on the Site (Fitzner and Gray 1991). The Gopher snake (*Pituophis melanoleucus*), yellow-bellied racer snake (*Coluber constrictor*), and western rattlesnake (*Crotalus viridis*) are commonly found on the Site (Burk *et al.* 2007).

Hanford is also home to a small number of native and non-native amphibians. Fitzner and Gray (1991) report that the Great Basin spadefoot (*Scaphiopus intermontana*), and Woodhouse’s toad (*Bufo woodhousei*) are considered to be common in riparian areas. TNC (1999)’s survey reported these species and also the tiger salamander (*Ambystoma tigrinum*) and bullfrog (*Rana catesbeiana*). The western toad (*Bufo boreas*) has also been previously documented at the Site (TNC 1999, Burk *et al.* 2007), is listed as a Species of Concern under the ESA, and is a Candidate for State listing.

Fish

The Hanford Reach supports 45 fish species spanning 12 families, five of which are represented by only one species, and one of which (*Petromyzontidae*) includes two local

species (Burk *et al.* 2007 as based on Gray and Dauble 1977). Fish species with the greatest economic importance include salmon (Chinook (*Oncorhynchus tshawytscha*), sockeye (*Oncorhynchus nerka*), and coho (*Oncorhynchus kisutch*)), and steelhead trout (*Oncorhynchus mykiss*) (DOE 2007a). Both the fall Chinook salmon and steelhead trout spawn in the Hanford Reach (Jamison 1982). Furthermore, “since 1962, the Hanford Reach spawning population has represented about 15 to 20% of the total fall Chinook escapement to the river. The destruction of other main-stem Columbia River spawning grounds by dams has increased the relative importance of the Hanford Reach spawning area” (*ibid.*).

Sport anglers also value the white sturgeon (*Acipenser transmontanus*), native mountain whitefish (*Prosopium williamsoni*), smallmouth bass, crappie (*Pomoxis spp.*), catfish (*Ictalurus punctatus*), walleye (*Sander vitreus*), and perch (*Perca flavescens*) (Jamison 1982). The Pacific lamprey (*Lampetra tridentata*), a Federal Species of Concern, travels through the Hanford Reach and has great cultural value to area tribes (Close 2000).

Of the species documented in the Hanford Reach, six are considered by the Federal and/or State government to be of particular conservation concern (bull trout (*Salvelinus confluentus*), leopard dace (*Rhinichthys flacatus*), mountain sucker (*Catostomus platyrhynchus*), river lamprey (*Lampetra ayresi*), spring-run Chinook (*Oncorhynchus tshawytscha*), steelhead). Additionally, Spring-run Chinook salmon are listed as endangered under the ESA, while bull trout and steelhead are both listed as threatened (Appendix C).

Birds

Surveys conducted between 1994 and 1999 documented 221 species of birds on the Hanford Site, bringing the total of known avian species at Hanford to 258 (TNC 1999). A number of reports including Ennor (1991), Fitzner and Gray (1991), and Landeen *et al.* (1992) provide inventories of birds that have been documented breeding, wintering, or migrating through the Hanford Site. Downs *et al.* (1993) focuses in particular on summarizing information regarding species of particular conservation concern.

Ferruginous hawks, red-tailed hawks (*Buteo jamaicensis*), and Swainson’s hawks (*Buteo swainsoni*) are commonly observed nesting on the Hanford Site, and feed primarily on small to medium-sized mammals (Downs *et al.* 1993). Other raptors commonly found breeding on Site include the Northern harrier (*Circus cyaneus*) and burrowing owl. Other common species at Hanford include sage sparrows (*Artemisiospiza belli*), western meadowlark (*Sturnella neglecta*) (the most abundant bird of the Columbia River plain shrub-steppe), and a wide variety of songbirds including the eastern kingbird (*Tyrannus tyrannus*), horned lark (*Eremophila alpestris*), barn swallow (*Hirundo rustica*), black-billed magpie (*Pica hudsonia*), common raven (*Corvus corax*), American robin (*Turdus migratorius*), European starling (*Sturnus vulgaris*), white-crowned sparrow (*Zonotrichia leucophrys*), dark-eyed junco (*Junco hyemalis*), red-winged blackbird (*Agelaius phoeniceus*), house finch (*Haemorhous mexicanus*), and house sparrow (*Passer domesticus*) (Landeen *et al.* 1992). Game bird species present on the Hanford Site

include the mourning dove (*Zenaida macroura*), California quail (*Callipepla californica*), ring-necked pheasant (*Phasianus colchicus*), Hungarian partridge (*Peridix perdix*), and chukar partridge (*Alectoris chukar*) (Downs *et al.* 1993).

Thirty-nine species of native birds within the Columbia Basin Ecoregion that are considered to be shrub-steppe dependent have been documented at Hanford. In addition, eight species of regional management concern that breed in steppe or shrub-steppe habitats were documented by Saab and Rich (1997) breeding at Hanford, including black-throated sparrow (*Amphispiza bilineata*), sage sparrow, sage thrasher (*Oreoscoptes montanus*), Brewer's blackbird (*Euphagus cyanocephalus*), Brewer's sparrow (*Spizella breweri*), lark sparrow (*Chondestes grammacus*), loggerhead shrike (*Lanius ludovicianus*), and western meadowlark (TNC 1999).

Eighteen species of birds documented on the Hanford Site are of special conservation concern, including the American white pelican (*Pelecanus erythrorhynchos*), the peregrine falcon, and the greater sage grouse (*Centrocercus urophasianus*), which is a Candidate for ESA listing (Appendix C).

Mammals

Approximately forty species of mammals have been identified as certain or potential residents of the Hanford Site (Fitzner and Gray 1991; Appendix C). The Great Basin pocket mouse (*Perognathus parvus*) and deer mouse (*Peromyscus maniculatus*) are the most abundant species on the Site (Downs *et al.* 1993), and are important prey for snakes, coyotes, raptors, badgers, and other species. The Northern pocket gopher (*Thomomys talpoides*) is also considered to be abundant (Fitzner and Gray 1991) though it is not commonly observed by humans (Downs *et al.* 1993).

Three lagomorph species – Nuttall's cottontail (*Sylvilagus nuttallii*), white-tailed jackrabbit (*Lepus townsendii*) and black-tailed jackrabbit (*Lepus californicus*) - are currently found at Hanford.

Four species of ungulates have been reported at Hanford. Elk and mule deer are both observed commonly on Site, while white-tailed deer (*Odocoileus virginianus*) are only occasionally documented. Researchers documented several pronghorn antelope (*Antilocapra americana*) sightings between 1978 and 1981, but the species has since been considered extirpated from the Site.

Four families of carnivores are represented at Hanford. The coyote, the sole representative of the family *Canidae*, is the most abundant large carnivore on-site (Downs *et al.* 1993). Bobcats (*Lynx rufus*) represent the *Felidae* family at Hanford, and are generally associated with rock outcroppings and canyons. Six species of the *Mustelidae* family occur at Hanford, with the badger and striped skunk (*Mephitis mephitis*) being the most abundant, according to Fitzner and Gray (1991).⁵² Minks

⁵² Although Fitzner and Gray (1991) list badgers as being common to the Site, Downs *et al.* (1993) suggest that their population size is unknown.

(*Mustela vison*), short-tailed weasels (*Mustela erminea*), long-tailed weasels (*Mustela frenata*), and river otters have also been documented on Site, though less commonly. Raccoons (*Procyon lotor*) represent the *Procyonidae* family at Hanford (Fitzner and Gray 1991). None of these species has been studied extensively on Site.

Two species of shrew, the vagrant shrew (*Sorex vagrans*) and the Merriam's shrew (*Sorex merriami*), have been documented at Hanford, though both are considered to be uncommon (Fitzner and Gray 1991). The Townsend's ground squirrel was also once common across the Site (Fitzner and Gray 1991).

Bats are well-represented at Hanford. Six species of bats have been observed on the Site, and several, including the pallid bat (*Antrozous pallidus*) and two species of myotis bats (*Myotis spp.*) are frequently associated with buildings in the 100 and 200 Areas.

Of the mammalian species that have been documented at Hanford, five are of special conservation concern, including the black-tailed jackrabbit, white-tailed jackrabbit, Merriam's shrew, Townsend's ground squirrel (*Spermophilus townsendii*), and Washington ground squirrel (*Spermophilus washingtoni*). The Washington ground squirrel is also a Candidate for ESA listing.

AIR

Air resources are defined as: “those naturally occurring constituents of the atmosphere, including those gases essential for human, plant, and animal life” (43 CFR § 11.14(b)). Although injury to air is sometimes assessed in the context of a natural resource damage assessment, the atmosphere is generally considered to be a pathway for the movement and re-suspension of contaminants by which other natural resources may be exposed to hazardous substances. Operations at Hanford are known to have emitted hazardous substances. At this time, the Trustees are focusing on air as a pathway, but may consider formally addressing injury to this resource in the future.

3.4 ECOLOGICAL SERVICES Each of the natural resources described above provides a variety of ecological services (human use services, including tribal connections to Site natural resources, are described in Chapter 4). According to the DOI regulations, services are defined as:

...the physical and biological functions performed by the resource including the human uses of those functions. These services are the result of the physical, chemical, or biological quality of the resource (43 CFR § 11.14 (nn)).

For example, rivers provide habitat for numerous aquatic plant and animal species. Riverbanks and riparian habitats provide protective cover, spawning, and nursery habitat for aquatic and terrestrial biota, aid in nutrient cycling, maintain hydrologic flows, and improve water clarity by promoting sedimentation of particulate matter. Phytoplankton and zooplankton serve as prey for aquatic invertebrates and help to cycle nutrients in aquatic habitats. Salmon also contribute to nutrient cycling—their post-spawning carcasses provide an influx of nutrients to the Columbia River ecosystem. Fish, amphibians, and reptiles help to control insect populations and serve as prey for higher

trophic level organisms, such as birds and mammals. Terrestrial habitat provides nesting and denning habitat for a suite of species, as well as flood control during storm events.

The resources described in this chapter are ecologically interdependent and provide interdependent services (43 CFR § 11.71(b)(4)). For example, Safriel and Adeel (2005) describe the interactions of dry land natural resources and their services, for example:

- “[S]oil formation and soil conversion are key supporting services of dryland ecosystems, the failure of which is one of the major drivers of desertification”;
- Nutrient cycling “supports the services of soil development and primary production through the breakdown of dead plant parts (thus enriching the soil with organic matter) and the regeneration of mineral plant nutrients... Unlike non-drylands, where soil microorganisms are major players in nutrient cycling, macrodecomposers such as termites, darkling beetles (*Tenebrionidae*),⁵³ and other invertebrates (many of which are soil dwellers) that are less water-sensitive become important for nutrient cycling”; and
- “The numerous dryland plant species of different growth forms jointly provide a package of services through their ground cover and structure, which provide the drylands’ most important services of water regulation and soil conservation... [and] In many arid and semiarid areas, this biodiversity of ‘vegetation cover’ and biological soil crusts is linked to a diversity of arthropod species that process most of the living plant biomass, constituting the first link of nutrient cycling.”

These and other services sustain a functioning ecosystem by supporting essential hydrological, geomorphological, and ecological processes.

3.5 PRELIMINARY DETERMINATION OF RECOVERY PERIOD Existing data indicate that natural resource services have been lost due to Site-related contamination. As described in the DOI NRDA regulations, this Plan includes a preliminary estimate of the time needed for affected natural resources to recover (43 CFR § 11.31(a)(2)). This recovery period is “either the longest length of time required to return the services of the injured resource to their baseline condition [i.e., the condition in which they would have been had the release not occurred] or a lesser period of time selected by the authorized official and documented in the Assessment Plan” (43 CFR § 11.14(gg)).

Recovery period estimates must be based on the best available information and, where appropriate, may be based on cost-effective models. More specifically, information may come from one or more of the following sources, as applicable: published studies on the same or similar resources; the experience of managers or resource specialists with the injured resource or with similar discharges elsewhere; and field and laboratory data from the assessment and control areas (43 CFR § 11.73(c)(1)).

⁵³ Darkling beetles are present at the Hanford Site (Rogers *et al.* 1978).

In estimating recovery times, Trustees consider factors such as ecological succession patterns in the area; the growth or reproductive patterns, life cycles, and ecological requirements of biota involved, including their reaction or tolerance to the hazardous substance involved; the bioaccumulation and extent of hazardous substances in the food web; and the chemical, physical, and biological removal rates of the hazardous substance from the media involved, including the nature of any potential degradation or decomposition products (43 CFR § 11.73(c)(2)).

For example, some contaminants released from the Site are expected to have extremely high persistence in Site media. Site activities resulted in the discharge of over 200,000 kg of uranium to the ground in the 200 and 300 Areas (Corbin *et al.* 2005 as cited in Zachara *et al.* 2007). These actions created large groundwater plumes of uranium, and at least one such plume “continues to grow in size” (Hartman *et al.* 2007 as cited in Zachara *et al.* 2007). Uranium does not decay over appreciable timeframes: the U-238 isotope⁵⁴ makes up the large majority by mass of natural uranium and has a half-life of about 4.5 billion years, whereas U-234 and U-235 have half-lives of approximately 240,000 years and 700 million years, respectively (ATSDR 1999). These lengthy half-lives indicate that uranium’s specific activity is relatively low compared to radionuclides with shorter half-lives. However, if not physically removed, it will persist for a very long time; and, uranium is also chemically toxic.

Overall, the soil and groundwater beneath Hanford contain approximately 1.8 million curies of radioactivity as of 2000 (Gephart 2003).⁵⁵ Furthermore, contamination of groundwater from single-shelled tanks has been substantial and is ongoing; sixty-seven tanks have or are suspected to have leaked up to 1 million gallons of waste (DOE 2010).

Radionuclides and other contaminants including hexavalent chromium have been released to the Columbia River, particularly between 1944 and 1971 (Gephart 2003). Groundwater travel time from the 200 Area to the river is uncertain but likely ranges from a few years to several decades. Travel times for contaminants subject to retardation by ion exchange and adsorption could be on the high end of that range, such as uranium, strontium-90, and chlorinated hydrocarbons. Over time, high flow rates in the Columbia River have diluted contaminant concentrations in water and sediment in the Hanford Reach and total discharge of groundwater into the River ranges from 0.08 to 2.8 cubic meters per second (0.001 percent of the average Columbia River flow). However, the variability in discharge rates along the River is not well known (DOE 2011c). Additionally the influx of contaminants from groundwater is ongoing, and as long as that

⁵⁴ An isotope is defined as a nuclide of an element having the same number of protons but a different number of neutrons. Nuclide is a general term applicable to all atomic forms of an element. Nuclides are characterized by the number of protons and neutrons in the nucleus, as well as by the amount of energy contained within the atom (<http://www.epa.gov/radiation/glossary/index.html>).

⁵⁵ This figure does not include contained wastes, such as those in tank farms. In total, it is estimated that about 430 million curies of human-made radioactivity remain on site (as of 2000) (Gephart 2003).

persists, it may adversely affect exposed biota, particularly those with life stages associated with river sediments and those exposed in areas of groundwater upwelling.

The potential for ongoing exposure to river biota is therefore at least as long as the groundwater travel time. Preventing contaminated groundwater from reaching the Columbia River is one of the main cleanup goals (DOE 2010). As part of Hanford's 2015 Vision, DOE, EPA and the Washington Department of Ecology hope to prevent contamination from reaching the River by 2015 by decommissioning, deactivating, decontaminating, and demolishing more than 235 facilities, remediating over 300 waste sites, and sending approximately 4.6 million tons of waste and debris to the Hanford Environmental Restoration Disposal Facility. These types of actions will shorten the time required for resources to recover to their baseline condition.

As mentioned, some Hanford contaminants may persist for thousands of years, including those with long half-lives such as the uranium isotopes, plutonium-239 (half-life of 24,100 years), and technetium-99 (half-life of 211,000 years), and carbon tetrachloride, a persistent contaminant in groundwater at Hanford. Based on an evaluation of the existing literature documenting the limited natural degradation rates of many Site contaminants and their resulting persistence in the environment, the Trustees' preliminary determination of the recovery period is that it will likely be at least hundreds of years before recovery will be achieved.

CHAPTER 4 | NATURAL RESOURCE HUMAN USE SERVICES

4.1 NATURAL RESOURCE SERVICES PROVIDED TO TRIBAL COMMUNITIES

In addition to the suite of ecological services described in Chapter 3, trust natural resources in the study area also provide a wide range of human use services. The release of hazardous contaminants from Hanford Site operations has potentially impacted people's use of natural resources, and the well-being they derive from such uses. Measures of the change in human use of a natural resource can be used to quantify natural resource injury (i.e., quantifying the loss in services provided by natural resources to humans), and can support selection and scaling of specific restoration actions to restore the scale and quality of human uses of natural resources. This section describes natural resource services provided to tribal and non-tribal communities that will be considered by the Trustees in conducting the Hanford injury assessment.

Indigenous peoples inhabited the landscape that became the Hanford Site from time immemorial. In the mid-19th century, various tribes in the region reserved rights to access the Hanford Site for traditional use purposes through Treaties with the United States. These traditional uses include the right to access natural resources at this site. Native Americans were still living in accordance with traditional beliefs and practices at Hanford when the Site was established in 1943, and were among those evicted when the U.S. government took control of the area (CTUIR 2012). After that time, little to no access was granted to indigenous groups for many years. More recently, increased, but still limited, access has been allowed. For example, today Native Americans use resources and conduct religious ceremonies in accessible areas at the Site (Yakama PAS). Note that the Federal government maintains a special trust relationship with Indian tribes pursuant to various treaties, statutes, Executive Orders, judicial decisions and other legal instruments. Inherent in the relationship is an enforceable fiduciary responsibility to the Yakama Nation, the Confederated Tribes of the Umatilla Indian Reservation, and the Nez Perce Tribe, to protect their rights and resources. (R. Jim, Yakama Nation). Indigenous peoples may utilize natural resources to an extent and in ways that are different from the general population (Harper *et al.* 2002, Nadasdy 2003, Turner 2005). In addition, the role that natural resources play in the culture of these indigenous communities may differ from that of the general population. "Culture" in this context encompasses the lived experiences and all of the material and spiritual relationships that indigenous peoples have with all of the elements of the natural world. Drawing on published anthropological research, *culture* in the context of this Plan incorporates *practice*, which consists of the everyday activities of the people on the land. As stated by the Nez Perce,

"The most appropriate way to understand our cultural values is to view our cultural practices conducted today on our landscape. They reflect a complex tradition showing high regard for the land. There isn't a daily activity of a

traditional lifestyle that doesn't have oral traditions telling how the activity is part of the land and plays a role in taking care of the land” (Nez Perce 2010).

The Yakama Nation underscores the importance of the Hanford Site and environs as follows:

“The Yakama subsistence lifestyle, including fishing, hunting, and plant gathering; use of traditional foods, medicines, and materials; sweathouse use, feasts, and other cultural practices, depends upon safe, unrestricted access to clean natural resources in the Hanford Assessment area year round in perpetuity” (Yakama 2010).

In general, natural resources and associated ecosystem services provide cultural services including, but not limited to, provisioning, regulating, cultural, and supporting services to tribal members. Thus, cultural service loss can encompass adverse changes in three broad areas of a tribe’s natural resource-based cultural practices, including, but not limited to: (1) Tribal economies (in terms of food, money, and livelihoods, etc.); (2) Tribal knowledge (languages, values, teachings, etc.); and (3) Tribal spiritual values (ceremonies, sacred histories, places, etc.).

As a result of differences in the nature and extent of services tribal members and their communities derive from the environment – and differences in the way in which changes in these services affect indigenous communities — it may be necessary to describe and quantify service losses for tribal communities separately from service losses to the general public. Given these differences, specific restoration actions may also be required to fully compensate the public for losses in indigenous community services.⁵⁶ Exhibit 4-1 provides a preliminary matrix of natural resources, ecosystem services associated with

“[P]rominent landforms such as Rattlesnake Mountain, Gable Mountain, and Gable Butte, as well as various sites along and including the Columbia River, remain sacred. American Indian traditional cultural places within the Hanford Site include, but are not limited to, a wide variety of places and landscapes: archaeological sites, cemeteries, trails and pathways, campsites and villages, fisheries, hunting grounds, plant gathering areas, holy lands, landmarks, important places in Indian history and culture, places of persistence and resistance, and landscapes of the heart (Bard 1997). Because affected tribal members consider these places sacred, many traditional cultural sites remain unidentified.” (*Hanford Site National Environmental Policy Act (NEPA) Characterization (2007) and CTUIR (2012)*).

these resources, and examples of tribal uses of these resources at Hanford. This list is not

⁵⁶ Any Federal undertaking that has the potential to affect Federally-listed (and/or eligible for listing) cultural resources, including Traditional Cultural Properties (TCP), must be evaluated, as mandated under the National Historic Preservation Act (NHPA) Section 106. Such actions would include restoration decisions associated with NRDA. As such, identification of TCPs within Federal jurisdiction must first occur, as mandated under NHPA Section 110 within the area of potential affect for the Federal undertaking.

intended to be all-inclusive; identification of specific sites or uses is not intended to undervalue other areas and uses that are not listed. In addition, the Trustees continue to work to refine and expand this matrix. Recognizing that this matrix is a simplification of a complex association of tribal values with natural resources, it is intended to illustrate and classify the critical links that exist between natural systems and tribal uses at Hanford. As such, it provides context for understanding the range and complexity of tribal uses of and values for this site and its resources, and for the studies proposed in Chapter 7 to address tribal lost use. This exhibit is organized according to “Natural Resource Categories,” which include resources that are likely to have been injured at Hanford: surface water, groundwater, geological resources, biological resources, and air. For each type of natural resource, there are several “Ecosystem Service Categories,” as defined by the Millennium Ecosystem Assessment and National Academy of Sciences (Millennium Ecosystem Assessment 2005). These categories are: cultural and amenity, provisioning, regulating, and supporting and habitat.⁵⁷ For each category, there are multiple “Associated Tribal Services” that are beneficial and of value to tribal members.⁵⁸ Finally, for each tribal service, examples are listed of “Specific Tribal Uses” at Hanford. The Tribal Narratives (which can be found in the Administrative Record) articulate in more detail the specific tribal uses of resources at Hanford.

As noted in Exhibit 4-1, specific physical areas at the Hanford Site carry particular cultural importance to the Yakama, CTUIR, and Nez Perce. As stated in the Hanford Site National Environmental Policy Act (NEPA) Characterization (Duncan 2007) and reiterated by CTUIR (2012),

“prominent landforms such as Rattlesnake Mountain, Gable Mountain, and Gable Butte, as well as various sites along and including the Columbia River, remain sacred. American Indian traditional cultural places within the Hanford Site include, but are not limited to, a wide variety of places and landscapes: archaeological sites, cemeteries, trails and pathways, campsites and villages, fisheries, hunting grounds, plant gathering areas, holy lands, landmarks, important places in Indian history and culture, places of persistence and resistance, and landscapes of the heart (Bard 1997). Because affected tribal members consider these places sacred, many traditional cultural sites remain unidentified” (Duncan 2007).

Despite the fact that many sites are not identified, as of 1997, over 1,500 cultural resource sites and isolated finds, as well as 531 buildings and structures⁵⁹ have been documented

⁵⁷ Ecosystem services that are market-mediated (i.e., can generally be monetarily valued) include provisioning, regulating, and supporting services; while those that are generally non-market-mediated include cultural/amenity services, such as subsistence, recreation, education, ceremonial, and artistic services (Chan *et al.* 2011).

⁵⁸ Service benefits that are generally market-mediated include employment, material, activity, and aesthetic benefits; while those that are generally non-market-mediated include benefits associated with place/heritage, spiritual, inspiration, knowledge, existence/bequest, option, social capital/cohesion, and identify (Chan *et al.* 2011).

⁵⁹ These figures include a small number of sites from early settlers and the Manhattan Project Era.

on the Hanford Site (Duncan 2007). Such sites include pit house villages, open campsites, spirit quest monuments (rock cairns), hunting camps, game drive complexes, and quarries in nearby mountains and rocky bluffs; hunting/kills sites, and small temporary camps near perennial sources of water (Duncan 2007). Forty-nine cultural resource sites have been listed on the National Register of Historic Places, most of which are associated with Native American sites (Duncan 2007).

EXHIBIT 4-1 HANFORD TRIBAL SERVICES MATRIX

| NATURAL RESOURCE CATEGORIES ¹ | ECOSYSTEM SERVICE CATEGORIES ² | ASSOCIATED TRIBAL SERVICES ³ | EXAMPLES OF SPECIFIC TRIBAL USES ^{3, 4} | | |
|--|--|--|--|---------------------|---|
| Surface water (includes sediment and hyporheic zone) | Cultural & Amenity | Water supply (subsistence, ceremonial, spiritual) | Life-giving source | | |
| | | | Drinking water (feasts) | | |
| | | | Sweat lodge purification | | |
| | | River features (subsistence, ceremonial) | Sweat lodge sites | | |
| | | | Fishing camp sites | | |
| | Provisioning | Water supply | Coyote Rapids (spiritual site) | | |
| | | | Drinking water (daily) | | |
| | | | Bathing, cleaning water | | |
| | | | Regulating | Water purification | Clean water (less disease) |
| | | | | Flood control | Stable shoreline (fishing/gathering area) |
| Supporting & Habitat | Climate regulation | Stable climate (maintaining habitat for species collected) | | | |
| | | Aquatic/riparian habitat for sacred plants/animals | Plant/animal collection for subsistence food, medicine, materials, ceremony | | |
| Groundwater (includes springs and seeps) | Cultural & Amenity | Water supply (subsistence, ceremonial, spiritual) | Salmon and other fish | | |
| | | | Life-giving source | | |
| | | | Drinking water (feasts) | | |
| | Provisioning | Water supply | Sweat lodge water (e.g., Rattlesnake Ridge springs) | | |
| | | | Drinking water (daily) | | |
| | | | Bathing, cleaning water | | |
| | Regulating | Water security | Clean water availability | | |
| | Geological (includes surface soil, vadose zone, dust, and rocks) | Cultural & Amenity | Spiritual sites, sacred grounds, landmarks and landscape features, traditional use areas | Burial Grounds | |
| | | | | Archeological sites | |
| | | | | Mooli Mooli | |
| Gable Butte | | | | | |
| Gable Mountain | | | | | |
| Rattlesnake Mountain | | | | | |
| Columbia River Islands | | | | | |
| White Bluffs | | | | | |
| Other spirit quest areas | | | | | |

| NATURAL RESOURCE CATEGORIES ¹ | ECOSYSTEM SERVICE CATEGORIES ² | ASSOCIATED TRIBAL SERVICES ³ | EXAMPLES OF SPECIFIC TRIBAL USES ^{3, 4} |
|--|--|--|---|
| | | | Sweat Lodges along river |
| | | Sweat lodge rocks | |
| | | Solitude, quiet, dark for meditation and ceremony; spiritual connection to Mother Earth | |
| | | Cultural/religious ceremonies, feasts, traditional uses | |
| | | Traditional ecological knowledge, information, education, observation, language, inspiration, community cohesion, heritage | Historical places, names, songs, stories, calendar |
| | | | Language, linguistic landmarks, mnemonics |
| | | | Cultural recognition / association |
| | | | Heritage, multi-generational ties |
| | | | Treaty rights education |
| | | | Environmental restoration/stewardship education/jobs |
| | Traditional Cultural Properties (TCPs) | | |
| | Scenic vistas, recreational experience, trails | | |
| | Social-economic opportunities | | |
| | Areas for barter, trade, reciprocity | | |
| | Provisioning | Raw materials (subsistence, medicinal, sacred) | Rocks and clay for building material |
| | | | Soil to white-wash buildings |
| | | | Clay for mud baths |
| | | | Ground (dirt floor) for sweat lodge |
| | | | Ground (dirt floor) for ceremonies, dancing |
| | | | Soil for healing wounds |
| Regulating | Erosion control | Stable soils, dust reduction | |
| | Nutrient cycling | Fertile soils (habitat for foods collected) | |
| | Supporting & Habitat | Terrestrial habitat for sacred plants/animals | Plant/animal collection for subsistence food, medicine, materials |
| Key species habitat | | Elk/deer and other wildlife | |

| NATURAL RESOURCE CATEGORIES ¹ | ECOSYSTEM SERVICE CATEGORIES ² | ASSOCIATED TRIBAL SERVICES ³ | EXAMPLES OF SPECIFIC TRIBAL USES ^{3, 4} |
|--|---|--|---|
| Biological (includes aquatic, riparian, and terrestrial wildlife, birds, fish, shellfish, invertebrates, plants, fungus, microbes) | Cultural & Amenity | Traditional ecological knowledge, information, education, observation, language, inspiration, community cohesion, heritage | Wildlife, hunting information and skills Fish, fishing information and skills Plant identification, gathering information Traditional foods and medicines knowledge Nutrition, health education Cultural recognition / association Treaty rights education Environmental restoration & stewardship education and careers Materials for barter, trade, reciprocity Aesthetics, existence, viewing, ecotourism |
| | Provisioning | Gathered foods and medicines (subsistence, healing, sacred) | Hemp, chokecherry, balsamroot as examples Berry A, Berry B, ... Herb A, Herb B, ... Roots A, Root B, Pine tea, sage (medicine) Fir, willow, flowers used in sweat lodges |
| | | Hunted and fished animals (clothing/blankets, subsistence, healing, sacred) | Deer Elk Rabbit Other wildlife salmon Other fish |
| | | Raw materials (sacred, subsistence use, shelter) | Plant parts for fishing poles Salmon drying racks Cedar bark for baskets Tule for mats Plant and animal parts for sweat lodge Wood for burning (fuel, sweat lodge) Wood for buildings |
| | | Ornamental use (spiritual, artistic) | Animal parts (hide) for clothing, shoes Animal parts (bones, teeth, shells) for jewelry Plant/animal parts for hats, pigments/dyes |

| NATURAL RESOURCE CATEGORIES ¹ | ECOSYSTEM SERVICE CATEGORIES ² | ASSOCIATED TRIBAL SERVICES ³ | EXAMPLES OF SPECIFIC TRIBAL USES ^{3, 4} |
|---|---|---|--|
| | Regulating | Biological control | Infestation control |
| | | Waste treatment | Predator/prey population control |
| | Supporting & Habitat | Biodiversity, food web | Nutrient cycling |
| | | | Culturally important species |
| Air | Cultural & Amenity | Information, education, observation, language | Viewshed |
| | Provisioning | Clean air supply | Respiration |
| | Regulating | Climate regulation | Stable air patterns |
| <p>Notes:</p> <ol style="list-style-type: none"> 1. Natural resources potentially injured at Hanford, as listed in DOI regulations, include surface water/sediment, groundwater, geologic resources, biological resources, and air. 2. Ecosystem services are the benefits to ecosystem functions, including provisioning, regulating, supporting, and cultural services; listing of these ecosystem services is not necessary to demonstrate the direct link between injured resources and tribal lost services, but illustrates the interconnectedness of ecosystem health and human services. 3. Sources of information include: Human Use Technical Working Group (TWG) Services Matrix and Publics Matrix; and Tribal Narratives provided by CTUIR, Nez Perce, and Yakama Nation. 4. Specific uses reflect tribal values associated with subsistence, culture, education, preservation, health and well-being, recreation, and business/economic services. 5. Note that some of these services may not change as a result of natural resource injury, but are referenced to provide a broad overview of the services provided by these resources. | | | |

4.2 NON-TRIBAL HUMAN USE SERVICES

There are a variety of non-tribal human uses that may have been impacted by the presence of contaminants from Hanford Site operations. In particular, the Trustees have considered past, current, and potential future impacts to recreation (both water-based and land-based) and social welfare changes due to changes in agriculture in the Hanford region (i.e., changes in producer or consumer surplus associated with agricultural products). The Trustees have also considered the nature, extent, and timing of past, present, and expected future resource use limitations due to institutional controls associated with the presence of hazardous contaminants at the Site.

Based on review of existing information, the Trustees are proposing a study to fully describe the past, current, and future geographic and temporal scope of contaminant-related institutional controls which could impact human use of natural resources at the Site. This study is described in Chapter 7.

At this time the Trustees are not proposing additional study of the effect of site releases on agricultural behaviors or a detailed study of recreational behavior. Below we summarize the information on which these determinations were based.

RECREATION

Assessment Area Recreational Opportunities

Hanford Reach National Monument

Hanford Reach National Monument currently provides the public with access to over 57,000 acres of land (US FWS 2011a). The Monument lands support a variety of recreation ranging from wildlife-dependent recreational activities such as hunting, to water-based recreational activities such as boating. This section provides an overview the most commonly pursued recreational activities, including discussion of where the activities occur, when they occur, and what factors influence recreational demand for them.

- **Fishing:** With the Hanford Reach being the last free flowing stretch of the Columbia River in the United States, it has become a very popular recreational fishing resource among fishermen in the Pacific Northwest. The Reach provides excellent fishing opportunity for anglers who wish to pursue sport fishing for fall Chinook salmon, steelhead, sturgeon, whitefish, and small-mouth bass (US FWS 2008). Most fishing occurs from motorized boats, though there is also some fishing done from non-motorized boats and from the river banks. The peak fishing seasons for some species, and especially for fall Chinook salmon, can feature heavy congestion at boat launches both within and downstream of the Monument (US FWS 2008). In addition to angler effort in the river, there is also a small amount of angler effort that occurs in the WB-10 ponds of the Wahluke Unit, which are part of the Columbia Basin Irrigation Project.
- **Hunting:** The Monument offers visitors the opportunity to hunt a variety of mammalian and avian species in riparian and shrub-steppe habitats during the fall and winter hunting seasons. The species open to hunting on the Monument are deer, elk, goose, duck, coot, dove, snipe and all upland game birds (US FWS

2011b). For the Monument, areas open or potentially open to hunting include all or portions of the Ringold Unit, Saddle Mountain, Wahluke, and Columbia River Corridor (US FWS 2002 & US FWS 2011b). Non-waterfowl hunting includes the Saddle Mountain Unit, the Ringold Unit, part of the Wahluke Unit, and all areas of the Columbia River Corridor Unit that are downstream of the Saddle Mountain National Wildlife Refuge (US FWS 2002 & US FWS 2011b).

- **Boating:** The Hanford Reach stretch of the Columbia River Corridor Unit offers opportunities for recreational participants to pursue both motorized and non-motorized boating. While boating in the Reach is primarily driven by angling demand, an increasing number of visitors are pursuing boating for alternative purposes such as scenery and wildlife observation (US FWS 2008). Visitors can also participate in boating-related recreation activities such as water-skiing, personal watercraft use (i.e., jet skiing), and commercial tours of the river. There are three main boat-launching areas on the Monument and several boat-launching areas downstream, including one near the Ringold Fish Hatchery, that have the potential to provide access to the Monument.⁹¹ With boating being primarily driven by angling demand, peak boating seasons closely mirror the peak fishing seasons, with heavy congestion occurring at boat launches during the summer sturgeon season and the fall Chinook salmon and steelhead season (US FWS 2008).
- **Wildlife Observation and Photography:** The four publicly accessible units of the Monument offer significant opportunities for visitors to view and photograph nature. The Monument offers a diverse range of scenic habitats and provides a home to over 240 bird species and more than 40 mammal species throughout the year (US FWS 2008).
- **Environmental Education and Interpretation:** The Monument does not have any formal educational or interpretive programs at this time; however, US FWS accommodates these activities as well as scientific research on the Monument when practical. Schools, nature appreciation groups, and research groups can access the Monument for field trips or biological research projects, and the Arid Lands Ecology Reserve Unit “provides unique settings for other research-oriented projects including an observatory and an underground gravitational research lab” (US FWS 2002).
- **Horseback Riding, Biking, Swimming, Camping, and Hiking:** All of these activities occur on the Monument, consistent with resource management restrictions. Though camping is technically prohibited on the Monument, an exception is that some camping does occur upstream of the Vernita Bridge, especially during peak fishing seasons (US FWS 2008).

Downstream of Hanford Reach National Monument

Downstream of the Monument, the Columbia River continues to provide recreational opportunities. Several miles downstream of the Monument is the McNary Dam; this dam

⁹¹ The downstream boat launches are discussed in the downstream recreation review below.

creates a reservoir-environment in the Columbia River that is known as “Lake Wallula.” Information available for recreation activities that occur downstream of the Monument along Lake Wallula is provided by the U.S. Army Corps of Engineers’ (USACE) Natural Resource Management System (NRMS). Though the NRMS was discontinued after 1999, the database does provide comprehensive information for visitation data through 1999. This section will review recreation information to the extent that it is available.

A host of recreation sites are available along Lake Wallula, including parks, beaches, boat launches, a visitor information center, and a National Wildlife Refuge. Collectively, these recreation sites allow recreation users to pursue activities similar to those that occur on Hanford Reach National Monument.

- **Fishing:** The downstream portion of the Columbia River between the Monument and the dam provides opportunity for anglers pursuing sport fish and attempting to avoid the fishing season congestion that occurs in parts of the Hanford Reach.
- **Hunting:** McNary National Wildlife Refuge, specifically the Wallula Unit, Peninsula Unit, the Two Rivers Unit, and the Burbank Slough Unit, provides hunting opportunities to recreational users of the Lake Wallula area (USACE 2011b).
- **Boating:** With boating congestion in some stretches of the Hanford Reach during peak fishing seasons, Lake Wallula provides important recreational resources both in terms of available boat launch facilities and additional area open to boating.
- **Wildlife Observation and Photography, Camping, Horseback Riding, Hiking, Biking, and Swimming:** Several Lake Wallula recreation sites offer year-round opportunities to pursue wildlife observation and birding. Recreational users can observe a diverse range of species and habitats at these sites. Several recreational sites in the Lake Wallula area offer seasonal or year-round camping (USACE 2011b). These sites also offer day-use areas with amenities such as picnic benches and recreational opportunities such as hiking or biking, so campers have ample opportunity to participate in a diverse range of recreational activities. Equestrians can use the Lewis and Clark Commemorative Trail and designated trails on the McNary National Wildlife Refuge. The Lake Wallula area offers plentiful opportunities for hiking in a diverse range of environments. Bicycling can be pursued on roads throughout the Lake Wallula area, and two recreation sites particularly single out bicycling as a popular recreational pursuit: Chiawana Park and Hood Park (USACE 2011b). Several Lake Wallula recreation sites offer visitors the opportunity to swim in the Columbia River (USACE 2011b).

Contaminant Effects on Recreation

Impacts of contamination on recreational opportunities can manifest in a variety of ways, ranging from fish consumption advisories, to hunting advisories, to closures of sites and facilities. Under DOI’s NRDA regulations, to the extent that contamination causes changes to available services in terms of recreational quality, public access, and recreation demand, these changes may be compensable (43 CFR § 11.71(e)).

To examine how contamination may be affecting human use recreation at the Monument and downstream of the Monument, this section reviews available contamination information for the region as it pertains to recreational activities, recreational quality, and public access.

- **Cleaned up sites:** For the Hanford Reach National Monument, the Rattlesnake, Saddle Mountain and Wahluke Units had been historically contaminated from activities related to the Hanford Site. These locations have since been cleaned up and are now able to “support unrestricted use” (EPA *et al.* undated).⁹²
- **Saddle Mountain Lakes:** There is evidence that the Saddle Mountain Lakes, in the Saddle Mountain National Wildlife Refuge, suffer from contamination due to the presence of “DDT-related compounds” (EPA *et al.* undated). This water body is part of the Columbia Basin Irrigation Project, and as such, is potentially exposed to non-Hanford contamination sources. Because Saddle Mountain National Wildlife Refuge is managed for research and education-related activities, and is therefore closed to most public access, this contamination does not pose significant loss of recreational resources. However, the US FWS may wish to open Saddle Mountain pond in the future.
- **Columbia River Shoreline:** There are “hot spots” of contamination along the Columbia River shoreline due to activities related to the Hanford Site. Most of the shoreline of the Hanford Site is accessible to visitors up to the mean high water mark, “except in those areas where reactor and reactor-related cleanup is ongoing” (EPA *et al.* undated). Contamination of the shoreline includes contaminated groundwater in locations near the former reactor sites. Water quality sampling has determined that groundwater underlying the stretch of river between river mile 363 and river mile 356 is contaminated above drinking water standards due to contamination from central Hanford (EPA *et al.* undated). This contaminated groundwater can enter the river through seeps and springs, and although it is unlikely that visitors could ingest enough water to be harmful, it is best not to consume water from the Columbia River within the Hanford Reach National Monument (EPA *et al.* undated).⁹³ However, as of 1996, the Columbia River Systems Operation Review found that “no water quality problems affecting recreational suitability are known to exist in the Hanford Reach” of the Columbia River (USACE 2011a).
- **Biota Monitoring Program:** The US Department of Energy (USDOE) “maintains a comprehensive environmental monitoring system” on the Hanford Site and in the Hanford Reach National Monument (EPA *et al.* undated). This monitoring system tests game species, including waterfowl and fish, for evidence of contamination. Results from the monitoring program indicate that “consumption of wildlife and fish harvested from the Monument does not pose a threat to humans” (EPA *et al.*

⁹² This designation by the EPA does not necessarily preclude injury under DOI NRDA regulations.

⁹³ Washington State has designated the Columbia River in this area as Class A (i.e., suitable for raw drinking water); and the USFWS advice relates to the Hanford Site shoreline only.

undated); except for wildlife in the Saddle Mountain Lakes, as noted above, which are potentially exposed to offsite contamination.

- **‘Class A’ Designation:** According to the 2008 Comprehensive Conservation Plan and Environmental Impact Statement, Washington State rates the water quality of the Hanford Reach stretch of the Columbia River as “Class A”. Class A waters are suitable for essentially all uses, including raw drinking water, primary-contact recreation, and wildlife habitat” (US FWS 2008). Note that the “Current Uses and Restrictions at Hanford Reach National Monument” document produced by the US Fish and Wildlife Service, mentioned above, refers only to the shoreline of Hanford Reach, as possibly contaminated above safe drinking water standards.
- **USGS Measurements:** To measure the Hanford Site’s contribution to contamination of the Hanford Reach and Columbia River waters downstream of the Reach, it is important to analyze water quality upstream of the Hanford Site as well as downstream of the Site. In 2002, the USGS measured a limited set of water quality parameters at stations upstream and downstream of the Monument. While this sampling effort was limited and did not test for all Hanford potential contaminants of concern, results indicated that water quality parameters such as total dissolved solids and dissolved oxygen “were well within EPA standards” (US FWS 2008). Further, “there were no statistically significant differences between upstream and downstream samples for these parameters” (US FWS 2008).
- **Ongoing DOE Monitoring:** The US Fish and Wildlife Service coordinates with the USDOE environmental monitoring system and factors the results of this program into determining regulations pertaining to public access of the Monument. At present, “a visitor will not be exposed to elevated levels of Hanford derived contaminants which could become a health issue unless they access specific areas illegally or perform activities that are prohibited on the [Monument]” (EPA *et al.* undated). Thus, DOE believes that, as long as visitors are following US FWS regulations when pursuing recreational activities on the Monument, they will not be exposed to contaminants at levels that pose a human health risk.
- **Closed areas on Hanford Site:** Much of the Hanford Site remains closed to visitors, precluding recreational activities in these areas. An inventory of the nature and geographic scope of institutional controls related to hazardous contaminant releases that could impact past, present, or future human uses of the Site is one of the studies described in Chapter 7.

Conclusion on Lost Recreational Services

The Trustees have identified the potential for loss in recreational opportunities, or the values the public holds for such activities, associated with the release of hazardous contaminants from Hanford Site operations. For example, it is possible that some anglers, hunters and other recreators in the region avoid or otherwise modify their behavior due to concern about contaminants in this area. However, the Trustees are unaware of any studies conducted to-date that have identified such impacts on recreator behavior. As

such, and given the limited scope of these impacts combined with public access to numerous substitute opportunities and sites, the Trustees are not currently proposing further study. As a result, no studies of recreator behavior are proposed in this Plan.

However, as mentioned above and given the potential that some recreators and other members of the public could be restricted from use of natural resources due to site-related institutional controls related to hazardous substance releases, the Trustees are proposing a study to inventory the nature and extent of such controls (see Chapter 7). This information may form a measure of the scale of lost human use of the Hanford Site, or may identify the need for a more focused study of lost human use of the Site.

AGRICULTURE

Agriculture is one of the key industries in the State of Washington, with the food and agriculture industry accounting for 160,000 jobs and contributing 12 percent of the state's economic output (WSDA 2011a). The state's 39,500 farms produced \$8.25 billion in agricultural output in 2010 (USDA 2011). Further, it is estimated that "each dollar of farm gate receipts has a multiplier effect of 2 to 3 times throughout the state's economy," meaning that the 2010 receipts of \$8.25 billion resulted in total economic impacts for the State of Washington ranging from \$16.5 billion to \$24.75 billion (WSDA 2009).⁹⁴ In 2009, for the four Washington counties identified in the Final Comprehensive Conservation Plan and Environmental Impact Statement (US FWS 2008), Franklin County produced \$467 million worth of crops on 891 farms, Grant County produced \$1.19 billion worth of crops on 1,858 farms, Adams County produced \$344 million worth of crops on 272 farms, and Benton County produced \$526 million worth of crops on 1,630 farms (WSDA 2011b).

In terms of agricultural commodity groups, the top five products of Washington's agricultural industry (with 2010 gate receipts in parentheses) are apples (\$1.44 billion), milk (\$950 million), wheat (\$925 million), potatoes (\$654 million), and cattle (\$568 million) (WSDA 2011b). For the Washington counties surrounding the Hanford Site, Franklin County agricultural production focuses on potatoes, apples and hay; Grant County farm production focuses on apples, cattle, and potatoes, Adams County farm production focuses on potatoes, wheat, and apples, and Benton County farm production focuses on potatoes, apples, and grapes (WSDA 2011b).

Additionally, West Lake, now classified as a waste site under CERCLA, was historically a source of good quality water for livestock. Currently, West Lake and its basin is a contaminated and highly saline habitat, most likely because of the evaporation of water from the pond and the accumulation of dissolved solids during Hanford operations (Burk *et al.* 2007).

The Trustees have applied available information to determine if releases of contaminants from Hanford Site operations have impacted the value of farm products or farm land in study region. The Trustees found no evidence that farm products from this region have been reduced in value, or that significant acreages of agricultural lands have been

⁹⁴ Gate receipts are the price of the product as sold by a farm.

rendered inarable due to the presence of contaminants from Hanford. Thus, no studies of injuries to agricultural services are included in this Plan.

CHAPTER 5 | CONFIRMATION OF EXPOSURE AND INJURY ASSESSMENT PROCESS

This chapter provides a brief overview of the exposure of natural resources to contaminants of potential concern (COPCs) at the Hanford Site and the subsequent injury assessment process. As the available information on these subjects is vast, this report does not attempt to comprehensively characterize all relevant information but rather aims to broadly and generally characterize the state of knowledge on these topics, while meeting the requirements of assessment plan content as set forth in 43 CFR § 11.31.

5.1 CONFIRMATION OF EXPOSURE The DOI's NRDA regulations require that at least one of the natural resources identified as potentially injured "has in fact been exposed to the released substances" (43 CFR §11.37(a)). A natural resource has been exposed to hazardous substances if "all or part of [it] is, or has been, in physical contact with... a hazardous substance, or with media containing... a hazardous substance" (43 CFR § 11.14(q)). The regulations also state that "whenever possible, exposure shall be confirmed by using existing data" (43 CFR § 11.37(b)(1)). This Plan confirms that a variety of potentially injured resources have been exposed to multiple contaminants of potential concern, including radionuclides, metals, and organic compounds.

A substantial body of information demonstrates past and ongoing exposure of the Hanford Site's natural resources to contaminants of concern; much of the information has been documented in the Yakama Nation and CTUIR's pre-assessment screens (Ridolfi 2006, CTUIR 2007). The scale of documented releases of contaminants to the air, soil, surface water, and groundwater is, on its face, sufficient evidence of exposure. Furthermore, vast datasets have documented the past and, in some cases, ongoing presence of contaminants in Site media. Examples of data confirming exposure of surface water, sediment, geological, groundwater, and biological resources to Site-related contaminants are described below.

SURFACE WATER

Contaminated liquid wastes were discharged directly into the Columbia River during Hanford operations starting in 1944, when B Reactor operations began (Hall 1991). Uranium from the 300 area was released to the river due to seepage and dike failures. Additionally, reactor effluent water released to the River contained radioactive contaminants such as zinc-65, chromium-51, iodine-131, tritium, cesium-137, and strontium-90 (Hall 1991).

Surface water samples collected from the 100 and 300 areas of the Hanford Reach of the Columbia River in the 1990s and 2000s have exceeded the 0.006 mg/L EPA Drinking

Water Standard (DWS) for antimony and the 0.005 mg/L DWS for cadmium (Industrial Economics and Ridolfi 2012).

SEDIMENT

Two lines of evidence confirm exposure of sediments to Site-related contaminants. First, sediment samples collected along the shoreline of the Columbia River adjacent to Hanford contained concentrations of radioactive contaminants including cobalt, strontium, cesium, europium, and plutonium higher than at upstream (i.e., reference) locations (Cooper and Woodruff 1993, as cited in Gephart 2003b). Second, a suite of contaminants in assessment area sediment frequently exceed concentrations above which adverse effects on biota are likely. For example, average chromium concentrations in the 1990s and 2000s range from approximately 12 mg/kg to over 40 mg/kg in the 100 and 300 areas of the Columbia River and downstream of the Site. These levels exceed sediment quality guidelines, indicating the potential for adverse impacts on benthic invertebrates (MacDonald *et al.* 2000).⁹⁵

GEOLOGICAL (SOIL)

As described above, contaminated liquid and solid wastes were released directly to Hanford Site soils in ditches, trenches, cribs, and storage tanks. Soils beneath Hanford have been estimated to contain 1.8 million curies of radioactivity and 100,000 to 300,000 tons of chemicals (Gephart 2003b).⁹⁶

In the 2011 Site monitoring report, soil samples from locations near facilities and operational areas generally had higher radionuclide concentrations than samples from more distant locations, and were significantly higher than concentrations at off-site locations (DOE 2011d). In addition, hexavalent chromium levels exceed published concentrations indicating adverse effects to earthworms. For example, average hexavalent chromium concentrations in the 100-BC, 100-K, 100-DH, and 200 areas exceed the 0.34 mg/kg, ecological soil screening level protective of soil invertebrates (LANL 2008, as cited in DOE 2011b).⁹⁷

GROUNDWATER

Hazardous substances released to soils have leached into the groundwater at the Hanford Site. Since the early 1950s, groundwater samples have been collected and analyzed from hundreds of groundwater monitoring wells across the Site. Major groundwater contaminants include carbon tetrachloride, hexavalent chromium, cyanide, iodine-129, nitrate, strontium-90, trichloroethene, tritium, and uranium. These plumes have a combined area in excess of 186 km² (DOE 2011c). Remedial activities are in place for

⁹⁵ The cited thresholds are used for illustrative purposes. This injury assessment plan includes a study comparing contaminant concentrations in sediments with literature-based adverse effects thresholds, and threshold selection is part of that effort.

⁹⁶ The full extent of soil and sediment contamination due to transport in air and deposition is unknown. See the potential for long-term injury study in Chapter 7.

⁹⁷ The cited threshold is used for illustrative purposes. This injury assessment plan includes a study comparing contaminant concentrations in soils with literature-based adverse effects thresholds, and threshold selection is part of that effort.

some, but not all locations. For instance, pump-and treat systems, as well as a soil-vapor extraction system, continue to remove contaminants from the groundwater and vadose zone beneath the 200 areas (DOE 2011d). Furthermore, contaminants have not only reached groundwater but have moved laterally with groundwater into the Columbia River (DOE 2011c).

Some examples of exceedances reported in the most recent DOE annual monitoring report include chromium exceedances of the EPA Drinking Water Standard of 100 µg/L in parts of the 200 West, 100-K, and 100-D areas as well as hexavalent chromium exceedances of the Washington State cleanup standard of 48 µg/L and the aquatic water quality criterion of 10 µg/L in almost all of the 100 areas. In the 100-NR-2 operable unit, strontium-90 concentrations exceeded EPA's DWS of 8 pCi/L, manganese concentrations exceeded the 50 µg/L DWS in several wells, and total petroleum hydrocarbon is a contaminant of concern for a CERCLA interim action (DOE 2011c). Additionally, in the 100-FR-3 operable unit, nitrate concentrations have been documented in excess of the 45 mg/L DWS (DOE 2011c).

Additionally, upwellings in the Hanford Reach of the Columbia River introduce groundwater contaminants to the River and to aquatic biota. Although the nature and extent of groundwater upwelling contamination is unknown, upwelling samples have documented hexavalent chromium, strontium-90, tritium, and uranium concentrations in excess of drinking water standards (Hulstrom and Tiller 2010).

BIOLOGICAL

A number of studies have documented the exposure of biota to Site-related contamination. Efforts to-date have focused mainly on vegetation, fish, and mammals. For example, the 2010 Hanford Site Environmental Report reported elevated levels of radionuclides in vegetation samples collected near Hanford facilities compared to off-site locations (DOE 2011d). The 2002 EPA fish contaminant survey documented contamination due to metals, pesticides, PCB congeners, dioxins, and furans in white sturgeon from the Hanford Reach (EPA 2002a).⁹⁸ In addition, small mammals have been analyzed for contamination, including radiological contamination, and preliminary Trustee analysis suggests that levels of mercury, PCBs, and uranium in mice collected near operational areas exceeded adverse effect concentrations from the literature.⁹⁹ Additionally, strontium-90 was detected in rabbits, deer, and elk (DOE 2011d, Price 1988).

⁹⁸ Note that, some of the contaminants studied in the EPA survey may not be entirely attributable to Hanford operations.

⁹⁹ The cited exceedances are noted for illustrative purposes. This injury assessment plan includes a study comparing contaminant concentrations in biotic tissues with literature-based adverse effects thresholds, and threshold selection is part of that effort.

5.2 INJURY DETERMINATION

As described above, natural resources within the assessment area have been and continue to be exposed to both historical pollution and the continuing release of contaminants to Site resources. This chapter demonstrates injury to trust resources resulting from this contamination, which motivates and provides additional weight of evidence for studies proposed in Chapter 7.

Determination of injury to natural resources, an essential part of the injury assessment process, consists of documentation that there is: (1) a viable pathway for the released hazardous substance from the point of release to a point at which natural resources are exposed to the released substance, and (2) that injury of site-related resources (i.e., surface water, sediment, soil, groundwater, biota) has occurred as defined in 43 CFR § 11.62.

PATHWAY

The DOI NRDA regulations define ‘pathway’ to be “the route or medium through which oil or a hazardous substance is or was transported from the source of the discharge or release to the injured resource” (43 CFR § 11.14(bb)), and indicate that pathway may be determined “by either demonstrating the presence of the oil or hazardous substance in sufficient concentrations in the pathway resource or by using a model that demonstrates that the conditions existed in the route and in the oil or hazardous substance such that the route served as the pathway” (43 CFR § 11.63(a)(2)). The regulations identify several methods for establishing pathway if existing information is not adequate for this purpose.

During the pathway determination phase, the Trustees will document how Site-related contaminants move through the environment. Specifically, the movement of contaminants from the source (i.e., the Site) to the environment will be determined. The pathway determination phase will also establish how contaminants move into the food web and then from one species to another.

In general, natural resources can be exposed to hazardous substances through both abiotic and biotic pathways. Abiotic components of pathways include processes such as volatilization, evaporation, aeolian transport, infiltration, runoff, flooding, and irrigation. Biotic pathways include dermal contact; respiration and inhalation; ingestion of food, water, or soils; uptake from soils by plants; decomposition of plants and animals; and the distribution of hazardous substances by the physical movement of biota (biotic vectors). For example, contaminated soils may expose groundwater through infiltration mechanisms, or the air through aeolian transport. Contaminated groundwater may enter the hyporheic zone and then expose surface water and sediments, which may in turn lead to the exposure of aquatic biota.

Response actions also may inadvertently facilitate contaminant transport. For example, pump and treat and re-injection systems that are designed to treat a specific contaminant may inadvertently transport and disperse other contaminants (e.g., tritium; Peterson *et al.* 2002).

The Trustees have developed a preliminary conceptual site model (Stratus 2009) that identifies and describes numerous pathways through which contaminants released on-site could injure natural resources and adversely impact the ecological and human use

services they provide. In addition, data showing Site-related contaminants in surface water, sediment, groundwater, soils, plants, invertebrates, fish, birds, and mammals within the assessment area (as described above) support this assertion.

Conducting assessment studies specifically to address pathway issues is most important in circumstances where the source of contamination observed in the study area is not obvious (e.g., releases from some combination of multiple entities, general anthropogenic activities, and/or natural sources). At Hanford, site activities clearly are the sole or predominant source of much of the observed contamination. That said, for certain hazardous substances, natural and/or off-site anthropogenic sources likely contribute to some extent. Several studies in this assessment plan are designed to help Trustees better understand the extent of these contributions. In particular, this plan includes several studies in which contaminant concentrations in various media are compared to thresholds. These studies include an analysis of baseline concentrations (i.e., the concentrations that would be present but for Hanford Site-related releases).

In addition, this assessment plan includes a study to assess the spatial distribution of patterns in surficial soils, which in combination with information on significant aerial releases and historic wind patterns, will help Trustees better identify areas more/less likely to have been exposed to potentially injurious contaminant concentrations. This assessment plan also includes an exposure study for wild terrestrial birds. Many studies include measurements of contaminants in site media and/or in the tissues of site organisms. All these studies will contribute to the Trustees' understanding of the pathways through which natural resources may have been exposed to Hanford Site contaminants. As assessment activities progress, the Trustees may or may not decide to pursue additional studies to support the establishment of pathways between Hanford Site releases and natural resources.

DETERMINING INJURY

Injuries to trust resources, as defined in the DOI NRDA regulations at 43 CFR §11.62, generally fall into three categories.

- The first category establishes injury based on the exceedance of regulatory standards or criteria. This may include exceedance of established standards (e.g., water quality standards) or the existence of advisories limiting/banning the consumption of contaminated biota (e.g., fish consumption advisories).
- The second category establishes injury based on adverse changes in an organism's viability. Changes in viability that constitute injuries include: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including impaired reproduction), and physical deformations.
- The third category establishes injury to a natural resource when concentrations of a hazardous substance are sufficiently high in that natural resource to cause injury to another natural resource.

Chapter 6 provides additional details on the regulatory definitions of injury for each trust resource.

The Trustees have identified a set of natural resources found within the assessment area on which to focus this assessment. Resources were chosen based on their relative and/or cumulative importance to the healthy functioning of the ecosystem, abundance within the assessment area, and the feasibility of conducting COPCs exposure and/or toxicity studies on each resource. As described in the following sections, at this time the Trustees are evaluating potential injury to surface water, sediments, soils, various biota associated with these resources, and groundwater. This list of resources may be modified as assessment activities proceed and additional information becomes available.

For each selected resource, the Trustees will gather existing information about past, present, and predicted future concentrations of COPCs and compare these data to known criteria, standards, guidance values, or other thresholds that, if exceeded, indicate that injury to the resource exists or is likely to exist. In addition, the Trustees will review existing site-specific community structure and toxicity studies for biota. The Trustees will review these studies in the context of the natural resource damage assessment and use the findings to determine whether injury has occurred or is likely to occur in any portion of the study area.

As part of this effort, the Trustees will assess whether sufficient data exist to adequately characterize injury to Trust resources. “Adequacy” in this context means the data provide a sound and sufficient basis to characterize injuries for purposes of establishing the scale and scope of required restoration. As described in the preceding section, studies have determined that Site-related contaminants are transported via surface water, groundwater, and air flow, and bioaccumulative contaminants are transported through a complex food web. Although considerable past effort has been undertaken to describe contaminant exposure across many resources, for some resources the available data are limited. For example, the spatial distribution of soil data in terrestrial habitats on-site may be insufficient to characterize the extent of contamination. As such, the Trustees have identified additional studies, described in Chapter 7, which are intended to fill in data gaps associated with characterizing the extent of contamination.

5.3 INJURY QUANTIFICATION

Once it has been determined that natural resources have been injured, quantification of that injury is undertaken to establish a basis for scaling restoration and determining damages. Injuries to natural resources can be quantified in terms of the actual measured loss of the specific resource(s), and/or the services that the injured resource would have provided had the release not occurred.¹⁰⁰ Ecological services include the services provided by natural resources, such as “food and fresh water... the climate and the air we breathe” (Millennium Ecosystem Assessment 2005).

¹⁰⁰ The Trustees may choose to quantify injury in units of resource, where the services provided by those resources are understood to be related to the scale of the available resource or where it is not feasible or cost-effective to quantify the human use or ecological service loss.

As described in the DOI regulations:

“In the quantification phase, the extent of the injury shall be measured, the baseline condition of the injured resource shall be estimated, the baseline services shall be identified, the recoverability of the injured resource shall be determined, and the reduction in services that resulted from the discharge or release shall be estimated.” (43 CFR § 11.70(c))

Injury quantification will consider the effect of remedial activities in the assessment area on the return of injured natural resources to their baseline condition.

BASELINE

In order to quantify injuries, the baseline conditions of the affected resources and associated services must be established. Baseline is “the condition or conditions that would have existed at the assessment area had the discharge of oil or release of the hazardous substance under investigation not occurred” (43 CFR § 11.14(e)). As required by the DOI regulations, the Trustees anticipate determining “the physical, chemical, and biological baseline conditions and the associated baseline services for injured resources at the assessment area” (emphasis added) and quantifying injury based on a reduction in these services (43 CFR § 11.72(a)).

Baseline conditions may be established based on the review of historical, pre-release data and information, or on reference locations that exhibit similar physical, chemical and biological conditions as the assessment area, excluding contamination (43 CFR § 11.72). The fact that releases of hazardous substances have occurred within the assessment area prior to the establishment of regular or standardized approaches for the collection of physical, chemical and biological data may necessitate the use of suitable reference locations in lieu of historical data for purposes of baseline determination.

In general, the characterization of baseline conditions will take place within the context of specific injury studies. For instance, studies that compare contaminant measurements in site media to thresholds will include an evaluation of what baseline concentrations would likely have been but for the Hanford Site releases. In particular, “upgradient” locations may be used for characterization of surface water and groundwater baseline conditions, and background soil concentrations could be used to establish baseline for geological resources. Field studies of biota, and studies using site media, will consider baseline through examination of suitable reference areas, and experimental laboratory studies (e.g., spiked exposure toxicity studies) will consider baseline through the use of control experiments.

“Baseline” also incorporates the ecosystem and human use services that would be provided by natural resources but-for injury (holding all other factors constant). For example, an aquifer that was not potable prior to contamination would have a different baseline condition than one that was potable. In this example, the injury assessment would consider the baseline level of human use services that would have been provided but for the release of hazardous substances.

ECOLOGICAL INJURY QUANTIFICATION

As described in Chapter 3, each trust resource provides a variety of ecological services, ranging from protective cover to nutrient cycling, food web sustainability to flood control. The Trustees currently propose to quantify injury to natural resources within assessment area aquatic and terrestrial habitats on a habitat basis, considering changes in injury over time. For example, the Trustees may apply habitat equivalency analysis (HEA), a commonly applied, well-accepted method that involves quantification of losses over space and time that is specifically identified in the DOI NRDA regulations (43 CFR § 11.83(c)(2)). Quantification of ecological losses will focus on endpoints that are considered the most biologically relevant (i.e., endpoints that most directly impact a resource's ability to function and provide services) such as growth, reproduction, and survival of biota, but may also include evaluation of other measures of health and organism viability.

The Trustees note that injuries to certain resources may be quantified individually (e.g., resources which are unique or of special concern, such as locally rare, threatened or endangered species, or require that restoration be scaled based on individual quantification of injuries, etc.). The Trustees are in the process of identifying whether any such resources have been impacted by exposure to Site-related contamination.

GROUNDWATER INJURY QUANTIFICATION

The DOI regulations provide guidance on the steps to follow in quantifying groundwater injury (43 CFR § 11.71). In addition to determining a volume of injured groundwater, the Trustees will also quantify, "...the effect of the discharge or release in terms of the reduction from the baseline condition in the quantity and quality of services ... provided by the injured resource...." (43 CFR § 11.70). In terms of services provided, all waters and uses must meet the standard for "committed use" and all uses must be "...reasonably probable, not just in the realm of possibility. Purely speculative uses of injured resources are precluded from consideration in estimating damages" (43 CFR § 11.84).

In the context of damage assessment, a range of hydrological metrics have been used to quantify injury, representing proxy measures for the services provided by groundwater. For example, groundwater can be quantified either as a "stock" or a "flow." These metrics include the three dimensional volume of the plume(s) combined with measures of porosity, the volume previously extracted, and calculated or modeled sustainable or "safe" yield (the amount of water that can be withdrawn from a given aquifer without depleting it over time). Because groundwater provides a range of services, the particular metric chosen to quantify services will relate to the types of services the Hanford Trustees understand to be adversely affected.

In some cases quantification of the volume of injured groundwater over time may not be necessary to establish damages and scale restoration. For example, a plume may effectively preclude groundwater use in a community. In such a case the loss in services is insensitive to the particular plume dimensions. Specifically, the DOI regulations at 11.71 state that "The effects of a discharge or release on a resource may be quantified by directly measuring changes in services provided by the resource, instead of quantifying

changes in the resource itself.” This approach is stated as being valid when three conditions hold:

- “(1) The change in the services from baseline can be demonstrated to have resulted from the injury to the natural resource;
- (2) The extent of change in the services resulting from the injury can be measured without also calculating the extent of change in the resource;
- and,
- (3) The services to be measured are anticipated to provide a better indication of damages caused by the injury than would direct quantification of the injury itself.” (43 CFR § 11.71(f))

Once the volume of injured groundwater has been quantified (if necessary), the next step in the injury quantification process is to consider what, if any, services have been impacted by the release of hazardous substances. This step is necessary since the goal is to restore, replace or acquire the equivalent of injured natural resources and the services they provide to their baseline condition. The scope of services that may have been lost as a result of groundwater injury will depend on a variety of factors, including baseline quality, hydrological limitations that could impact the usability of the resource, policy and regulatory limitations unrelated to the release of a hazardous contaminant, access limitations, regional water supply and demand balances, etc. For example, a plume may exist in an area that requires residences to hook-up to a public water supply (i.e., precludes private wells) for reasons unrelated to the presence of a plume. In some cases the information required to develop an inventory of lost services will exist. In others, it may be necessary to conduct primary research to determine the extent to which service flows have been lost as a result of injury to groundwater resources.

Injuries Resulting From Exposure of Other Natural Resources to Contaminated Groundwater

Under the DOI regulations, injury to groundwater can be demonstrated based on concentrations of hazardous substances sufficient to cause injury to surface water, air, geological or biological resources. While this definition of injury may be applicable in a range of cases, some trustees choose to evaluate groundwater as a pathway, and quantify the injuries resulting from groundwater contamination as losses to the exposed resources (rather than the groundwater itself). For example, where groundwater transports contaminants to surface water, exposing fish to those contaminants, injury could be assessed as service losses incurred by fish. The Trustees are still evaluating which methodology is most appropriate for this Site.

Addressing Contamination of the Vadose Zone and Geological Resources

As described in Chapter 3, the movement of moisture in the Hanford vadose zone is the primary driving force for the migration of Site-related contaminants to groundwater (Burk *et al.* 2007). While moving through the vadose zone, contaminants can become “stuck” (i.e., adsorbed and/or absorbed by the soil matrix), then releasing to groundwater over an extended period of time (Freeman *et al.* 2001).

The DOI regulations list geologic resources (i.e., soil) as a separate category of natural resources, and suggest quantification of injury to such resources in terms of “[t]he volume of geologic resources that may act as a source of toxic leachate.” (43 CFR § 11.71 (k)(3)) Thus, while trustees can choose to assess injuries and damages to the vadose zone, in practice vadose zone contamination has been treated by trustees as a pathway and reservoir of contaminants. The Trustees are in the process of reviewing existing information to determine which methodology is most appropriate for this Site.

Existing Data and/or Primary Research

Whether existing data will be sufficient to complete a groundwater damage assessment for the Hanford Site is yet to be determined. For example, depending on the approach followed and information obtained regarding service losses, it may turn out that precise determination of plume dimensions or other characteristics will not be required. Currently, the Trustees are working with USGS to review the DOE Hanford plume maps to determine if the maps are sufficiently accurate for assessment purposes.

LOST HUMAN USE SERVICES QUANTIFICATION

As described in Chapter 4, a variety of human uses are thought by the Trustees to have been affected by the presence of contaminants released from Hanford operations. At this time the Trustees are focusing on human use losses to tribal communities; due to the nature of public access and resource availability at Hanford, non-tribal human use losses are expected to be relatively modest, and are therefore not included in this Plan.

“Tribal lost services” refer to a loss in natural resource services of importance to a tribal Trustee entity or members, for which separate natural resource restoration actions are likely to be needed. As stated in Chapter 4, as a result of differences in the nature and extent of services tribal members and their communities derive from the environment -- and differences in the way in which changes in these services affect indigenous communities -- it may be necessary to describe and quantify service losses for tribal communities separately from service losses to the general public. That is, specific restoration actions may be required to fully compensate the public for losses in indigenous community services.

The techniques available to assess changes in tribal member uses of the environment in the context of natural resource damage assessment are less well-developed (and have been applied less frequently) than the techniques used for other categories of natural resource services. As a result, damage assessments involving tribal lost use of natural resources have generally relied on similar methods as applied to other service categories (modified and supplemented to reflect unique circumstances of tribal member use), or on methods applied to assess other impacts on tribal cultures (e.g., land claims, cultural impact assessment, etc.).

Examples of such methods, which have been applied to measure service losses to indigenous communities in the context of natural resource damage assessment include but are not limited to:

- **Assessment of changes in cultural services.** This includes assessment and analysis of changes in levels of traditional knowledge, cultural practices, and relationships resulting from shifts in the use of natural resources caused by the presence of hazardous contaminants. Such an analysis is generally based on applied anthropological and ethnographic approaches.
- **Direct assessment of loss of resource use.** This can involve application of revealed preference techniques, user surveys, existing data, etc. For example, assessment of the number of individuals who previously utilized a site, the nature and frequency of that use, substitution or alternative behaviors, and the expected recovery period for the activity.
- **Habitat and resource equivalency.** This involves the use of resource-based measures to quantify the level of service loss under the assumption that ecological service losses are a proxy measure of cultural service losses.
- **Stated preference.** This involves the use of surveys to elicit tribal attitudes and preferences towards an injured resource.

These approaches may be used in combination to assess changes in services resulting from the release of hazardous contaminants to the environment. Each of these approaches, all of which are available to the Hanford Trustees, is discussed in greater detail below.

Assessment of Changes in Cultural Services

One approach for conducting cultural service loss assessment is to inventory and evaluate the existing documentary record related to tribal uses of and services provided by natural resources. This would include consideration of all of the relevant information held by the participating tribal communities that can be located and accessed from other archives. These sources would include scientific reports and academic studies on historic tribal use and traditional cultural context; tribal environmental philosophy and ethnographic descriptions of land and river-based practices; newspaper and media reports on environmental and health issues affecting the communities; studies on the health and social status of the communities; transcripts of oral narratives, etc.

The goal of this type of assessment is to evaluate and organize the existing information so that it can be analyzed in ways that are supported by, and consistent with, the criteria and ethics of standard social science research practice, the conventions of the best strategies of community-based participatory research, and the most advanced ethnographic approaches. The ultimate objective is to gain as complete an understanding as possible (using documentary sources) of the community and its interactions with the natural environment and how these behaviors have changed over time and in response to the presence of hazardous contaminants.¹⁰¹ In this context, primary documents would be

¹⁰¹ Cultural changes can impact a community in terms of time; social cohesion; the intergenerational transfer of knowledge and identity and of the speaking/use of indigenous languages; their economic self-sufficiency; and even the maintenance of the population on the territory. For example, in a recent assessment a tribal trustee developed seven cultural indicators affected by changes in ecosystem services over time. These indicators relate to water, fishing, and the use of the river; horticulture, farming, and basket-making; medicine plants and healing; hunting and trapping; well-being of children, youth

given priority as they provide more validity than secondary sources as meaningful indicators of change and service flow interruption. Ultimately, all of the materials in the available record could be assessed for their relative contribution to the objectives of the work: understanding the nature and scope of interruptions to ecosystem service flows within the affected communities due to the presence of hazardous contaminants. The goal is to produce an assessment record that meets the needs of the natural resource damage assessment process and is sound and valid from a social scientific perspective, but is also consistent with the communities' values and traditions to assure that the results are accepted.

Although this approach draws heavily on the existing evidentiary base, it also involves identification and consideration of data gaps. Where appropriate and required, primary research efforts such as oral history research, can be applied to focus on gathering information directly from people who had used and who continue to use the natural resources and to ask them directly how their knowledge of environmental contamination affected their cultural practice.

The principal strengths of the applied indigenous community research methodology includes utilization of existing information to the fullest extent possible; applying approaches to organization and review of available information that are well-accepted; recognizing the complex relationship between indigenous communities and natural resources; explicitly considering baseline factors; and enhancing the probability of community acceptance of the results. The principal weaknesses involve the time and cost to implement the work, the need for information that may be considered confidential or proprietary, and the challenge of quantifying results such that they can be used to support restoration scaling using evidence that is typically qualitative in nature.

Direct Assessment of Loss of Resource Use

Some impacts on tribal uses of natural resources may be relatively limited in geographic scope and/or temporal scope. Others may be of a magnitude that may not warrant a substantial research effort, or may be very well-defined (e.g., the loss of access to a culturally significant area for a limited period of time). In these cases direct assessment of lost use can provide a basis for assessing service losses.

The strengths of this approach are its simplicity: the direct measure of changes in use to establish service losses, the ability to control for baseline factors in the assessment, and the fact that the information required to conduct such an assessment is generally available with limited additional effort. The principal disadvantage is the failure of the approach to see changes recognizing the complex relationship between indigenous communities and natural resources.

and family; food security and sustainable livelihoods; and transmission of community knowledge to future generations. For each of the indicators, measures of ecosystem impairment were causally linked (where relevant) to cultural injury or interruption of resource services.

Habitat and Resource Equivalency

Resource equivalency methods may be used to define the level of service losses that have resulted from the release of hazardous contaminants, serving as proxy measures for cultural service losses. In such cases a biological measure of resource injury (such as the presence of phytotoxicity) is assumed to provide a better indication of lost services than direct measures of changes in a tribal member's behavior.

The benefit of a habitat or resource-based approach to scaling cultural losses is that it is relatively easy to conduct, can be explicitly designed to address baseline issues, and avoids potential confidentiality issues. The principal weaknesses is that the service loss measures developed are not a direct measures of the change in services but an estimate based on the contaminant concentration levels, and the method may fail to address the complex relationship between indigenous communities and natural resources.

Stated Preference

Stated preference approaches involve the application of public opinion surveys to elicit information from individuals regarding their use of a resource, and/or attitudes and preferences towards an injured resource or restoration strategy. For example, the Trustees may use a survey to understand the frequency with which tribal members fish or hunt, the species they target, consumption rates, etc. Such surveys might be applied as a direct approach to service loss quantification, or might be combined with the approaches discussed above.

In a few cases stated preference methods have been applied to directly assign economic values to foregone cultural use (Duffield 1999). That is, these studies provide economic measures of the value of lost services, without necessarily defining the nature and extent of the loss of use or cultural harm.

The strength of the stated preference methods is the ability to pose to a respondent any hypothetical alternative scenario (i.e., the method is not limited to observing behaviors under actual conditions). While more flexible than revealed preference approaches, stated preference surveys can be costly and time consuming to administer, and may not be consistent with tribal policies or values. As a result, researchers often look to apply revealed preference methods to assess changes in human use of natural resources, since such methods are generally less controversial and pose fewer challenges. Revealed preference studies, however, typically address a narrower set of values than stated preference.

Combination Approaches

As previously noted, the approaches outlined above may be conducted independently, or combined in order to assess tribal lost use services.

As described in Chapter 7, within this Plan the Trustees will consider a study that relies on existing information to define the type and scale of tribal lost use, and based on that study determine if additional research is needed to support injury quantification.

REMEDICATION-RELATED IMPACTS

As described in Appendix A, extensive remediation has taken place on the Hanford Site since the early 1990s when cleanup became the Hanford mission. These remedial activities include but are not limited to the removal of contaminated soils which involves disposal of wastes, backfilling, and revegetation, groundwater pump and treat systems, demolition of inactive facilities, groundwater monitoring, and the transfer and remediation of liquid tank wastes. Hanford remediation has focused on cleaning up the solid and liquid wastes, decontaminating and demolishing facilities, and preventing groundwater contamination from reaching the Columbia River.

Adverse impacts to natural resources as a result of remediation-related activities are compensable under the DOI regulations. For instance, on the Hanford Site, remediation equipment staging areas and waste disposal areas have resulted in the loss of habitat and ecological services. The use of trucks and the creation of roads to provide access to demolition and de-contamination sites as well as the destruction of plants and soil resources when contamination is removed have resulted in the temporary loss of ecological services.

Chapter 7 provides a list of proposed studies that may be called for to complete the Injury Assessment. This set of studies includes an assessment of the nature and extent of injury resulting from remediation-related activities. The analysis will be conducted based on an assessment of the extent of lost habitat services, described over time (e.g., number of acres of habitat services lost, for some period of time).

CHAPTER 6 | DEFINITION OF INJURY

As described in Chapter 5, one essential component of injury assessment is the determination of injury. Because the Trustees are conducting this natural resource damage assessment effort in accordance with the DOI regulations at 43 CFR Part 11, they plan to “determine that an injury has occurred based upon the definitions provided in this section for surface water, groundwater, air, geological, and biological resources” (43 CFR § 11.62(a)). These definitions are identified below.

6.1 SURFACE WATER

Surface waters include both waterways and waterbodies as well as their associated bed and bank sediments. Injury to surface water “has resulted from the discharge of oil or release of a hazardous substance if one or more of the following changes in the physical or chemical quality of the resource is measured:

- (i) Concentrations and duration of substances in excess of drinking water standards as established by sections 1411–1416 of SDWA, or by other Federal or state laws or regulations that establish such standards for drinking water, in surface water that was potable before the discharge or release;
- (ii) Concentrations and duration of substances in excess of water quality criteria established by section 1401(1)(D) of SDWA, or by other Federal or state laws or regulations that establish such criteria for public water supplies, in surface water that before the discharge or release met the criteria and is a committed use, as the phrase is used in this part, as a public water supply;
- (iii) Concentrations and duration of substances in excess of applicable water quality criteria established by section 304(a)(1) of the CWA, or by other Federal or state laws or regulations that establish such criteria, in surface water that before the discharge or release met the criteria and is a committed use, as that phrase is used in this part, as a habitat for aquatic life, water supply, or recreation. The most stringent criterion shall apply when surface water is used for more than one of these purposes;
- (iv) Concentrations of substances on bed, bank, or shoreline sediments sufficient to cause the sediment to exhibit characteristics identified under or listed pursuant to section 3001 of the Solid Waste Disposal Act, 42 U.S.C. 6921; or
- (v) Concentrations and duration of substances sufficient to have caused injury as defined in paragraphs (c), (d), (e), or (f) of this section to ground water, air, geologic, or biological resources, when exposed to surface water, suspended sediments, or bed, bank, or shoreline sediments” (43 CFR § 11.62(b)(1)).

Under DOI's NRDA regulations, the bed, bank, and shoreline sediments, including suspended sediments, are also considered to be part of the surface water resource. The Trustees intend to evaluate the concentrations of chemicals of potential concern in sediments to assess the degree to which these substances may be causing adverse effects to exposed aquatic species.

The DOI NRDA regulations define injury to surface water sediments in several ways. In general, these sediments are determined to be injured when:

- a) Concentrations of substances on bed, bank or shoreline sediments are sufficient to cause the sediment to exhibit characteristics identified under or listed pursuant to section 3001 of the Solid Waste Disposal Act, 42 U.S.C. 6921 (43 CFR § 11.62(b)(1)(iv)); or
- b) Other natural resources (for example, biological resources) become injured as a consequence of exposure to the sediments (43 CFR § 11.62(b)(1)(v)).

6.2 **GROUNDWATER**

Injury to groundwater “has resulted from the discharge of oil or release of a hazardous substance if one or more of the following changes in the physical or chemical quality of the resource is measured:

- (i) Concentrations of substances in excess of drinking water standards, established by sections 1411–1416 of the SDWA, or by other Federal or state laws or regulations that establish such standards for drinking water, in ground water that was potable before the discharge or release;
- (ii) Concentrations of substances in excess of water quality criteria, established by section 1401(1)(d) of the SDWA, or by other Federal or state laws or regulations that establish such criteria for public water supplies, in ground water that before the discharge or release met the criteria and is a committed use, as the phrase is used in this part, as a public water supply;
- (iii) Concentrations of substances in excess of applicable water quality criteria, established by section 304(a)(1) of the CWA, or by other Federal or state laws or regulations that establish such criteria for domestic water supplies, in ground water that before the discharge or release met the criteria and is a committed use as that phrase is used in this part, as a domestic water supply; or
- (iv) Concentrations of substances sufficient to have caused injury as defined in paragraphs (b), (d), (e), or (f) of this section to surface water, air, geologic, or biological resources, when exposed to ground water” (43 CFR § 11.62(c)(1)).

- 6.3 GEOLOGICAL** Soils are geologic resources. Injury to these resources occurs “if one or more of the following changes in the physical or chemical quality of the resource is measured:
- (i) Concentrations of substances sufficient for the materials in the geologic resource to exhibit characteristics identified under or listed pursuant to section 3001 of the Solid Waste Disposal Act, 42 U.S.C. 6921;
 - (ii) Concentrations of substances sufficient to raise the negative logarithm of the hydrogen ion concentration of the soil (pH) to above 8.5 (above 7.5 in humid areas) or to reduce it below 4.0;
 - (iii) Concentrations of substances sufficient to yield a salt saturation value greater than 2 millimhos per centimeter in the soil or a sodium adsorption ratio of more than 0.176;
 - (iv) Concentrations of substances sufficient to decrease the water holding capacity such that plant, microbial, or invertebrate populations are affected;
 - (v) Concentrations of substances sufficient to impede soil microbial respiration to an extent that plant and microbial growth have been inhibited;
 - (vi) Concentrations in the soil of substances sufficient to inhibit carbon mineralization resulting from a reduction in soil microbial populations;
 - (vii) Concentrations of substances sufficient to restrict the ability to access, develop, or use mineral resources within or beneath the geologic resource exposed to the oil or hazardous substance;
 - (viii) Concentrations of substances sufficient to have caused injury to ground water, as defined in paragraph (c) of this section, from physical or chemical changes in gases or water from the unsaturated zone;
 - (ix) Concentrations in the soil of substances sufficient to cause a toxic response to soil invertebrates;
 - (x) Concentrations in the soil of substances sufficient to cause a phytotoxic response such as retardation of plant growth; or
 - (xi) Concentrations of substances sufficient to have caused injury as defined in paragraphs (b), (c), (d), or (f), of this section to surface water, ground water, air, or biological resources when exposed to the substances” (43 CFR § 11.62(e)).

- 6.4 BIOLOGICAL** Injury to biological resources occurs “if concentration of the [hazardous] substance is sufficient to:
- (i) Cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations; or

- (ii) Exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342, in edible portions of organisms; or
- (iii) Exceed levels for which an appropriate state health agency has issued directives to limit or ban consumption of such organism” (43 CFR § 11.62(f)).

The methods used to determine injury to a biological resource need to satisfy several acceptance criteria:

- (i) “The biological response is often the result of exposure to oil or hazardous substances. This criterion excludes biological responses that are caused predominately by other environmental factors such as disturbance, nutrition, trauma, or weather. The biological response must be a commonly documented response resulting from exposure to oil or hazardous substances.
- (ii) Exposure to oil or hazardous substances is known to cause this biological response in free-ranging organisms. This criterion identifies biological responses that have been documented to occur in a natural ecosystem as a result of exposure to oil or hazardous substances. The documentation must include the correlation of the degree of the biological response to the observed exposure concentration of oil or hazardous substances.
- (iii) Exposure to oil or hazardous substances is known to cause this biological response in controlled experiments. This criterion provides a quantitative confirmation of a biological response occurring under environmentally realistic exposure levels that may be linked to oil or hazardous substance exposure that has been observed in a natural ecosystem. Biological responses that have been documented only in controlled experimental conditions are insufficient to establish correlation with exposure occurring in a natural ecosystem.
- (iv) The biological response measurement is practical to perform and produces scientifically valid results. The biological response measurement must be sufficiently routine such that it is practical to perform the biological response measurement and to obtain scientifically valid results. To meet this criterion, the biological response measurement must be adequately documented in scientific literature, must produce reproducible and verifiable results, and must have well defined and accepted statistical criteria for interpreting as well as rejecting results.”

Additionally, injury determination must:

“be based upon the establishment of a statistically significant difference in the biological response between samples from populations in the assessment area and in the control area. The determination as to what constitutes a statistically significant difference must be consistent with the quality assurance provisions of the Assessment Plan. The selection of the control area shall be consistent with the guidance provided in § 11.72 of this part.”

Several specific biological responses already determined to meet the above criteria are identified in the regulations, and can be found at (43 CFR § 11.62(f)(4)). These responses include the following (paraphrased):

- (i) *Category of injury—death.* Five biological responses for determining when death is a result of exposure to the discharge of oil or release of a hazardous substance meet the acceptance criteria.
 - (A) Brain cholinesterase (ChE) activity
 - (B) Fish kill investigations
 - (C) Wildlife kill investigations
 - (D) *In situ* bioassay
 - (E) Laboratory toxicity testing
- (ii) *Category of injury—disease.* One biological response for determining when disease is a result of exposure to the discharge of oil or release of a hazardous substance has met the acceptance criteria.
 - (A) *Fin erosion.*
- (iii) *Category of injury—behavioral abnormalities.*
 - (A) Clinical behavioral signs of toxicity.
 - (B) Avoidance.
- (iv) *Category of injury—cancer.* One biological response for determining when cancer is a result of exposure to the discharge of oil or release of a hazardous substance has met the acceptance criteria.
 - (A) Fish neoplasm
- (v) *Category of injury—physiological malfunctions.* Five biological responses for determining when physiological malfunctions are a result of exposure to the discharge of oil or release of a hazardous substance have met the acceptance criteria.
 - (A) Eggshell thinning
 - (B) Reduced avian reproduction
 - (C) Cholinesterase (ChE) enzyme inhibition
 - (D) Delta-aminolevulinic acid dehydratase (ALAD) inhibition
 - (E) Reduced fish reproduction
- (vi) *Category of injury—physical deformation.* Four biological responses for determining when physical deformations are a result of exposure to the discharge of oil or release of a hazardous substance have met the acceptance criteria.
 - (A) Overt external malformations
 - (B) Skeletal deformities

- (C) Internal whole organ and soft tissue malformation
- (D) Histopathological lesions.

6.5 AIR Injury to air resources occurs “if one or more of the following changes in the physical or chemical quality of the resource is measured:

- (i) Concentrations of emissions in excess of standards for hazardous air pollutants established by section 112 of the Clean Air Act, 42 U.S.C. 7412, or by other Federal or state air standards established for the protection of public welfare or natural resources; or
- (ii) Concentrations and duration of emissions sufficient to have caused injury as defined in paragraphs (b), (c), (e), or (f) of this section to surface water, ground water, geologic, or biological resources when exposed to the emissions.”

6.6 LINKING INJURY STUDIES TO DOI REGULATIONS The injury assessment studies that are currently proposed to support assessment of terrestrial, aquatic, geological, and groundwater injuries, as well as human use service losses are described in detail in Chapter 7. The exhibit below outlines the specific DOI natural resource damage assessment regulations associated with each study.

EXHIBIT 6-1 LINKING INJURY ASSESSMENT PLAN STUDIES TO DOI NATURAL RESOURCE DAMAGE ASSESSMENT REGULATIONS

| INJURY/DAMAGES DETERMINATION/QUANTIFICATION APPROACH | DOI NRDA REGULATIONS INJURY DEFINITION | DOI NRDA REGULATIONS DEFINITION COMPONENTS |
|--|---|---|
| SURFACE WATER AND SEDIMENTS | | |
| Comparison of surface water data to injury thresholds | Threshold exceedances 43 CFR § 11.62(b)(1) | Hazardous contaminant concentrations are in excess of applicable water quality criteria 43 CFR § 11.62(b)(1)(i-iii) |
| Comparison of sediment data to effects thresholds | | Contaminant concentrations sufficient to cause injury to groundwater, soil, or biota when exposed to sediments 43 CFR § 11.62(b)(1)(v) |
| Review of Hanford sediment and pore water toxicity studies | | |
| Benthic invertebrates: sediment toxicity testing | | |
| SOILS | | |
| Comparison of soil data to effects thresholds | Sufficient to cause injury 43 CFR § 11.62(e) | Concentrations in the soil of substances sufficient to cause a toxic response to soil invertebrates 43 CFR § 11.62(e)(9) |
| Soils geospatial evaluation | | Concentrations sufficient to cause injury to other resources when exposed to the substances 43 CFR § 11.62(e)(11) |
| Review of Hanford soil toxicity studies | | Concentrations sufficient to cause injury to other resources when exposed to the substances 43 CFR § 11.62(e)(11); concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i) Statistical significant difference in mortality between population samples and controls 11.62(f)(4)(i)(E). |
| Nematode toxicity testing | | Concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i); statistical significant difference in mortality between population samples and controls 11.62(f)(4)(i)(E); and/or concentrations in the soil of substances sufficient to cause a phytotoxic response 43 CFR § 11.62(e)(10) |
| Native plant toxicity testing | | NA |
| Impacts of remedial activities | Recoverable damages include any increase in injuries as a result of response actions 43 CFR § 11.15(1) | NA |
| VADOSE/GEOLOGICAL | | |
| Characterize vadose zone contamination and potential for long-term injury to groundwater and surface water | Sufficient to cause injury 43 CFR § 11.62(e) | Concentrations sufficient to cause injury to other resources when exposed to the substances 43 CFR § 11.62(e)(11) |
| Evaluation of existing vadose zone models | Quantify injury in terms of the reduction from baseline services 43 CFR § 11.70-11.73 | Quantifying injured groundwater 43 CFR § 11.71(i) and Source and pathway and injury quantification |
| GROUNDWATER | | |
| Developing comprehensive database and comparison to injury | Injury to groundwater, threshold | Concentrations in excess of water quality criteria and drinking water |

Final Hanford Natural Resource Damage Assessment Injury Assessment Plan

| INJURY/DAMAGES DETERMINATION/QUANTIFICATION APPROACH | DOI NRDA REGULATIONS INJURY DEFINITION | DOI NRDA REGULATIONS DEFINITION COMPONENTS |
|---|--|---|
| thresholds | exceedances 43 CFR § 11.62 (c)(1) | standards 43 CFR § 11.62(c)(1)(i-iii) |
| Groundwater upwellings | Quantify injury in terms of the reduction from baseline services 43 CFR § 11.70-11.73 | Concentrations sufficient to cause injury to biological resources when exposed to groundwater 43 CFR § 11.62(c)(1)(iv) |
| Define the legal, political, and economic environment for baseline services provided by groundwater | | Baseline services determination 43 CFR § 11.72 |
| Review of contaminant plume mapping | | Determining areal extent of hazardous substances in water or geologic materials within the assessment area 43 CFR § 11.71(i)(1) |
| Vertical distribution of contaminant plumes | | Determining vertical extent of released substances 43 CFR § 11.71(i)(2) |
| Geology of Columbia riverbed | | Quantifying injured groundwater 43 CFR § 11.71(i) and concentrations sufficient to cause injury to biological resources when exposed to groundwater 43 CFR § 11.62(c)(1)(iv) |
| Synoptic sampling of river corridor wells | | |
| Validity, limitations to existing Hanford groundwater models | | Quantifying injured groundwater 43 CFR § 11.71(i) |
| Quantification of injured groundwater volume and time dimensions | | |
| BIOTA | | |
| Comparison of biological tissue data to adverse effects thresholds | Concentrations sufficient to cause injury to biota 43 CFR § 11.62(f)(1-4) | Concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i) |
| Assessment of plant community health | | Concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i) and/or statistical difference between assessment area and control areas 43 CFR § 11.62(f)(3) |
| Assessment of terrestrial invertebrate abundance | | |
| Mussels: Distribution, abundance, and histopathology | | |
| Prickly sculpin habitat use | | |
| Assessment of avian abundance and diversity | | |
| Small mammal population assessment | | Concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i) and/or statistical significant difference in mortality between population samples and controls 11.62(f)(4)(i)(E) |
| Mussels: Toxicity testing | | |
| Early life stage sculpin, white sturgeon, and rainbow trout toxicity testing | | Concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i) and/or statistical significant difference in mortality between in situ populations and controls 11.62(f)(4)(i)(D) |
| Chinook salmon artificial redd evaluation | | |
| Mussels: Caged (<i>in situ</i>) study | | Concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i); to cause avoidance 43 CFR § 11.62(f)(iii)(B); and/or groundwater upwelling contamination sufficient to cause injury to biota 43 CFR § 11.62(b)(v) and 11.62(c)(iv) |
| Chinook salmon spawning habitat evaluation | | |
| Great Basin pocket mouse: carbon tetrachloride and histopathology | Concentrations sufficient to cause adverse changes in viability 43 CFR § 11.62(f)(1)(i); statistical difference between assessment populations and control populations 11.62(f)(3); and/or physical deformations | |

| INJURY/DAMAGES DETERMINATION/QUANTIFICATION APPROACH | DOI NRDA REGULATIONS INJURY DEFINITION | DOI NRDA REGULATIONS DEFINITION COMPONENTS |
|--|---|---|
| | | 11.62(f)(4)(vi) |
| Evaluation of exposure in Hanford Site avian species | Determination of exposure pathways 43 CFR § 11.63 | Establish pathway 43 CFR § 11.63(a-f) |
| HUMAN USE | | |
| Ethnographic Study to Identify Traditional Cultural Properties at Hanford | <i>Study does not link directly to an injury definition, but provides information to support restoration planning.</i> | NA |
| Inventory of institutional controls related to the release of hazardous contaminants | Quantification of service reductions 43 CFR § 11.71 | In quantifying changes in natural resource services, services include provision biological resources, recreation, and other products or services used by humans 43 CFR § 11.71(e) |
| Assess tribal service losses | | |
| Current resource characterization for restoration of tribal losses | | |
| ALL RESOURCES | | |
| Treatment of non-detects in studies analyzing existing data | <i>Study does not link directly to an injury definition, but provides information necessary to conduct studies analyzing existing data.</i> | NA |
| Quantification of lost aquatic, terrestrial, geological, groundwater, and human use services | Quantify injury in terms of the reduction from baseline services 43 CFR § 11.70-11.73 | Quantifying lost natural resource services 43 CFR § 11.71(a) |

CHAPTER 7 | INJURY ASSESSMENT STUDIES

7.1 In order to advance the injury assessment process, the Trustees plan to undertake a series of studies that will inform both determination and quantification of injury to natural resources resulting from Site-related contamination. Damage determination studies designed to provide information to help the Trustees identify and scale restoration needed to address natural resource injuries, including the cost of such restoration, will be addressed in a separate Restoration and Compensation Determination Plan (to be developed at a later date, in accordance with 43 CFR § 11.81).

INTRODUCTION

This chapter describes the studies that the Trustees are presently undertaking or are considering at this time. The selected efforts represent the Trustees' best understanding of the studies that may be necessary to identify and quantify injury to site natural resources and their services. The Plan is not intended to limit other studies that may be undertaken in the course of the assessment, but represents the current best judgment of the Hanford Trustees regarding the types of studies that are needed to advance the assessment. The Trustees recognize that other studies may become necessary or advisable, as the assessment proceeds. For instance, focused pathway studies may be needed to the extent that the Trustees identify uncertainties regarding the source of specific contaminants associated with identified injuries. The Trustees may also choose to evaluate specific natural resources in greater detail. For example, the Pacific lamprey is a species of exceptionally high cultural value to indigenous peoples in the region, as are many other natural resources. As new information becomes available during the course of this assessment the Trustees may choose to pursue additional assessment activities.

Note that the inclusion of a study within this Plan does not guarantee that it will be undertaken -- the Hanford Trustees may determine that some of these efforts are not needed, or may have lower priority -- and studies not included within the Plan may be deemed necessary at a later date as more information becomes available. For example, some studies may not be needed if reasonable assumptions can be made, balancing the cost of additional research or sampling against the expected gain in information from a particular study. As such, this Plan provides a starting point from which the Trustees will begin to prioritize study efforts and implement the injury assessment process. As these efforts progress and additional information is generated, the Trustees may modify this Plan, and may provide amendments to this Plan for public review and comment.

EFFORTS TO DATE

A number of Trustee efforts have led to the selection of the particular studies included in this chapter. The Hanford Natural Resource Trustee Council was formed in 1993 and provided technical advice to DOE regarding response activities. More recently, the Trustees have been meeting on a monthly basis to discuss Hanford assessment activities. There are six technical working groups (TWGs) that focus on more technical analyses including the aquatic, terrestrial, groundwater, human use, restoration, and source and pathway TWGs. Specifically, the Hanford TWGs have conducted preliminary evaluations of geo-coded sediment and fish contaminant data to determine resources at risk, developed a number of species profiles, which summarize and evaluate historical contaminant data on Hanford species of concern, conducted research on contaminant sources and resource use of several ponds and ditches on Hanford, evaluated groundwater contaminant plume maps, and began developing the Hanford Natural Resource Restoration Plan which addresses early restoration and restoration project evaluation criteria.

The Trustees held a number of workshops and expert panels to explore different methods for injury assessment as well as key questions on the effects of contamination at Hanford. Workshop and panel topics included data management, quality assessment, ecosystem service valuation, human use services and service flows in natural resource damage assessments, compiling toxicity thresholds, injury to aquatic biota in the Hanford Reach, groundwater contaminant upwellings, the integration of groundwater and vadose zone analyses, and the effects of radionuclides on biota at Hanford.

With contractor support, the Trustees have completed a number of large technical analyses including a compilation and evaluation of natural resource information and historical contaminant concentrations from the Hanford Site, an analysis and summary of key data gaps, and a preliminary estimate of injury at the Site. Together, these analyses have helped the Trustees to evaluate existing information and identify injury studies that will fill data gaps and allow the Trustees to determine and quantify injury at the Hanford Site.

INJURY STUDIES

Initial injury determination and quantification activities will entail the evaluation of existing data. Some data evaluation efforts are underway: for example, the Trustees have begun examining contaminant data in the Hanford Environmental Information System

Quality Assurance

The Trustees recognize the importance of data quality, including the need to both understand and document the quality of existing data as well as ensuring the quality of newly generated data. Work plans for individual studies will include Quality Assurance Project Plans that will describe data quality-related measures that will be undertaken as part of study implementation. Chapter 8 provides more information on quality assurance management in the context of this natural resource damage assessment.

(HEIS) database.¹¹⁹ Future efforts will focus on a more comprehensive evaluation of available contaminant concentration data and other information. This approach will ensure that the Trustees utilize the substantial amount of existing data on the nature and extent of contamination.

The availability of such a large volume of existing information, however, presents challenges in data management, and in recognition of these challenges, the Trustees have developed a Quality Assurance Management Plan (HNRTC 2011b) and a Data Management Plan (HNRTC 2011a). The purpose of these documents, and of data and quality management activities in general, is to establish and adhere to a methodology governing the collection, collation, evaluation and management of all environmental data and related information to help ensure the integrity of the data, such that the data collections and applications undertaken by the Trustees are of known and acceptable quality, are scientifically valid and legally defensible.

In addition to evaluating existing information, the Trustees have identified a number of potential studies to provide new information to support injury determination and quantification. These studies are summarized in Exhibit 7-1 and ES-1, and described below in more detail. These studies address aquatic resources, terrestrial resources, vadose zone/geological resources, groundwater, human use, and data management.

Identification of Traditional Cultural Properties at Hanford

Before field studies or other studies undertaken at the Hanford Site begin, Traditional Cultural Properties (TCPs) must be identified. Any Federal undertaking that has the potential to affect Federally-listed (and/or eligible for listing) cultural resources, including TCPs, must be evaluated, as mandated under the National Historic Preservation Act (NHPA) Section 106. TCPs cannot be discovered through archaeological or historical research alone. The existence and significance of such locations can only be ascertained through interviews with knowledgeable users of the area or through other forms of ethnographic research.

Studies of environmental media (i.e., groundwater, soils, sediment) generally focus on comparisons of observed and forecast future contaminant concentrations with injury or effects thresholds. Human use studies focus on understanding the likely extent of institutional controls related to contaminant releases that may limit public use of the site, as well as understanding the manner and extent to which contaminants have affected tribal use of the site and services derived from natural resources at the site. Proposed studies of biota are intended to examine the ecological impacts to native species and communities due to exposure to hazardous contaminants released from site operations.

¹¹⁹ Existing databases include, but are not limited to, HEIS, the Columbia River Component historic database, the Columbia River Component Data Summary Report for the Remedial Investigation of Hanford Site Releases to the Columbia River, the River Corridor Baseline Risk Assessment GiSdT database, and the Near-Field Monitoring Program's collection effort, reported through the Environmental Release Summary database.

The range and types of biota studies are particularly varied. Consistent with DOI NRDA guidance, they include laboratory and field studies; these two categories each have advantages and disadvantages. Field studies have a distinct advantage in that they comprehensively reflect the cumulative effects of contaminants present at a site, however complex those mixtures may be. Because field studies examine biota under natural conditions, these organisms are also exposed to other natural stressors (food foraging, predators, disease, temperature fluctuations, etc.). Organisms may be more sensitive to contaminants when faced with such natural stressors. However, natural systems are typically highly variable, making it difficult to detect differences in organisms or populations in a study area as compared to reference areas, even if such differences exist. Field studies have other limitations. Obtaining adequate sample sizes can be challenging, depending on the study organism and endpoint(s). In addition, even if effects are observed in a field study, it can be difficult to persuasively determine the causality of the effect: site contaminants could be responsible, or arguably, other site-specific factors (differences in habitat type, prey availability, predator pressure, disease prevalence) may contribute to, or could be responsible for, the observed effects.

In contrast, laboratory studies address causality directly. For example, spiked exposure studies (i.e., studies in which biota are exposed to a specific level of a contaminant) can demonstrate that specific contaminants cause specific effects, albeit under controlled, laboratory conditions. Laboratory studies are limited in that they do not fully mimic field conditions. Also, testing all contaminant combinations or exposures that may occur under field conditions is frequently not technically or financially possible.

In situ studies and laboratory toxicity studies that use site media combine features of both lab and field studies. *In situ* experiments expose organisms to actual site mixtures of contaminants under actual field conditions (e.g., variable water flow and temperatures, parasite exposure, etc.) but may not fully replicate field conditions—for instance, organisms are frequently protected from predation by virtue of being caged. Laboratory toxicity experiments with site media expose organisms comprehensively to whatever site-specific, potentially complex mixture of contaminants is present, but they do so under conditions that are controlled in other ways (e.g., temperature, food availability, etc.).

Because the various types of potential biotic injury studies have different -- and often complementary -- advantages and disadvantages, the Trustees have selected a variety of approaches to evaluate injury.

In all cases, individual study plans will be developed by the Trustees and principal investigators prior to study implementation. These individual study plans will detail the approaches to be followed, including actions to assure data quality. These study plans will undergo peer review, to provide assurance that the study designs will provide the information required by the assessment.

To help guide future assessment efforts, the Trustees have grouped the proposed studies into three priority categories. The assignment of a study to a particular category is based on Trustee judgments regarding: cost effectiveness; technical study sequencing requirements; likelihood of demonstrating injury; likely contribution to the selection and

scaling of restoration alternatives; and/or anticipated concerns of the public. The three categories are:

1. Nearer-term priorities,
2. Middle-term priorities, and
3. Longer-term priorities.

The first of these -- the nearer-term priorities -- includes studies that are presently ongoing, prerequisites for subsequent work, and/or expected to generate information of significant use in refining future study designs. The second category includes those that are more likely to identify substantive injuries, are anticipated to address concerns of the public, and/or are expected to contribute the most towards informing the selection and scaling of restoration alternatives. The third category includes studies that depend on the prior completion of other efforts, and those that are presently expected to present more difficult technical issues.

As noted previously, both the conduct and timing of these studies will depend on the specific needs of the assessment, resource and funding limitations, and other factors.

EXHIBIT 7-1 OVERVIEW OF INJURY ASSESSMENT STUDIES

| RESOURCE / USE | STUDY | STATUS | CATEGORY | GENERAL APPROACH |
|--------------------------------|--|-----------|----------|---|
| AQUATIC | | | | |
| SURFACE WATER | Comparison to injury thresholds | Ongoing | 1 | Comparison of observed surface water concentrations to regulatory water quality criteria |
| SEDIMENT | Comparison to effects thresholds | Ongoing | 1 | Comparison of sediment concentrations to literature-based adverse effects thresholds and guidelines to inform understanding of the potential severity and magnitude of effects |
| AQUATIC BIOTA (GENERAL) | Comparison to effects thresholds - tissues | Ongoing | 1 | Compare site-specific contaminant data in biota tissue to literature-based adverse effects thresholds to inform understanding of potential severity and magnitude of effects |
| | Review of Hanford sediment and pore water toxicity studies | Potential | 1 | Evaluate results of existing studies of toxicity to trust resources to identify evidence of injury |
| BENTHIC INVERTEBRATES | Sediment toxicity testing | Potential | 2 | Evaluate toxicity of Site sediments to benthic invertebrates |
| MUSSELS | Distribution, abundance, and histopathology | Potential | 3 | Collect data on mussel community health; determine correlations between community metrics, habitat quality, and presence of contaminants; assess histopathological endpoints |
| | Toxicity testing | Ongoing | 1 | Evaluate toxicity of a sub-set of contaminants to mussels, including native and sensitive species |
| | Caged (<i>in situ</i>) study | Potential | 3 | Evaluate direct toxicity of contaminants in surface water and sediment to mussels <i>in situ</i> |
| FISH | Chinook salmon spawning habitat evaluation | Potential | 2 | Compare habitat characteristics and contaminant concentrations, including chromium, at known and potential spawning locations to determine whether contamination influences spawning site selection and avoidance |
| | Chinook salmon artificial redd evaluation | Potential | 3 | Assess effects of chromium-contaminated, and other contaminated groundwater upwellings on salmon development, using artificial redds |
| | Prickly sculpin habitat use | Potential | 2 | Estimate relative abundance and density of sculpin; evaluate population size/age structure in areas exposed to contaminated groundwater versus reference sites |
| | Early life stage sculpin, white sturgeon, and rainbow trout toxicity testing | Potential | 3 | Expose early life stage sculpin and sturgeon in the laboratory to waterborne chromium and other contaminants |

Final Hanford Natural Resource Damage Assessment Injury Assessment Plan

| RESOURCE / USE | STUDY | STATUS | CATEGORY | GENERAL APPROACH |
|------------------------------------|---|-----------|----------|---|
| AQUATIC RESOURCES | Quantification of lost aquatic ecological services | Potential | 1 | Compile aquatic resource information and analyze to quantify lost services |
| TERRESTRIAL | | | | |
| SOIL | Comparison to effects thresholds | Ongoing | 1 | Compare concentrations of contaminants in soil with literature-based toxicity thresholds to inform potential severity and magnitude of effect |
| | Geospatial evaluation | Potential | 1 | Geospatial evaluation of patterns in soil data to identify areas more/less likely to have been exposed to potentially injurious contaminant concentrations, and areas where additional sampling may be useful |
| TERRESTRIAL BIOTA (GENERAL) | Comparison to effects thresholds - tissues | Ongoing | 1 | Compare site-specific contaminant data in biota tissue to literature-based adverse effects thresholds to inform potential severity and magnitude of effect |
| | Review of Hanford soil toxicity studies | Potential | 1 | Evaluate results of existing studies on soil toxicity to identify evidence of injury |
| PLANTS | Native plant toxicity testing | Potential | 3 | Evaluate potential phytotoxic effects of Site soils |
| | Assessment of plant community health | Potential | 3 | Compare health of plant communities at Hanford Site to suitable reference locations |
| INVERTEBRATES | Nematode toxicity testing | Potential | 3 | Evaluate the suitability of site soil as a habitat for biota |
| | Assessment of terrestrial invertebrate abundance | Potential | 2 | Assess abundance and (possibly) diversity of species/species groups across contaminant gradients; examine correlations between metrics and measures of contaminant exposure |
| BIRDS | Assessment of avian abundance and diversity | Potential | 2 | Assess abundance and diversity of birds across contaminant gradients using visual and auditory metrics; examine correlations between metrics and measures of contaminant exposure |
| | Evaluation of exposure in Hanford Site avian species | Potential | 2 | Evaluate exposure of avian species to contaminants |
| MAMMALS | Small mammal population assessment | Potential | 2 | Identify impacts of contaminant exposure on small mammal community population |
| | Great Basin pocket mouse: carbon tetrachloride and histopathology | Potential | 3 | Evaluate effects of carbon tetrachloride exposure on small burrowing mammals at Hanford Site |
| TERRESTRIAL RESOURCES | Impacts of remedial activities | Potential | 1 | Compilation of information describing the general type, timing, location, and spatial extent of activities; estimation of severity of impacts on habitat |

| RESOURCE / USE | STUDY | STATUS | CATEGORY | GENERAL APPROACH |
|------------------------------|---|-----------|----------|---|
| | Quantification of lost terrestrial ecological services | Potential | 1 | Compile terrestrial resource information and analyze to quantify lost services |
| VADOSE/GEOLOGICAL | | | | |
| GEOLOGICAL RESOURCES | Characterize vadose zone contamination and potential for long-term injury to groundwater and surface water due to contaminants that have been released to the vadose zone | Potential | 1 | Utilize available information and model outputs to develop an understanding of the likely nature, extent, and timing of natural resource injury, and lost services that could occur as a result of vadose zone contamination. |
| | Evaluation of existing vadose zone models | Potential | 2 | Assess ability and limitation of existing models to quantify vadose zone contamination flux |
| GROUNDWATER | | | | |
| GROUNDWATER RESOURCES | Developing comprehensive database and comparison to injury thresholds | Ongoing | 1 | Create a comprehensive Hanford groundwater database for Trustee use in injury determination and quantification; use information in database to compare observed groundwater concentrations to regulatory water quality criteria |
| | Review of contaminant plume mapping | Ongoing | 1 | Evaluate methods and results of current groundwater contaminant plume mapping at Hanford |
| | Define the legal, political, and economic environment for baseline services provided by groundwater ¹¹⁸ | Potential | 1 | Describe services provided by groundwater at Hanford Site under baseline conditions; analyze how these services have been impacted by contaminants |
| | Validity and limitations to existing Hanford groundwater models | Potential | 1 | Verify validity of existing Hanford groundwater models in quantifying currently injured groundwater, as well as understanding of past and future nature and extent of groundwater contamination. |
| | Groundwater upwellings | Potential | 2 | Characterize distribution, frequency, and volumetric flow rate of contaminant upwellings in Columbia River, as pathway to potential injury to biota |
| | Synoptic sampling of river corridor wells | Potential | 2 | Sample selected river corridor wells at varying river stages to determine impact of river stage on groundwater depth readings |

¹¹⁸ This study to define the legal, political, and economic environment of baseline groundwater services should be done prior to other groundwater studies.

| RESOURCE / USE | STUDY | STATUS | CATEGORY | GENERAL APPROACH |
|------------------------|---|-----------|----------|---|
| | Vertical distribution of contaminant plumes | Potential | 1 | Construct monitoring wells in key areas for sampling to identify depth of significant plumes |
| | Geology of Columbia riverbed | Potential | 3 | Drill boreholes on river islands, develop seismic and electromagnetic profiles, and perform geophysical surveys to determine the presence of plumes near and beneath the river as well as ongoing potential for contaminant migration |
| | Quantification of injured groundwater volume and time dimensions | Potential | 1 | Quantify groundwater affected by contaminant release across Site |
| HUMAN USE | | | | |
| TRIBAL USE | Ethnographic study to identify Traditional Cultural Properties | Potential | 1 | Identify Traditional Cultural Properties at the Hanford Site |
| | Assess tribal service losses | Potential | 1 | Identify service losses to tribal use not accounted for in other studies |
| | Current resource characterization to allow for restoration of lost tribal services | Potential | 1 | Characterize and monitor contaminant concentrations in natural resources to verify potential for restoration of tribal services |
| INSTITUTIONAL CONTROLS | Inventory of institutional controls related to the release of hazardous contaminants, and description of associated limits on human use of the site | Potential | 1 | Inventory the nature and geographic scope of institutional controls related to hazardous contaminant releases that could impact past, present or future human use of the site. |
| ALL RESOURCES | | | | |
| DATA MANAGEMENT | Treatment of non-detects in studies analyzing existing data | Potential | 1 | Evaluate a variety of options for handling non-detect sample results within each analysis. |

7.2 AQUATIC RESOURCES

The Hanford Site has a lengthy operational and remedial history, and as part of that history, a number of ecological, toxicological, and other studies have produced information of potential use in the injury assessment. The studies included in this Injury Assessment Plan build on available information from past efforts and are intended to address key data gaps and/or remaining uncertainties. The following paragraphs briefly describe such prior research in order to characterize the larger scientific context into which the proposed studies will fit.

OVERVIEW OF EXISTING SITE AQUATIC RESOURCE DATA

Available information about the Hanford Site's aquatic natural resources that is of most relevance to a natural resource damage assessment includes but is not limited to: (a) measurements of hazardous substances in site media (surface water, pore water, sediments) and in the tissues of aquatic organisms, (b) information about species presence/absence at various locations; (c) results of toxicity testing of specific biota with site media and site contaminants, (d) population and community investigations, and (e) other research exploring potential contaminant-related effects at the Site (e.g., reproductive studies, histopathological evaluations, biota condition assessments, behavioral assessments, etc.).

Measurements of Hazardous Substances

The Trustees have identified at least seven partially overlapping databases that contain many measurements of concentrations of hazardous substances in site media and biotic tissues. The Hanford Environmental Information System (HEIS) database contains the largest numbers of samples of soils, surface water, biota, and groundwater, while other databases contain larger numbers of sediment and pore water samples. HEIS continues to be developed, and may eventually serve as the repository for virtually all site sampling efforts, past and ongoing. A substantial effort has been underway within this past year to add more data to HEIS; as this effort progresses, it may become increasingly less necessary to rely on other compilations of contaminant information. In addition to HEIS, databases with information on aquatic samples include: (a) the Columbia River Component historic database, (b) the Columbia River Component Data Summary Report for the Remedial Investigation of Hanford Site Releases to the Columbia River (WCH 2011), and (c) the River Corridor Baseline Risk Assessment GiSdT database.

Although the number of measurements of contaminants in site abiotic and biotic media is large, many challenges remain in effectively using these data in the context of an assessment. These challenges include but are not limited to: the variety of sampling efforts (and associated sampling objectives) associated with the datasets; the need to understand quality assurance issues associated with the various datasets; analytic issues associated with non-detect values¹¹⁹; and the absence of sample characterization information in many cases (e.g., sampling depths and geographic coordinates). Studies that rely on this information (e.g., those involving comparisons of measured

¹¹⁹ "Non-detect values" refers to the contaminant concentration value reported when the true concentration was lower than the testing method employed is able to detect.

concentrations with thresholds) will need to address these issues during the detailed study design and implementation stages.

Toxicity Testing

Trustees frequently include toxicity testing among site assessment activities. Some such testing has been conducted: in particular, the River Corridor Baseline Risk Assessment (DOE 2011b) presents the results of site-specific toxicity tests with site media. RCBRA tests include assessments of sediment toxicity to pak choi, and to the amphipod *Hyaella azteca*, as well as assessments of pore water toxicity to the daphnid *Ceriodaphnia dubia* and to the frog *Xenopus laevis*. The results of these efforts provide information that may be valuable in the context of an assessment; however, preliminary review of the approach and results suggests that they may have important limitations associated with their use (see discussion in “Benthic Invertebrates: Sediment Toxicity Testing”). Altogether, the Trustees plan to undertake additional review of the RCBRA’s toxicity testing results and may pursue additional toxicity testing of site media, as described in “Benthic Invertebrates: Review of Hanford Toxicity Studies” and “Benthic Invertebrates: Sediment Toxicity Testing”.

Species Distribution and Population/Community Characterization Information

In a natural resource damage assessment, Trustees may choose to evaluate species distributions and population or community metrics to evaluate the extent to which hazardous substances may have affected biota at this level of ecological organization.

Some information on these topics is available: for example, Mueller *et al.* (2001) presents the results of a mussel survey of the Hanford Reach, documenting the species composition, densities, and distribution of native freshwater mussels along the Benton County shoreline of the Hanford Reach. The authors found several shells of the western pearlshell but concluded that “the species appears to be largely absent from its historical range” (Mueller *et al.* 2011). Based in part on this study’s results, the Trustees believe that additional mussel work may be useful in identifying the potential sensitivity of native unionid species to site contaminants in the laboratory and under field conditions (see “Mussels: Distribution, Abundance, and Histopathology”, “Mussels: Toxicity Testing”, and “Mussels: Caged (*in situ*) Study”).

The River Corridor Baseline Risk Assessment (DOE 2011b) presents community-level information on aquatic communities, which was gathered using a rock basket deployment technique. Baskets were deployed in association with three groundwater plumes (chromium, uranium, and strontium-90) as well as at locations between the areas of most direct plume influence, and at reference locations. The authors conclude that “For most RCBRA study sites, results for aquatic community measures were as high as or higher than upstream reference sites with similar habitat characteristics.” The Trustees have reservations about the defensibility of this conclusion and in the future may choose to more formally and carefully review both the study design and its results. The Trustees may also choose to conduct additional benthic invertebrate community health assessment (e.g., using different geographic scope and/or sample sizes, different technical methods, and/or using more sophisticated statistical approaches to more carefully control for

confounding factors). However, at the current moment, such a study represents a lower priority effort and is not included in the Injury Assessment Plan at present.

DOE's Ecological Monitoring and Compliance Project (EMC), which until 2011 was managed by Pacific Northwest National Laboratory (PNNL) and is now managed by Mission Support Alliance (MSA), includes information on aquatic species locations including but not limited to salmon and steelhead redd counts, amphibian occurrences including call responders, and clam counts. For purposes of natural resource damage assessment, this information may be useful in identifying likely locations of biota in the event that future field studies on these species are pursued, but it is not likely to be useful for direct injury determination purposes as the program has not been designed to definitively identify species absence, or to quantify population-level metrics such as abundance.

Other authors have also developed and/or compiled general information on aquatic species presence at the Hanford Site (e.g., Fitzner and Gray 1990, CRCIA 1998, TNC 1999, TNC 2003, Duncan 2007).

Histopathological Investigations

The Trustees may examine organisms for evidence of physiological injuries including but not limited to histopathological impacts. Some site-specific histopathology information on aquatic biota has been collected in recent years, although most study efforts appear to be subject to certain limitations. For example, PNNL's databases include histology information for certain biota collected between 2002 and 2005 (i.e., 3 bass, 1 adult bullfrog, 1 tadpole bullfrog, 3 suckers, 830 clams, 33 sculpin, 68 crayfish, and 7 whitefish). The Trustees have not identified reports that describe the sampling methods, sampling design, and/or discuss the results.¹²⁰

Larson *et al.* (2008) describes a November 2003 to February 2005 *in situ* investigation on exposure of the (non-native) Asiatic clam, *Corbicula fluminea*, to contaminants in the 300 Area. Growth, survival, and tissue conditions were evaluated at two nearshore locations, one of which was associated with contaminated groundwater upwelling, and the other was an upstream reference location. The authors did not identify any effects of contaminant exposure; however, growth overall was poor (negative), which the authors attribute to the type of tubing in which the clams were contained. The study's results may not be representative of results under natural conditions.

DOE (2011b) discusses results of sampling in 2006 and 2007 for mussels, sculpin, juvenile suckers, and for Asian clams *in situ*:

- In mussels, the authors found statistically increased observations between study site versus reference site organisms, in two of the 20 measurements: digestive cell vacuolation severity and degraded mantle condition. This study was limited to six study sites and three reference sites.

¹²⁰ The Hanford Site Environmental Report for Calendar Year 2003 (Poston *et al.* 2004) states that other than radiological results in clams, "Analyses for other species and biological components were still under development when this report was prepared." Subsequent annual environmental reports also do not appear to present the results of this sampling.

- In sculpin, the authors found statistically increased fish length and weight among study site versus reference area fish. The authors also found four out of 22 histopathological measurements to differ between study and reference sites: the number of liver parasites and the number of muscle granulomas was higher among site fish, and the number of encysted parasites in gills and kidneys were higher among reference fish.
- In Asian clams (a non-native species), the authors found statistically increased observations between study site versus reference site organisms, in two of the 19 measurements: the incidence of digestive system epithelial cell shedding, and reproductive system follicle cyst presence. These clams were exposed *in situ* for periods of 3 or for 7 to 8 months.

Finally, as part of a white sturgeon workshop, Kiser (2010) preliminarily reports histology information associated with several tissues from 30 white sturgeon, including 25 from the Hanford Reach and five from a reference area above Wanapum Dam. External and internal anomalies were observed in about 15 percent of all sturgeon, including reference area fish. Tissue histopathology also indicated abnormalities in all fish sampled, including those from the reference area. The observed histopathology was consistent with a chronic viral, bacterial, or chemical stressor. Gonadal observations include inflammation, degeneration, and oocyte necrosis, potentially indicating reproductive impairment. Metal concentrations were “generally low” except for mercury; radionuclide concentrations were “infrequently” detected and were “near detection limits” (*ibid.*). Concentrations of total DDT and PCBs were elevated within the study area fish tissues. The workshop’s conclusions include that, despite the long lifespan of the species and its potential for exposure to higher past contaminant concentrations, “There is considerable uncertainty regarding the likelihood of detecting historical histological impacts [on white sturgeon].”

Chinook Salmon Investigations

A number of efforts have examined NRDA-relevant endpoints in this species. The results are complex: some studies found little evidence of effects on the evaluated endpoints, while others suggest potential impacts. This complexity likely arises from several sources, including differences in the endpoints examined, differences in the life stage examined, and differences in other exposure characteristics used in the study (e.g., water hardness levels, field vs. lab exposure, study duration, etc.). As for all studies in this Assessment Plan, the selected Chinook salmon studies are intended to build on available information from past efforts and to address key data gaps and/or remaining uncertainties.

The following paragraphs summarize past work on Hanford Reach Chinook salmon, and also summarize studies on laboratory Chinook exposed to Hanford Site contaminants, noting those efforts whose findings are most closely linked to the new studies described in this Assessment Plan.

Hanford Reach Chinook salmon field investigations include those conducted by Tiller *et al.* (2004), who collected 100-D and 100-H Area juvenile fall Chinook salmon over three sampling events, to assess chromium body burdens, fish length, weight, and histology.

The authors did not detect statistically significant differences in chromium levels in the tissues of fish from the 100-Areas compared to upstream Vernita Bridge fish. Overall, the authors found “no impact” in the 100-Area fish compared to these reference fish. The authors observed no gross morphological anomalies in any fish, and also found “no indications of any tissue damage in any of the specimens examined.” That said, the histology sample sizes were small: although up to 20 fish were collected per site and sampling event, “up to 10” specimens per location (total of 29 fish) were subject to histological assessment.

Lab studies have investigated the potential for site contaminants to affect traditional ecotoxicological endpoints relating to reproduction as well as the survival and growth of early life stage Chinook. As a group, these studies have produced mixed results. For example, Farag et al. (2006b) assessed the potential for chromium to affect fertilization under exposure conditions “similar to those of the Hanford Reach”; the tested concentration ranges (0 to 266 µg/L) did not affect this endpoint. Concentrations of hexavalent chromium in Hanford Reach pore water have been measured as high as 632 µg/L (Hope and Peterson 1996).

Olson and Foster (1956) exposed Chinook salmon to hexavalent chromium concentrations of 0 to 184 µg/L for seven months, starting at the egg stage. No significant mortality occurred during the egg stage, but by the end of the fry¹²¹ stage, significantly fewer fish survived at the 184 µg/L and 80 µg/L concentrations. Growth retardation was “a more sensitive index of toxicity than mortality” and was “probably significant in the group exposed to 0.016 ppm [16 µg/L].”

Farag et al. (2000) examined the effects of chromium on early life-stages (eyed egg through swim up, plus a holding period of 30 days after swim up), to monitor development, physiological function, growth, and survival. Aqueous chromium concentrations of 5 to 120 µg/L did not significantly affect the assessed endpoints. Referencing Olsen and Foster (1956), discussed above, the authors note that their findings are “similar to early studies conducted at Hanford that showed alevins¹²² to be tolerant to chromium exposure until after the initiation of exogenous feeding and swim-up, when mortality increased dramatically.”

Patton *et al.* (2007) also evaluated the effects of chromium exposure on early life stage Chinook (eyed egg through swim up), exposing these fish to Hanford Site groundwater diluted with Columbia River water. The exposure produced final hexavalent chromium concentrations from 0.79 to 260 µg/L. These authors also found no effects of these exposures on survival, growth, development rate, weight, or length.

¹²¹ Fertilized Chinook salmon eggs are deposited in gravel bed depressions (redds); these eggs hatch into alevins, which use the remaining yolk sac to grow. Alevins remain associated with the gravel and eventually emerge (“swim up”) from the gravel as fry, the first free-swimming and exogenous feeding stage of the salmon’s life cycle.

¹²² See previous footnote.

Farag *et al.* (2006a) found that concentrations of 24 and 54 µg Cr/L for 105 days did not affect growth or survival of Chinook parr;¹²³ however, after increasing concentrations to (a) 120 and (b) 266 µg/L, respectively, weight was decreased under treatment (a), and survival was reduced in treatment (b). The authors also reported fish health impairments in both treatments, as evidenced by kidney lesions and biochemical changes.

Other studies have suggested that site contaminants may have behavioral impacts on this species. For example, DeLonay *et al.* (2001) found that Chinook parr are capable of detecting and avoiding water with chromium concentrations of ≥ 54 µg/L (80 mg/mL hardness); at a higher level of hardness, 200 mg/mL, intended to simulate Hanford groundwater, the parr failed to avoid chromium concentrations of up to 266 µg/L. Behavioral changes constitute an injury under DOI's NRDA regulations; furthermore, if salmon avoid chromium-containing water in the field, the contamination may effectively reduce the area of usable habitat for these fish.

Some information suggests the potential for contaminant-related avoidance behavior in the field: Geist (2000) reports that spawning salmon used areas of hyporheic upwelling where the specific conductance indicated a surface water source of the upwelling, whereas they did not use hyporheic discharge zones where the source was groundwater.¹²⁴

The behavioral findings are among those suggesting that additional research on Chinook salmon is appropriate for the Hanford assessment (e.g., see "Fish: Chinook Salmon Spawning Habitat Evaluation." The Trustees are also considering a field-based (*in situ*) investigation of potential impacts to early life stages (see "Fish: Chinook Salmon Artificial Redd Evaluation"). Organisms may sometimes experience adverse effects to contaminants under field conditions that are not evident from laboratory-based exposures, conducted under much more controlled conditions.

Additional Investigations

Additional site-specific field research, on potential contaminant-related effects to aquatic and aquatically-linked biota, include:

- **A 2005 pilot study on bullfrog and Woodhouse's toad malformations** in animals from two Hanford Reach slough/backwater pools. The authors found a "relatively low" rate of malformations (Poston *et al.* 2006).
- **Canada geese reproduction.** Fitzner *et al.* (1991) note that nearly four decades of research on the nesting ecology and behavior of this species have been conducted. Fertility rates in the 1950s and 1960s found reproductive rates "as high or higher than in areas not supporting nuclear operations." Simmons *et al.* (2010) summarizes Canada goose research at Hanford, concluding that radiological dose rates were "well below applicable guidelines" and that

¹²³ Parr represent the life stage between the fry and smolt stages. Parr have vertical markings on their sides.

¹²⁴ Dissolved oxygen was higher in the surface water discharge areas, but concentrations in both areas were higher than levels needed for egg/alevin survival. Contaminant concentrations were not measured.

maximum concentrations of a variety of other metals “met or fell below existing toxicological benchmarks, suggesting minimal risk... from exposure.”

- **Great blue heron reproduction.** Despite heavy metal concentrations, Tiller *et al.* (2005) found that in 1996, reproductive health of *A. herodias* nesting along the Hanford Reach to be one of the highest reported in the United States. The authors note that there has been a decline in the numbers of active nests from 94 in 1983 to 37 in 1999, attributing this change to increased human activity near nest trees, wind toppling of trees used as nesting sites, and low subadult/survival ratios (Rickart and Tiller 2003 as cited in Tiller *et al.* 2005).

IMPLICATIONS OF EXISTING DATA FOR INJURY ASSESSMENT

Given the description above of available information on contaminant exposure and potential aquatic injuries, the following injury assessment studies have been identified to fill important data gaps. Phase 1 priorities for aquatic injury assessment focus on organizing the information necessary to better understand aquatic resource exposure and to help guide work plan development for later stages of the injury assessment. Phase 1 priorities therefore include estimating the level and extent of surface water, pore water, sediment and aquatic biota tissue contamination, estimating baseline contaminant concentrations in site media, and reviewing the existing sediment and pore water ecotoxicity testing studies. Phase 2 and 3 priorities encompass further efforts that would help the Trustees refine their understanding of potential aquatic injuries. In particular, Phase 2 and 3 efforts include: conducting additional laboratory toxicity testing, gathering information about population and community attributes, conducting *in situ* assessments to evaluate the effects of exposure to site media on aquatic biota, and collecting information on the health of aquatic biota.

SURFACE WATER: COMPARISON TO INJURY THRESHOLDS

Objectives: (1) To determine injuries to surface water resources based on comparisons of measured and/or modeled concentrations of Site COPCs to regulatory water quality standards or criteria. (2) To identify COPCs that may be most strongly associated with potential injuries (e.g., by virtue of having a greater magnitude and/or exceedance of effects thresholds). (3) To identify locations with higher or lower levels of exposure to hazardous substances, to help inform site selection in potential future injury studies.

Need/Rationale: Surface water is a key natural resource, providing habitat to numerous aquatic biota species as well as providing services to humans. Contaminant concentrations in excess of certain levels (e.g., Washington State water quality standards) generally indicate that an injury has occurred under DOI’s NRDA regulations (43 CFR § 11.62(b)(1)(i) through (iii); see Chapter 6).¹²⁵

¹²⁵ Chapter 6 provides complete definitions of injury to natural resources, including injury determination. Exceedances of certain concentration thresholds is a key component of these definitions but is not the only requirement that must be satisfied.

Comparing contaminant concentrations in surface waters to regulatory water quality standards or criteria is a cost-effective and widely used approach to evaluate potential surface water injuries. Furthermore, making comparisons can also help document the existence of a pathway between sources of releases and receptors, and/or may suggest that additional field or lab studies on certain biological receptors/locations/contaminant combinations may be appropriate.

Approach: The study will focus on the Hanford Reach of the Columbia River and appropriate reference locations. The first component of this task will involve assembling and evaluating available surface water and pore water data, and incorporating it into the Trustees' natural resource damage assessment database in accordance with the Data Management Plan and the Quality Assurance Management Plan (HNRTC 2011a, 2011b). Although many measurements of surface water COPCs are available, a comprehensive assessment database has not been developed.

The Trustees will also determine the water quality criteria and standards (e.g., Federal drinking water standards, state water quality criteria) against which sample concentrations will be evaluated.

This study will include an evaluation of baseline conditions, which will include to the extent possible a characterization of the concentration ranges of hazardous substances expected to be present in surface waters but for Hanford Site releases. As part of this evaluation, contaminants will be identified as having one or more of the following origins: natural sources, Hanford Site operations, and/or other anthropogenic sources.

SEDIMENT: COMPARISON TO EFFECTS THRESHOLDS

Objectives: (1) To determine potential past, current, and future injuries to sediment resources based on comparisons of measured sediment COPC concentrations to regulatory standards and literature-based effects thresholds. (2) To identify COPCs that may be most strongly associated with potential injuries (e.g., by virtue of having a greater magnitude and/or exceedance of effects thresholds). (3) To identify locations with higher or lower levels of exposure to hazardous substances, to help inform site selection in potential future injury studies.

Need/Rationale: Sediments provide essential habitat for aquatic plants, mussels and other invertebrates, and fish (e.g., species such as salmon use the river bed as spawning habitat). Comparing sediment contaminant concentrations to appropriate adverse impact thresholds is a cost-effective, widely used approach to identify potential sediment injuries.

Although comparing measured concentrations to literature-based thresholds is not generally, in itself, sufficient to determine injury in accordance with the DOI regulations,¹²⁶ such analyses can inform the Trustees' understanding of the nature and

¹²⁶ Chapter 6 provides complete definitions of injury to natural resources, and sediments are considered to be part of the surface water resource (43 CFR § 11.14(pp)). Injury to sediments is most commonly determined when sediments are sufficiently contaminated to have caused injury to other natural resources (43 CFR § 11.62(b)(1)(v)).

extent of potential injury. For example, within the context of a cooperative assessment, such comparisons can provide a basis for reaching agreement on injury determination and/or quantification assumptions. These comparisons can also help document the existence of a pathway between sources of releases and receptors, and/or may suggest that additional field or lab studies on certain biological receptors/locations/contaminant combinations may be appropriate. They may also help to identify those COPCs that are the largest drivers of injury (e.g., based on the magnitude and/or extent of threshold exceedances).

Approach: The study will focus on the Hanford Reach of the Columbia River and appropriate reference locations. The first component of this task will involve assembling and evaluating available data, and incorporating it into the Trustees' natural resource damage assessment database in accordance with the Data Management Plan and the Quality Assurance Management Plan (HNRTC 2011a, 2011b). Although many measurements of sediment COPCs are available, a comprehensive assessment database has not been developed.

The second component of this study requires identification of adverse effects thresholds—i.e., Site-specific and/or generic values from the literature—against which the Trustees will compare contaminant concentrations from the database described above. Potential thresholds identified to date include Washington State sediment quality criteria, as well as literature-based sediment quality guidelines. Building off the preliminary work done by the Trustees, and supplemented by additional literature review and/or the results of toxicity testing, the Trustees will develop sediment thresholds for each COPC.

This study will include an evaluation of baseline conditions, which will include to the extent possible a characterization of the concentration ranges of hazardous substances expected to be present in Hanford Reach sediments but for Hanford Site releases. As part of this evaluation, contaminants will be identified as having one or more of the following origins: natural sources, Hanford Site operations, and/or other anthropogenic sources.

AQUATIC BIOTA: COMPARISON TO EFFECTS THRESHOLDS - TISSUES

Objectives: (1) To determine potential past, current, and future injuries to aquatic biota based on comparisons of measured tissue COPC concentrations to literature-based effects thresholds. (2) To identify COPCs that may be most strongly associated with potential biotic injuries (e.g., by virtue of having a greater magnitude and/or exceedance of effects thresholds). (3) To identify species and/or locations with higher or lower levels of exposure to hazardous substances, to help inform site selection in potential future field studies of aquatic biota.

Need/Rationale: Biologic resources, including aquatic organisms, are trust resources that provide a suite of essential ecological services. Comparison of COPC tissue concentrations to appropriate adverse impact thresholds is a cost-effective, widely used approach to identify potential biological injuries. With certain exceptions, comparisons of measured concentrations in tissues to thresholds is not usually in itself sufficient to

determine injury in accordance with the DOI regulations;¹²⁷ nevertheless, such analyses can inform the Trustees' understanding of the nature and extent of potential biological injuries. Within the context of a cooperative assessment, these kinds of comparisons can provide a basis for reaching agreement on injury determination and/or quantification assumptions. These studies can also help document the existence of a pathway between sources of releases and receptors, and/or may suggest that additional field or lab studies on certain biological receptors/locations/contaminant combinations may be appropriate. They may also provide help identify those COPCs that may be the largest drivers of injury (e.g., based on the magnitude and/or extent of threshold exceedances).

Approach: The study will focus on the Hanford Reach of the Columbia River and appropriate reference locations. The first component of this task will involve assembling and evaluating available data, and incorporating it into the Trustees' natural resource damage assessment database in accordance with the Data Management Plan and the Quality Assurance Management Plan (HNRTC 2011a, 2011b). Although measurements of COPC concentrations in biota exist, a comprehensive assessment database has not been developed. Therefore, the Trustees will create a database, ensuring that data are normalized, contain location information where possible, and are presented in consistent units (e.g., convert radiological concentrations to internal radiological dose estimates). This effort may also identify species of interest for which additional data collection may be warranted.

The second component of this study requires identification of adverse effects thresholds—i.e., Site-specific and/or generic from the literature—against which the Trustees will compare contaminant concentrations from the database described above. Building off preliminary work done by the Trustees, and supplemented by additional literature review and/or results of toxicity testing, the Trustees will develop injury thresholds for COPCs and species/species guild of potential concern.

This study will include an evaluation of baseline conditions, which will include to the extent possible a characterization of the concentration ranges of hazardous substances expected to be present in selected Hanford Reach biota but for Hanford Site releases. As part of this evaluation, contaminants will be identified as having one or more of the following origins: natural sources, Hanford Site operations, and/or other anthropogenic sources.

¹²⁷ Chapter 6 provides complete definitions of injury to natural resources. Injury to biological resources can occur when concentrations of hazardous substances exceed action or tolerance levels established under the Food, Drug, and Cosmetic Act (43 CFR § 11.62(f)(1)(ii)), or when concentrations exceed levels for which an appropriate state health agency has issued directives to limit or ban consumption of an organism (43 CFR § 11.62(f)(1)(iii)). However, no such consumption limits or bans have been issued, and for many Hanford Site COPCs, no action or tolerance levels have been established. For these and other reasons, the Trustees expect that this study will focus on comparing COPC tissue concentrations with literature-based adverse effects thresholds, in particular those associated with potential injuries to biota as defined in 43 CFR § 11.62(f)(1)(i)).

AQUATIC BIOTA: REVIEW OF HANFORD SEDIMENT AND PORE WATER TOXICITY STUDIES

Objective: To determine what conclusions may be drawn with respect to injury determination and quantification for sediments and sediment-associated biota based on existing sediment and pore water toxicity testing data.

Need/Rationale: Sediment and pore water toxicity testing are common components of natural resource damage assessments, undertaken to determine the extent to which sediments are injured by virtue of causing injury to other natural resources (see 43 CFR § 11.62(b)(1)(v)). This effort focuses on the toxicity of Hanford Reach sediments to benthic invertebrates. Some contaminants adhere to sediments particularly, and sediment-associated invertebrates are an important part of many freshwater food webs. Reliance on existing information can be a cost-effective way to determine injury, and thus the Trustees propose to evaluate existing testing approaches and results to determine whether available data are of sufficient quantity and quality to meet assessment needs.

Approach: Documentation of reduced survival, growth, reproduction or other adverse effects arising from exposure of biota to hazardous substances in Site sediments relative to reference sediments is an injury under DOI NRD regulations. The benthic community is a key natural resource, forming the base of the aquatic food chain. Sediment toxicity testing has been undertaken in the past at Hanford. For example, DOE (2011b) reports the results of testing of 49 nearshore aquatic sites, and states that 28-day bioassays with *H. azteca* found reduced survival at study sites compared to reference sites, and that *C. dubia* exposed to pore water collected under “low river flow” conditions experienced reduced reproduction compared to reference sites. However, the Trustees have identified limitations associated with these previous studies (see “Invertebrate Sediment Toxicity Testing” below). Therefore, this study will involve a significantly more detailed and rigorous review of available information, documenting, compiling and summarizing these and potentially other studies undertaken at Hanford that evaluated the toxicity of Site sediments to biota. This effort will also include a careful review of these results from an NRD perspective. This work will involve evaluation of test acceptability, assessment of test relevance, and determination of adequacy of spatial coverage. It may also involve re-evaluation of test information using alternate approaches (e.g., alternate statistical analyses), and as appropriate, will result in developing conclusions on the interpretation of existing data in the context of injury determination and quantification for natural resource damage assessments.

BENTHIC INVERTEBRATES: SEDIMENT TOXICITY TESTING

Objective: To evaluate the toxicity of sediments from the Hanford Site to selected benthic invertebrates.

Need/Rationale: This effort will support an injury determination to benthic invertebrates and associated sediments (e.g., see 43 CFR § 11.62(b)(1)(v)), and may inform injury quantification efforts. Measurements of contaminants in the tested sediments will also contribute to the Trustees’ pathway determination for sediments and associated biota. As noted previously, collecting river sediments and subjecting them to toxicity testing using

standardized test organisms is a common component of many natural resource damage assessments. Some COPCs adhere to sediments, and the sediment-associated invertebrates that may be exposed to sediment-associated COPCs are an important part of many freshwater food webs.

The River Corridor Baseline Risk Assessment (RCBRA DOE 2011b) reported the results of some Site-specific toxicity tests with Site media, including sediments and pore waters. However, there are some key limitations of the data presented in DOE 2011b. For example, there are potential concerns with the control data in the 28-day *H. azteca* tests. In sediment toxicity testing using *H. azteca*, the negative control samples should achieve over 80 percent survival (Ingersoll *et al.* 2008) and such criteria are also often applied to reference sediment samples (MacDonald *et al.* 2012); however, Figure 6-35 in DOE (2011b) indicates that at least some reference site samples did not meet this criterion, and, hence, data from certain locations may not be relevant for evaluating sediment toxicity. A closer evaluation may help explore the extent to which this issue may or may not affect a determination about overall test acceptability. In addition, longer tests, such as the 42-day reproduction tests in amphipods, tend to represent a more sensitive endpoint than 28-day tests examining survival and growth. Lastly, the *C. dubia* pore water bioassays exposure duration was limited to seven days, and therefore, potential effects of longer-term exposures are unclear.

The Trustees are interested in conducting additional aquatic invertebrate sediment toxicity testing, designed to ensure appropriate, comprehensive site selection to reflect the diversity of habitat and contamination regimes present, and to use longer-term exposures to more thoroughly explore the potential for chronic effects.

Approach: The specific approach to this study will be defined by the Trustees and the principal investigators in a detailed work plan. However, at this time the Trustees believe that the target organisms could include amphipods (*H. azteca*) in 42-day tests, and midges (*Chironomus dilutis*) in 53 to 60-day tests. Endpoints could include survival, growth, biomass, and reproduction. In addition, it may be desirable to evaluate the viability of F1 (offspring) amphipods and midges produced by the exposed F0 (parental) generation. The results of such tests have, in some cases, supported the development of injury thresholds that are more protective than those based on survival or biomass evaluated in short-term toxicity tests (e.g., 10-day for midge and 28-day for amphipods).

Sediment characteristics, including contaminant concentrations in sediments and pore waters, will also be measured. As part of these efforts, the Trustees will need to select appropriate reference locations from which the baseline condition of sediment resources can be established. Where contaminant concentrations are to be measured, investigators should select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

MUSSELS: DISTRIBUTION, ABUNDANCE, AND HISTOPATHOLOGY

Objectives: To collect information on mussel community health, and to determine whether correlations exist between these metrics and either habitat characteristics or measures of exposure to contaminants.

Need/Rationale: This study results will help the Trustees determine whether native mussels in the Hanford Reach have been injured due to exposure to Site contaminants in accordance with 43 CFR § 11.62(f)(1)(i) and 11.62(f)(3), and the extent of such injury. Measurements of contaminants in the site media will also contribute to the Trustees' determination of exposure pathways to these receptors.

Mussels provide freshwater ecosystems with a wide range of important ecological services. Not only do they serve as a food resource for aquatic and terrestrial predators, they also filter particulate matter from the water column, improving water quality. Their shells provide biogenic habitat, and their nutrient excretion supports the benthic invertebrate community (Spooner and Vaughn 2006). Mussels are also indicators of the ecological health of surface water communities. Their immobile nature (as adults) helps ensure that their status reflects local environmental conditions. In addition, mussels require suitable host fish for parts of their life cycle. The ability of mussels to thrive in a particular area therefore may provide an indirect indication of the status of the host fish community.

The Hanford Reach mussel community has undergone significant change. Mueller *et al.* (2011) evaluated the species, distribution, and densities of native freshwater mussels in the Hanford Reach. Four species of native mussels were identified, of which the western and Oregon floaters (*Anodonta kennerlyi* and *Anodonta oregonensis*) were most abundant. The California floater (*Anodonta californiensis*) was the next most abundant, while the formerly-abundant western pearlshell (*Margaritifera falcata*) appears to have been extirpated. This species has also been in decline regionally (WCH 2008, Appendix F). Potential causes of decline include physical/chemical habitat alterations, thermal stress, availability of host fish, competition with non-native species, and the presence of contaminants. Pauley (1961, 1967, 1968) (as cited in Ingersoll *et al.* 2012) found high levels of pedunculated tumors in *Anodonta* in the Hanford Reach.

Approach: The Trustees will design a study that will examine mussel community characteristics (potentially including abundance, diversity, and age structure) in areas within the Hanford Reach thought to be potentially influenced by contaminant plumes from upwelling groundwater, as well as in reference areas. Semi-quantitative or quantitative sampling methods may be employed. Both live and dead unionids will be targeted for collection. Collected mussels may be subject to histopathological analysis (i.e., to identify lesions, tumors, or other deformities).¹²⁸

¹²⁸ As noted previously, DOE (2011b) reported results of a limited investigation of mussel histopathology, assessing six study sites and three reference sites. The authors found statistically increased observations between study site versus reference site organisms, in two of the 20 measurements: digestive cell vacuolation severity and degraded mantle condition. The Trustees will consider the design and evaluation of the DOE (2011b) histopathological study in more detail as part of

Live animals not retained for histology, contaminant measurements, or for use as voucher specimens, will be returned to their collected location. Sediment and pore water samples will also be collected for purposes of environmental and contaminant characterization, and habitat characteristics will be documented. Where contaminant concentrations are to be measured, investigators should select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

MUSSELS: TOXICITY TESTING

Objectives: The objectives of this study (Ingersoll *et al.* 2012) are to:

- Determine the sensitivity of a native mussel (*M. falcata*) to hexavalent chromium relative to a related commonly tested freshwater mussel surrogate (*Lampsilis siliquoidea*) in water-only exposures;
- Evaluate the sensitivity of *M. falcata* and *L. siliquoidea* to hexavalent chromium in combination with other stressors (uranium, nitrate, and thermal stress) in water-only exposures;
- Determine the concentration of hexavalent chromium in water-only exposures in which these mussels are adversely affected, as defined under DOI's NRDA regulations (see 43 CFR § 11.62(f)(1)(i) and 11.62(f)(4)(i)(E)).

Need/Rationale: This study results will help the Trustees determine whether native mussels in the Hanford Reach have been injured due to exposure to hexavalent chromium alone or in combination with other stressors, and will potentially help the Trustees quantify any identified injury.

As noted above, mussels are sentinels of freshwater community health, and Hanford Site contaminants may have played a role in alterations to this community over the years. Toxicity testing has the potential to identify clear cause-effect linkages between contaminant/stressor exposure and effects. Available information suggests that one site contaminant, hexavalent chromium, can have adverse effects on some freshwater mussels. In particular, the sensitivity of juvenile mussels (*Anodonta imbecillis*) to chromium has been tested and the 96-hour median lethal effect concentration (LC50) was found to be 39 µg/L in relatively soft water and 618 µg/L in relatively hard water (Keller and Zam 1991 as cited in Ingersoll *et al.* 2012).¹²⁹ When combined with mercury, the chromium 48-hour LC50 was lowered from 295 µg/L to 170 µg/L (*ibid.*).

Approach: The first step in this study involves methods development, focusing on the collection and culture of *M. falcata*. If these efforts are sufficiently successful, acute toxicity testing with *M. falcata* and *L. siliquoidea* will proceed, with chromium alone or with chromium along other stressors representative of the Site.

determining whether, and how, to conduct additional mussel histopathological evaluations in the context of this broader mussel study effort.

¹²⁹ "Lethal effect concentration" refers to the concentration of a contaminant associated with fatality in 50 percent of the test organisms.

Should acute toxicity tests demonstrate one or more of the secondary stressors in combination with hexavalent chromium to be synergistic or additive to hexavalent chromium toxicity, chronic toxicity tests with hexavalent chromium and those stressors will then be performed with either *M. falcata* or *L. siliquioidea*. The choice of which of the two mussel species in which to conduct subsequent, chronic studies will be determined based on the success in propagating or conducting toxicity tests with *M. falcata*, which is the preferred species.

MUSSELS: CAGED (IN SITU) STUDY

Objectives: To determine whether *in situ* exposure to the Hanford Reach environment adversely affects the health of unionid mussels.

Need/Rationale: Depending on the results of previous mussel research, the Trustees may pursue this *in situ* study using native and/or surrogate unionids, to support a determination of injury to mussels (e.g., see 43 CFR § 11.62(f)(1) and 11.62(f)(4)(i)(D)). *In situ* studies allow for the exposure of organisms to site conditions, including the physical, chemical, and biological stressors normally present at a site, but do so in a controlled manner that allows for real-time comparisons of effects on selected species of a known life stage and initial condition. Measurements of contaminants in the site media will also contribute to the Trustees' determination of exposure pathways to these receptors. If pursued, this study may support a quantification of injury to mussels by helping identify areas where site conditions are/are not adequate for mussels.

Approach: Native and/or surrogate unionid mussel species may be employed. The selected species will be deployed in appropriate enclosures, to locations within the Hanford Reach thought to be potentially influenced by contaminant plumes from upwelling groundwater. Mussels will also be deployed in reference areas for comparative purposes. Potential endpoints include survival, growth, histopathological condition, and contaminant uptake (i.e., tissue chemistry). Sediment and pore water samples from the study sites will also be collected for purposes of environmental characterization. Habitat characteristics of those sites will be documented.

FISH: CHINOOK SALMON SPAWNING HABITAT EVALUATION

Objective: The purpose of this study is to examine whether contaminants are influencing Chinook salmon (*Onchorhynchus tshawytscha*) spawning habitat selection in the Hanford Reach.

Need/Rationale: Chinook salmon are considered to be injured if their behavior is altered by the presence of contaminants (e.g., see 43 CFR § 11.62(f)(1)). In addition, areas of sediments/groundwater upwelling that are contaminated to the extent that salmon avoid them are also determined to be injured (see 43 CFR § 11.62(b)(v) and 11.62(c)(iv)). It is anticipated that this study will help determine injury to these resources and may also provide information useful in quantifying these injuries (e.g., the size of the affected areas), if present. Measurements of contaminants in the site media will also contribute to the Trustees' determination of exposure pathways to this species.

Chinook salmon are a species of exceptionally high ecological and human use value and are a high priority for the Trustees. Chinook salmon are known to seek out specific types of habitat for purposes of spawning. Redd locations are routinely monitored within the Hanford Reach, and Site-specific models have been developed to identify the characteristics of Chinook salmon spawning habitat in the Hanford Reach (e.g., Geist and Dauble 1998, Geist *et al.* 2000, Geist *et al.* 2006). These models have identified water depth, velocity, substrate, and slope as important discriminators of spawning habitat.

In addition to these habitat characteristics, research has suggested that certain other variables also differed between spawning and no-spawning reaches: in particular, "the permeability, specific discharge, and vertical hydraulic gradient were all higher in [the] spawning reach than in [the] non-spawning reach" of the Columbia River (Geist *et al.* 2006).

Groundwater upwelling, in particular, may influence habitat use by Chinook salmon, and a behavioral change due to contaminants is considered an injury under DOI's regulations (43 CFR § 11.62(f)(1)). For example, Geist (2000) reports that spawning salmon used areas of hyporheic upwelling where the specific conductance indicated a surface water source of the upwelling, whereas they did not use hyporheic discharge zones where the source was groundwater. Dissolved oxygen was higher in the surface water discharge areas, but concentrations in both areas were higher than levels needed for egg/alevin survival. Contaminant concentrations were not measured; however, it is possible that contaminants in upwelling groundwater may be rendering some otherwise suitable spawning habitat undesirable.

Of note, chromium is a known contaminant in Hanford Site groundwater. As noted previously, DeLonay *et al.* (2001) found Chinook parr to be able to detect and avoid water with low concentrations of chromium, and also found that the parr spent less time in waters with higher concentrations of chromium. Laboratory-based avoidance constitutes an injury under DOI's NRDA regulations (43 CFR § 11.62(f)(iii)(B)); of note, however, this effect depended on water hardness. Farag *et al.* (2006a) found exposure of juvenile Chinook salmon to concentrations of 120 µg/l or more were associated with impaired growth, while exposure to concentrations of 266 µg/l were associated with reduced survival.

Approach: Potentially suitable spawning habitat (e.g., as identified in existing models) in the Hanford Reach will be identified. Known redd locations will be compared with these areas of potentially suitable habitat, and from these comparisons, study locations will be selected. The selected locations will include areas of previously-known spawning as well as areas without a known spawning history. Contaminant concentrations in all study areas, including concentrations in upwelling groundwater, will be measured, as will other habitat characteristics thought to be important in salmon habitat spawning site selection. This may involve revising existing habitat use models to determine whether their performance in predicting redd locations is improved when contaminant measurements are included. Where contaminant concentrations are to be measured, investigators should select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that

have been identified as injurious. The Trustees note that prior to implementation of this study, it may be necessary to gather more information, such as generating data to refine existing substrate maps.

FISH: CHINOOK SALMON ARTIFICIAL REDD EVALUATION

After the evaluation of Chinook salmon spawning habitat discussed above, the Trustees may pursue an *in situ* study using artificial redds.

Objective: This study's objective is to ascertain the effect of exposure to contaminants in upwelling groundwater on Chinook salmon eggs and alevins.

Need/Rationale: If salmon do not consistently avoid areas with contamination (e.g., if upwelling of contaminated groundwater is intermittent and does not occur during redd site selection), salmon eggs may be subject to contaminant exposure from sediments and upwelling groundwater, and may be injured from this exposure (e.g., see 43 CFR § 11.62(f)(1) and 11.62(f)(4)(i)(D)). This study will, therefore, also support a determination of injury to salmon, and inform injury and quantification. Measurements of contaminants in the site media will also contribute to the Trustees' determination of exposure pathways to this species.

Of note, the earliest, redd-associated Chinook life stages may not be as sensitive to the effects of chromium as later life stages: Farag *et al.* (2000) and Patton *et al.* (2007) evaluated but did not find effects on these life stages from chromium exposures designed to approximate those in Hanford pore water. However, it is also possible that contaminants may cause effects under (typically more stressful) field conditions, which are not identified under the more controlled conditions of the laboratory.

Approach: Artificial Chinook salmon redds will be constructed at sites possessing characteristics thought to be favorable as spawning habitat. These sites should include areas with recent spawning activity as well as areas without known recent spawning activity. Areas of suspected groundwater upwelling will be specifically targeted. Fertilized eggs will be placed in Vibert boxes within the artificial redds. Redds will be monitored for endpoints including but not necessarily limited to hatching success, fry survival, and growth. Non-contaminant related habitat characteristics will be documented, as will measures of contaminant exposure, and upwelling of surface or groundwater.

FISH: PRICKLY SCULPIN HABITAT USE

Objective: To estimate the relative abundance, density, and age structure of sculpin, in areas exposed to contaminated groundwater compared to reference locations.

Need/Rationale: This study will support an injury determination for sculpin, a forage fish occupying a ecological niche distinctly different from those occupied by other species proposed for evaluation in this assessment plan, and will provide data that will inform injury quantification in accordance with 43 CFR § 11.62(f)(1)(i) and 11.62(f)(3). Measurements of contaminants in the site media will also contribute to the Trustees' determination of exposure pathways to this species.

The prickly sculpin is a suitable fish to study in part because it can serve as a surrogate for other species of conservation concern. For example, the mottled sculpin (*Cottus*

bendirei) is a Federal species of concern and is listed on Oregon Biodiversity Information Center's list of rare, threatened, and endangered species of Oregon (Kagan and Christy 2010). In addition, sculpins have been used as indicators of stream health (Besser *et al.* 2007, Yearley 2000). Sculpins are bottom dwellers and typically remain close to the substrate (Brown 2005). Adult sculpin build nests of eggs on the underside of rocks in the fast-moving streams in which they live. Once the eggs hatch, the fry drop to the bottom of the nest (Brown 2005). At this time, the fry still have a yolk sac and are about five mm long. The adult male sculpin tending the nest continues to fan the fry, aerating the eggs and keeping them free of silt, until the yolk sacs are absorbed, about two weeks after hatching (Brown 2005). The fry then disperse and grow into juveniles. Consequently, both the adults and early life stage fish have the potential for significant exposure to contaminants in sediments or in upwelling groundwater.

Some studies have found sculpin to move tens of meters or less over the course of a month or more (Petty and Grossman 2004, Petty and Grossman 2010). As a small fish with a limited home range, sculpin are likely to be exposed to COPCs for greater periods of time and will reflect the local conditions more precisely than species with larger ranges (Besser *et al.* 2007, Van Verst *et al.* 1998). Sculpin can be used to demonstrate the worst-case exposure for fish in a given area and can be used to estimate exposure to fish-eating biota (Van Verst *et al.* 1998). Sculpins have also been reported to be more sensitive to certain metals than are salmonids and other larger fish (Besser *et al.* 2007).

Some information on Hanford Reach sculpin has been collected: The River Corridor Baseline Risk Assessment (DOE 2011b) reports the collection, through electrofishing, of sculpin in nearshore fine sediments and gravel-pebble substrate areas. Sculpin were caught at 26 locations thought to be affected by contaminated groundwater plumes and seven areas thought to be unaffected by site contamination. These fish were subject to disease and histopathological evaluations¹³⁰ as well as contaminant analyses (liver and kidneys), weight, and length measurements. The authors found four out of 22 measurements to differ between study and reference sites: the number of liver parasites and the number of muscle granulomas was higher among site fish, and the number of encysted parasites in gills and kidneys were higher among reference fish.

Approach: Potentially suitable sculpin habitat will be identified. These areas are expected to be nearshore, as sculpin are often (but not always) found at depths of less than 0.5 meters (Hendricks 1997, Becker 1983). Electrofishing will be used to capture fish. Sculpin will be quickly identified to species, measured and weighed. Habitat information will also be documented, including measurements of prey availability as research has suggested this to be an important factor affecting sculpin presence (Petty and Grossman 1996), as will contaminant concentrations in site media. The Trustees may choose to use a mark-recapture model to estimate population size. The Trustees may also elect to phase this study to better understand the ability of the study to achieve its objectives, prior to deciding to proceed with a full-scale implementation effort.

¹³⁰ Disease and physiological deformations are injuries in accordance with 43 CFR § 11.62(f)(1)(i); histopathological effects are specifically noted as an injury in 43 CFR § 11.62(f)(vi).

FISH: EARLY LIFE STAGE SCULPIN, WHITE STURGEON, AND RAINBOW TROUT TOXICITY TESTING

Objectives: To determine the sensitivities of a representative sculpin species, the white sturgeon, and rainbow trout to waterborne site contaminants including chromium, both alone and in combination with other stressors (e.g., uranium and nitrate).

Need/Rationale: This study will help evaluate the extent to which chromium, and potentially other stressors associated with the Hanford Site, may injure sculpin and/or sturgeon in accordance with 43 CFR § 11.62(f)(1)(i) and 11.62(f)(4)(i)(E). As a laboratory study, it is suited towards identifying the causality of potential injuries.

As noted previously, adult and early life stage sculpin live in close proximity to sediments. Early life stages of fish are frequently among the most sensitive to contaminant exposure, and in the Hanford Reach, chromium or other contaminants in upwelling groundwater may be reaching areas where sculpin spawn. In addition, sculpin have been reported as being more sensitive to certain metals than are salmonids and other larger fish, and have been extirpated from some streams due to elevated metal concentrations (Besser *et al.* 2007, Kunz *et al.* 2005, Dorts *et al.* 2010, Allert *et al.* 2005).

The white sturgeon's life cycle also puts eggs and larval stages in close association with sediments: after fertilization, eggs remain attached to the substrate for approximately seven to 11 days before hatching, dependent upon water temperature (UCWSRI 2002, Wydoski and Whitney 2003). Hatched larvae leave the substrate during a swim-up phase, which lasts approximately five to six days, during which time they disperse. After dispersal, larvae seek shelter in substrate and remain hidden for approximately 20 to 25 days until their yolk-sac is absorbed. Upon absorption of their yolk-sac, young white sturgeon emerge from the substrate to seek food (UCWSRI 2002). During these early life stages, fertilized sturgeon eggs and larvae may be exposed to contaminants in upwelling groundwater.

Rainbow trout are the same species as the anadromous steelhead trout, a fish of environmental and human use importance that spawns in the Hanford Reach (Jamison 1982). Early life stages, including eggs and larvae, are closely associated with sediments. Fry emerge from redds two to three weeks after hatching. Stevens and Chapman (1984) found significantly reduced survival to hatch at 89 µg/L of trivalent chromium. Subadult rainbow trout were found to have an avoidance threshold for chromium of 28 µg/L (Anestis and Neufeld 1986 as cited in DeLonay *et al.* 2001), although avoidance thresholds increased linearly with levels of pre-exposure. Measured concentrations of hexavalent chromium in Hanford Reach pore water have ranged from nondetectable to 632 µg/L (Hope and Peterson 1996).

Approach: Standard methods are available for sculpin, white sturgeon, and rainbow trout toxicity testing. Sculpin will be field-collected, and adults spawned in the laboratory to provide embryos and/or fry for use in toxicity testing. Standard methods (e.g., ASTM E1241) will be used to conduct toxicity tests. For white sturgeon, fertilized eggs can be obtained from hatchery sources, and alevins and fry can be used in standard toxicity tests. Rainbow trout eggs and juveniles can also be obtained from hatchery sources. Toxicity

testing will include exposure to chromium alone or with chromium along other stressors representative of the Site. Chronic exposure tests may be preferred, as these are considered to more closely reflect field conditions.

AQUATIC RESOURCES: QUANTIFICATION OF LOST AQUATIC ECOLOGICAL SERVICES

Objective: The objective of this study is to quantify the ecological services that have been lost as a result of injury to aquatic resources in the past and potential loss of ecological services in the future as a result of Site-related contamination.

Need/Rationale: In order to determine the scale and type of restoration actions required to compensate the public, the Hanford Trustees will need to understand the scale and scope of lost services.

Approach: This study involves two phases. The first phase consists of compiling information obtained from the aquatic resource studies mentioned above. This information will likely include the degree to which sample concentrations exceed identified injury thresholds, toxicity information on the adverse effects of varying levels of contamination, as well as ecological information (e.g., the abundance or distribution of aquatic species, habitat usage by species of concern). The second phase consists of analyzing the compiled data in order to quantify the geographic and temporal scope of lost ecological services in the past and potential losses in the future due to Site-related contamination. This will involve developing a relationship between contaminant concentrations and the severity of corresponding adverse effects on aquatic resources. The relationship will likely be based on information published in the literature, and data from site-specific studies on the toxicity of contaminants of concern, as well as information on habitat usage, species abundance, and species diversity. Site-specific contaminant concentrations will then be compared to the developed relationship in order to determine the extent to which Site aquatic resources have been injured (i.e., determine the estimated service loss).

7.3 TERRESTRIAL RESOURCES

The Hanford Site has a lengthy operational and remedial history, and as part of that history, a number of ecological, toxicological, and other studies have provided information of potential use in the injury assessment. The studies included in this Injury Assessment Plan build on available information from past efforts and are intended to address key data gaps and/or remaining uncertainties. The following paragraphs briefly summarize key data that have resulted from past investigations of the Site's terrestrial resources and are intended to generally characterize the larger research context into which the proposed studies will fit.

OVERVIEW OF EXISTING SITE TERRESTRIAL RESOURCE DATA

Available information about the Hanford Site's terrestrial natural resources that is of most relevance to a natural resource damage assessment includes but is not limited to: (a) measurements of hazardous substances in soils and in the tissues of terrestrial organisms, (b) information about species presence/absence at various locations; (c) results of toxicity testing of specific biota with site media and site contaminants, (d) population and

community investigations, and (e) other research exploring the potential for contaminant-related effects at the Site (e.g., reproductive studies, histopathological evaluations, biota condition assessments, behavioral assessments, etc.).

Measurements of Hazardous Substances

As noted previously, the Trustees have identified at least seven partially overlapping databases that contain many measurements of concentrations of hazardous substances in site media and biotic tissues. The Hanford Environmental Information Systems (HEIS) database contains the largest numbers of samples of soils and biota. HEIS continues to be developed, and HEIS may eventually serve as the repository for virtually all site sampling efforts, past and ongoing. A substantial effort has been underway within this past year to add more data to HEIS; as this effort progresses, it may become increasingly less necessary to rely on other compilations of contaminant information. In addition to HEIS, databases with information on terrestrial natural resources include: (a) the Columbia River Component historic database, (b) the Columbia River Component Data Summary Report for the Remedial Investigation of Hanford Site Releases to the Columbia River (WCH 2011), and (c) the River Corridor Baseline Risk Assessment GiSdT database.

A review of the entries in these databases suggests that, of the non-domestic terrestrial biota, mammals (e.g., mule deer, cottontail rabbit, black-tailed jackrabbit, mouse species) have been the most frequently sampled. Information on contaminant concentrations in wild terrestrial birds appears to be particularly sparse (recognizing that some measurements are available for pheasant and quail). The limited availability of exposure information on a broader range of wild avian species is a key factor behind the Trustees' inclusion of the study "Birds: Evaluation of Exposure to Hanford Site Avian Species."

The number of measurements of contaminants in site soils is large; however, many challenges remain in effectively using these data, as well as the terrestrial biota data, in the context of a natural resource damage assessment. Challenges include but are not limited to: the variety of sampling efforts (and associated sampling objectives) associated with the datasets; the need to understand quality assurance issues associated with the various datasets; analytic issues associated with non-detect values; and the absence of sample characterization information in many cases (e.g., sampling depths and geographic coordinates). Studies that rely on this information will need to address these issues during the detailed study design and implementation stages.

Species Distribution and Population/Community Characterization Information

In a natural resource damage assessment, Trustees may choose to evaluate species distributions and population or community metrics to evaluate the extent to which hazardous substances may have affected biota at these levels of ecological organization.

Some information on these topics is available: for example, the River Corridor Baseline Risk Assessment (DOE 2011b) reports community assessment results for terrestrial vegetation and for small mammals. While these data are potentially useful for assessment purposes, preliminary Trustee review has identified important limitations associated with these efforts. For example, the upland plant community comparisons are limited to remediated areas and reference sites; furthermore, site selection was intentionally biased

towards sites with an established vegetative community (to ensure an adequate sample collection for contaminant analysis purposes). The scope of a natural resource damage assessment is not limited to remediated locations or to areas where recovery may be better. Community evaluations of unremediated locations, without a bias towards higher ecological quality sites, is important so that Trustees can better understand the extent to which Hanford contaminants in site soils may be affecting or may have affected terrestrial communities.

The RCBRA's small mammal community results also warrant careful scrutiny. This study's primary objectives did not encompass characterizing small mammal community parameters in detail. Only a single campaign's worth of data were collected, which—as recognized by DOE (2011b)—significantly limits the study's ability to characterize population or community attributes. For more discussion on these topics, see “Plants: Assessment of Plant Community Health” and “Mammals: Small Mammal Population Assessment”).

DOE's Ecological Monitoring and Compliance Project (EMC) has also included the collection information on terrestrial species. Until 2011, the EMC Project was managed by Pacific Northwest National Laboratory (PNNL) and now is managed by Mission Support Alliance (MSA). The collected information primarily includes observations of species locations and dates; it includes but is not limited to species such as elk, deer, eagles, sage sparrows, and raptors (e.g., nest locations). For purposes of natural resource damage assessment, this information may be useful in identifying likely locations for biota in the event that future field studies on these species are pursued, but it is not likely to be useful for direct injury determination purposes as the program has not been designed to definitively identify species absence, or to quantify population-level metrics such as abundance.

Other authors have also developed and/or compiled general information on terrestrial species presence at the Hanford Site (e.g., Fitzner and Gray 1991, Downs *et al.* 1993, TNC 1999, Sackschewsky and Downs 2001, and Duncan 2007).

Considering the needs of the injury assessment and limitations on available information, this assessment plan includes studies such as “Invertebrates: Assessment of Terrestrial Invertebrate Abundance”, “Birds: Assessment of Avian Abundance and Diversity”, and “Mammals: Small Mammal Population Assessment,” which are intended to help fill data gaps with respect to terrestrial species population/community characteristics at Hanford.

Toxicity Testing

Trustees frequently include toxicity testing among site assessment activities. Some such testing has been conducted with site media. For example, the River Corridor Baseline Risk Assessment (DOE 2011b) presents the results of soil toxicity tests on Sandberg's bluegrass and on the nematode, *C. elegans*. The results of these efforts provide information that may be valuable in the context of a natural resource damage assessment; however, preliminary review of the approach and results suggests that they also are subject to the same types of limitations as noted above for the RCBRA vegetative and small mammal community data, most especially their focus on testing soils from remediated locations and from areas with healthier vegetative communities.

Altogether, the Trustees plan to undertake additional review of the RCBRA's toxicity testing results and may pursue additional toxicity testing of site media, as described in "Terrestrial Biota: Review of Hanford Toxicity Studies", "Invertebrates: Nematode Toxicity Testing," and "Plants: Native Plant Toxicity Testing" below.

Histopathological Investigations

The Trustees may examine organisms for evidence of physiological injuries including (but not limited to) histopathological impacts. Site-specific information on histopathology of terrestrial species appears to be limited. One such study is an assessment of adult male mule deer reproductive health. In particular, in response to observations of adult male deer with atypical antlers, Tiller *et al.* (1997) conducted research that found these deer to have infertile, atrophied testicles. The authors stated that radiation, natural aging, infectious agents, and genetics were ruled out as causes, while other stressors including heavy metals, herbicides/pesticides/insecticides were unlikely to be causative agents. Plant and fungal toxins were not evaluated.

The study "Mammals: Great Basin Pocket Mouse – Carbon Tetrachloride and Histopathology" is intended to provide histopathological data on a species that, has a burrowing mammal, has a very different life history than the mule deer, and that may be particularly exposed to carbon tetrachloride in Hanford Site soils.

Additional Investigations

DOE (2011b) evaluated reproduction in cliff swallows, eastern kingbirds, and western kingbirds, but the authors note that predation was sufficiently high as to render interpretation impossible.

In the future, the Trustees may choose to pursue additional avian assessment studies; however, to inform any such potential future research, the Trustees intend to first complete the study "Birds: Evaluation of Exposure to Hanford Site Avian Species."

IMPLICATIONS OF EXISTING DATA FOR INJURY ASSESSMENT

Given the description above of available information on contaminant exposure and potential terrestrial injuries, the following injury assessment studies have been identified to fill important data gaps. Phase 1 priorities for terrestrial injury assessment focus on organizing the information necessary to better understand aquatic resource exposure and to help guide work plan development for later stages of the injury assessment. Phase 1 priorities therefore include estimating the level and extent of soil and terrestrial biota tissue contamination, estimating baseline contaminant concentrations in soils and biotic tissues, conducting a geostatistical spatial analysis of soil data contaminant concentrations, reviewing the existing soil ecotoxicity testing studies, and assessing the impacts of site remedial activities. Phase 2 and 3 priorities encompass further efforts that would help the Trustees refine their understanding of potential terrestrial injuries. In particular, Phase 2 and 3 efforts include: conducting additional laboratory toxicity testing, gathering information about terrestrial population and community attributes, gathering additional exposure data where gaps are evident, and collecting information on the health of terrestrial biota.

SOILS: COMPARISON TO EFFECTS THRESHOLDS

Objective: (1) To determine potential past, current, and future injuries to soil resources and terrestrial biota based on comparisons of measured soil COPC concentrations to literature-based effects thresholds. (2) To identify COPCs that may be most strongly associated with potential injuries (e.g., by virtue of having a greater magnitude and/or exceedance of effects thresholds). (3) To identify locations with higher or lower levels of exposure to hazardous substances, to help inform site selection in potential future studies.

Need/Rationale: Soils are a key natural resource, providing habitat to numerous terrestrial species. Comparison of contaminant concentrations in soils to appropriate adverse impact thresholds is a cost-effective approach commonly undertaken to evaluate the likelihood and potential severity of injury to soils. While comparisons of measured concentrations in soils to thresholds is not, in itself, sufficient to determine and quantify injury in accordance with the DOI regulations, such analyses inform the Trustees' understanding of the nature and extent of potential injury. Within the context of a cooperative assessment, these kinds of comparisons can provide a basis for reaching agreement on injury determination and/or quantification assumptions. These studies can also help document the existence of a pathway between sources of releases and receptors, and/or may suggest that additional field or lab studies on certain biological receptors/locations/contaminant combinations may be appropriate.

Approach: The study will focus on the Hanford Site and appropriate reference locations. The first component of this task will involve assembling and evaluating available data, and incorporating it into the Trustees' natural resource damage assessment database in accordance with the Data Management Plan and the Quality Assurance Management Plan (HNRTC 2011a, 2011b). Although data on soil concentrations exist, a comprehensive database is not currently available.

The second component of this study requires identification of adverse effects thresholds—i.e., Site-specific and/or generic values from the literature, against which the Trustees will compare contaminant concentrations from the database described above. Building off the preliminary work done by the Trustees, and supplemented by additional literature and/or the results of toxicity testing, the Trustees will develop injury thresholds for each COPC.

This study will include an evaluation of baseline conditions, which will include to the extent possible a characterization of the concentration ranges of hazardous substances expected to be present in Hanford Site soils but for Hanford Site releases. As part of this evaluation, contaminants will be identified as having one or more of the following origins: natural sources, Hanford Site operations, and/or other anthropogenic sources.

SOILS: GEOSPATIAL EVALUATION

Objectives: (1) To identify which surficial soils of the Hanford Site are either more or less likely to have been exposed to potentially injurious contaminant concentrations, and (2) to identify areas where additional soil sampling may be necessary to adequately characterize surficial soil contamination for natural resource damage assessment purposes.

Need/Rationale: The Trustees are concerned that available documentation of releases of hazardous substances associated with Site operations may not be complete. They are specifically concerned about the potential for past aerial emissions to have resulted in the contamination of surficial soils, which may in turn expose biota. The Trustees wish to better understand how comprehensive available information is with respect to surficial soil contaminant concentration measurements, and to evaluate whether—considering typical wind patterns, for example—the spatial extent of sampling is sufficient to have likely identified areas of concern from an assessment perspective. This study will also contribute to the Trustees’ determination of exposure pathways to soils.

Approach: The Trustees will work closely with a geostatistician and potentially with additional experts to evaluate available surficial soil contaminant concentration data. The exact approach to be used will be selected by the principal investigator(s) in close coordination with the Trustees, but may include:

- Exploratory analyses of available soil data for visual evaluation of spatial patterns, as well as confirmation of known and potential source locations;
- Global and local, directional and omni-directional variogram analyses of selected soil data for determining spatial correlations along specific directions of interest, such as those aligned with dominant wind directions;
- Estimation techniques designed to identify “hot spots” (i.e., contiguous areas with expected contaminant concentrations in excess of specific thresholds based on selected tolerable errors and/or confidence), such as areas with sparse data situated downwind of dominant wind directions, as confirmed by directional variograms as well as contiguous areas with expected contaminant concentrations below specific thresholds, but upper confidence concentrations in excess of specific thresholds based on selected tolerable errors and/or confidence.

Any “hot spot” areas, if identified, might be reasonable sites to target in field studies of terrestrial biota. Similarly, locations where soil sampling data are sparse but where typical wind patterns, as confirmed by directional variograms, suggest that aeolian transportation may have been more likely, could be identified as priority areas for additional soil sampling, to ensure that significant areas of potential terrestrial contaminant exposure and injury are not overlooked.

The study will mainly focus on surficial soils for two reasons: first, because surficial soil concentrations will drive exposures for most terrestrial biota, and second, because aerially deposited contaminants are more likely to be present in the more surficial strata. Of note, however, the need to categorize soil samples by depth may present a technical challenge. In the largest two Hanford Site soil databases identified (i.e., HEIS and GiSdT), sampling depth information is not specified for roughly 80 percent of the soil samples. It may be possible to determine approximate depths of samples through use of sampling method information and/or coordination with the entities responsible for the original sample collection. The appropriateness and reliability of such approximations would be evaluated during the exploratory and variogram analyses of investigated soil data.

Finally, the Trustees recognize that this effort will be informed by an understanding of the locations and general types of known aerial contaminant releases: this knowledge may suggest that specific analysis of the spatial patterns of particular contaminants in particular areas should be prioritized, e.g., given priority to directional variogram analyses of soil data in certain parts of the Site. However, the focus of this analysis will be on drawing conclusions based on available measurements of hazardous substances in surficial soils, rather than reconstructing the history of the Site's aerial emissions.

The Trustees note that, as a result of this effort, additional sampling of soils and/or associated biota may be warranted. For example, the results of this effort in combination with results from the "Current Resource Characterization to Allow for Restoration of Lost Tribal Services" may suggest that sampling of plants used by Tribes for food or medicinal purposes is needed.

TERRESTRIAL BIOTA: COMPARISON WITH INJURY THRESHOLDS - TISSUES

Objectives: (1) To determine potential past, current, and future injuries to terrestrial biota based on comparisons of measured tissue concentrations of COPCs to literature-based effects thresholds. (2) To identify COPCs that may be most strongly associated with potential biotic injuries (e.g., by virtue of having a greater magnitude and/or exceedance of effects thresholds). (3) To identify species and/or locations with higher or lower levels of exposure to hazardous substances, to help inform site selection in potential future field studies of aquatic biota.

Need/Rationale: Biologic resources, including terrestrial organisms, are trust resources that provide a suite of essential ecological services. Comparison of COPC tissue concentrations to appropriate adverse impact thresholds is a cost-effective, widely used approach to identify potential biological injuries. While comparisons of measured concentrations in tissues to thresholds is not, in itself, sufficient to determine and quantify injury in accordance with the DOI regulations,¹³¹ such analyses can inform the Trustees' understanding of the nature and extent of potential injury. Within the context of a cooperative assessment, these kinds of comparisons can provide a basis for reaching agreement on injury determination and/or quantification assumptions. These studies can also help document the existence of a pathway between sources of releases and receptors, and/or may suggest that additional field or lab studies on certain biological receptors/locations/contaminant combinations may be appropriate. They may also provide help identify those COPCs that may be the largest drivers of injury (e.g., based on the magnitude and/or extent of threshold exceedances).

Approach: This study will focus on the Hanford Site and appropriate reference areas. The first component of this task will involve assembling and evaluating available data,

¹³¹ Chapter 6 provides complete definitions of injury to natural resources. Injury to biological resources can occur when concentrations of hazardous substances exceed action or tolerance levels established under the Food, Drug, and Cosmetic Act (43 CFR § 11.62(ii)), or when concentrations exceed levels for which an appropriate state health agency has issued directives to limit or ban consumption of an organism (43 CFR § 11.52(iii)). However, no such consumption limits or bans have been issued, and for many Hanford Site COPCs, no action or tolerance levels have been established. For these and other reasons, the Trustees expect that this study will focus on comparing COPC tissue concentrations with literature-based adverse effects thresholds, in particular those associated with potential injuries to biota as defined in 43 CFR § 11.62(f)(i)).

and incorporating it into the Trustees' natural resource damage assessment database in accordance with the Data Management Plan and the Quality Management Plan (HNRTC 2011a, 2011b). Although data on contaminant concentrations in biota exist, a comprehensive database is not currently available. The Trustees are presently creating a database, ensuring that data are normalized, contain location information where possible, and are presented in consistent units (e.g., convert radiological concentrations to internal radiological dose estimates). This effort may also identify species of interest for which additional data collection may be warranted.

The second component of this study requires identification of adverse effects thresholds, Site-specific and/or generic from the literature, against which the Trustees will compare contaminant concentrations from the database described above. Building off the preliminary work done by the Trustees, and supplemented by additional literature and/or results of toxicity testing, the Trustees will develop injury thresholds for COPCs and species/species guild of potential concern.

This study will include an evaluation of baseline conditions, which will include to the extent possible a characterization of the concentration ranges of hazardous substances expected to be present in selected Hanford Site biota but for Hanford Site releases. As part of this evaluation, contaminants will be identified as having one or more of the following origins: natural sources, Hanford Site operations, and/or other anthropogenic sources.

TERRESTRIAL BIOTA: REVIEW OF HANFORD SOIL TOXICITY STUDIES

Objective: To determine what conclusions may be drawn with respect to injury determination and quantification for terrestrial biota, based on existing toxicity testing data.

Need/Rationale: Soil toxicity testing is a common component of natural resource damage assessments, undertaken to determine the extent to which soils are injured by virtue of causing injury to other natural resources (see 43 CFR § 11.62(e)(11)). This effort focuses on the toxicity of Site-specific terrestrial biota. Reliance on existing information can be a cost-effective way to determine injury, and Trustees are well-served to evaluate existing testing approaches and results to determine whether available data are of sufficient quantity and quality to meet assessment needs.

Approach: Documentation of reduced survival, growth, reproduction or other adverse effects arising from exposure of vegetation and/or other biota to hazardous substances in Site soils relative to reference soils is an injury under DOI NRD regulations. Terrestrial soils are a key natural resource, providing habitat for plants and invertebrates that form the base of the terrestrial food chain.

Soil toxicity testing has been undertaken at Hanford. For example (as summarized in Exhibit 7-2), DOE (2011b) reports the results of testing of Sandberg's bluegrass to determine whether remediated waste sites presented an ecological risk to the growth and development of this native grass species. DOE (2011b) also reports the results of 24-hour toxicity tests examining the effects of remediated site and reference soils on the

nematode, *C. elegans*. In both sets of experiments, study sites were selected from amongst 85 remediated waste sites documented to have been cleaned up to Interim Record of Decision requirements, representing a mix of minimally disturbed sites as well as highly disturbed sites remediated with backfill.

Some preliminarily identified limitations on these efforts are described below (in “Plants: Native Plant Toxicity Testing” and “Invertebrates: Nematode Toxicity Testing”). This study will involve a significantly more detailed and rigorous review of available information, documenting, compiling and summarizing these and potentially other studies undertaken at Hanford that evaluated the toxicity of Site sediments to biota.

This study will compile and summarize studies undertaken at Hanford that evaluated the toxicity of Site soils to biota. The results of these studies will be carefully reviewed from an NRD perspective. This work should involve evaluation of test acceptability, assessment of test relevance, and determination of adequacy of spatial coverage. It may also involve re-evaluation of test information using alternate approaches (e.g., alternate statistical analyses). The results of this analysis will provide a basis for recommending additional studies that will fill critical data gaps.

PLANTS: NATIVE PLANT TOXICITY TESTING

Objective: To evaluate the toxicity of soils from the Hanford Site to key native plant species.

Need/Rationale: As noted previously, collecting site media and subjecting them to toxicity testing using standardized test organisms is a common component of many natural resource damage assessments. To the extent soil toxicity is shown to exist in such testing, it provides evidence supporting an injury determination to both plants and site soils (e.g., see 43 CFR § 11.62(f)(1)(i), 11.62(f)(4)(i)(E), and 11.62(e)(11)). Measurements of contaminants in the tested soils will also contribute to the Trustees’ determination of exposure pathways. Study results may inform injury quantification efforts as well.

Healthy plants and plant communities are a critical requirement for proper ecosystem function. Plants are the base upon which the terrestrial food web is structured. Injury to the foundation of the food web can disrupt the interactions between all subsequent trophic levels, fundamentally changing the dynamics of the ecosystem. Thus, the health of the ecosystem as a whole is closely tied to the health of the vegetative community. Plants also serve other important ecosystem functions as nesting habitat and cover, which many other terrestrial species depend upon for survival and reproduction.

Plants are subject to contaminant exposure both through direct contact and uptake or absorption of soil-bound contaminants, as well as through exposure to radiation emitted by contaminated soil. Exposure to contaminants can affect germination, growth, and other endpoints. Standard toxicity tests have been developed and widely used to identify how site-specific contaminated media affects these endpoints as compared to media collected from reference sites and/or artificial media.

The RCBRA (DOE 2011b) includes results of toxicity testing of Sandberg's bluegrass (*Poa secunda*, a native species) to selected site soils. This effort falls short of meeting Trustee assessment needs for several reasons. First, testing in upland areas was limited to seven upland *remediated* waste sites plus three reference sites;¹³² however, the scope of a natural resource damage assessment is not limited to remediated locations. For assessment purposes, toxicity testing of unremediated locations is important so that Trustees can better understand the extent to which Hanford contaminants in site soils may be affecting terrestrial plants. Evaluating unremediated areas may also inform Trustees about possible past impacts to vegetative communities at sites prior to their remediation.

The RCBRA's site selection method further reduces the utility of the bluegrass toxicity testing results for a natural resource damage assessment. Specifically, the selection of upland sites was intentionally biased towards areas of good ecological recovery –i.e., areas with an established vegetative community. This bias was intended to ensure adequate vegetative sample collection for contaminant analysis. However, this study design choice makes it impossible to fully understand the extent to which Hanford contaminants in site soils may be affecting native plants: areas with poorer recovery may have soils with greater phytotoxicity but were not tested.

Finally, and independent of the previous considerations, the number of sites evaluated is small, particularly given the large number and disparate history of waste sites and contamination regimes at Hanford. Overall, the scale of the study effort may not be sufficiently comprehensive to adequately characterize the potential phytotoxicity of Hanford vegetation to site soils. For all these reasons, the Trustees believe that additional soil toxicity testing of vegetation is warranted.

Approach: After evaluating available toxicity test data generated and collected for the Hanford Site Risk Assessments from an NRD perspective, additional toxicity tests may be warranted. Ideally, this study could include a diversity of sites representing off-site control sites, remediated sites, and those where cleanup actions have not yet been implemented. The sites should also represent the likely range of contaminant conditions and mixes to which flora have been exposed over the years.

Study elements are expected to include the identification of test species, the selection of test media (soil) across a range of contaminant concentrations, the use of appropriate control growth media, and the measurement of endpoints. Endpoints may include seedling survival, seedling height, tissue chlorosis and necrosis, numbers of leaves, above- and below-ground biomass, and photosynthetic activity, among others. Soil properties will also be measured. Where contaminant concentrations are to be measured, investigators will take care to select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

¹³² Sandberg's bluegrass toxicity testing also included soils from eight riparian "study" sites and eight "rare plant" sites, as well as three riparian reference sites (DOE 2011b).

PLANTS: ASSESSMENT OF PLANT COMMUNITY HEALTH

Objective: To evaluate the health of the plant communities across the Hanford Site in comparison to suitable reference locations.

Need/Rationale: This study will support an injury determination to Hanford Site plants and may inform injury quantification efforts to this community in accordance with 43 CFR § 11.62(f)(1)(i) and 11.62(f)(3). Measurements of contaminants in site soils and plant tissues will also contribute to the Trustees' pathway determination for these natural resources.

As noted previously, healthy plants and plant communities are a critical requirement for proper ecosystem function. Toxic substances have the potential to reduce cover and to cause changes in plant community structures. Contamination can result in significant changes to the composition and health of plant communities.

The RCBRA (DOE 2011b) included a plant cover and diversity survey; however, this effort falls short of meeting Trustee assessment needs for several reasons.

First, RCBRA community evaluations in upland areas focused on 20 *remediated* sites and 10 reference sites;¹³³ however the scope of a natural resource damage assessment is not limited to remediated locations. For assessment purposes, Trustees are interested in understanding the extent to which Hanford contaminants in site soils may be affecting terrestrial plant communities. Evaluating unremediated areas may also inform Trustees about possible past impacts to vegetative communities at sites prior to their remediation.

The RCBRA's site selection method further reduces the utility of the vegetative community testing results for a natural resource damage assessment. Specifically, the selection of upland sites was intentionally biased towards areas of good ecological recovery –i.e., areas with an established vegetative community. This bias was intended to ensure adequate vegetative sample collection for contaminant analysis. However, this study design choice makes it impossible to fully understand the extent to which Hanford contaminants in site soils may be affecting native plants: areas with poorer recovery may have soils with greater were excluded from the study.

Finally, and independent of the previous considerations, the number of sites evaluated is modest, particularly given the large number and disparate history of waste sites and contamination regimes at Hanford. Overall, the scale of the study effort may not be sufficiently comprehensive to adequately characterize the potential phytotoxicity of Hanford vegetation to site soils. For all these reasons, the Trustees believe that additional soil toxicity testing of vegetation is warranted.

Approach: The Principal Investigator(s) responsible for detailed study design will consider in detail the results of the DOE (2011b) plant community work as well as other relevant information as part of developing a carefully-designed survey of plant community health. The study will provide information relevant to evaluating the extent

¹³³ Plant communities in riparian areas were also assessed, at eight "study" sites, eight "rare plant" sites, and three reference sites.

to which plant communities may have been affected by contaminant releases from Hanford. The Trustees will measure the occurrence, composition, and density of plant cover at (and near) an appropriate number of operational and other contaminated areas, and in suitable reference areas. Samples may be gathered to measure contaminant concentrations in tissues and soils, and/or for genetic analysis to evaluate measures of genetic damage consistent with radiation exposure. Soil properties will also be measured.

Ideally, the selected sites should also represent the likely range of contaminant conditions and mixes to which flora have been exposed over the years. Where contaminant concentrations are to be measured, investigators should select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

INVERTEBRATES: NEMATODE TOXICITY TESTING

Objectives: To evaluate the toxicity of soils from the Hanford Site to selected nematodes.

Need/Rationale: As noted previously, collecting site media and subjecting them to toxicity testing using standardized test organisms is a common component of many natural resource damage assessments. To the extent soil toxicity is shown to exist, it provides evidence supporting an injury determination to both nematodes and site soils (e.g., see 43 CFR § 11.62(f)(1)(i), 11.62(f)(4)(i)(E), and 11.62(e)(11)). Measurements of contaminants in site soils will also contribute to the Trustees' pathway determination for soils and soil-associated biota. Study results may inform injury quantification efforts as well.

The nematode is a ubiquitous roundworm that lends itself well to soil toxicity testing, and a standard toxicity test, ASTM E2172-01, is widely used for expressly this purpose. The availability of information on this organism, of a standard test for toxicity, and ease of study make the nematode an ideal potential soil toxicity test organism.

DOE (2011b) includes results of toxicity testing of the nematode *C. elegans* to selected site soils. DOE (2011b) did not identify statistically significant differences in survival between nematodes exposed to upland study site soils and those exposed to reference site soils; however, the tested upland sites were limited to 20 remediated waste sites and 10 reference sites.¹³⁴ Importantly, the scope of a natural resource damage assessment is not limited to remediated locations. Toxicity testing of unremediated locations is important if Trustees are to understand the extent to which terrestrial invertebrates may be (or may have been) affected by the Site's releases of hazardous substances.

Approach: After evaluating available toxicity test data from an NRD perspective, as recommended previously, additional toxicity tests may be warranted. Ideally, this study could include a diversity of sites representing off-site control sites, remediated sites, and those where cleanup actions have not yet been implemented. The selected exposure

¹³⁴ DOE (2011b) also reports the results of *C. elegans* soil toxicity testing at 11 riparian sites adjacent to known contaminated media, seven riparian sites located between operational areas, and three riparian reference sites.

regimes should also represent the likely range of contaminant conditions and mixes to which terrestrial invertebrates have been exposed over the years, to the extent possible. The testing could potentially include longer exposures (to be more representative of chronic conditions), and/or might include both lethal and sub-lethal endpoints, such as survival, reproductive success, movement, and/or feeding (Sochová *et al.* 2006). Where contaminant concentrations are to be measured, investigators will take care to select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

INVERTEBRATES: ASSESSMENT OF TERRESTRIAL INVERTEBRATE ABUNDANCE

Objective: To evaluate the abundance of certain terrestrial insects, and potentially spiders, across one more gradients of contamination at Hanford, and examine correlations between these metrics and measures of contaminant exposure. Invertebrate community diversity may also be assessed.

Need/Rationale: This study will support an assessment of injury to terrestrial invertebrates in accordance with 43 CFR § 11.62(f)(1)(i) and 11.62(f)(3). Measurements of contaminants in site soils and terrestrial invertebrates will also contribute to the Trustees' pathway determination for these natural resources.

A healthy invertebrate community is fundamental to a healthy ecosystem. Terrestrial invertebrates are prey for small mammals and birds and provide essential ecological services (e.g., pollination). Invertebrate macro-decomposers, such as darkling beetles (*Tenebrionidae*) provide essential nutrient-cycling services in dry land areas (Safriel and Adeel 2005). Møller and Mousseau (2009) reported negative relationships of the abundance of spiderwebs, grasshoppers, dragonflies, bumblebees, and butterflies with background radiation exposure.

Approach: A survey of insect health will evaluate the extent to which insect abundance may have been affected by contaminant releases from Hanford. The Trustees will measure the abundance of insects an appropriate number of operational and other contaminated areas, and in suitable reference areas. Physical samples may be gathered to measure contaminant concentrations in tissues and soils, and/or for genetic analysis to evaluate measures of genetic damage consistent with radiation exposure. Invertebrate sampling may include above- and/or below-ground measures, and could include visual standard point counts, soil sample collection with subsequent processing/sieving and organism identification, pitfall traps, and/or other methods. Habitat characteristics (e.g., soil properties, litter, vegetation characteristics) will be documented at survey sites, as will other information (e.g., external radiation levels).

Ideally, the selected sites should represent the likely range of contaminant conditions and mixes to which terrestrial insects have been exposed over the years. Where contaminant concentrations are to be measured, investigators should select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

BIRDS: ASSESSMENT OF AVIAN ABUNDANCE AND DIVERSITY

Objective: To evaluate the abundance¹³⁵ and diversity of terrestrial birds, across one or more gradients of contamination at Hanford and examine correlations between these metrics and measures of contaminant exposure.

Need/Rationale: This study will support an assessment of injury to the avian community, in accordance with 43 CFR § 11.62(f)(1)(i) and 11.62(f)(3). Measurements of contaminants in site soils will contribute to the Trustees' pathway determination for these natural resources.

A healthy bird community is also fundamental to a healthy ecosystem. Møller and Mousseau (2007) found relationships between species richness, abundance, and population density of forest birds—particularly those eating soil invertebrates—in relation to Chernobyl radiation. Møller and Mousseau (2010) reported negative relationships of the abundance of birds with background radiation exposure, reporting that of the taxa evaluated, birds and mammals showed the strongest effects of radiation exposure. Birds in particular “appear to be the most efficient indicator of low-level radiation” (*ibid.*).

Approach: A survey of bird community status will evaluate the extent to which the avian community may have been affected by contaminant releases from Hanford. The Trustees will measure the abundance of birds in an appropriate number of operational and other contaminated areas, and in suitable reference areas. Specific methods may include line transects or point counts, documenting birds through visual and auditory means. Physical samples may be gathered to measure contaminant concentrations in soils. This study may also include the collection and genetic analysis of bird tissue samples to evaluate measures of genetic damage consistent with radiation exposure. Habitat characteristics will be documented at survey sites, as will other information (e.g., time of day, weather, radiation levels).

Ideally, the selected sites should represent the likely range of contaminant conditions and mixes to which terrestrial insects have been exposed over the years. Where contaminant concentrations are to be measured, investigators should select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

BIRDS: EVALUATION OF EXPOSURE TO HANFORD SITE AVIAN SPECIES

Objective: To evaluate the exposure of selected avian species to Hanford Site contaminants, as indicated through measurements of contaminants in eggs.

Need/Rationale: Birds can be exposed to contaminants in the environment through direct digestion of contaminated media (e.g., water) or, more often, through dietary pathways (i.e., consumption of contaminated food items), yet relatively few direct measurements of contaminants in wild avian tissues are available. Data are especially few for terrestrial birds. This study will contribute to the establishment of a complete pathway between

¹³⁵ In this context, “abundance” is intended to encompass any of several potential measures of population size, including abundance, relative abundance, or occupancy.

contaminant sources and avian receptors in accordance with 43 CFR § 11.63, and may suggest future lines of inquiry with respect to injury assessments of particular species. Focusing on eggs is particularly appropriate, as early life stages tend to be the most susceptible to the effects of many contaminants.

Surveys conducted between 1994 and 1999 documented 221 species of birds on the Hanford Site, bringing the total of known avian species at Hanford to 258 (TNC 1999). Of note, not all documented species breed onsite, and it is only onsite breeders that would be investigated in this study.

Approach: Bird egg analysis can provide a direct indication of contamination to which an organism has been exposed. For this study, the principal investigator(s) will select a suite of bird species based upon criteria including the species' life histories, the technical feasibility of egg collection, and the anticipated abundance of nests onsite and at reference locations. To the extent possible (e.g., without inflicting undue mortality on the population), sufficient numbers of eggs of each species will be collected to allow for statistically rigorous analysis of concentrations of multiple COPCs. Eggs will be collected from a diversity of nests located across areas in various conditions to allow for comparison between locations (e.g., remediated areas, un-remediated areas, and reference areas).

Eggs will be tested for selected COPCs, likely focusing on both lipophilic organic contaminants (as these may be maternally deposited into the yolk), as well as metals that are expected to partition preferentially to shells. Detection limit and sample volume restrictions may result in the need to composite eggs within nests prior to analysis, and will likely limit the total number of contaminants that can be analyzed within a given sample.

We note that depending on the species and COPCs, it may also be appropriate to collect blood and/or feather samples, as recommended by the principal investigators.

MAMMALS: SMALL MAMMAL POPULATION ASSESSMENT

Objectives: To evaluate the abundance¹³⁶ of one or more small mammalian species, across one or more contaminant gradients at Hanford, and to evaluate correlations between measures of contaminant exposure and population metrics.

Need/Rationale: This study will inform an injury determination for one or more small mammalian species in accordance with 43 CFR § 11.62(f)(1)(i) and 11.62(f)(3). Measurements of contaminants in site soils and mammalian tissues will also contribute to the Trustees' pathway determination for these natural resources.

Small mammals serve an important ecological role in food webs, commonly consuming plants and sometimes invertebrates, thereby filling the role of a primary (or secondary) consumer. Small mammals may themselves be prey to carnivorous mammals and predatory birds. Møller and Mousseau (2010) reported negative relationships of the

¹³⁶ In this context, "abundance" is intended to encompass any of several potential measures of population size, including abundance, relative abundance, or occupancy.

abundance of mammals with background radiation exposure, reporting that of the taxa evaluated, mammals and birds showed the strongest effects of radiation exposure.

Significant differences in relative abundance, or occupancy, between un-remediated affected sites and control sites can be indicative of a population-level injury to a species.¹³⁷ Although difficult to demonstrate in upper-trophic level species with expansive home-ranges, population-level impacts may be more readily identified in smaller mammals that can be easily collected and studied in the field, and that are associated with a small home-range. The RCBRA (DOE 2011b) collected small mammals for the purpose of comparing tissue concentrations in study sites versus references sites. However, this study was designed to support exposure studies in mid-trophic level and broad-ranging species, rather than to identify population-level impacts to small mammals between sites. Further, as recognized by DOE (2011b), the availability of only a single campaign's worth of data collection for the small mammal community significantly limits its usability in drawing conclusions on population-level endpoints such as relative abundance, occupancy, or density. One candidate species for this population assessment is the Great Basin pocket mouse. This mouse, primarily an herbivore, is an important native species that serves as prey for many species of animals. As a burrowing mammal, it may be exposed to contaminants present below the surface, and it is the most abundant small mammal found at the Hanford Site (Downs *et al.* 1993). Other mammalian species may also be considered.

Samples may be gathered to measure contaminant concentrations in tissues and soils, and/or for genetic analysis to evaluate measures of genetic damage consistent with radiation exposure. Habitat characteristics will be documented at survey sites, as will other information (e.g., external radiation levels).

Approach: The Principal Investigator(s) responsible for detailed study design will consider in detail the results of the DOE (2011b) small mammal community work as well as other relevant information as part of developing a multi-season field study. This study will examine differences in abundance and density, relative abundance, and/or occupancy of this species between various sites including partially or completely remediated sites, sites where remediation has not yet begun, and suitable reference areas. Study methods may include traps, canine scent surveys, and/or other approaches.

MAMMALS: GREAT BASIN POCKET MOUSE - CARBON TETRACHLORIDE AND HISTOPATHOLOGY

Objective: To evaluate whether Hanford Site Great Basin pocket mice may have been injured as a result of exposure to carbon tetrachloride.

Need/Rationale: This study will inform an injury determination for the Great Basin pocket mouse, a common burrowing mammal found at the Hanford Site in accordance with 43 CFR § 11.62(f)(1)(i), 11.62(f)(3), and 11.62(f)(4)(vi). Measurements of

¹³⁷ Of note, injury can be determined at the individual organism level: the DOI NRDA regulations do not require injury at the population level to be present in order for an injury determination to be made.

contaminants in site soils will also contribute to the Trustees' pathway determination for soils and associated mammals.

As a burrowing mammal, the Great Basin pocket mouse may be more likely than other animals to be exposed to carbon tetrachloride, one of the soil-associated site COPCs. Carbon tetrachloride's primary toxic effect in mammals is hepatotoxicity, causing liver tumors and general liver damage.

Approach: This study will include collecting Great Basin pocket mice from areas known or thought to be subject to higher levels of carbon tetrachloride, as well as from reference areas, to determine whether mice from contaminated locations have a higher incidence of pathology of the liver (and potentially other organs). It is recommended that contaminant levels, including carbon tetrachloride, be simultaneously collected. Because carbon tetrachloride is subject to "rapid clearance from exposed organisms" (ASTDR 2005), measurements of the exposure of mice to this contaminant may be more accurately made through evaluation of levels in site media rather than in tissues.

TERRESTRIAL RESOURCES: IMPACTS OF REMEDIAL ACTIVITIES

Objective: To identify and quantify impacts to terrestrial habitats associated with Site remediation activities.

Need/Rationale: This effort will support the determination and quantification of injury associated with those remedial activities that address Site contamination, in accordance with 43 CFR § 11.15(1).¹³⁸

Approach: Quantifying injury to the terrestrial habitat due to site remedial activities will require the Trustees to identify, organize, and summarize extensive information related to several parameters. Specific questions to be addressed with this study include:

- What remedial activities have occurred on site that have generated injuries that are recoverable under CERCLA? Such site activities are likely to include but may not be limited to: landfill construction (for hazardous materials), road development, borrow pit use, and capping, all of which are actions likely to result in temporary or permanent adverse impacts to terrestrial habitat.
- Where are or have these activities been located, and what is the spatial extent of the disturbed or injured habitat? As part of this question, Trustees will consider ancillary disturbance that may have occurred away from the remediation site itself (e.g., associated borrow pits, or roads leading to the site).
- When did remediation of the site begin, and when is full recovery of the habitat's services expected to be restored?
- What was the condition of the site prior to restoration (i.e., what level of services was it providing) and what is the anticipated condition when restoration is complete?

¹³⁸ In particular, Trustees may recovery injuries "that are reasonably unavoidable as a result of response actions taken or anticipated."

As part of this study, it will also be important to evaluate the likely severity of the identified remedial activities on the affected area(s). Close collaboration with DOE will be important to ensure the accuracy and completeness of information on which this analysis will rely. The Trustees propose to use HEA (discussed in Chapter 5) to quantify these injuries.

TERRESTRIAL RESOURCES: QUANTIFICATION OF LOST TERRESTRIAL ECOLOGICAL SERVICES

Objective: The objective of this study is to quantify the ecological services terrestrial resources (soil and terrestrial biota) have lost in the past and may lose in the future as a result of Site-related contamination.

Need/Rationale: In order to determine the scale and type of restoration actions required to compensate the public, the Hanford Trustees will need to understand the scale and scope of lost services.

Approach: This study involves two phases. The first phase consists of compiling information obtained from the geological and terrestrial biota studies mentioned above. This information will likely include the degree to which sample concentrations exceed identified injury thresholds, toxicity information on the adverse effects of varying levels of contamination, as well as ecological information (e.g., the abundance or distribution of terrestrial species, species community health). The second phase consists of analyzing the compiled data in order to quantify the geographic and temporal scope of ecological services terrestrial resources have lost in the past and may lose in the future. This will involve developing a relationship between Site-related contaminant concentrations and the severity of adverse effects experienced by terrestrial resources as a result of the contamination. The relationship will likely be based on literature information and data from site-specific studies on the toxicity of contaminants of concern as well as information on habitat usage, species abundance, and species diversity. Site-specific contaminant concentrations will then be compared to the developed relationship in order to determine the extent to which Site terrestrial resources have been injured (i.e., determine the estimated service loss).

7.4 VADOSE/ GEOLOGICAL RESOURCES

Remedial activities began on the Hanford Site in the early 1990s. These activities have focused on groundwater and soil contamination in the Columbia River corridor. After remedial activities are complete in the river corridor, the focus will switch to the Central Plateau and contamination in the 200 Areas. The majority of Hanford's solid waste burial grounds and underground liquid waste storage tanks are found in the 200 Areas; hence, there is significant vadose (deep soils) contamination in this area. The vadose zone includes soil resources between the surface soils (which are assessed as part of the terrestrial resources described above) and the groundwater resources (described below). Vadose zone soils, and other geological resources, are typically assessed by Trustees as a source and pathway for contamination to groundwater and aquatic resources. Due to the significant volume of contaminants currently present in the Hanford vadose zone, and the potential for these contaminants to injure groundwater and aquatic resources, we include

vadose zone/geological resources as a separate resource category to be addressed in the assessment.

Deep vadose zone contamination poses some of the most difficult remediation challenges for the protection of groundwater at the Hanford Site (Chronister 2011). Recently, Hanford officials have been working to integrate groundwater and vadose zone remedial activities and adopt a holistic cleanup approach (Goswami 2011). However, the potential for vadose contamination to impact groundwater resources and ultimately move towards the Columbia River is not well characterized. The study described below is intended to characterize contamination in the vadose zone and the potential for injury due to vadose contamination, based on existing information and models.

OVERVIEW OF EXISTING SITE VADOSE ZONE DATA

Available information on the Hanford Site's vadose zone resources that is of most relevance to the injury assessment includes (a) measurements of hazardous contaminants in the vadose zone, and (b) information on the stratigraphy and geology of the vadose zone.

Measurements of Hazardous Substances and Geology of Vadose Zone

Numerous studies have been conducted at the Hanford Site to characterize the stratigraphy and geology of the vadose zone as well as contamination in the vadose zone. Vadose zone soil data are collected and monitored using geophysical logging of boreholes and soil-vapor monitoring (Hartman 2000). The quantity, location, and movement of vadose contamination and moisture are documented through the borehole monitoring (Hartman 2000). The Groundwater/Vadose Zone Integration Project established a Characterization of Systems Task to organize a set of data, parameters, and conceptual models that could be used to estimate contaminant migration and impacts in the vadose zone. Freeman *et al.* 2001 provides a catalog of data sources describing hydraulic properties important in characterizing the vadose zone. Gee and Ward (2001) found that the transport of a vadose zone plume was controlled by distinct horizontal sedimentary layers at the six and 12 meter depths, and that a change from coarse to fine sand caused significant lateral spreading of the plume. Conceptual models of the vadose zone need to include two- or three- dimensional aspects of transport to adequately capture vadose transport (Gee and Ward 2001).

Vadose zone monitoring and sampling has continued in recent years with carbon tetrachloride soil vapor monitoring in the 200 West Area, tank farm vadose characterization, borehole sampling in C Tank Farm, surface geophysical exploration in part of the S Tank Farm, and geophysical logging as described in the 2010 Monitoring Report (DOE 2011c). More recently, a new operable unit has been created for the deep vadose zone (200-DV-1) to allow for a centralized focus and systematic approach to the challenges presented by the contamination in the deep vadose zone (DOE 2010a). In addition, a site-wide groundwater and vadose zone project was planned by Washington State Department of Ecology Nuclear Waste Program to expedite cleanup of soil and groundwater to be implemented from July, 2011 through June, 2013 (Goswami 2011). The main objectives of the project include developing site-wide groundwater and vadose

zone strategy, policy, and integration, deep vadose zone science and technology, and site-wide well installation, monitoring, and decommissioning.

IMPLICATIONS OF EXISTING DATA FOR INJURY ASSESSMENT

Given the information above on the level of existing relevant vadose zone data, the following injury assessment studies have been identified to fill data gaps. Phase 1 priority for the assessment of vadose zone (geological) resources focuses on characterizing vadose zone contamination and the potential for long-term injury to groundwater and surface water resources due to contaminants that have been released to the vadose zone, as described below. The phase 2 priority study in this section encompasses efforts to evaluate current vadose zone models.

CHARACTERIZING VADOSE ZONE (GEOLOGICAL RESOURCE) CONTAMINATION AND THE POTENTIAL FOR LONG-TERM INJURY TO GROUNDWATER AND SURFACE WATER DUE TO CONTAMINANTS THAT HAVE BEEN RELEASED TO THE VADOSE ZONE

In developing this Injury Assessment Plan the Trustees have considered available information on the nature and extent of hazardous contaminants in the environment resulting from releases from Hanford operations. The Trustees have also considered information that can be used to establish the level of past, current, and likely future natural resource injuries and service losses resulting from these releases. There is, however, a great deal of uncertainty as to the potential for long-term future natural resource injuries and services losses that could result from sources of contamination at the site that are not well-characterized. There is also a great deal of uncertainty regarding the likely nature and effectiveness of future remedial actions in addressing these sources of contamination.

In particular, despite current uncertainty, it is estimated that a substantial portion of the hazardous substance inventory at the Hanford Site remains in the vadose zone, so understanding this potential injury is of great importance. For example, there are several existing sources of hazardous contaminants in the vadose zone in the Central Plateau of the Hanford Site (DOE 2011c, Chronister 2011, Goswami 2011). These sources of potential injury may not be fully removed as part of the ongoing site cleanup, pending final cleanup decisions.

Objective: The purpose of this study will be to utilize available information and model outputs to develop an understanding of the likely nature, extent, and timing of natural resource injury, and lost services that could occur in the long-term future. The output of this effort will be subject to significant uncertainties, which should be described in the resulting white-paper and briefing.

Need/Rationale: The Trustee Council will need to determine the expected duration of ongoing injuries, as well as the potential that additional injury could occur in the future, as a result of ongoing sources of contaminants that are not being addressed by ongoing or planned remedial activities. Based on this information, the Trustees may be able to make assumptions about the nature, extent, and timing of future injury, or will identify the need for additional studies to define the nature and extent of such injury.

Approach: A team will be assembled to develop a whitepaper for presentation to the Trustee Council on this topic. This whitepaper will describe (1) significant sources of contaminants in the vadose zone and other geological resources that are not currently addressed by ongoing or planned remedial activities; (2) what is known about the potential future fate of these contaminants; (3) what the likely fate of these contaminants implies for future injury to groundwater resources and the environment of the Columbia River. No new data collection or modeling will be conducted as part of this effort.

EVALUATION OF EXISTING VADOSE ZONE MODELS

Objective: The objective of this study or expert panel is to assess the ability and limitation of currently used models to quantify vadose zone contamination flux in order to determine whether the models can be used to accurately predict the impact of vadose contamination on groundwater resources.

Need/Rationale: A variety of models are used by DOE to quantify contamination flux in the vadose zone. Verifying the accuracy of these models may allow the Hanford Trustees to make an informed decision on whether to rely on the results of the models to help estimate the quantity of injured groundwater in the vadose zone and the impact vadose zone contamination may have on groundwater resources.

Approach: Contamination in the vadose zone is an important component in determining groundwater injury at the Hanford Site due to the threat vadose zone contamination poses to the underlying groundwater resource. An independent evaluation of the models used at Hanford to quantify contamination flux in the vadose zone could provide additional information on the validity of these models, and the re-modeling of vadose zone contamination using three-dimensional models could strengthen understanding of the Hanford vadose zone.

The purpose of this study is to perform an independent assessment of Hanford vadose zone models. A limited-area field experiment within the 200 East Area was conducted to study vadose zone contamination; water was injected into the vadose zone and migration tracked with boreholes. The movement of the injected water was analyzed by comparing simulated distributions of the water using three different simulation tools: 1) upscaling, 2) cokriging/artificial neural network (ANN), and 3) transition probability (TP)/Markov chain (MC) to observe spatial and temporal evolution of the moisture plume. Since moisture retention and unsaturated hydraulic conductivity measurements are sparse, these methods are used to model moisture flow. However, this field injection experiment was very limited in area and volume, and therefore provides data on the unsaturated zone specific only to the zone of the experiment, which represents a miniscule portion of the total Site vadose zone impacted by Site contaminants. Additional vadose zone injection tests and simulations at different locations within the Site will provide information on the quantitative hydraulic properties of the vadose zone across the Site. Such experiments will be relatively costly and time-consuming; therefore, a cost/benefit analysis should be done to determine the net value of such tests. It will be useful to compare the physical hydraulic properties of the previous injection test zones to other important vadose zone

areas of the Site (i.e., grain size distribution, saturated hydraulic conductivity, and porosity).

7.5 GROUNDWATER The Hanford Site has a lengthy operational and remedial history, and as part of that history, a number of existing groundwater studies provide information of potential use in the injury assessment. The studies included in this Injury Assessment Plan either review or build on available information from past efforts and are intended to address key data gaps and/or remaining uncertainties.

Several of the groundwater studies described below could be very costly to conduct. As such, to provide information to support a decision on whether to undertake such studies, the Trustees propose to first complete an analysis of the legal, political, economics, and hydrological contexts which define the baseline for groundwater at the Hanford site. This will include developing a general understanding of the scope and scale of services that may have been lost. This understanding will inform the decision to conduct additional groundwater characterization efforts. In addition, in some cases the Trustees may reach a determination that the information which would be provided by a study will be limited; in those cases, the Trustees may choose to rely on reasonable assumptions in place of values or information developed through primary research.

To provide context for the proposed groundwater injury studies, the following paragraphs briefly summarize key data that have resulted from past investigations of the Site's groundwater resources and are intended to generally characterize the larger research context into which the proposed studies will fit.

OVERVIEW OF EXISTING SITE GROUNDWATER RESOURCE DATA

Available information about the Hanford Site's groundwater resources that is of most relevance to the injury assessment includes but is not limited to: (a) measurements of hazardous substances in groundwater and the vadose zone, (b) measurements of the areal and vertical extent of groundwater contamination, including groundwater plume maps, (c) measurements of aquifer porosity, adsorption effects and matrix diffusion effects, and (d) information on the extent of groundwater upwellings in the Columbia River.

Measurements of Hazardous Substances

As noted previously, the Trustees have identified at least seven partially overlapping databases that contain many measurements of concentrations of hazardous substances in site media. The Hanford Environmental Information Systems (HEIS) database contains the largest numbers of samples of groundwater and soils. HEIS continues to be developed, and HEIS may eventually serve as the repository for virtually all site sampling efforts, past and ongoing. A substantial effort has been underway within this past year to add more data to HEIS; as this effort progresses, it may be possible to rely less on other compilations of contaminant information. In addition to HEIS, databases with information on groundwater resources include the Columbia River Component historic database and the River Corridor Baseline Risk Assessment GiSdT database.

A review of the entries in these databases indicates that existing groundwater samples are distributed across the Site but concentrated around the operational areas (100, 200, and 300 areas). The number of measurements of contaminants in site groundwater is large; however, challenges remain in effectively using these data in the context of the injury assessment. These challenges include but are not limited to: variations in sampling efforts (and associated sampling objectives) associated with the datasets; the level of quality assurance associated with the various datasets; analytic issues associated with non-detect values; and the absence of readily available sample characterization information in some cases (e.g., sampling depths and geographic coordinates). Studies that rely on this information (e.g., those involving comparisons of measured concentrations with thresholds) will need to address these issues through careful study design and implementation.

Measurements of Areal and Vertical Extent

Groundwater resources on Site have been monitored since the late 1940s. Samples are collected monthly, quarterly, or semiannually in wells near regulated waste units, and less frequently from wells farther away from waste sites and operational areas (Hartman 2000). Thousands of samples have been collected from hundreds of monitoring wells, piezometers, and aquifer tubes, distributed across the Site.

The Department of Energy uses groundwater sampling data along with knowledge of Site hydrogeology, waste disposal practices, and chemical characteristics to develop groundwater contaminant distribution maps. These maps are presented in the annual groundwater monitoring reports. Contaminant plume maps have been delineated over the past 30 years, based on information from thousands of samples. However, in many places, there are gaps of two miles or more between wells. Thus, when sampling data are mapped and interpreted for delineating plume boundaries, interpolated concentration contours may be subject to large uncertainty in some locations. The Hanford Trustees are currently working with the USGS to review existing plume maps and estimate their accuracy for assessment purposes (see the “Review of Contaminant Plume Mapping” study described below).

Plume area is one of several parameters needed to reliably estimate the volume of contaminated groundwater; another important parameter is the vertical distribution of plumes. Limited data exists on the vertical extent of plumes. Within the past two to three years, multiple-depth samples have been collected in numerous wells in the Central Plateau (200 Areas) and in the 100 Areas along the Columbia River. Data on the vertical distribution of strontium-90 in the 100-N operable unit, nitrate in 200-BP-5 unit, numerous contaminants in the 200-UP-1 unit, carbon tetrachloride and technetium-99 in the 200-ZP-1 unit, and uranium and trichloroethene in the 300 area is reported in the 2010 Annual Monitoring Report (DOE 2011c). However, data gaps remain pertaining to contaminants and locations which have not yet been characterized, the available data are based on a limited number of wells, and the wells have not been sampled for long enough to establish reasonable temporal trends.

Porosity, Adsorption, and Matrix Diffusion Effects

As part of groundwater monitoring at the Hanford Site for the past 30 years, hundreds of reports have been produced describing the results of hydrogeologic investigations. The Department of Energy used a groundwater model used in the 200 Areas for evaluating potential remediation options, which estimated a 15 percent effective porosity (Central Plateau Version 3 MODFLOW Model, ECF-Hanford-10-0371, 2010). Additionally, Cole *et al.* 1997 reports effective porosity values estimated from specific yields obtained from well-aquifer tests in the range of approximately one to 40 percent, laboratory measurements of porosity ranging from 19 to 41 percent, and tracer tests indicating porosities ranging from one to 25 percent.

Some dissolved contaminants, particularly cations such as strontium-90, adsorb to aquifer mineral grain surfaces. This phenomenon can significantly increase the potential for continued contamination of the groundwater as the adsorbed contaminants dissolve into the water. There has been considerable work at Hanford addressing adsorption processes. For instance, distribution coefficients (i.e., the ratio of concentrations at equilibrium) for a number of contaminants including uranium and strontium-90 are reported in Cole *et al.* 1997.

Additionally, molecular diffusion of dissolved contaminants into low-permeability clay/silt lenses and layers can affect contaminant migration patterns. This process, referred to as matrix diffusion, has an effect similar to that of adsorption/desorption in slowing contaminant migration and delaying remedial actions, such as pump-and-treat systems. Unlike adsorption, matrix diffusion impacts all dissolved contaminants in a similar manner.

Information on Extent of Upwellings

The Trustees have particular interest in the current and expected future movement of contaminated groundwater to the Columbia River. There are a number of known upwelling locations, where Hanford groundwater releases into the Columbia River. As part of the Remedial Investigation of Hanford Site Releases to the Columbia River, surface water, pore water, and sediment samples were collected from 2008 through 2010 to help characterize groundwater upwellings (Hulstrom and Tiller 2010). Upwelling locations were located and mapped using conductivity and temperature measurements. Upwellings were found to be non-uniformly distributed and varied by water depth, season, and proximity to the shoreline (Hulstrom and Tiller 2010). Sampling results also documented hexavalent chromium, strontium-90, tritium, and uranium concentrations in excess of water quality guidelines in both nearshore and offshore locations. However, sampling effort was limited, and further study may be necessary to determine the potential adverse effects from contaminated groundwater upwellings (see “Groundwater Upwelling” study below).

IMPLICATIONS OF EXISTING DATA FOR INJURY ASSESSMENT

Given the information above on the level of existing relevant groundwater data, the following injury assessment studies have been identified to fill important data gaps. Phase 1 priorities for groundwater injury assessment focus on organizing the information necessary to estimate the level and extent of groundwater contamination and the

associated restoration requirements, including reviewing existing contaminant maps, reviewing groundwater models, determining the vertical extent of certain plumes, defining the context of baseline groundwater services, and quantifying contaminated groundwater. Phase 2 and 3 priorities encompass further efforts that would help the Trustees refine their understanding of potential groundwater injuries including characterizing the interaction between groundwater and the Columbia River and the impact of vadose zone contamination.

DEVELOPING A COMPREHENSIVE DATABASE AND COMPARISON TO INJURY THRESHOLDS

Objectives: (1) To create a comprehensive groundwater database; (2) to determine injuries to groundwater resources based on comparisons of measured and/or modeled concentrations of Site COPCs to regulatory water quality standards or criteria; (3) to identify COPCs that may be most strongly associated with potential injuries (e.g., by virtue of having a greater magnitude and/or exceedance of effects thresholds); and, (4) to identify locations with higher levels of hazardous substances, to help inform site selection in potential future injury studies.

Need/Rationale: Groundwater is a key natural resource, providing services to humans and serving as a pathway for the movement of contaminants to other resources. Contaminant concentrations in excess of certain levels (e.g., EPA maximum contaminant levels) generally indicate that an injury has occurred under DOI's NRDA regulations (43 CFR § 11.62(c)(1)(i) through (iv)); see Chapter 6).¹³⁹

A comprehensive database will allow the Hanford Trustees to compare the influence of well location and depth on contaminant plume concentration data in order to make an informed decision on the reliability of well sampling data for use in drawing contaminant plume maps.

In addition, comparing contaminant concentrations in groundwater to regulatory water quality standards or criteria is a cost-effective and widely used approach to evaluate potential groundwater injuries. Furthermore, making comparisons will also contribute to the Trustees' determination of exposure pathways between sources of releases and receptors.

Approach: The study will focus on groundwater beneath the Hanford Site, groundwater upwellings in the Hanford Reach of the Columbia River, and appropriate reference locations. The first component of this task will involve assembling and evaluating available data, and incorporating it into the Trustees' natural resource damage assessment database in accordance with the Data Management Plan and the Quality Assurance Management Plan (HNRTC 2011a, 2011b). Although many measurements of groundwater COPCs are available, a comprehensive database for use in damage assessment has not been developed. Developing a comprehensive groundwater database

¹³⁹ Chapter 6 provides complete definitions of injury to natural resources, including injury determination. Exceedances of certain concentration thresholds is a key component of these definitions but is not the only requirement that must be satisfied.

involves gathering and organizing data records and information on groundwater wells, depth, and associated contaminant concentrations. Much of the groundwater data is available in HEIS, and could be collected from HLAN (with QA/QC of the metadata), but this task will involve determining if the HEIS database is comprehensive and sufficient for injury assessment purposes. This database will also allow the Trustees to analyze the impact of well data quality including well siting, construction, and screened interval location, on the sampling and modeling of contaminant plumes to ultimately decide if well data meets injury assessment needs.

The Trustees will also determine the water quality criteria and standards (e.g., Federal drinking water standards, state water quality criteria) against which sample concentrations will be evaluated.

Lastly, this study will require an evaluation of baseline conditions, which will include a characterization of the concentration ranges of hazardous substances expected to be present in groundwater but for Hanford Site releases. As part of this evaluation, contaminants will be identified as having one or more of the following origins: natural sources, Hanford Site operations, and/or other anthropogenic sources. In some cases this determination will require new analysis; in other cases available information will be sufficient to make a baseline determination.

REVIEW OF CONTAMINANT PLUME MAPPING

Objective: To review and evaluate existing contaminant plumes, including determining whether the contaminant plume map generation method(s) being used by the DOE and contractors is sufficiently accurate for groundwater injury assessment purposes as defined under 43 CFR § 11 as well as whether additional plume maps need to be generated.

Need/Rationale: An assessment of the DOE plume maps is underway and will allow the Hanford Trustees to determine the need for a study to generate revised contaminant plume maps. If DOE plume maps are deemed appropriate for assessment purposes, the Hanford Trustees will be able to use these maps to move forward in assessing the quantity of injured groundwater. If the plume maps are not deemed appropriate for assessment purposes, the Trustees can begin to assess the need for an additional study to map plumes; this might involve the development of an alternative groundwater plume model to estimate various plumes' full extent and volume which would incorporate information on wells, contaminant data, and hydrostratigraphy that are deemed appropriately representative of the Site.

Approach: The Trustees, through the USGS, are evaluating the methods and results of current groundwater contaminant plume mapping at Hanford used to prepare illustrations and ancillary information presented in the annual Hanford Site Groundwater Monitoring and Performance Reports. As a critical part of the evaluation, USGS will independently regenerate groundwater contaminant plume maps, areas, and volumes from original monitoring and hydrogeologic data; evaluate the uncertainty of the original data; and determine the sources of uncertainty in the data that most substantially influence uncertainty in plume maps, areas, and volumes. Once this effort is complete, the Trustees

can determine whether the current maps are appropriate for the natural resource damage assessment and whether any additional maps need to be drawn.

DEFINE THE LEGAL, POLITICAL, AND ECONOMIC ENVIRONMENT FOR BASELINE SERVICES PROVIDED BY GROUNDWATER

Objective: The objective of this study is to describe the services that will be provided by groundwater at the Hanford Site under baseline conditions and how these services have been impacted by the release of hazardous contaminants.

Need/Rationale: An understanding of the baseline services provided by groundwater at the Hanford Site, in the context of political, legal, and economic setting is necessary to determine how the services have been affected by the release of contaminants. Once the baseline services and how services have been affected has been determined, the Trustees will be able to identify and scale appropriate restoration projects to restore or replace those lost services. As noted above, this information will also help support decisions regarding the value and need for additional groundwater injury studies.

Approach: This study should be undertaken prior to other groundwater studies, in order to provide the necessary context on groundwater baseline services which will help scope subsequent studies. This study will involve the development of a white paper that describes the services that will be provided by groundwater at the Hanford Site under baseline conditions, and how those services have been impacted by contamination. The paper should address the full range of services, including use, non-use, and in situ services. This paper should also address the institutional, policy, legal, economic, and hydrological factors that define how groundwater will have been used absent contamination.

VERIFYING VALIDITY AND LIMITATIONS TO HANFORD GROUNDWATER MODELS

Objective: To verify the validity of Hanford groundwater models, to support a quantification of groundwater injuries.

Need/Rationale: A variety of models are used by DOE to estimate current, past, and future injured groundwater. Verifying the accuracy and validity of these models may allow the Hanford Trustees to make an informed decision on whether to rely on the results of the models to help estimate the quantity of injured groundwater on Site.

Approach: In a natural resource damage assessment, injury to groundwater resources can be quantified in physical units, such as an annual sustainable yield, a flux, or as a volume. Models frequently play a critical role in this quantification: data on past contaminant levels may be few or absent (but may be approximated through models), and models are also necessary to estimate future concentrations. Groundwater computer models have been applied at the Hanford Site to examine and simulate groundwater flow patterns, water budgets, aquifer responses to hydraulic stresses, migration of contaminant plumes, and the performance of groundwater remediation systems. These models are helpful in interpolating hydrogeologic conditions between wells, conducting sensitivity analyses regarding data gaps, prioritizing future data gathering steps, testing remediation alternatives, and in assessing exposures and groundwater injury under

various assumed scenarios. In general, MODFLOW (a groundwater flow modeling code), coupled with a contaminant transport code, and STOMP are the modeling codes generally used at Hanford.

Since these models are essential in estimating contaminant plume volumes, an independent assessment of the groundwater models used at Hanford will provide additional validation of the current assumptions, parameters, and application of the models including what they should not be used for, and if the models are being used and applied appropriately. This validation process could be accomplished through the use of an expert panel. Note that the panel may require a significant amount of time to review existing information and to come to a consensus opinion.

GROUNDWATER UPWELLINGS

Objective: To characterize the distribution, frequency, and volumetric flow rate of a few known contaminant upwellings in the Columbia River in order to assess the potential for exposure pathways and injury to aquatic biota.

Need/Rationale: Defining the distribution, frequency, and volume of a few known contaminant upwellings in the Columbia River will allow the Hanford Trustees to estimate the potential adverse effects to aquatic biota in the River, in accordance with 43 CFR § 11.62(c)(iv) , as well as the need for further study.

Approach: Groundwater upwellings in the Columbia River can adversely or beneficially affect aquatic biota, depending on contaminant levels in the upwelling water. However, the nature, extent, frequency, and volume of these upwellings is not well known. Hanford Site groundwater upwellings have been studied through pore water sampling as well as sediment and surface water sampling, the results of which can be found in the Field Summary Report for Remedial Investigation of Hanford Site Releases to the Columbia River, WCH-380 (Hulstrom and Tiller 2010). An assessment of groundwater upwelling pore water data is presented in the Data Quality Assessment Report for the Remedial Investigation of Hanford Site Releases to the Columbia River, WCH-381 (Hulstrom 2010). Samples were taken from the 100-B/C, 100-K, 100-N, 100-D, 100-H, 100-F, Hanford townsite, and 300 Area to characterize groundwater upwellings, and upwellings were found in all study areas (Hulstrom and Tiller 2010). However, groundwater upwellings were not uniformly distributed across the study areas and changed with water depth, season, and proximity to the shoreline (Hulstrom and Tiller 2010).

Although the above mentioned sampling of pore water, surface water, and sediment has provided information on chromium upwellings in the Columbia River, questions remain on the distribution, frequency, and volumetric flow rates of upwellings and past estimates could be strengthened based on new information. Improvements to the digital elevation model for the Columbia River channel, new detail on the stratigraphy near the river, and riverbed pore water sampling results could be used to further the accuracy of chromium upwelling estimates. Additionally, more precise measurements of net gains or losses in river discharge rates along the reaches impacted by Site groundwater could also further Trustee understanding of Hanford upwellings. A chromium upwellings study could more

accurately characterize the spatial and temporal distribution of known upwellings, area and resources influenced by the known seeps, as well the frequency of seepages.

The Trustees may also elect to phase this study to better understand the ability of the study to achieve its objectives, prior to deciding to proceed with a full-scale implementation effort.

SYNOPTIC SAMPLING OF RIVER CORRIDOR WELLS

Objective: To sample selected river corridor wells at varying river stages to determine the influence of river stage on groundwater depth readings.

Need/Rationale: Understanding the effect of river stage on groundwater depth readings will allow the Hanford Trustees to decide whether well readings near the river are accurate and appropriate for use to estimate plume maps and pathways near and beneath the river for the purposes of groundwater injury determination and quantification.

Approach: The Columbia River stage changes drastically within short time periods and could affect groundwater well readings. Understanding the relationship between groundwater depth and river stage will help to determine the reliability of groundwater data for developing plume maps, and whether the river stage and therefore timing of groundwater sampling significantly affects groundwater plume estimates. Samples will be taken from multiple wells within one hour and from wells at high, middle, and low river stages to determine the impact of the river on well water levels.

VERTICAL DISTRIBUTION OF CONTAMINANT PLUMES

Objective: To construct additional multi-depth monitoring wells in key areas of several of the major plumes and to sample the wells for several years in order to obtain information on the vertical depth of the significant plumes to inform injured groundwater volume calculations.

Need/Rationale: Information on the vertical depth of many major plumes on the Hanford Site is lacking. This study will provide additional information on the vertical depth of contaminant plumes, useful information used to estimate the volume of contaminant plumes for injury assessment.

Approach: One of the major uncertainties in assessing injured groundwater volumes on Site is the sparseness of vertical sampling data within all of the significant contaminant plumes. There are a limited number of samples from different depths within some areas of plumes in the 200 West, 200 East, 300-FF-5, 100-HR-3, and 100-NR-2 operational units (as described in the 2010 Groundwater Monitoring Report, DOE 2011c). However, vertically spaced sampling has been done only since 2009, which is insufficient temporal coverage, as well as spatial coverage to enable accurate delineation of three-dimensional plume configurations. Without adequate three-dimensional data, assumptions must be made regarding plume boundaries which can result in over-estimates of injured groundwater volumes. Additional collection of spatial and temporal plume thickness data will increase the accuracy of plume volume estimates. This will likely require construction of several more multi-depth monitoring wells at key areas of several plumes,

and an additional period of sampling of the multi-depth wells for a number of years. Installation and monitoring of several more multi-depth monitoring wells will be highly costly.

GEOLOGY OF COLUMBIA RIVER BED

Objective: The objective of this study is to characterize the geology of the Columbia River bottom in order to determine the potential impact of plumes near and beneath the river and contaminant upwellings in the River, as well as the potential for contaminants to migrate into groundwater on the non-Hanford side of the River.

Need/Rationale: Information on the geology of the Columbia River will allow the Trustees to more accurately map groundwater plumes near the River as well as determine any potential for groundwater plumes to affect riverine resources or locations on the non-Hanford side of the River (i.e., the Trustees will be able to more accurately determine the scale and scope of groundwater injury near the River). However, groundwater upwelling characterization (described in the upwelling study above) may provide adequate information for assessment purposes; thus, this study is a lower priority for the Trustees and may need to be re-evaluated after the upwelling is completed.

Approach: The geologic stratigraphy of the Columbia River bottom is not well known. Faults and other geologic structures can offset the hydrostratigraphic units, complicating interpretation of groundwater flow under the river. Drilling boreholes on river islands, seismic and electro-magnetic profiles, and geophysical surveys across the River could define the river bed stratigraphy, provide information to compare hydrostratigraphy and bank geology, provide information for correlating and interpreting geology between wells, and aid in the interpretation of groundwater flux and riverine upwellings. Measurement of hydraulic heads beneath the River bed will help define three-dimensional hydraulic gradients under the River.

QUANTIFY INJURED GROUNDWATER VOLUME AND TIME DIMENSIONS

Billions of gallons of contaminated wastes have been discharged on the Hanford Site, resulting in contaminated groundwater above drinking water standards. The groundwater on Site provides a range of services, which have been impacted due to the contamination. The metric chosen to quantify these losses depends on the type of services affected.

Objective: The objective of this study is to quantify injured Hanford groundwater resources.

Need/Rationale: The Trustees will need to understand the quantity of injured groundwater in order to determine the scale of lost services and the types of restoration projects required to restore those losses.

Approach: This study requires an understanding of the range and type of services impacted by groundwater contamination on the Hanford Site. Once these services are identified, the quantity of injured groundwater can be calculated using a stock volume, flux volume or sustainable yield approach as appropriate. Once the injured groundwater is quantified, and the Trustees have an understanding of groundwater baseline, the scale of lost services and type of required restoration projects can be determined.

7.6 TRIBAL USE As noted in Chapter 4, there are a range of tribal use services provided by natural resources that may have been impacted by releases from Hanford Site operations. While there is a large amount of available information on indigenous peoples use of the site (e.g., information which is used to inform decisions on whether remedial actions will disturb culturally important sites), the Trustees are unaware of any studies that have been done to assess the impacts of the presence of hazardous contaminants on current tribal use of natural resources. This information will be required to complete service quantification for this category of lost use. This information will inform the Trustees understanding of the scale and scope of benefits of potential primary restoration, as well as the scale and scope of any required compensatory restoration. In addition, while there are numerous ongoing efforts to characterize the nature and extent of contamination at the site (see below), the Trustees believe that a more focused effort to determine if additional characterization would allow for greater use of site resources by tribal community members is needed.

ETHNOGRAPHIC STUDY TO IDENTIFY TRADITIONAL CULTURAL PROPERTIES AT HANFORD

Objective: This study will identify Traditional Cultural Properties (TCPs) within the Federal government’s jurisdiction of the Hanford assessment area. Any Federal undertaking that has the potential to affect Federally-listed (and/or eligible for listing) cultural resources, including TCPs¹⁴⁰, must be evaluated, as mandated under the National Historic Preservation Act (NHPA) Section 106. Such actions could include assessment and restoration decisions associated with natural resource damage assessments. As such, identification of TCPs must first occur, as mandated under NHPA Section 110. Therefore, this study will be conducted to identify TCPs within the “area of potential effect” (APE) for the assessment, which is a Federal undertaking at Hanford. This effort will support assessing Tribal Lost Services and making decisions regarding the scale and scope of primary and compensatory restoration.

Need/Rationale: In compliance with NHPA Sections 106 and 110, DOE must identify the properties within their jurisdiction that qualify for listing as cultural resources in the National Register. While archeological sites may not be affected by the injury assessment, TCPs could be affected by the decisions made within the process. TCPs are generally eligible under any (or all) of the following three criteria (of four total):

- Property is associated with events that have made a significant contribution to the broad patterns of our history.
- Property is associated with the lives of persons significant to our past.
- Property has yielded or may be likely to yield information important in prehistory or history.

¹⁴⁰ For further definition, refer to the National Park Service National Register (NR) Bulletin 38 (Guidelines for Evaluating and Documenting Traditional Cultural Properties).

A property (TCP) must maintain integrity, which is “the ability of a property to convey its significance” (NR Bulletin 15). There are seven aspects of integrity: location, design, setting, material, workmanship, feeling, and association. If a project compromises or may compromise any of these characteristics that give a property significance, it is considered to be adversely affecting the property.

The Trustees recognize that multiple activities and actions at the Hanford Site may trigger requirements under the NHPA: in particular, actions outside of the NRDA may generate the need for information on TCPs. The Trustees acknowledge that NRDA may or may not be the correct legal and financial structure within which to pursue these activities, and that further discussions are needed to determine the best method to accomplish the work. That acknowledged, the Trustees have included this study in this Plan in recognition of the importance of TCP identification, and to note that the natural resource damage assessment may provide an opportunity to systematically address concerns regarding the impacts of site operations and cleanup, including the assessment and subsequent restoration actions, on TCPs.

The association of a TCP with the traditional belief system and culture of a Native American group is a characteristic that gives it significance. “A traditional cultural property then, can be described generally as one that is eligible for inclusion in the national register because of its association with cultural practices or beliefs of a living community that (a) are rooted in that communities history, and (b) are important in maintaining and continuing cultural identity of the community” (NR Bulletin 38). TCPs are culturally significant for a number of reasons for Native American groups: locational setting (including associated natural resources such as water, soil, plants, etc.), feeling, and association. By not fully restoring a TCP or installing institutional controls (e.g., when leaving contamination in place) that prohibit the Affected Tribes from utilizing the TCP, the association, setting, and feeling have been adversely effected. Adverse effects to TCPs must be mitigated.

Approach: TCPs cannot be discovered through archaeological or historical research alone. The existence and significance of such locations can only be ascertained through interviews with knowledgeable users of the area or through other forms of ethnographic research (NR Bulletin 38).

This study to identify TCPs is needed to determine if any properties that are within the project area (the entire Hanford site) will be adversely affected by the injury assessment and other related NRD activities. This study must be conducted by a trained professional meeting Secretary of Interior Standards.

ASSESS TRIBAL SERVICE LOSSES

As discussed in Chapter 4, there are a range of services provided by natural resources to tribal communities. These services may have been diminished in quality, or interrupted, by the presence of contaminants released by Hanford operations. As a result, specific restoration actions may be required to address these service losses. In Chapter 5, we discuss several approaches that could be used to assess the nature and extent of tribal service losses associated with contaminant releases. This information could be used to support Trustee decision-making regarding the scale and scope of primary and compensatory restoration.

Objective: This study is intended to identify natural resources and the nature and extent of services that they provide which are important to the health, welfare, economy, tradition, and cultural integrity of tribal members in the assessment area. Tribal lost services will then be assessed by selecting and implementing appropriate approach(es) to fill data gaps and determine Tribal service loss associated with Hanford contaminant releases. This information ultimately will be used to support decision-making regarding the scale and scope of potential primary and compensatory restoration for lost tribal use services.

Need/Rationale: Natural resources in the Hanford assessment area provide many services to tribal members in ways that are distinct from the general public, including social, cultural, spiritual, medicinal, recreational, and subsistence services, uses, and values. Examples include collecting sacred or medicinal plants; participating in subsistence and ceremonial fishing, hunting, and gathering; conducting ceremonial drinking, bathing, and sweating; and using sacred grounds for meetings, ceremonies, and spiritual recognition. The lives of tribal members are intricately linked to the natural resources in the assessment area; when one or more resource, such as surface water, plants or animals, is contaminated by the releases of hazardous substances, the ability of the environment to support subsistence and traditional uses can be diminished.

The resources that are used by tribal members, particularly those that support the cultural integrity and continuity of each Tribe, must be identified, including those that would have

“The Yakama subsistence lifestyle, including fishing, hunting, and plant gathering; use of traditional foods, medicines, and materials; sweathouse use, feasts, and other cultural practices, depends upon safe, unrestricted access to clean natural resources in the Hanford Assessment area year round in perpetuity”
(Yakama, 2010).

existed and been used by tribal members in the absence of Hanford releases. Compilation of existing materials and a critical review of the documentary record will identify what data are most useful and necessary for the injury assessment to identify the link between Hanford contaminants, injured resources, and service losses. During this process, the Tribes will propose and then undertake approaches they deem appropriate for collecting additional information and assessing changes in the use of natural resources by tribal members that have occurred as a result of the presence of contaminants from Hanford operations. This effort will distinguish changes in natural resource services to Tribes at

the Hanford site that are unrelated to contaminant releases from those that are the result of the presence of contaminants.

Approach: Tribal Trustees will collectively or independently develop and implement individual study plan(s) to: 1) review available information related to tribal services, 2) assess the nature and extent of tribal lost services, and 3) develop information to determine the appropriate scope and scale of restoration options to restore such losses. This effort will need to address confidentiality of tribal information. The following specific tasks will be identified in the individual study plan(s), which may be customized according to the needs of each Tribe:

- Identify, compile, and review existing literature and historical data as they relate to natural resources and associated tribal services now and prior to Hanford contaminant releases (i.e., baseline), including historical reports, scientific papers, oral histories, etc.
- Evaluate the compiled information and determine what sensitive information shall not be released, what information is necessary for assessing tribal service loss (and may require data sharing agreements), and what information is still missing that will help link Hanford contaminants to injured resources and changes in tribal behaviors and services. This effort will result in identification of the information needed (and data available) to assess the nature and extent of tribal lost services and restoration selection and scaling.
- Evaluate and select sound approach(es) to fill gaps and assess tribal lost services, while protecting confidential information.

Following these plans, one or more studies will be implemented to assess tribal lost services due to the release of contaminants, as distinct from other factors that have led to changes in tribal use of resources over time, and identify restoration options and scaling.

CURRENT RESOURCE CHARACTERIZATION TO ALLOW FOR RESTORATION OF LOST TRIBAL SERVICES

Tribal community member use of natural resources at the Hanford Site may be limited by concerns over exposure to hazardous contaminants. While numerous efforts are ongoing to characterize the nature and extent of contamination at the Site (discussed below), the scope of these efforts (geographic, temporal, resource specific) may not be sufficient for tribal community members to make informed decisions regarding their use of resources at the Site. As such additional monitoring and sampling may be needed to allow for restoration of lost services.

As noted, a variety of programs are in place to characterize the nature and extent of contamination in Hanford Site resources, including:

- **Environmental Surveillance Project.** Part of Mission Support Alliance's Public Safety and Resource Protection program, this project monitors the concentrations of radionuclides and chemical and metal contaminants in environmental media including air, surface water, sediment, soil, natural

vegetation, agricultural products, fish, birds, and mammals. Monitoring occurs on the Hanford Site, as well as at several offsite locations. External radiation levels are also monitored. Data from this program are reported regularly in the annual Hanford Site Environmental Monitoring Reports (MSA 2012a). Currently, the annual budget for this project is approximately \$2,100,000 (DOE 2012c).

- **Soil and Groundwater Remediation Project.** Managed by the CH2M HILL Plateau Remediation Company (CHPRC), this project includes sampling and monitoring of groundwater and soil on-site to characterize distribution of contamination and evaluate the effectiveness of remediation activities (CHPRC, 2012; Poston *et al.* 2010).
- **Drinking Water Monitoring Project.** This program conducts routine monitoring of drinking water supplies on the Hanford Site to ensure compliance with the Safe Drinking Water Act (Poston *et al.* 2010).
- **Biological Control Program.** The biological control program was established to limit the environmental impact of radioactively contaminated or otherwise undesirable plants and animals. As part of this program, radiological surveillance is done to help characterize the extent and distribution of contaminated biota and soil (Poston *et al.* 2010).
- **Near-Facility (Near Field) Environmental Monitoring.** This program monitors environmental media, as well as external radiation levels, around DOE facilities that have released, or have the potential to release, radioactive or hazardous contaminants. Monitored sites include areas around nuclear facilities (e.g., 100-N reactor and the Plutonium Finishing Plant), and waste storage and disposal facilities (e.g., burial grounds and trenches). Resources monitored include soil, air and vegetation (Poston *et al.* 2010). Although this monitoring is currently managed under the Environmental Surveillance Project, historically the annual budget for this monitoring was approximately \$500,000 (DOE 2012c).
- **Washington State Department of Health Hanford Environmental Radiation Oversight Program.** This Department of Health program's primary responsibility is providing oversight of DOE monitoring programs designed to characterize the impact of releases of radiation on the public and the environment. The program is itself not intended to provide comprehensive characterization of site contamination, but rather to independently verify the characterization work being done by DOE. Results of the program's work are published annually in a Data Summary Report (WDOH 2012). Currently, the annual budget for this oversight program is approximately \$764,000 (DOE 2012c).
- **Hanford Long-Term Stewardship Program.** This program was established to manage DOE's post-cleanup obligations. One of the key activities of the program will be the surveillance and maintenance of physical remedies and

institutional controls to ensure continued protection of human health and the environment (DOE 2010b).

- **CERCLA Five-Year Reviews.** The five-year review process required under CERCLA calls for additional characterization of sites where contaminants remain at levels that preclude unrestricted use of an area. Additionally, it will evaluate the effectiveness of completed remedies to determine if those remedies continue to be protective of the public and the environment. These reviews will be conducted by the Hanford Long Term Stewardship Program (DOE 2010b).
- **Ecological Monitoring Project.** Part of Mission Support Alliance's Public Safety and Resource Protection program, this project monitors the abundance, condition, and distribution of biota on the Hanford Site. Note that this program is focused on population-level conditions of biotic resources, rather than concentrations of contaminants within individual specimens (MSA 2012b).

In addition to the established long-term monitoring programs described above, several recent and on-going efforts have included comprehensive characterization of the Hanford Site and its resources. These efforts include:

- **Remedial Investigation of Hanford Site Releases to the Columbia River:** Samples collected between 2008 and 2010 (and approximate sample numbers reported in the Columbia River Component [CRC] database) included aquifer tubes (3,000), pore-water (400), surface water (600), sediment (1,200), soil (100), and fish (1,000) (DOE 2010c).
- **River Corridor Baseline Risk Assessment (RCBRA):** Samples collected as part of the RCBRA (and approximate sample numbers reported in the Guided Interactive Statistical Decision Tools [GiSdT] database) included soil/sediment (9,500), surface water, including seeps, springs, aquifer tubes, and pore-water (3,500), groundwater (13,000), and biota (200) (Neptune and Company Inc. 2009).

This study will consider these existing characterization efforts, assuring that additional characterization is not duplicative of these efforts.

Objective: There are a number of ongoing efforts to characterize and monitor contaminant concentrations within the Hanford study area. This study will define how to better organize and present this information for use by the tribal publics as well as the general public. It will also identify where additional characterization of contaminant concentrations would allow for restoration of tribal lost services. This effort will require close coordination with tribal community members and resource managers to fully understand tribal concerns and information needs.

Need/Rationale: A significant concern of the tribal Trustees for natural resources at Hanford is an absence of sufficient characterization of contaminant concentrations in natural resources. This information is needed by tribal resource managers to inform decisions by tribal members who are interested in utilizing resources at Hanford, but want to assure that these uses are safe.

Approach: Following response actions and/or primary restoration efforts, characterization of natural resources will be required to monitor the safety of the natural resources and to allow for restoration of tribal services. Some of this characterization is already taking place, but additional actions may be needed. This additional characterization would include organization of existing information, as well as gathering of additional information on the nature and extent of residual contamination and condition of injured resources. The scope and scale of characterization required to restore tribal use of the site needs to be determined and compared against information from existing characterization efforts (e.g., determine what media to monitor, where to monitor (and density of samples), and frequency and duration of sampling).

In addition to any monitoring plans associated with remedial activities and long-term stewardship plans, which rely heavily on the expectation of institutional controls, additional characterization may be undertaken by the Tribes to verify whether tribal use services can be confidently resumed. This effort would include developing Sampling and Analysis and Quality Assurance Project Plans, conducting field sample collection and laboratory analysis activities for all resource types in terrestrial, riparian, and aquatic zones, and conducting adaptive management, as necessary.

The factors that will need to be determined in this study are:

- Do existing sampling and characterization efforts provide enough information and the right type of information to inform tribal member use?
- How would this information be better assessed and presented for use by tribal members in making decisions about resource use? What is the most effective means to communicate this information to the public?
- What additional information is needed? Over what time period?
- What is the most cost-effective means to obtain additional characterization information?

**7.7 OTHER
HUMAN USES**

As discussed in Chapter 4, based on review of existing information, the Trustees are proposing a study to fully describe the past, current, and future geographic and temporal scope of contaminant-related institutional controls which could impact human use of natural resources at the site. At this time the Trustees are not proposing additional study of the effect of site releases on agriculture or a detailed study of recreational behavior. While this information exists, it has not been compiled in a manner sufficient for injury quantification.

**INVENTORY OF INSTITUTIONAL CONTROLS RELATED TO THE RELEASE OF
HAZARDOUS SUBSTANCES, AND DESCRIPTION OF ASSOCIATED LIMITS ON HUMAN
USE OF THE SITE**

Objective: To determine the extent to which institutional controls at the Hanford site, past, current, and expected future, are related to the release of hazardous contaminants.

To define the geographic scope and nature of these controls, and describe the types of human uses that may be impacted.

Need/Rationale: The primary source of non-tribal lost human use opportunities at the Hanford site will be associated with institutional controls made necessary by the presence of hazardous contaminants released from site operations. These controls may relate to areas of the site that will be subject to access restrictions, as well as limitations on the use of specific resources (e.g., groundwater). These restrictions may result in quantifiable injury. Based on this study, the Trustees will be able to determine if additional analysis of the likely change in the scale and scope of human use of the site from baseline conditions is called for.

Approach: An inventory of institutional controls will be developed. These controls will be screened to determine if they are related to the presence of a hazardous contaminant released from Hanford operations. A set of maps will be developed that presents these controls, for past, present and expected future conditions. Once this inventory is completed, the nature of any expected change in human use will be described.

7.8 ALL RESOURCES TREATMENT OF NON-DETECTS IN STUDIES ANALYZING EXISTING DATA

Initial data evaluations conducted by the Trustees to date have determined that a substantial number of available records identifying contaminant concentrations in various media in key sites databases (specifically, HEIS) are identified as “non-detects.” The value that is reported for records that are identified as non-detects is dependent upon the type of reporting limit reported by the lab that conducted the analysis. Values reported may be the Adjusted Reporting Limit, Estimated Quantitation Limit, Instrument Detection Limit, Method Detection Limit, Practical Quantitation Limit, or Required Detection Limit (DOE 2007b). Occasionally the value may simply be reported as “0” or some number < 0 .

While it is not necessarily clear from the record documentation what value specifically is reported for each record, we can generally assume that the actual concentration of the contaminant in question is something less than the value reported. The issue of non-detects becomes particularly problematic in cases where the value being reported exceeds a selected injury threshold, numerically suggesting injury although the analyte was not detected. Initial data evaluations have identified this situation in a number of media/contaminant pairings, including antimony in sediment, mercury in fish tissue, and chromium in soil. Alternately, if a detection limit exceeds an identified injury threshold, a reported value (e.g., if listed as zero) may suggest an absence of injury, even though the actual analyte’s concentration may have exceeded an injury threshold.

Records identified as “non-detects” represent valuable historical information that cannot be replicated. Thus, the Trustees prefer not to simply remove these data from the analysis but rather wish to identify the most appropriate treatment of these. Although it can be tempting to simply use the reported value or use some proportion of the detection limit,

for most applications, “substitution” approaches have been severely critiqued (e.g., Helsel 2010).

Objective: Determine the most appropriate way to treat samples identified as non-detects within analyses that rely upon historical data, and develop recommendations for additional data collection efforts.

Need/Rationale: Because of the substantial number of contaminants measured as non-detects, the Trustees need to develop a method to both utilize these data and reduce uncertainty in data analyses.

Approach: For each study that relies upon the analysis of historical data, the Trustees will evaluate a variety of options for handling non-detect sample results within each analysis. As a detailed analysis of non-detect samples for every media type and contaminant in each individual study area will not be feasible, the Trustees may prioritize detailed evaluations of non-detects in cases where:

- The extent of non-detects included within the group of samples to be analyzed is substantial (e.g., > 30 percent of available samples); and/or,
- The reported value of non-detect samples frequently exceeds the lowest identified injury threshold for a given contaminant/media type pairing (e.g., the vast majority of PCB sediment samples are non-detects and the reported values are above injury thresholds); and/or,
- The detection/reporting/quantitation limit value (where known) exceeds the lowest identified injury threshold for a given contaminant/media type pairing; and/or,
- Other evidence (e.g., toxicity testing results) indicates that injury to a specific resource due to a given contaminant is likely.

Evaluation of existing samples identified as non-detects may also indicate that additional data collection is warranted to adequately characterize the present state of the resource. In these instances, investigators will take care to select laboratory methods whose detection limits are sufficiently low such that the lowest detectable concentration of a contaminant does not exceed levels that have been identified as injurious.

CHAPTER 8 | QUALITY ASSURANCE MANAGEMENT

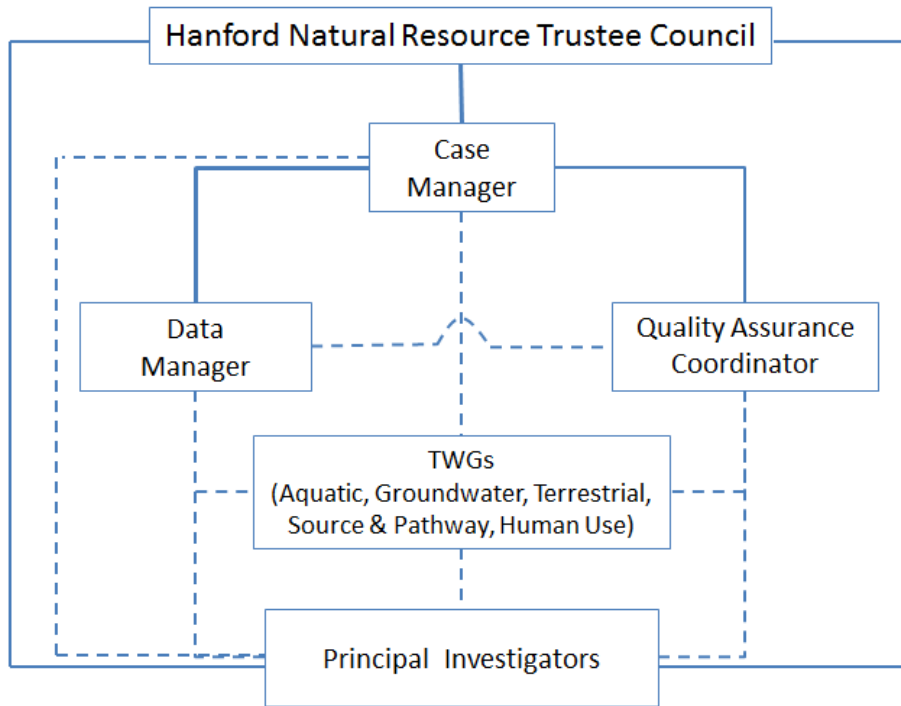
The DOI NRDA regulations require the Trustees to develop a Quality Assurance Plan (QAP) that “satisfies the requirements listed in the NCP and applicable EPA guidance for quality control and quality assurance plans” (43 CFR § 11.31(c)(2)). The Trustees recognize the importance of data quality: many of the management decisions involved in accomplishing the Hanford natural resource damage assessment ultimately require the use of environmental data. The collection, compilation, evaluation and reporting of environmental data are necessary to perform the functions of the assessment. It is necessary that the origin and quality of the data used to make these decisions is properly documented so that data gaps may be identified; assessments of the severity, location and extent of injury are accurate; and thus, appropriate decisions may eventually be made as to the needed type and scale of restoration actions.

The Hanford Trustees have developed a Quality Management Plan (QMP) in order to document the Trustees Quality Systems and to provide a blueprint for how the Trustees will plan, implement, and assess its Quality Systems for work performed by or on behalf of the Hanford Trustees. Consistent with EPA (2001), the Trustees’ QMP (HNRTC 2011b) presents the organizational structure, functional responsibilities of management and staff, lines of authority, and required interfaces for those planning, implementing, and assessing all activities conducted under the assessment. The following paragraphs summarize key elements of this Quality Management Plan, including the requirement that natural resource damage assessment work plans include project-specific Quality Assurance Project Plans (QAPPs).

8.1 PROJECT MANAGEMENT

Exhibit 8-1 shows the quality assurance management organization for the Hanford natural resource damage assessment.

EXHIBIT 8-1 QUALITY ASSURANCE MANAGEMENT ORGANIZATION FOR THE HANFORD NATURAL RESOURCE DAMAGE ASSESSMENT



*Solid lines – formal lines of authority
Dashed lines – advisory/coordination*

The Trustees have overall program management responsibilities for the natural resource damage assessment including data quality management. The Case Manager is responsible for the management and communication of specific quality assurance activities with advisory input from the Technical Working Groups (TWGs). TWGs also work closely with Principal Investigators in the technical design of work plans to help ensure that these documents meet the Trustees’ needs. The Data Manager is responsible for assembling documents and data collected in support of the assessment (both current and historical) in an accessible and complete format for assessment purposes. Principal Investigators are responsible for project-specific design and implementation of the quality assurance/quality control (QA/QC) activities. The Quality Assurance Coordinator oversees QA program implementation, contributing to the work plan development, data review, and documentation processes. Specific responsibilities of the Hanford Quality Assurance Coordinator include:

- Annually reviewing the Hanford natural resource damage assessment QMP, revising it if changes are necessary, and obtaining appropriate document approvals.
- Overseeing the verification and validation of the historical and newly acquired data for the Hanford assessment.
- Identifying and delegating responsibility for responding to specific QA/QC needs, and ensuring timely answers to requests for guidance or assistance including interpretation of the Quality Management Plan and providing guidance on compliance.
- Ensuring that all work plans, Quality Assurance Project Plans, and standard operating procedures (SOPs) are technically reviewed and approved prior to collection and/or analysis of environmental data.
- Ensuring that problems and deficiencies identified in technical audits and data assessments are resolved.

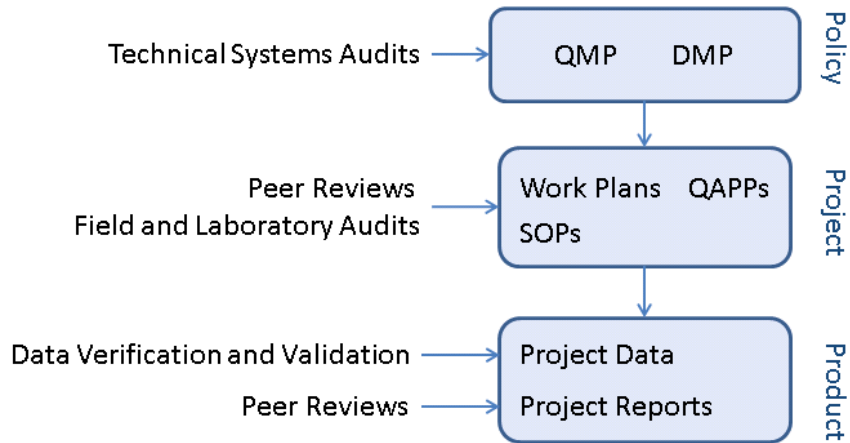
8.2 QUALITY SYSTEM DESCRIPTION

The goal of the Quality System is to ensure that the acquisition and use of environmental data, whether historical or generated under the oversight of the Hanford Trustees, includes sufficient up-front planning and review to ensure data quality is adequate to meet project goals. In order for any data to be useful for the natural resource damage assessment, the data must be of known and documented quality: it must have sufficient supporting documentation such that data users can evaluate whether the data meet their needs. This is achieved by ensuring that adequate quality assurance tools are used throughout the entire data collection and assessment process from initial planning through data usage. The tools used in the Quality System include:

- The Trustees’ Quality Management Plan (HNRTC 2011b);
- The Data Management Plan (HNRTC 2011a);
- Work plans including associated Quality Assurance Project Plans that may be developed to support assessment activities;
- Standard Operating Procedures;
- Peer reviews;
- Technical systems audits;
- Field and laboratory audits; and
- Data verification and validation.

Exhibit 8-2 depicts the relationships of these tools to one another. The Technical Working Groups, Data Manager, QA Coordinator, Principal Investigators and appropriate staff participate in and are responsible for the creation and implementation of each of these tools.

EXHIBIT 8-2 COMPONENTS OF THE QUALITY SYSTEM



Quality system components shall be consistent with, and supportive of, project objectives (e.g., they will have a graded approach as described in EPA 2001a). In other words, the level of application of quality system controls to an environmental data program can vary according to the intended use of the results and the degree of confidence needed in the quality of the results. For example, if historical data are being used to support planning for additional sampling, the degree of review and documentation may be less than the degree of review and documentation if historical data are to be used for injury determination.

Specifically, it is the responsibility of the QA Coordinator working with the TWG leads and Principal Investigators to ensure that the following objectives are achieved.

- All environmental data used and generated are of known and acceptable quality for the intended use. The data quality information developed with all environmental data is documented and available within the Data Management System (DMS).
- If new data are to be collected, the intended uses of the data are defined before the data collection effort begins so that appropriate QA measures can be applied to ensure a level of data quality commensurate with the project data objectives. The determination of this level of data quality takes into account the prospective data needs of secondary users. The assigned level of data quality, specific QA activities, and data acceptance criteria must be explicitly described in each individual Quality Assurance Project Plan.
- The general audit and data review procedures are stated during the planning process for the acquisition and use of any data used in the assessment process. The audits and data assessments should be documented and provided with the final data reports.

**8.3 DATA
GENERATION
AND
ACQUISITION**

NEW DATA GENERATION ACTIVITIES

All Hanford assessment projects that involve the generation of new environmental data (activities that involve the measurement, monitoring or collection of physical, chemical, or biological data) are required to document all aspects of their project's sampling design, sample collection, analysis, quality control, and data management activities in a work plan. Work plans should generally include, but are not necessarily limited to, the following elements:

- Cover page with title and date;
- Signatory page (including the Principal Investigator(s) and QA Coordinator);
- Background/introduction;
- Study measurement endpoints;
- Sampling design strategy (e.g., numbers and types of samples, sampling locations, sampling timing, and identification of analyses that will be conducted on the samples);
- Detailed methods, including new, study-specific SOPs or references to SOPs;
- A description of the statistical methods to be used in interpreting results;
- Provisions for health and safety, as applicable;
- Descriptions of all permissions needed to conduct the study (e.g., collection permits, paperwork documenting approval for work on-site at Hanford); and
- References.

Accompanying the work plan must be a study-specific QAPP that describes the methods for documenting and assessing environmental data, QA, QC, and other technical activities that must be implemented to ensure that the results of the work performed will satisfy the stated performance criteria. The QAPP should follow the EPA guidelines for QAPP preparation (EPA 2002b).

These work plans must be peer-reviewed, signed by the Principal Investigator(s) and the QA Coordinator, then approved by the Hanford Trustee Council. The QA Coordinator shall ensure appropriate QA/QC measures are included in all technical guidance documents. The Principal Investigator and the QA Coordinator are jointly responsible for the proper use of these documents, which is ensured through the training and audit processes. The Case Manager provides higher-level oversight to ensure documents are consistent with overall Trustee priorities.

HISTORICAL DATA ACQUISITION AND USE

If a historical dataset is identified that may be useful for formulating or performing a study, the request for potential inclusion of the dataset in the Trustees' DMS will be made through the development and submittal of a Data Acquisition Plan (DAP) as described in the Data Management Plan (HNRTC 2011a). Once implementation of the DAP has been approved by the Hanford Trustees, the dataset(s) will be obtained,

reviewed by the QA Coordinator, and assigned a QA Category, as described in the Trustees' Quality Management Plan (HNRTC 2011b).

Reports relying on historical data shall describe the data review procedures undertaken as part of report development, as well as the results of those efforts (i.e., whether or not specific data sets were included/excluded from use). The QA Coordinator shall advise as to the appropriate nature and type of data review procedures for use in connection with specific efforts.

**8.4 ASSESSMENT
AND
OVERSIGHT**

The appropriate type of assessment activity for particular projects will be determined during the planning process. Assessment tools include technical systems audits, laboratory and field audits, peer reviews, and data verification and validation. For evaluating particular activities, the work plan will describe the appropriate assessment tool and identify personnel responsibilities.

Data quality verification, validation, and assessment shall be consistent with *EPA Guidance on Environmental Data Verification and Data Validation (QA/G-8)* (EPA 2002c).

The QA Coordinator determines if appropriate actions have been implemented in response to assessment findings. The QA Coordinator, in a timely manner, determines the effectiveness of responses to assessments and maintains the documentation and correspondence relating to assessments and actions. Following any assessment event, the QA Coordinator prepares a written summation of needed changes and actions and then presents this summation in a timely manner to the Case Manager.

**8.5 DATA
VALIDATION
AND
USABILITY**

The purpose of data validation is to verify that the data are of known quality, are technically valid, are legally defensible, satisfy project objectives, and are usable for their intended purpose. Work plan Quality Assurance Project Plans shall describe the criteria that should be used for accepting, rejecting, or qualifying project data. Understanding the extent of validation of historic data is integral to evaluating their usability for natural resource damage assessment purposes and is an important aspect of the categorization of historical data described above. Overall, data quality verification and validation shall be consistent with *EPA Guidance on Environmental Data Verification and Data Validation (QA/G-8)* (EPA 2002c).

REFERENCES

- Abbotts and Weems. 2008. Hanford Remediation at 20 Years: A Glass Half-Empty. Remediation: Winter, 73-86.
- Alaska Statute (AS 41.17.950.). Alaska Statutes, Title 41, Chapter 17, Section 950. Definitions. Accessed at:
<<http://www.touchngo.com/lglcntr/akstats/statutes/title41/chapter17/section950.htm>.
- Allert, A.L., J.F. Fairchild, C.J. Schmitt, B.C. Poulton, W.G. Brumbaugh, and R.J. DiStefano. 2005. Longitudinal characterization of stream communities downstream of lead mining in the Missouri Ozarks. Abstracts, 26th Annual Meeting of SETAC North America Society of Environmental Toxicology and Chemistry, Baltimore, MD. November 13-17, p. 248.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for Ionizing Radiation. U.S. Department of Health and Human Services. September.
- Ballinger, M.Y. and R.B. Hall. 1991. A History of Major Hanford Facilities and Processes Involving Radioactive Material. Pacific Northwest Laboratory, PNL-6964 HEDR.
- Becker, G. 1983. Fishes of Wisconsin. Madison, Wisconsin: The University of Wisconsin Press.
- Besser, J.M., C.A. Mebane, D.R. Mount, C.D. Ivey, J.L. Kunz, I.E. Greer, T.W. May, and C.G. Ingersoll. 2007. Sensitivity of mottled sculpins (*Cottus bairdi*) and rainbow trout (*Oncorhynchus mykiss*) to acute and chronic toxicity of cadmium, copper, and zinc. Environmental Toxicology and Chemistry 24:1657-1665.
- Bjornstad, B.N. 2006. Past, Present, Future Erosion at Locke Island: Hanford Cultural Resources Project. Prepared for the U.S. Department of Energy under Contract DE-AC05-76RL01830.
- Brown, L. 2005. The Mottled Sculpin (*Cottus bairdi*). North American Native Fishes Association. Available at <<http://www.nanfa.org/articles/acmottledsculpin.shtml>>. Accessed November 28, 2010.
- Burk, K.W., Chamness, M.A., Fowler, R.A., Fritz, B.G., Hendrickson, P.L., Kennedy, E.P., Last, G.V., Poston, T.M., Sackschewsky, M.R., Scott, M.J., Snyder, S.F., Sweeney, M.D., and P.D. Thorne. 2007. Hanford Site National Environmental Policy Act (NEPA) Characterization. J.P. Duncan (Ed.). Pacific Northwest National Laboratory, PNNL-6415 Revision 18.
- Cary, A. "Washington will hold off on Hanford vit plant dispute resolution." Tri-City Herald.com. 4 Aug. 2012. Accessed 24 Oct. 2012 <[---

R-1](http://www.tri-</p></div><div data-bbox=)

cityherald.com/2012/08/04/2047964/washington-will-hold-off-on-hanford.html#storylink=misearch>.

- Chan, K.M.A., T. Satterfield, and J. Goldstein. 2011. Rethinking Ecosystem Services to Better Address and Navigate Cultural Values. *Ecol. Econ.* (2011), doi:10.1016/j.ecolecon.2011.11.011.
- Chronister, G.B. 2011. Strategies for Immobilization of Deep Vadose Contaminants at the Hanford Central Plateau (11503). Prepared for the U.S. Department of Energy, CHPRC-01182-FP Revision 0.
- Close, D.A. 2000. Pacific lamprey research and restoration project. Annual report 1998. Confederated Tribes of the Umatilla Indian Reservation, Pacific Lamprey Research and Restoration Project, Report to Bonneville Power Administration, Contract No. 00000248-1, Project No. 199402600, 94 electronic pages (BPA Report DOE/BP-00000248-1).
- Cohen, Felix S. 1982. Handbook of Federal Indian Law. Michie Bobbs-Merrill.
- Cole, C.R., Wurstner, S.K., Bergeron, M.P., Williams, M.D., and P.D. Thorne. 1997. Three-Dimensional Analysis of Future Groundwater Flow Conditions and Contaminant Plume Transport in the Hanford Site Unconfined Aquifer System: FY 1996 and 1997 Status Report. Pacific Northwest National Laboratory, PNNL-11801.
- Confederated Tribes of the Umatilla Indian Reservation (CTUIR). 2007. Preassessment Screen for the Hanford Facility. May 17.
- Confederated Tribes of the Umatilla Indian Reservation (CTUIR). 2012. CTUIR Tribal Ecosystem Services and Injury Assessment for Hanford NRDA. Draft Report to Hanford HNRTC. Prepared by Barbara Harper and Mathew Johnson. July 6.
- CRCIA. 1998. Screening Assessment and Requirements for a Comprehensive Assessment. Columbia River Comprehensive Impact Assessment. U.S. Department of Energy and Confederated Tribes of the Umatilla Indian Reservation. DOE/RL-96-16, Revision 1, UC 630.
- DeLonay, A. J., Brubaugh, W. G., Little, E. E., and L. Cleveland. 2001. Laboratory Evaluation of the Behavioral Avoidance-Preference Response of Chinook Salmon (*Oncorhynchus tshawytscha*) to Chromium in the Hanford Reach of the Columbia River, Washington, USA. Prepared for the Hanford Natural Resource Trustee Council. USGS Columbia Environmental Research Center.
- Dorts, J., P. Kestemont, M. Dieu, M. Raes, and F. Silvestre. 2010. Proteomic Response to Sublethal Cadmium Exposure in a Sentinel Fish Species, *Cottus gobio* [Abstract]. *Journal of Proteome Research*, American Chemical Society. Available at <<http://pubs.acs.org/doi/abs/10.1021/pr100650z?mi=sg7veh&af=R&pageSize=20&searchText=copper+dioxygenase>>. Accessed December 13, 2010.
- Downs, J.L. W.H. Rickard, C.A. Brandt, L.L. Cadwell, C.E. Cushing, D.R. Geist, R.M. Mazaika, D.A. Neitzel, L.E. Rogers, M.R. Sackschewsky, and J.J. Nugent. 1993. Habitat types on the Hanford Site: Wildlife and plant species of concern. Prepared

for the U.S. Department of Energy under Contract DE-AC06-76RLO 1830. PNL-8942. December.

- Downs, J.L., M.A. Simmons, J.A. Stegen, A.L. Bunn, B.L. Tiller, S.L. Thorsten, and R.K. Zufelt. 2004. Ecological Characterization Data for the 2004 Composite Analysis. PNNL-14884. Prepared for the U.S. Department of Energy under Contract DE-AC06-76RL018300. November.
- Duffield, J. et al. 1999. The Economic Value of Foregone Cultural Use: A Case Study of the Penobscot Nation. Final Report. June.
- Ennor, H.R. 1991. Birds of the Tri-Cities and Vicinity. Lower Columbia Basin Audubon Society, Richland, Washington.
- Farag, A.M., A.J. DeLonay, W.G. Brumbaugh, E.E. Little, L. Cleveland, and D.F. Woodward. 2000. The potential for chromium to adversely affect Chinook salmon (*Oncorhynchus tshawytscha*) in the Hanford Reach of the Columbia River, Washington, USA. Submitted to Dan Audet, U.S. Fish and Wildlife Service. 24 October. Available January 2013 at <http://www5.hanford.gov/pdw/fsd/AR/FSD0001/FSD0021/D8793346/D8793346_29024_63.pdf>.
- Farag, A.M., T. May, G. D. Marty, M. Easton, D. D. Harper, E. E. Little, L. Cleveland. 2006a. The effect of chronic chromium exposure on the health of Chinook salmon (*Oncorhynchus tshawytscha*). *Aquat. Toxicol.* 76: 246-257.
- Farag, A.M., D.D. Harper, L. Cleveland, W.G. Brumbaugh, and E.E. Little. 2006b. The potential for chromium to affect the fertilization process of Chinook salmon (*Oncorhynchus tshawytscha*) in the Hanford Reach of the Columbia River, Washington, USA. *Arch. Environ. Contam. Toxicol.* 50:575-579.
- Fitzner, R.E. and R.H. Gray, 1991. The Status, Distribution and Ecology of Wildlife on the U.S. DOE Hanford Site: A Historical Overview of Research Activities. *Environmental Monitoring and Assessment* 18:173-202.
- FLUOR. 2008. Technical Evaluation of the Interaction of Groundwater with the Columbia River at the Department of Energy Hanford Site, 100-D Area. SGW-39305.
- Freeman, E.J., R. Khaleel, and P.R. Heller. 2001. A Catalog of Vadose Zone Hydraulic Properties for the Hanford Site. Pacific Northwest National Laboratory, PNNL-13672.
- Future of Farming Project. 2008. "Competitive Advantages of Washington Agriculture - Current and Future." Accessed on the Web 11/7/2011. <<http://agr.wa.gov/fof/docs/Competitiveness.pdf>>.
- Gard, H.A. and R.M. Poet. 1992. "Archaeological Survey of the McGee Ranch Vicinity, Hanford Site, Washington." PNL, Richland, September 1992 PNL #8186

- Gee, G.W. and A.L. Ward. 2001. Vadose Zone Transport Field Study: Status Report. Pacific Northwest National Laboratory, PNNL-13679.
- Geist, D. R. 2000. Hyporheic discharge of river water into fall Chinook salmon (*Oncorhynchus tshawytscha*) spawning areas in the Hanford Reach, Columbia River. *Canadian Journal of Fisheries and Aquatic Sciences* 57: 1647-1656.
- Geist, D.R. and D.D. Dauble. 1998. Redd site selection and spawning habitat use by fall Chinook salmon: The importance of geomorphic features in large rivers. *Environmental Management* 22:655-669.
- Geist, D.R., E.V. Arntzen, Y.-J. Chien, T.P. Hanrahan, C.J. Murray, W.A. Perkins, M.C. Richmond, and Y. Xie. 2006. Spawning habitat studies of Hanford Reach fall Chinook salmon (*Oncorhynchus tshawytscha*). Prepared for U.S. Department of Energy, Bonneville Power Administration under contract no. 00000652. September.
- Geist, D.R., J. Jones, C.J. Murray, and D.D. Dauble. 2000. Suitability criteria analyzed at the spatial scale of redd clusters improved estimates of fall Chinook salmon (*Oncorhynchus tshawytscha*) spawning habitat use in the Hanford Reach, Columbia River. *Canadian Journal of Fisheries and Aquatic Sciences* 57:1636-1646. [Abstract only]
- Gephart, R.E. 2003. A short history of Hanford waste generation, storage, and release. PNNL-13605 Revision 4. October.
- Gerber, M.S. 2001. History of Hanford Site Defense Production (Brief). Prepared for the U.S. Department of Energy. February.
- Goswami, D. 2011. Sitewide groundwater and vadose zone project: Implementation plan for July 1, 2011 through June 30, 2013, Towards expedited cleanup of soil and groundwater at the Hanford Site. Washington State Dept. of Ecology Nuclear Waste Program.
- Gray, R.H. and D.D. Dauble. 1977. Checklist and relative abundance of fish species from the Hanford reach of the Columbia River. *Northwest Sci.* 51:208-215.
- Hall, R.B. 1991. Letter report: References for radioactive releases to the atmosphere from Hanford Operations, 1944-1957. PNL-7868 HEDR. November.
- Hall, J.A. (ed.). 1998. Biodiversity Inventory and Analysis of the Hanford Site, 1997 Annual Report. The Nature Conservancy of Washington, Seattle, Washington.
- Hallock, L.A. and McAllister, K.R. 2005. Pacific Treefrog. *Washington Herp Atlas*. <<http://www1.dnr.wa.gov/nhp/refdesk/herp/>>.
- Hartman, M.J. (Ed.). 2000. Hanford Site Groundwater Monitoring: Settings, Sources and Methods. Pacific Northwest National Laboratory, PNNL-13080, February.
- Hartman, M.J., Morasch, L.F., and W.D. Webber. 2001. Hanford Site Groundwater Monitoring for Fiscal Year 2000. Pacific Northwest National Laboratory, PNNL-13404. March.

- Hanford Natural Resource Trustee Council (HNRTC). 2000. Preassessment Screen Determination for the Hanford 1100 Area.
- Hanford Natural Resource Trustee Council. 2011a. Data Management Plan: Hanford Natural Resource Injury Assessment. Prepared by Industrial Economics, Inc. under U.S. Department of Energy Contract #DE-DT0001642. August 23. 42 pages.
- Hanford Natural Resource Trustee Council (HNRTC). 2011b. Quality Assurance Management Plan. Hanford Natural Resource Damage Assessment. Revision 0. Prepared by EcoChem, Inc. and Industrial Economics Inc. under U.S. Department of Energy Contract #DE-DT0001642. August 23.
- Hanford Natural Resource Trustee Council (HNRTC). 2011c. Guiding principles for protection of natural resources. Draft 4, March.
- Hanson, W.C. and R. L. Browning. 1959. Nesting Studies of Canada Geese on the Hanford Reservation, 1953-1956. *Journal of Wildlife Management* 23: 129-137.
- Harper, B. et al. 2002. The Spokane Tribe's multipathway subsistence exposure scenario and screen level RME. *Risk Analysis* 22(3):513-526.
- Hendricks, P. 1997. Status, Distribution, and Biology of Sculpins (Cottidae) in Montana: A Review. Montana Natural Heritage Program. Helena, MT. 22 pp. Available at <<http://mtnhp.org/animal/reports/fish/sculpin.html>>. Accessed November 30, 2010.
- Hope, S.J., and Peterson, R.E. 1996. Chromium in river substrate pore water and adjacent groundwater: 100-D/DR Area, Hanford Site, Washington. BHI-00778, Rev. 0. Prepared for the U.S. Department of Energy. Bechtel Hanford Inc., Richland, WA.
- Hulstrom, L.C. 2011. Data Summary Report for the Remedial Investigation of Hanford Site Releases to the Columbia River, Hanford Site, Washington. River Corridor Closure Contract, Washington Closure Hanford, WCH-398 Revision 0.
- Hulstrom, L.C. 2010. Data Quality Assessment Report for the Remedial Investigation of Hanford Site Releases to the Columbia River, Hanford Site, Washington. River Corridor Closure Contract, Washington Closure Hanford, WCH-381 Revision 1.
- Hulstrom, L.C., and B.L. Tiller. 2010. Field Summary Report for Remedial Investigation of Hanford Site Releases to the Columbia River, Hanford Site, Washington: Collection of surface water, pore water, and sediment samples for characterization of groundwater upwelling. River Corridor Closure Contract, Washington Closure Hanford, WCH-380 Revision 1.
- Industrial Economics, Inc. and Ridolfi Inc. 2012. Hanford Preliminary Estimate of Damages. Prepared for the Hanford Natural Resource Trustee Council.
- Ingersoll, C.G., C.D. Ivey, N.E. Kemble, D.R. Mount, J. Field, D.D. MacDonald, D. Smorong, and S. Ireland. 2008. Compilation of control performance data for laboratories conducting whole-sediment toxicity tests with the amphipod *Hyaella azteca* and the midge *Chironomus dilutus* (formerly *C. tentans*). Poster presented at SETAC North America 29th Annual Meeting, Tampa, Florida.

- Ingersoll, C., N. Wang, and B. Brumbaugh. 2012. U.S. Geological Survey (USGS) Quality Assurance Project Plan: Determining the effects of hexavalent chromium alone and with other stressors on native mussels inhabiting the Hanford Site located in eastern Washington State. Prepared for Joe Bartoszek, U.S. Fish and Wildlife Service. July 6.
- Jamison, J.D. 1982. Standardized input for Hanford Environmental Impact Statements Part II: Site description. PNL-3509 PT2, UC-11. July.
- Kagan, J. and J. Christy. 2010. Rare, Threatened and Endangered Species of Oregon. Oregon Biodiversity Information Center, Institute for Natural Resources Portland State University.
- Keller A.E., and S.G. Zam. 1991. The acute toxicity of selected metals to freshwater mussel, *Anodonta imbecilis*. *Environ Toxicol Chem* 10:539–546.
- Kiser, T. 2010. Continued discussion of 2009 fish sampling program. Overview of preliminary results of sturgeon contaminant burdens and histology. Hanford Large Sturgeon Sampling Expert Panel Workshop. August 4-5, 2010, Eastern Washington Field Office, Spokane, WA. Final Summary Report.
- Kunz, J.L., J.M. Besser, T.W. May, and C.G. Ingersoll. 2005. Toxicity of a Metal Mixture to Stream-Dwelling Sculpins and Crayfish. Poster presented at the 26th Annual Meeting of SETAC, 2005 13 – 17 November, Baltimore, Maryland, USA.
- Landeen, D.S., A.R. Johnson, and R.M. Mitchell. 1992. Status of Birds at the Hanford Site in Southeastern Washington. WHC-EP-0402, Revision 1. Westinghouse Hanford Company, Richland, Washington.
- Lee, D.R., D.R. Geist, K. Saldi, D. Hartwig, and T. Cooper. 1997. Locating Ground-Water Discharge in the Hanford Reach of the Columbia River. Pacific Northwest National Laboratory, PNNL-13404. Prepared for the U.S. Department of Energy. Contract DE-AC06-76RLO 1830
- MacDonald, D.D., J. Sinclair, M.A. Crawford, H. Prencipe, and M. Coady. 2012. Evaluation and interpretation of the sediment chemistry and sediment toxicity data for the Upper Columbia River. Prepared for Washington Department of Ecology Toxic Cleanup Program through Science Applications International Corporation. Bothell, Washington. Prepared by MacDonald Environmental Sciences Ltd., Nanaimo, British Columbia.
- Millennium Ecosystem Assessment. 2005. Multiple publications. Available online: <<http://www.maweb.org/en/index.aspx>>.
- Møller, A.P. and T.A. Mousseau. 2007. Species richness and abundance of forest birds in relation to radiation at Chernobyl. *Biology Letters of the Royal Society* 3: 483–486.
- Møller, A.P. and T.A. Mousseau. 2009. Reduced abundance of insects and spiders linked to radiation at Chernobyl 20 years after the accident. *Biology Letters*, doi:10.1098/rsbl.2008.0778.

- Møller, A.P. and T.A. Mousseau. 2010. Efficiency of bio-indicators for low-level radiation under field conditions. *Ecological Indicators*, doi:10.1016/j.ecolind.2010.06.013
- Morisset, Mason D. 1999. Recent Developments in Defining the Federal Trust Responsibility. April. Accessed July 5, 2012 at <<http://www.msaj.com/papers/43099.htm>>.
- Mueller R.P. and D.R. Geist. 1999. Steelhead spawning surveys near Lock Island, Hanford Reach of the Columbia River. PNNL-13055, Pacific Northwest National Laboratory, Richland, Washington.
- Mueller, R.P., G.K. Turner, B.L. Tiller, I.D. Welch, and M.D. Bleich. 2011. Assessment of the Species Composition, Densities, and Distribution of Native Freshwater Mussels Along the Benton County Shoreline of the Hanford Reach, Columbia River, 2004. PNNL-19933, Pacific Northwest National Laboratory, Richland, Washington.
- Nadasdy, P. 2003. *Hunters and Bureaucrats: Power, Knowledge and Aboriginal-State Relations in the Southwest Yukon*. Vancouver: UBC Press.
- Nez Perce Tribe. 2000. Preassessment Screen Determination for the Hanford 1100 Area (National Priority List) Site.
- Nez Perce Tribe. 2010. Nez Perce Perspective at Hanford. Prepared by: Gabriel Bohnee, ERWM Director, Jonathan Mathews, Environmental Specialist, Josiah Pinkham, Cultural Resource Specialist, Anthony Smith, Policy Analyst, and John Stanfill, Hanford Coordinator. Nez Perce Environmental Restoration Waste Management (ERWM). October 12.
- Newcomer, D.R. 1990. Evaluation of Hanford Site Water-Table Changes – 1980 to 1990. Pacific Northwest Laboratory, Richland, Washington. PNL-7498.
- Olson, P.A. and R.F. Foster. 1956. Effect of chronic exposure to sodium dichromate on young Chinook salmon and rainbow trout. In Hanford Biological Research Annual Report, 1955. Atomic Energy Commission, HW-41500, Richland, Washington.
- Palmerton Natural Resource Trustee Council (PNRTC). 2006. Palmerton Zinc Pile Superfund Site Natural Resource Damage Assessment Plan. February.
- Patton, G., D. Dauble, and C. McKinstry. 2007. Evaluation of early life stage fall Chinook salmon exposed to hexavalent chromium from a contaminated groundwater source. *Environ. Monit. Assess.* 133:285-294.
- Pauley, G.B. 1961. A tumorlike growth in the foot of a freshwater mussel (*Anodonta californiensis*). *J Fish Res Board Canada* 24:679-682.
- Pauley, G.B. 1967. Pacific northwest laboratory annual report for 1966 to the USACE Division of Biology and Medicine, Volume 1 Biological Sciences. BNWL-480, pp191-193.
- Pauley, G.B. 1968. Tumor incidence among freshwater mussel populations. In Pacific Northwest Laboratory Annual Report For 1967 to the USAEC Division of Biology

- and Medicine. Volume I. Biological Sciences, pp. 9.33-9.34. Eds. R. C Thompson, P. Teal, and E. G. Swezea. BNWL-714. Peterson, R.E., and M.P. Connelly. 2001. Zone of Interaction Between Hanford Site Groundwater and Adjacent Columbia River. Pacific Northwest National Laboratory, PNNL-13674.
- Peterson, R.E. and M.P. Connelly. 2001. Zone of Interaction Between Hanford Site Groundwater and Adjacent Columbia River: Progress Report for Groundwater/River Interface Task Science and Technology Groundwater/Vadose Zone Integration Project. Pacific Northwest National Laboratory, PNNL-13674.
- Peterson, R.E. and V.G. Johnson. 1992. Riverbank Seepage of Groundwater Along the 100 Areas Shoreline, Hanford Site. WHC-EP-0609, UC-703.
- Petty, J.T. and G.D. Grossman. 1996. Patch selection by mottled sculpin (*Pices: Cottidae*) in a southern Appalachian stream. *Freshwater Biology* 35:261-276.
- Petty, J.T. and G.D. Grossman. 2004. Restricted movement by mottled sculpin (*pices: cottidae*) in a southern Appalachian stream. *Freshwater Biology* 49:631-645.
- Petty, J.T. and G.D. Grossman. 2010. Giving-up densities and ideal pre-emptive patch use in a predatory benthic stream fish. *Freshwater Biology* 55:780-793.
- Poston, T.M., J.D. Downs, and R.L. Dirkes, (eds.). 2010. Hanford Site Environmental Report for Calendar Year 2009. PNNL-19455. September.
- Poston, T.M., R.W. Hanf, R.L. Dirkes, and L.F. Morasch (eds.). 2006. Hanford Site Environmental Report for Calendar Year 2005. PNNL-15892. September.
- Qafoku, N.P., P.E. Dressel, J.P. McKinley, E.S. Ilton, W. Um, C.T. Resch, R.K. Kukkadapu, and S.W. Peterson. 2011. Geochemical Characterization of Chromate Contamination in the 100 Area Vadose Zone at the Hanford Site (Part 2). Pacific Northwest National Laboratory. PNNL-17865. Prepared for the U.S. Department of Energy under Contract DE-AC06-76RL01830.
- Reidel, S.P., V.G. Johnson, and F.A. Spane. 2002. Natural gas storage in basalt aquifers of the Columbia Basin, Pacific Northwest USA: a guide to site characterization. Pacific Northwest national Laboratory, Richland, WA, PNNL-13962.
- Reidel, Stephen P. and Karl R. Fecht. 1994. Geologic Map of the Richland 1:100,000 Quadrangle, Washington
- Relander, C. 1986. *Drummers and Dreamers*. Caldwell, Idaho: The Caxton Printers.
- Ridolfi, Inc. 2006. Preassessment screen for the Hanford facility. Public review draft. Prepared for the Confederated Tribes and Bands of the Yakama Nation. October 18.
- Rogers, L.E., N. Woodley, J.K. Sheldon, and V.A. Uresk. 1978. Darkling beetle populations (*Tenebrionidae*) of the Hanford Site in Southcentral Washington. Battelle Pacific Northwest Laboratories, Richland, Washington. PNL-2465.
- Saab, V.A. and T.D. Rich. 1997. Large-scale Conservation Assessment for Neotropical Migratory Land Birds in the Interior Columbia River Basin. General Technical

- Report PNW-GTR-399. U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station, Portland, Oregon.
- Sackschewsky, M.R. and J.L. Downs. 2001. Vascular plants of the Hanford Site. PNNL-13688. Prepared for the U.S. Department of Energy under Contract DE-AC06-76RL01830. September.
- Safriel, U. and Z. Adeel. 2005. Chapter 22: Dryland Systems. In: Ecosystems and Human Well-Being: Current State and Trends. Millennium Ecosystem Assessment. Island Press, Washington, D.C.
- Selby, J.M. and J.K. Soldat. 1958. Summary of Environmental Contamination Incidents at Hanford 1952-1957. Hanford Atomic Products Operation, performed under Contract W-31-109-Eng-52 between the Atomic Energy Commission and General Electric Company, HW-54636.
- Shearer, J.P. 2012. Hanford Site Waste Management Units Report. CH2M Hill Plateau Remediation Company. DOE-RL-88-30, Revision 21. Prepared for the U.S. Department of Energy under Contract DE-AC06-08RL-14788.
- Sochová, I., J. Hofman, and I. Holoubek. 2006. Using nematodes in soil ecotoxicology. *Environment International* 32: 374-383.
- Spooner, D.E. and C.C. Vaughn. 2006. Context-dependent effects of freshwater mussels on stream benthic communities. *Freshwater Biology* 51:1016-1024.
- Stratus Consulting. 2009. Hanford Site Natural Resource Damage Assessment, Phase 1 Summary Report. Prepared for the Hanford Natural Resource Trustees. July 1.
- Stevens, D.G. and G.A. Chapman. 1984. Toxicity of trivalent chromium to early life stages of steelhead trout. *Environmental Toxicology and Chemistry* 3:125-133.
- Stratus Consulting Inc. (Stratus). 2009. Hanford Site Natural Resource Damage Assessment, Phase 1 Summary Report. Prepared for the Hanford Natural Resource Trustees.
- The Nature Conservancy (TNC). 1999. Biodiversity inventory and analysis of the Hanford Site. Final Report: 1994-1999. Prepared for the U.S Department of Energy under grant award DE-FG06-94RL12858. October.
- The Nature Conservancy (TNC). 2003. Biodiversity studies of the Hanford Site. Final Report. Prepared for the U.S Department of Energy and the U.S. Fish and Wildlife Service, Hanford Reach National Monument. August.
- Tiller B.L., G.W. Patton, D.D. Dauble, and T.M. Poston. 2004. Monitoring tissue concentrations of chromium and fish condition in juvenile fall chinook salmon from the Hanford Reach of the Columbia River. PNNL-14473, Pacific Northwest National Laboratory, Richland, WA.
- Turner, N. 2005. *The Earth's Blanket: Traditional Teachings for Sustainable Living*. Washington: University of Washington Press.

- Upper Columbia White Sturgeon Recovery Initiative (UCWSRI). 2002. Upper Columbia White Sturgeon Recovery Plan. November.
- U.S. Army Corps of Engineers (USACE). 2011b. "Lake Wallula Overview." Accessed on the Web 11/5/2011.
<http://www.nww.usace.army.mil/corpsoutdoors/siteMenu.asp?lake_id=100>. ¹⁴¹
- U.S. Department of Agriculture (USDA). 2011. "Washington's 2010 Agricultural Value Second Highest on Record." Press Release in conjunction with National Agricultural Statistics Service (NASS). 2011. Accessed on the Web 11/7/2011.
<<http://agr.wa.gov/AgInWa/docs/2010WaAgValuesUSDAPressRelease.pdf>>
- U.S. Department of Energy (DOE). Multiple Years. Annual Environmental Reports.
- U.S. Department of Energy (DOE). 1987. Final Environmental Impact Statement, Disposal of Hanford Defense High-Level, Transuranic and Tank Wastes, Hanford Site, Richland, Washington. DOE/EIS-0113, Vol. I-III, U.S. Department of Energy, Washington, D.C.
- U.S. Department of Energy (DOE). 1988. Consultation Draft: Site Characterization Plan, Reference Repository Location, Hanford Site, Washington. DOE/RW-0164, U.S. Department of Energy, Washington, D.C.
- U.S. Department of Energy (DOE). Multiple Years (1996-present). Hanford Site Groundwater Monitoring Reports.
- U.S. Department of Energy (DOE), U.S. Department of the Interior (Bureau of Land Management, Bureau of Reclamation, and U.S. Fish and Wildlife Service), Nez Perce Tribe Department of Environmental Restoration and Waste Management, and the Confederated Tribes of the Umatilla Indian Reservation. 1999. Final Hanford Comprehensive Land-Use Plan Environmental Impact Statement (HCP EIS), Hanford Site, Richland, Washington.
- U.S. Department of Energy (DOE). 2002. History of the Plutonium Production Facilities at the Hanford Site Historic District, 1943-1990. U.S. Department of Energy, Richland Operations Office, DOE/RL-97-1047. June.
- U.S. Department of Energy (DOE). 2006a. The Second CERCLA Five Year Review Report for the Hanford Site. DOE/RL-2006-20 Revision 1.
- U.S. Department of Energy (DOE). 2007a. Risk Assessment Report for the 100 Area and 300 Area Component of the River Corridor Baseline Risk Assessment. DOE/RL-2007-21. Draft A. June.
- U.S. Department of Energy (DOE). 2007b. Hanford Environmental Information System (HEIS) Result Table Data Dictionary. November.

¹⁴¹ Information for specific Lake Wallula recreation sites, including recreation activities and facilities, can be found by clicking on specific sites in the recreation site menu on the left side of the "Lake Wallula Overview" page.

- U.S. Department of Energy (DOE). 2008. Remedial Investigation Work Plan for Hanford Site Releases to the Columbia River. DOE/RL-2008-11. September.
- U.S. Department of Energy (DOE). 2009. Central Plateau Cleanup Completion Strategy. DOE/RL-2009-81, Revision 0. September.
- U.S. Department of Energy (DOE). 2010. Hanford Site Cleanup Completion Framework. DOE/RL-2009-10, Revision 0. July.
- U.S. Department of Energy (DOE). 2010a. Long-range deep vadose zone program plan. DOE/RL-2010-89, Revision 0.
- U.S. Department of Energy (DOE). 2010b. Radionuclide Air Emissions Report for the Hanford Site, Calendar Year 2009. DOE/RL-2010-17 Revision 0.
- U.S. Department of Energy (DOE). 2011a. Columbia River Component Risk Assessment Volume I: Screening-Level Ecological Risk Assessment. DOE/RL-2010-117. Volume I, Part 2, Draft A. September.
- U.S. Department of Energy (DOE). 2011b. River Corridor Baseline Risk Assessment Volume I: Ecological Risk Assessment. DOE/RL-2007-21. Volume I, Part 1, Draft C. August.
- U.S. Department of Energy (DOE). 2011c. Hanford Site Groundwater Monitoring Report for 2010. Revision 0. DOE/RL-2011-01. August.
- U.S. Department of Energy (DOE). 2011d. Draft Hanford Site Third CERCLA Five-Year Review Report, Richland Operations Office. July.
- U.S. Department of Energy (DOE). 2011e. Hanford Site Environmental Report for Calendar Year 2010. 2011. TM Poston, JP Duncan, and RL Dirkes (Eds.). Prepared for U.S. DOE by personnel from Pacific Northwest National Laboratory. September.
- U.S. Department of Energy (DOE). 2011f. Projects & Facilities: 618-10 and 618-11 Burial Grounds. Accessed at <http://www.hanford.gov/page.cfm/618BurialGrounds> on October 18, 2012.
- U.S. Department of Energy (DOE). 2011g. Cleanup Progress at Hanford. Accessed on October 18, 2012 at <http://www.hanford.gov/news.cfm/DOE/Cleanup%20Progress%20at%20Hanford-04-2012.pdf>.
- U.S. Department of Energy (DOE). 2012. Hanford Site Cleanup Completion Framework. DOE/RL-2009-10, Revision 1, Draft. May.
- U.S. Department of the Interior (USDOI) and U.S. Fish and Wildlife Service (US FWS). "Hanford Reach National Monument Final Comprehensive Conservation Plan and Environmental Impact Statement. Adams, Benton, Grant and Franklin Counties, Washington." FWS Website for Hanford Reach National Monument. 2008. Accessed on the Web 11/7/2011. <http://www.fws.gov/hanfordreach/documents/finalccp/final-ccp.pdf>.

- U.S. Environmental Protection Agency (EPA). 2001a. EPA Requirements for Quality Management Plans (QA/R-2). EPA Requirements for Quality Management Plans (QA/R-2). EPA-240-B-01-002. Washington, D.C.
- U.S. Environmental Protection Agency (EPA). 2001b. USDOE First Five Year Review Report. Region 10, Hanford Project Office. April.
- U.S. Environmental Protection Agency (EPA). 2002a. Columbia River Basin Fish Contaminant Survey, 1996-1998. EPA 910-R-02-006.
- U.S. Environmental Protection Agency (EPA). 2002b. Guidance for Quality Assurance Project Plans (QA/G-5). EPA-240-R-02-009. Washington, D.C.
- U.S. Environmental Protection Agency (EPA). 2002c. Guidance on Environmental Data Verification and Data Validation (QA/G-8). EPA-240-R-02-004. Washington, D.C.
- U.S. Environmental Protection Agency (EPA). 2012c. National Priorities List (NPL). Available at: <<http://www.epa.gov/superfund/sites/npl/>>.
- U.S. Environmental Protection Agency (EPA), US Department of Energy, Washington Department of Health, and Washington Department of Energy. Undated. "Current Uses and Restrictions at the Hanford Reach National Monument." Accessed on the Web 11/7/2011.
<[http://yosemite.epa.gov/R10/CLEANUP.NSF/6ea33b02338c3a5e882567ca005d382f2f133ac95a7d2684882564ff0078b367/\\$FILE/National%20Monument.pdf](http://yosemite.epa.gov/R10/CLEANUP.NSF/6ea33b02338c3a5e882567ca005d382f2f133ac95a7d2684882564ff0078b367/$FILE/National%20Monument.pdf)>.
- U.S. Fish and Wildlife Service (US FWS). 2002. Hanford Reach National Monument Saddle Mountain National Wildlife Refuge Visitor Services Review. USFWS Website for Hanford Reach National Monument. July 2002. Accessed on the Web 11/7/2011. <<http://www.fws.gov/mcriver/documents/regulations/2011-hanford-reach.pdf>>
- U.S. Fish and Wildlife Service (US FWS). 2008. Hanford Reach National Monument Final Comprehensive Conservation Plan and Environmental Impact Statement. Adams, Benton, Grant, and Franklin Counties, Washington. August.
- U.S. Fish and Wildlife Service (US FWS). 2011a. Hanford Reach National Monument: Plan Your Visit. Available at <<http://www.fws.gov/hanfordreach/visit.html>>. Viewed on November 7, 2011.
- U.S. Fish and Wildlife Service (US FWS). 2011b. Hanford Reach National Monument Hunting Regulations. USFWS Website for Mid-Columbia River National Wildlife Refuges. 2011. Accessed on the Web 11/7/2011.
<<http://www.fws.gov/mcriver/documents/regulations/2011-hanford-reach.pdf>>.
- U.S. Fish and Wildlife Service (US FWS). 2011c. Hanford Reach National Monument: Wildlife and Habitats. Available at
<<http://www.fws.gov/hanfordreach/wildlife.html>>. Viewed on October 23, 2011.
- Van Verst, S.P., C.L. Albin, G.W. Patton, M.L. Blanton, T.M. Poston, A.T. Cooper, and E.J. Antonia. 1998. Survey of Radiological Contaminants in the Near-Shore

- Environment at the Hanford Site 100-N Reactor Area. Prepared for the U.S. Department of Energy under Contract DE-AC06-76RLO 1830. PNNL-11933, Pacific Northwest National Laboratory.
- Warren, J.L. 1980. Vegetation Maps of the Hanford Reach, Columbia River. Prepared for the U.S. Army Corps of Engineers, Seattle District, Seattle, Washington, by Pacific Northwest National Laboratory, Richland, WA.
- Washington Closure Hanford (WCH). 2008. Inter-Areas Component of the River Corridor Baseline Risk Assessment Sampling Summary. WCH-274, Revision 0. February.
- Washington Department of Fish and Wildlife (WDFW). "Regulations & Seasons: Summary of General Hunting Season Dates." Hunting Section of WDFW Website. 2011. Accessed on the Web 11/7/2011. <http://wdfw.wa.gov/hunting/regulations/summary_hunting_dates.html>
- Washington Native Plant Society. 2008. Threats to the Shrub-Steppe. <http://www.wnps.org/ecosystems/shrubsteppe_eco/threats.htm>. Viewed on October 23, 2011.
- Washington State Department of Agriculture (WSDA). "Agriculture: A Cornerstone of Washington's Economy." 2011. Accessed on the Web 11/7/2011. <<http://agr.wa.gov/AgInWa/>>.
- Washington State Department of Agriculture (WSDA). "Agriculture: A Cornerstone of Washington's Economy - Maps." 2011. Accessed on the Web 11/7/2011. <http://agr.wa.gov/AgInWa/Crop_Maps.aspx> />.
- Washington State Department of Agriculture (WSDA). "Washington Agriculture: Strategic Plan 2020 and Beyond." Future of Farming Project. 2009. Accessed on the Web 11/7/2011. <<http://agr.wa.gov/fof/docs/FoFStrategicPlan.pdf>>.
- Weiss, S.G. and R.M. Mitchell. 1992. A synthesis of ecological data from the 100 Areas of the Hanford Site. Prepared for the U.S. Department of Energy under contract DE0AC06-87RL10930. WHC-EP-0601. October.
- Wydoski, R.S. and R.R. Whitney. 2003. Inland Fishes of Washington. Second edition, revised and expanded. American Fisheries Society, in association with University of Washington Press.
- Yakama Nation. 2010. Yakama Nation Tribal Narrative. Hanford Site Natural Resource Damage Assessment, Human Use Technical Working Group. Yakama Nation ERWM. December 8.
- Yeardley, R.G. Jr. 2000. Use of small forage fish for regional streams wildlife risk assessment: Relative bioaccumulation of contaminants. Environmental Monitoring and Assessment 65:559-585.

Zachara, J., C. Liu, C. Brown, S. Kelly, J. Christensen, J. McKinley, J.A. Davis, J. Serne, E. Dresel, and W. Um. 2007. A site-wide perspective on uranium geochemistry at the Hanford Site. Pacific Northwest National Laboratory, PNN-17031. October.

APPENDIX A | THE FOUR HANFORD NPL SITES

On November 3, 1989, Hanford was added to the NPL as four separate sites: the 100 Area, 200 Area, 300 Area, and 1100 Area (see Exhibits B-1 through B-4, below).⁸³ In order to coordinate response actions, each of these sites was further subdivided into operable units (OUs), based on geographic area or common waste sources. A total of 1,200 waste management units have been identified throughout the Hanford Site and are grouped among the four NPL sites (DOE 2006a).

Cleanup efforts for the remaining Hanford Site contamination are organized into three major components: the River Corridor (including the 100 and 300 Areas), the Central Plateau (primarily the 200 Area), and tank waste. Cleanup of the Site is a particularly large and complex effort, dependent on many dozens of individual decision steps, stakeholder coordination, sustained funding, and the ability to address complex technical challenges. Full remediation of the NPL sites is expected to extend over the next 40 to 50 years; however, timelines are difficult to determine, due to the factors discussed above (DOE 2012).

Additional summary information describing the four Hanford NPL sites and the current status of remediation efforts is provided below. More detailed information can be found at <http://www.hanford.gov/>.

AREA DESCRIPTIONS

100 AREA

The 100 Area contains the remnants of Hanford's nine nuclear reactors, spread over six reactor sites (B/C, K, N, D, H, and F). The footprint covers about 26 sq. mi. of land area, 11 sq. mi. of which is contaminated (including groundwater contamination and waste disposal locations) (DOE 2011d). Much of the contamination is the result of the reactors discharging cooling water into various trenches, cribs, ponds, and other waste sites. Additional contamination sites include an array of structures such as buildings and buried pipelines, and two basins that currently store spent nuclear fuel from the reactors.

The 100 Area site is divided into 22 OUs. Five OUs address groundwater contamination, and the remaining contamination is grouped geographically into 17 OUs, which encompass the Area's 400 soil, structure, debris, or burial ground waste sites. With the exception of OUs that are designated as "isolated units" (IUs), the 100 Area OUs are associated with reactor areas (EPA 2001b).

⁸³ Note that the areas captured within the 100, 200, 300, and 1100 designations have changed slightly over time.

200 AREA

Used for chemical processing and waste management, the 200 Area NPL site covers about 75 sq. mi. of land area, and consists of large amounts of radioactive, hazardous, and mixed, soil and groundwater contamination. The 200 Area, also referred to as the Central Plateau, contains about 1,000 structures, including the Plutonium Finishing Plant (PFP), and five chemical processing facilities, called “canyon” facilities (T, B, U and S, or REDOX plant, and the Plutonium/Uranium Extraction (PUREX) plant). The approximately 700 soil waste sites, with associated structures and facilities, are divided into 23 soil OUs organized by discharge type (e.g., cooling water, solid waste) and waste site type (e.g., pond, crib, ditch). Additionally, there are four groundwater OUs, two each in the 200-East and 200-West areas.

The 200 Area is split into the Inner and Outer Areas. The Inner Area will be dedicated to waste management and containment of contamination from Hanford Site cleanup actions, and will remain under Federal ownership (DOE 2011d). The Outer Area includes the areas surrounding the Inner Area in the Central Plateau. Cleanup of the Outer Area to standards similar to those being achieved in the River Corridor is expected (DOE 2011d).

A key feature of the characterized waste in the 200 Area are the tank farms where highly radioactive liquid effluents are stored in single and double shelled underground tanks. There are 177 tanks, some of which have are known to have leaked (DOE 2011d). One of the double-shelled tanks is now known to be leaking, and a number of the single-shelled tanks are known or suspected to be leaking. 200 Area tank wastes remain a major clean up and remediation challenge (Abbotts and Weems 2008). Additionally, a number of trenches (associated with canyon facility operations), ponds (where cooling water was discharged in the Outer Area), and other solid waste burial grounds and pipelines are located in the 200 Area (DOE 2009).

300 AREA

The 300 Area NPL site consists of a quarter-mile industrial complex, with contamination stemming from multiple unlined liquid disposal areas, burial grounds, landfills, and other miscellaneous disposal sites. The site is split into three OUs, two that address soil contamination areas (300-FF-1 and 300-FF-2), and one that addresses groundwater contamination (300-FF-5). The primary contaminant in the 300 Area is uranium from the fuel fabrication process (EPA 2001b).

1100 AREA

The 1100 Area NPL site consists of two non-adjacent areas covering a total of just less than 5 sq. mi. The first area, located adjacent to the City of Richland, is split into three OUs (1100-EM-1, 1100-EM-2, and 1100-EM-3), and the second portion, located on the Arid Land Ecology (ALE) Reserve is a single OU (1100-IU-1). The portion of the site near Richland was historically used as a warehousing, vehicle maintenance, and transportation distribution center, and was known to be a dumping area for up to 15,000 gallons of waste battery acid from these operations. The site is located in close proximity to the Richland groundwater wells that supply drinking water to the City. The ALE site was a former NIKE missile base. Waste sites discovered in these OUs include the landfills, burn pits, solvent and fuel tanks, and a TCE groundwater plume (EPA 2001b).

HANFORD SITE AREAS NOT LISTED

Two additional numbered areas were excluded from NPL designation. The first of these, the 400 Area, contains the Fast Flux Test Facility (FFTF), a nuclear research and test reactor. The FFTF underwent destruction and demolition and was placed under long-term surveillance and maintenance in 2009. The 600 Area is defined as all portions of the Hanford Site not included in the 100, 200, 300, 400, or 1100 Areas. All waste sites potentially falling in the 600 Area were included in one of the other four NPL sites, due to proximity or likeliness to other waste sites (DOE 2006a).

In summary, over 40 years of plutonium production activity at Hanford resulted in (DOE 2011g):

- 586-sq.-mi. footprint requiring cleanup;
- 2,300 tons of spent nuclear fuel (stored near the Columbia River);
- 20 tons of leftover plutonium in the Plutonium Finishing Plant;
- 1,012 waste sites, 522 facilities, and nine plutonium production reactors (near the Columbia River) requiring cleanup;
- More than 100 sq. mi. of groundwater contaminated;
- 53 million gallons of waste in 177 underground tanks, 67 of which may have leaked;
- 15,000 cubic meters of buried or stored plutonium-contaminated waste on site;
- 850 waste sites and 970 facilities on the Central Plateau requiring cleanup.

EXHIBIT A-1 HANFORD NPL SITE DESIGNATIONS (DOE 2011D)

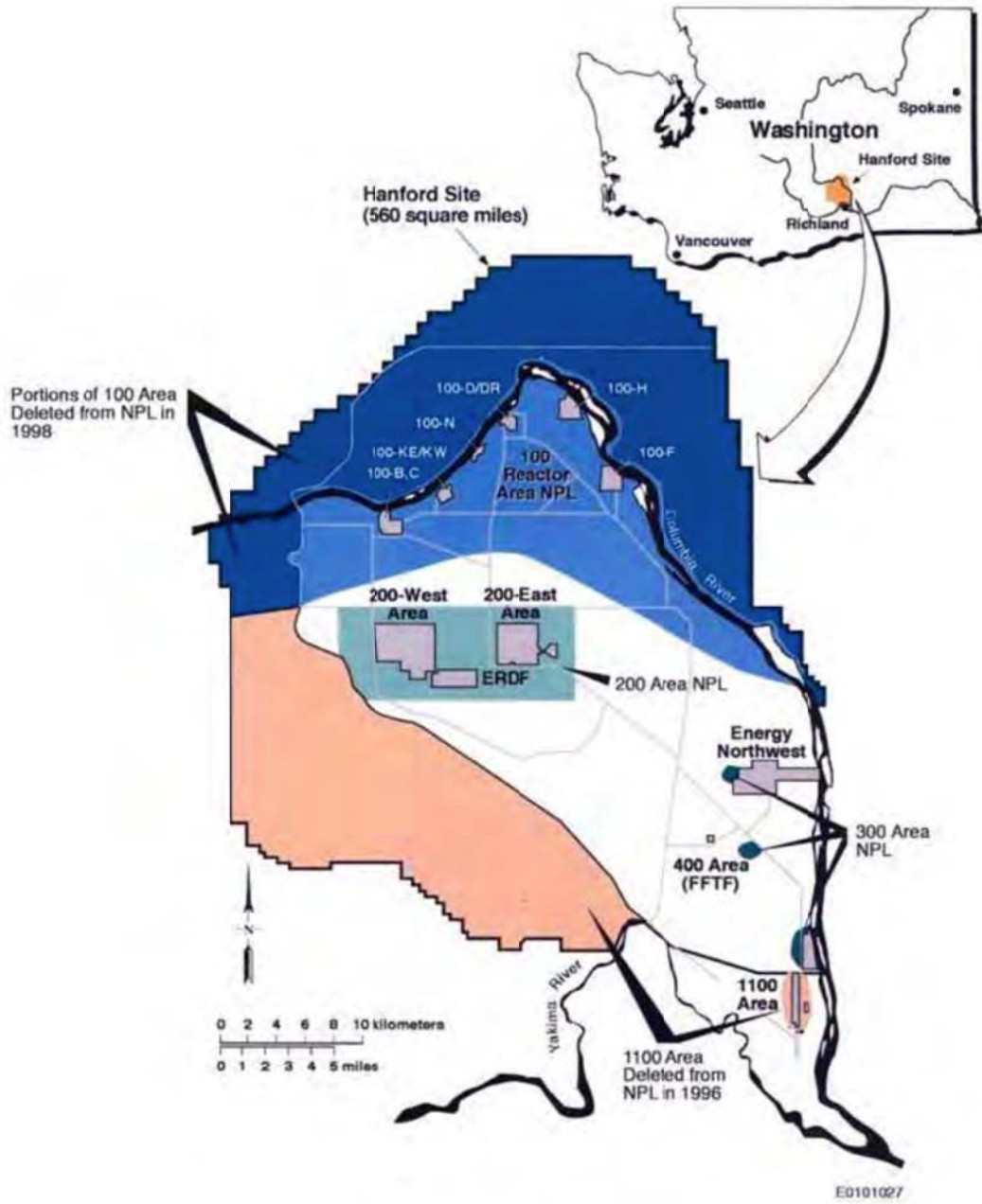
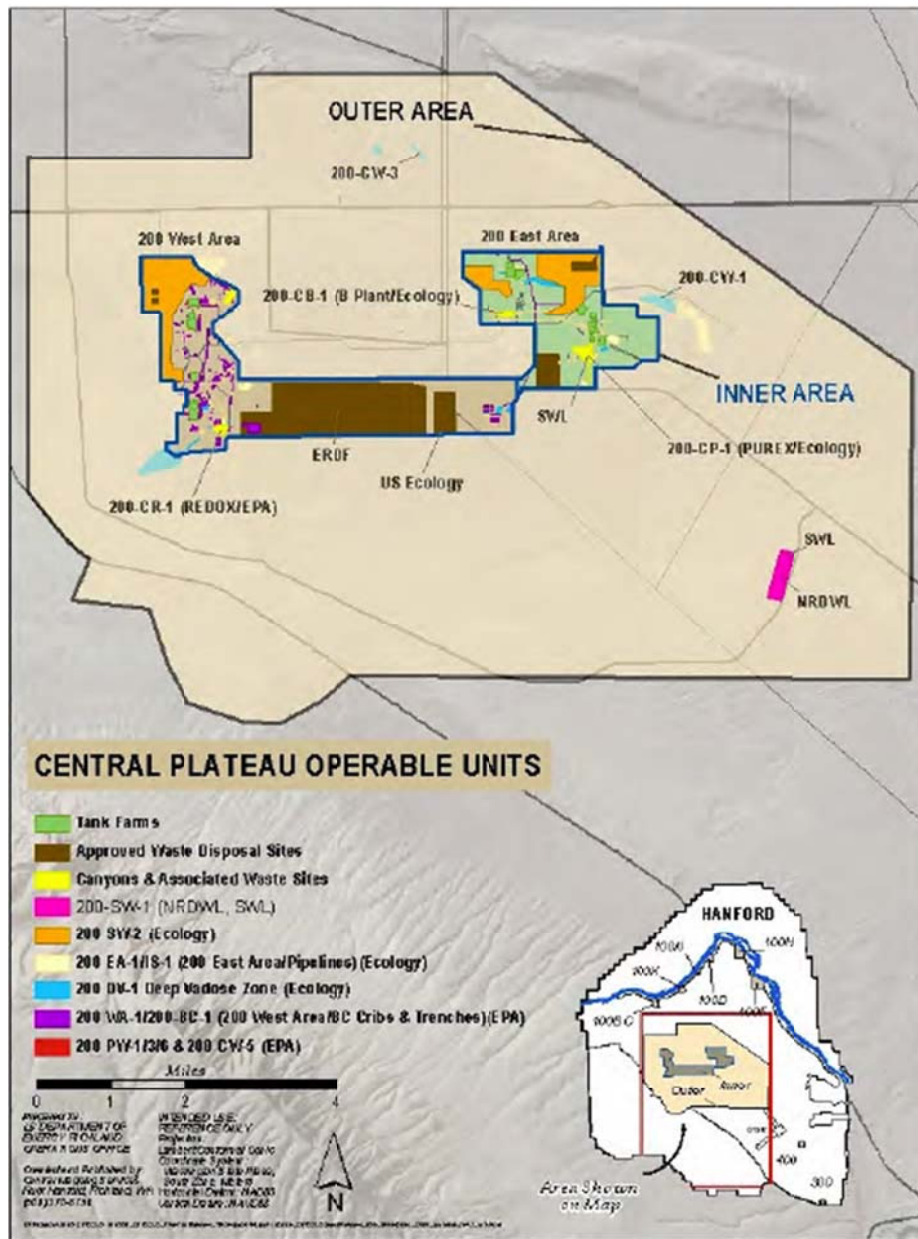
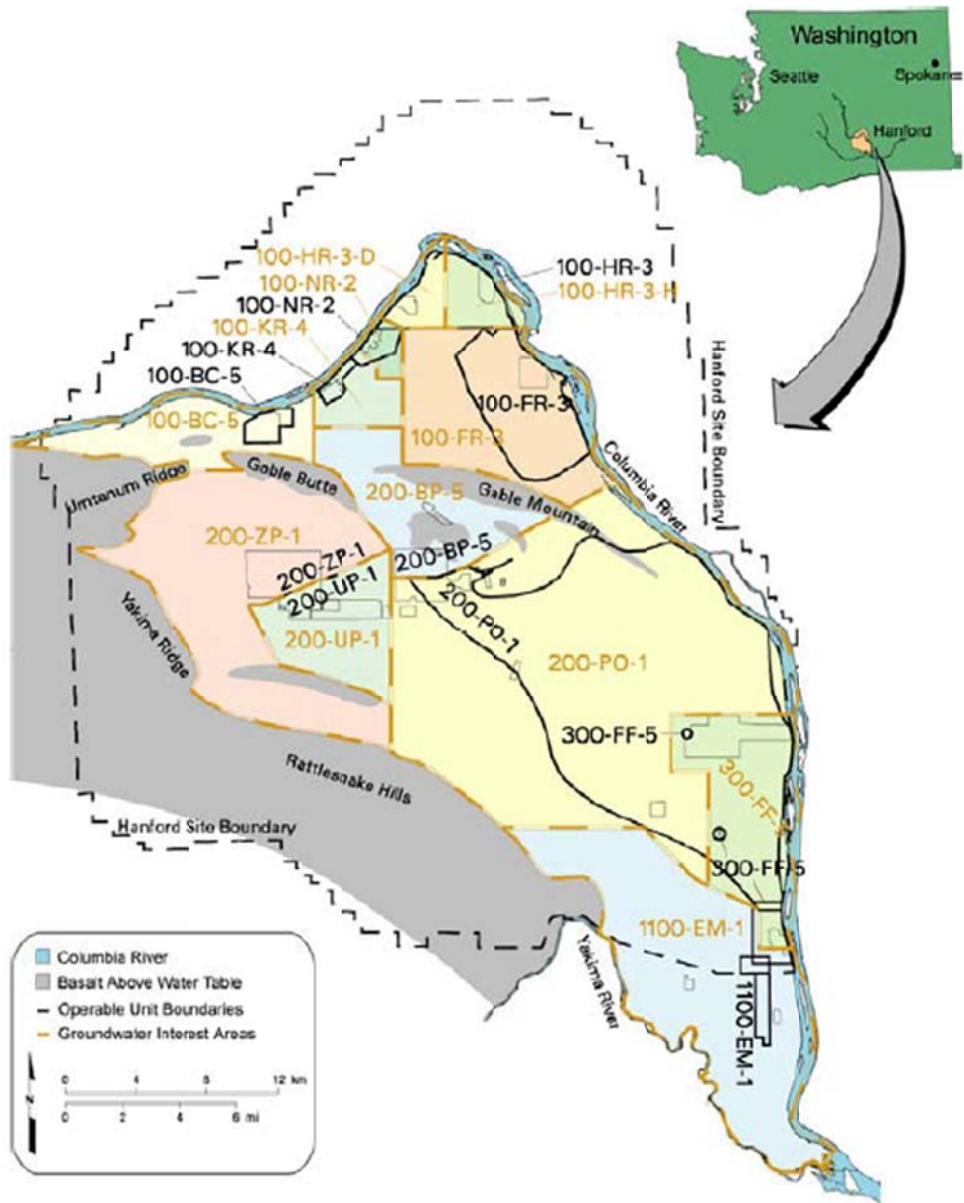


EXHIBIT A-3 CENTRAL PLATEAU SOURCE OPERABLE UNITS (DOE 2011D)



Note: Not all operable units are shown. Refer to Exhibit B-6, “200 Area Operable Units”, for a full list of operable units in the inner and outer areas.

EXHIBIT A-4 MAP OF GROUNDWATER OPERABLE UNITS IN THE HANFORD NPL SITES (DOE 2006B)



**SITE
ASSESSMENT
AND
REMEDATION**

After listing on the NPL, a remedial investigation/feasibility study (RI/FS) is performed. The RI is a mechanism for collecting data to characterize site conditions, determine the nature of the waste, assess HH&E risk, and test treatment options; the FS develops, screens, and evaluates alternative remedial actions. The RI/FS are conducted concurrently, each utilizing information from the other to maximize the efficiency of data collection. The RI/FS process is a five-phase approach: 1) scoping; 2) site characterization; 3) development and screening of alternatives; 4) treatability investigations; and 5) detailed analysis (EPA 2011).

From the information recorded in the RI/FS report, a Record of Decision (ROD) is generated. The ROD is a public document that explains which cleanup alternatives will be pursued. The ROD documents site history, site description, site characteristics, community participation, enforcement activities, past and present activities, contaminated media, contaminants present, scope and role of response action, and the remedy selected for cleanup.

A summary of remediation status by NPL Site and OU follows.

100 AREA

Closure of the 100 Area is planned based on five decision areas, encompassing boundaries beyond individual OUs and waste sites. These areas are the 100 B/C Area, 100-N Area, 100-D and H Areas, and the 100-F Area combined with 100-IU2/6. These decision areas include the entirety of the 100 Area NPL site (see Exhibit B-1) (DOE 2012, p. 29). Final RODs have not yet been issued for these decision areas. However, eight RODs for interim actions have been issued, and as of July 2011, RI/FSs were being developed in support of final RODs (DOE 2011d). Five interim RODs address soil contamination, one addresses K-Basins spent fuel removal, and two address groundwater. Multiple other CERCLA documents have been issued to address individual building demolition and cleanup of various 100 Area structures (DOE 2011d).

Decision documents have been issued for 100-HR-1, 100-IU-1, 100-HR-3, K Basins (100-KR-2), 100-NR-1 and 100-NR-2, and 100-BC-1. Additionally, “Remaining Sites” and “Burial Grounds” RODs have been issued to cover various waste sites within the 100 Area OUs. A “Remaining Sites” ROD, designed to be inclusive of all 100 Areas not already covered by an existing decision document, selects removal, treatment, disposal, backfill and re-vegetation for multiple 100 Area waste sites. Groundwater has been addressed in various pump-and-treat systems, while new methods (injection) are being tested for the 100-N Area.

To date, a majority of high-priority 100 Area sites have been remediated and backfilled with clean soil (DOE 2011d).

EXHIBIT A-5 100 AREA OPERABLE UNITS AND REMEDIATION STATUS

| ISOLATED UNITS (IUS) | | |
|----------------------|--|---|
| 100-IU-1 | Riverland Railroad Wash Station | Study and remediation selection complete |
| 100-IU-2 | White Bluffs Townsite Area | Study and remedy selection underway |
| 100-IU-3 | North Slope (Wahluke Slope) | Study not begun |
| 100-IU-4 | Buried Sodium Dichromate Drums | Study not begun |
| 100-IU-5 | Pickling Acid Cribs | Study not begun |
| 100-IU-6 | Hanford Townsite Area | Study and remedy selection underway |
| 100-B/C AREA | | |
| 100-BC-1, 100-BC-2 | Soil, Buildings, and Burial Grounds | Remedy construction complete |
| 100-BC-5 | Groundwater (Sr-90 tritium, nitrate, hexavalent chromium) | Final RI/FS work plan underway |
| 100-K AREA | | |
| 100-KR-1 | Principally Soil Sites Contaminated by Liquid Discharges | Remedy construction underway |
| 100-KR-2 | Soil, Buildings, and Burial Grounds | Remedy construction underway; K Basin ROD (removal of radioactive sludge) |
| 100-KR-4 | Groundwater (Sr-90, C-14, tritium, TCE, hexavalent chromium) | Remedy construction underway; pump-and-treat |
| 100-N AREA | | |
| 100-NR-1 | Soil, Buildings, and Burial Grounds | Remedy construction underway; Completion expected in 2012 |
| 100-NR-2 | Groundwater (Sr-90) | (Interim) Remedy construction underway; Previously conducted pump-and-treat, currently testing injection methods for a permeable reactive barrier |
| 100-D AREA | | |
| 100-DR-1 | Soil, Buildings, and Burial Grounds | Remedy construction complete |
| 100-DR-2 | Soil, Buildings, and Burial Grounds | Remedy construction underway |
| 100-H AREA | | |
| 100-HR-1 | Soil, Buildings, and Burial Grounds | Remedy construction complete |
| 100-HR-2 | Soil, Buildings, and Burial Grounds | Remedy construction underway |
| 100-HR-3 | Groundwater (chromium) | Remedy construction underway (pump and treat) |
| 100-F AREA | | |
| 100-FR-1 | Principally Soil Sites Contaminated by Liquid Discharges | Remedy construction underway |
| 100-FR-2 | Soil, Buildings, and Burial Grounds | Remedy construction complete |

| | | |
|--------------------------------|-------------|--|
| 100-FR-3 | Groundwater | Study and remedy selection underway; to be included in final ROD for 100-F, 100-IU-1 and 100-IU-2 (projected for 2012) |
| Sources: DOE 2011d, EPA 2012c. | | |

200 AREA

Closure plans for the 200 Area is organized into three major components: the Inner Area, the Outer Area, and groundwater. Each component contains smaller decision units, which are determined primarily based on geography and the status of existing decision documents. The extent of each of the main closure plan components are described as (DOE 2009):

- “Inner Area: The final footprint area of the Hanford Site that will be dedicated to waste management and containment of residual contamination and will remain under federal ownership and control.”
- “Outer Area: All Areas of the Central Plateau beyond the boundary of the Inner Area.”
- “Groundwater: Contaminant plumes underlying the Central Plateau and originating from waste sites on the Central Plateau.”

As of July 2011, the 200 Area had four RODs in place (DOE 2011d): an interim action and final ROD for the 200-ZP-1 OU (groundwater), and two final RODs to address the ERDF and contaminated soil removal at the 221-U Facility (a “canyon” site in the Inner Area).

Closure of the NRDWL/SWL is included in cleanup actions for the 200 Outer Area (DOE 2009).

EXHIBIT A-6 200 AREA OPERABLE UNITS AND REMEDIATION STATUS

| 200-PW-1, 200-PW-3, 200-PW-6, AND 200-CW-5 GROUNDWATER OPERABLE UNITS | | |
|---|---|--|
| 200-PW-1 | Plutonium/Organic-Rich Waste; large-scale carbon tetrachloride contamination, and radionuclides in soil | RI/FS high priority |
| 200-PW-3 | | |
| 200-PW-6 | | |
| 200-CW-5 | U Pond/Z Ditches Cooling Water; large-scale carbon tetrachloride contamination, and radionuclides in soil | RI/FS high priority |
| EAST INNER AREA | | |
| 200-EA-1 | Other unassigned 200 East Area waste sites | RI/FS in process |
| 200-IS-1 | East Inner Area, pipelines and associated components | RI/FS in process |
| WEST INNER AREA | | |
| 200-BC-1 | 200 West Inner Area cribs and trenches | RI/FS workplan underway |
| 200-WA-1 | Other unassigned 200 West Inner Area waste sites | RI/FS workplan underway |
| CANYONS AND ASSOCIATED WASTE SITES (CENTRAL PLATEAU, INNER AREA) | | |
| 200-CU-1 | U Plant "Canyon" Building | Final remedial actions scheduled for 2011 |
| 200-CB-1 | B Plant (original fuel separation facility) | |
| 200-CP-1 | PUREX Plant | |
| 200-CR-1 | REDOX Plant (S Plant) | Demolished; Materials shipped to ERDF and WIPP; Remedial action for below grade structure to occur |
| T Plant | 221-T Canyon Building | Still in operation; Receiving K Basin sludge |
| PFP | Plutonium Finishing Plant (PFP) | Readied for demolition |
| CENTRAL PLATEAU OUTER AREA | | |
| 200-CW-1 | Gable Mountain/B-Ponds and Ditches Cooling Water | Study and remedy selection underway |
| 200-CW-3 | 200 North Cooling Water | Interim ROD issued; Remedy construction complete |
| 200-OA-1 | B/C Controlled Area unplanned release from B/C cribs | Study not begun |
| GROUNDWATER | | |
| 200-ZP-1 | Groundwater contamination in northern 200 West Area (Contaminant plumes: carbon tetrachloride) | Conducting pump-and-treat; Natural attenuation |
| 200-UP-1 | Discharge to five liquid waste disposal sites in 200 West Area (Contaminant plumes: U, Tc-99) | Conducting pump-and-treat |

| | | |
|--------------------------------|--|---|
| 200-PO-1 | Groundwater contamination in southern portion of 200 East Area from PUREX liquid waste disposal and B-Plant (Contaminant plumes: tritium and I-129) | Study and remedy selection underway |
| 200-BP-5 | Groundwater contamination plumes associated with B-Plant operations in 200 East Area and 600 Area north of 200 East (Contaminants of concern: Tc-99, Co-60, Sr-90, Cs-137, Pu-239/240) | RI/FS issued; Study and remedy selection underway |
| OTHER OUS | | |
| 200-DF-1 | Environmental Restoration Disposal Facility (ERDF) | Hanford remediation waste disposal site |
| 200-DV-1 | Deep Vadose/Tank waste | Study not begun |
| 200-CS-1 | Chemical Sewer | Study and remedy selection underway |
| 200-SW-2 | Radioactive Landfills and Dumps | Study and remedy selection underway |
| Sources: DOE 2011d, EPA 2012c. | | |

300 AREA

Final RODs have been issued for the 300-FF-1 and 300-FF-2 OUs, and portions of the contamination in 300-FF-5 are covered in the ROD for 300-FF-1. Remedial actions have been completed at 300-FF-1 and are still underway at 300-FF-2 (DOE 2011d). Remedial actions underway for 300-FF-5 (groundwater) include monitoring and natural attenuation.

The primary remediation action is removal of contaminated soil and debris, treating the material, disposing in long-term waste management facilities (mainly ERDF), and backfilling and re-vegetating the areas where possible.

Remaining future cleanup and closure will be covered under a single ROD, which will include nearby 600 Area waste sites (618-10 and 618-11 burial grounds, which are considered to be two of Hanford’s most challenging remediation projects) (DOE 2012, DOE 2011f).

1100 AREA

Remediation of the 1100 Area was completed in 1996, and the area was removed from the NPL (groundwater monitoring north of Richland continues) (DOE 2006a).

SUMMARY

In summary, achievements in clean up and remediation of environmental impacts that have been accomplished to date include the following milestones (Abbotts and Weems 2008):

- Active cleanup footprint has been reduced from 586 sq. mi. to 201 sq. mi.;
- All spent fuel, previously stored near the River, has been moved to dry storage;
- All plutonium left in PFP has been stabilized and shipped off-site;

- Five of the nine reactors have been cocooned, and associated facilities demolished, with two additional underway;
- 636 waste sites have been remediated, and 310 facilities have been demolished;
- 5.8 billion gallons of groundwater have been treated, leaving 65 of the original 100 sq. mi. of contaminated groundwater remaining;
- All pumpable liquids and two million gallons of solids have been transferred from single- to double-shelled tanks, and construction of Waste Treatment Plant for underground tank waste is underway;
- Over 11,500 of an original 15,000 cubic meters of plutonium-contaminated waste buried on site has been retrieved;
- 81 of an original 850 waste sites and 337 of an original 970 facilities in the Central Plateau have been remediated and demolished;
- DOE continues to advance site closure activities. Priority projects include (Poston 2010):
 - Restoring the Columbia River Corridor with an expected completion date of 2015, providing a basis for closure reviews of the 100 and 300 Areas by independent experts;
 - Completing tank waste treatment, closing underground storage tanks, and completing construction of the Tank Waste Treatment and Immobilization Plant (WTP, which will process and stabilize radioactive waste on site),⁸⁴
 - Continuing cleanup of and protecting groundwater resources, and;
 - Treating and disposing of mixed low-level waste and retrieve transuranic waste for shipment offsite.

⁸⁴ The WTP's planned date for full operation has been 2019 (<http://www.hanford.gov/page.cfm/WTP>, viewed 24 October 2012). However, DOE informed the State of Washington that it may not be able to meet deadlines set forth in its 2010 consent decree with the State, including the deadline for the vitrification plant to be fully operational by the end of 2022 (Cary 2012).

**APPENDIX B | ECOTOXICITY SUMMARIES FOR SELECTED
CONTAMINANTS**

TABLE OF CONTENTS

| | |
|-----|----------------------|
| B1 | Carbon Tetrachloride |
| B2 | Cesium-137 |
| B3 | Chromium |
| B4 | Iodine-129 |
| B5 | Mercury |
| B6 | PCBs |
| B7 | Plutonium |
| B8 | Strontium-90 |
| B9 | Technetium-99 |
| B10 | Tritium |
| B11 | Uranium |

CARBON TETRACHLORIDE (CCl₄) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Carbon tetrachloride (CCl₄) is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. CCl₄, which is primarily of anthropogenic origin, is a volatile organic chemical (VOC),⁸⁷ existing as a vapor in the air at room temperature (EPA 2010).

II. SOURCES Although marine algae, oceans, volcanoes, and drill wells have been cited as natural sources of CCl₄ (Gribble 1994, as cited in ASTDR 2005), the majority of CCl₄ in the environment comes from “direct release to the atmosphere during production, disposal, or use of the compound” (ASTDR 2005).

Historically, the primary anthropogenic use of CCl₄ was in the production of chlorofluorocarbons (CFCs) (NLM 2003; Rossberg 2002, as cited in EPA 2010).⁸⁸ After the 1970s, until 1986, the primary use of CCl₄ was as a grain fumigant (ASTDR 2005). Beginning in January 1996, the production and import of CCl₄ was banned in most developed countries (including the U.S.A.).

At Hanford, CCl₄ was used widely as a cleaning agent and solvent, “including for degreasing equipment and machinery parts...[and] in the refining process during the separation of plutonium,” (Peterson 2007). More specifically, CCl₄ was used at Hanford to “recover plutonium isotopes” (Markwiese *et al.* 2008). Over the years that plutonium was produced at the site, hundreds of cubic meters of liquid CCl₄ waste were discharged to soil. This resulted in groundwater contamination and a dispersed CCl₄ vapor plume in the subsurface (vadose zone) (Markwiese *et al.* 2008) that is “spreading within a thick, unconfined aquifer” (Williams 2007). It is estimated that “363,000 to 580,000 L of liquid CCl₄ and other co-contaminants in spent solvent extraction mixtures were discharged to the ground during the plutonium refining operations at Hanford between 1955 and 1973. The used solvent was discharged to the soil column through a system of drain fields and trenches (cribs) that covered an area of <0.02 km²” (Williams 2007).

⁸⁷ “Volatile organic compounds (VOC) means any compound of carbon, excluding carbon monoxide, carbon dioxide, carbonic acid, metallic carbides or carbonates, and ammonium carbonate, which participates in atmospheric photochemical reactions, except those designated by EPA as having negligible photochemical reactivity” (40 CFR 5.100). “Volatile organic compounds, or VOCs are organic chemical compounds whose composition makes it possible for them to evaporate under normal... atmospheric conditions of temperature and pressure” (US EPA 2010)

⁸⁸ The Consumer Products Commission banned the use of CFCs in consumer products in the 1970s (EPA 2010 p. 4).

III. ENVIRONMENTAL CHEMISTRY FATE AND TRANSPORT

In soils, “CCl₄ is expected to evaporate rapidly ... due to its high vapor pressure and may migrate into groundwater due to its low soil adsorption coefficient” (ASTDR 2005).

Although many VOCs are unstable in air, CCl₄ is stable in the troposphere (the lowest layer of Earth’s atmosphere), persisting for 30-50 years (OxyChem 2010). “CCl₄ does not readily dissociate in the lower atmosphere, nor is it easily washed out by rainfall,” (Peterson 2007) Eventually, CCl₄ rises and undergoes photolysis in the upper atmosphere (stratosphere) (EPA 2010), creating chlorine radicals and trichloromethyl radicals which are destructive to the ozone layer (Peterson 2007). The primary chemical degradation products of CCl₄ are chloroform, methylene chloride, and chloromethane (Markiwese 2008).

Within soils, CCl₄ is highly mobile. The K_{oc}⁸⁹ values for CCl₄ from various sources range from 48.89 (soils with organic carbon content of 0.66 percent) to 143.6 (soils with organic carbon content of 1.49 percent) (ASTDR 1992, as cited in Irwin 1997), and the average K_{oc} value is 71 (NLM 2003, as cited in EPA 2010).

Carbon tetrachloride’s mobility in soils allows it to pass through to “lower soil horizons and groundwater” (NLM 2003, as cited in EPA 2010). However, CCl₄ is only “slightly soluble” in water (Peterson 2007). The Log K_{ow}⁹⁰ value for CCl₄ is 2.73 (EPA 1995, as cited in Sample et al 1997).

There is potential for CCl₄ to biodegrade in both soil and water under both aerobic and anaerobic conditions (NLM 2003; US EPA 1996b; Semprini 1995, as cited in EPA 2010): “Biodegradation may occur in groundwater, but will be very slow, [6-12 months under aerobic conditions and 7-28 days under anaerobic conditions, Peterson 2007], compared with evaporation” (OxyChem 2010). The half-life⁹¹ of CCl₄ in river water is between 0.3 and 3 days, depending upon the water movement (EuroChlor 1999).

The distribution of CCl₄ throughout the Hanford environment is “complex because of its potential to migrate either as a dense nonaqueous phase liquid (DNAPL), in the gaseous state, and/or dissolved in water” (Williams 2007). Simulations of CCl₄ contaminant flow to the Columbia River at the Hanford site have determined that sorption and abiotic degradation are critical in predicting the future movement of CCl₄ from the 200 West Area to the river (Bergeron and Cole 2005 as cited in Williams 2007).

⁸⁹ The organic carbon adsorption coefficient (K_{oc}) is a measure of the tendency for organic substances to be adsorbed by soil or sediment; this parameter is substance-specific and is largely independent of soil properties (Duffus *et al.* 2007).

⁹⁰ “The octanol-water partition coefficient (K_{ow}) is the ratio of the concentration of a chemical in octanol and in water at equilibrium and at a specified temperature. Octanol is an organic solvent that is used as a surrogate for natural organic matter. This parameter is used in many environmental studies to help determine the fate of chemicals in the environment” (USGS 2010).

⁹¹ Half-life here refers to the transfer of CCl₄ from aquatic systems to the atmosphere through volatilization and calculations are based on the value of the Henry’s law constant (EuroChlor 1999).

Calculations based on groundwater, soil–gas concentration and well venting data from the Hanford site, show that approximately 12 percent of the original carbon tetrachloride inventory was estimated to be in the vadose zone, 21 percent was lost to the atmosphere, and 1 to 2 percent was dissolved in the upper 10 m of the unconfined aquifer beneath the 200 West Area (Swanson *et al.* 1999; Rohay and Johnson 1991, as cited in Williams 2007). The remaining 65 percent of the original inventory is unaccounted for and may be held as residual DNAPL in the soil pores of both the vadose zone and groundwater (Swanson *et al.* 1999, as cited in Williams 2007).

BIOACCUMULATION POTENTIAL

CCl₄ has a low potential to bioconcentrate (Hoffman *et al.* 1990, as cited in Irwin 1997): it does not readily bioaccumulate in either plants or animals (EuroChlor 1999; Peterson 2007). However, K_{oc} values for carbon tetrachloride suggest that bioaccumulation is at least possible under conditions of constant exposure and may occur in occupational settings or in people living at or near hazardous waste sites (ASTDR 1992, as cited in Irwin 1997). In a comprehensive search done for the carbon tetrachloride entry in the 1997 Environmental Contaminants Encyclopedia, “[n]o data were located on the biomagnifications of carbon tetrachloride” (ASTDR 1992, as cited in Irwin 1997). In addition, “Limited data indicate that CCl₄ has a low tendency to bioconcentrate in the food chain even though it is a lipophilic compound” (Neely *et al.* 1974, Peason and McConnell 1975, as cited in ASTDR 2005). This can be explained physiologically: “since most animals readily metabolize and excrete carbon tetrachloride following exposure biomagnification is not expected” (ASTDR 1992, as cited in Irwin 1997). In other words, “rapid clearance from exposed organisms” (ASTDR 2005) prevents CCl₄ from bioaccumulating across trophic levels (EPA 2010).

Experimentally derived bioconcentration factors (BCFs) also indicate that CCl₄ will not “bioconcentrate appreciably in aquatic...organisms” (EuroChlor 1999; NLM 2003 as cited in EPA 2010). Reported BCFs were 17.38 and 30.2 in trout and bluegill sunfish, respectively (ASTDR 1992, as cited in Irwin 1997), and the maximum BCFs listed for fish is 69.95 (Sample *et al.* 1996). In 1980, Kenaga reported a bioconcentration factor (predicted from water solubility) of 14 (calculated), and determined experimentally that this bioconcentration factor was 18 (Kenaga 1980, as cited in Irwin 1997). These numbers are all consistent with a substance with low bioaccumulation potential which is generally defined as BCF < 250 (Kenaga 1980, Abdullah *et.al.* 2007).

ACCUMULATION WITHIN SPECIFIC TISSUES

It has been shown that CCl₄ preferentially migrates toward tissues that have a high fat content (EPA 2010), and that it “does tend to become concentrated in fatty tissues” (ASTDR 2005). CCl₄ is absorbed quickly by organisms and distributed widely throughout tissues (especially to the tissues with high lipid content), but is also excreted quickly (EPA 2010). Animal studies have shown that under differing conditions, 34–75% of carbon tetrachloride leaves the body in expired air, 20–62% leaves the body in feces, and only low amounts leave the body in the urine. Animal

studies also suggest that it may take weeks for the remainder of the compound in the body to be eliminated, especially that which has entered the body fat (ASTDR 2005). Tissues commonly examined in both terrestrial and aquatic animals as indicators of exposure (in laboratory experiments) include liver, fat, brain, heart, gills, muscle, and blood (reviewed in Irwin 1997).

In plants, some data indicate that CCl₄ does not migrate to tissue with higher fat content: CCl₄ “residue in wheat germ with a high fat content was found to be less than 50% of that found in bran,” (Hayes 1982 as cited in Irwin 1997).

IV. TYPICAL MAJOR EXPOSURE ROUTES

For aquatic biota, dermal absorption is an important CCl₄ exposure route (ANL 1996). For terrestrial biota, exposure can “occur by breathing carbon tetrachloride present in the air, by drinking water contaminated with carbon tetrachloride, or by getting soil contaminated with carbon tetrachloride on the skin” (ASTDR 2005). In fact, “toxic amounts may be absorbed through the skin to cause chronic health effects” (OxyChem 2010).

Furthermore, VOCs “in soil represent a potentially significant exposure pathway to fossorial wildlife through the inhalation of contaminated subsurface burrow air” (Carlsen 1996; USEPA 2003, as cited in Markwiese 2008). At Hanford, burrowing mammals may be more at risk for exposure to CCl₄ than other organisms because of the CCl₄ soil content at some areas of the site.

V. ECOTOXICITY

CCl₄ is a known hepatotoxin (Alexeeff & Kilgore 1983, Manubisan *et al.* 2007). Other toxic effects occur but generally appear to be secondary: “an extensive body of scientific data indicates that carbon tetrachloride-induced liver carcinogenicity appears to be secondary to toxic effects of the chemical. Assays for mutagenicity and other genotoxic effects have primarily been negative or have produced evidence of effects only at high, cytotoxic concentrations” (Manubisan *et al.* 2007). Similarly, although “subchronic/chronic exposure by various routes also results in damage to respiratory, cardiac, neural and reproductive/fetal tissues and in reduced body weight, [these occur] generally at doses greater or equal to those producing hepatic effects” (OxyChem 2010).

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

There are no known beneficial or protective properties attributed to CCl₄.

MECHANISM(S) AND LOCI OF TOXICITY

A 2007 review of CCl₄ mode of action states that “the primary site of toxicity and carcinogenesis is the liver. Carbon tetrachloride consistently causes liver toxicity, resulting in fatty degeneration, cellular necrosis, fibrosis and cirrhosis. This occurs in multiple species and through multiple routes of exposure” (Manibusan *et al.* 2007). More recent information expands upon this: “The liver and kidney are the primary sites of induced CCl₄ toxicity. More specifically, target organs for CCl₄ damage are

the central nervous system, liver, kidney, lungs, eyes, heart, skin” (OxyChem 2010). CCl₄ is readily absorbed by the gastrointestinal tract in humans and animals (EPA 2010).

There is substantial evidence that the first step in biotransformation of CCl₄ is “reductive dehalogenation: reductive cleavage of one carbon-chlorine bond to yield chloride ion and the trichloromethyl radical” (Reinke and Janzen 1991; Tomasi *et al.* 1987; McCay *et al.* 1984; Mico and Pohl 1983; Slater 1982; Poyer *et al.* 1980, 1978; Lai *et al.* 1979 as cited in EPA 2010). It is this free radical that forms the trichloromethyl peroxy radical, the primary initiator of the liver damage that occurs from exposure to CCl₄ (Boll *et al.* 2001a; McCay *et al.* 1984; Rao and Recknagel 1969 as cited in EPA 2010).

FACTORS AFFECTING TOXICITY

High fat diets and malnutrition greatly enhance CCl₄ uptake (NAS 1978, as cited in Irwin, 1997; Sagai 1978, as cited in Irwin 1997). Though this is not expected to be an issue relevant to biota exposed in the natural environment, ethanol consumption also enhances CCl₄ uptake and hepatotoxicity (NAS 1978, as cited in Irwin, 1997; Reynolds *et al.* 1982, as cited in Irwin 1997). Other biological factors that enhance toxic effects include respiration rate (e.g., in the case of burrowing mammals or other species exposed through inhalation), and age. Mammals that are in utero or in infancy are particularly sensitive to CCl₄ exposure because it may cross the placenta and may be excreted in breast milk (OxyChem 2010). Blain *et al.* (1999)

PLANTS

There has been relatively little investigation of the effects of CCl₄ exposure on plants other than algae.

AQUATIC INVERTEBRATES AND FISH

CCl₄ exposure has a wide range of toxic effects on aquatic invertebrates and fish in laboratory experiments. Rainbow trout fed diets containing 3,200 and 12,800 ppm CCl₄ developed hepatomas (4 out of 44 at the lower dose level and 3 out of 34 at the higher dose level) after 20 months whereas no tumors were found in the controls (IARC 1972, as cited in Irwin 1997). A 1979 study by Weber *et al.* (as cited in ERED) on immature rainbow trout demonstrated a range of effects at varying doses (292-6,400 mg/kg wet weight concentration in fish tissue), including physiological measures of liver function, body weight increases, inflammation of intestines and peritoneal lining, mottled liver and spleen; hemorrhage, and death.

Studies performed in conditions that more closely mimic field/environmental conditions have found CCl₄ exposure to be less toxic, when compared to results from laboratory studies such as those listed above. In 1980, Carroll *et al.* evaluated exposure of immature bluegill to CCl₄. The fish were exposed to environmentally realistic CCl₄ levels by absorption (whole body), and there was no effect on mortality (Carroll *et al.* 1980, as cited in ERED). The Pesticide Action Network (PAN)

Pesticide Database contains a list of toxic effects of CCl₄ on a variety of aquatic organisms (Exhibit 1) (Kegley *et al.* 2010).

EXHIBIT 1 PAN PESTICIDE DATABASE INFORMATION

| SPECIES GROUP | SPECIES INCLUDED | NO. OF STUDIES | EFFECTS NOTED | ACUTE TOXICITY RANGE |
|-------------------------|--|----------------|---|---------------------------------------|
| Cnidaria | Hydra (<i>Hydra attenuata</i>) | 1 | Abnormal Growth | - |
| Crustaceans | Fairy shrimp (<i>Streptocephalus proboscideus</i>) | 1 | Mortality | Not acutely toxic |
| Echinoderms | Sea urchin (<i>Paracentrotus lividus</i>) | 1 | Developmental changes (fertilized eggs) | - |
| Fish | Rainbow trout (<i>Oncorhynchus mykiss</i>), Medaka, high-eyes (<i>Oryzias latipes</i>), Bluegill (<i>Lepomis macrochirus</i>), Zebra danio (<i>Danio rerio</i>), Fathead minnow (<i>Pimephales promelas</i>), Carp (<i>Leuciscus idus melanotus</i>), English sole (<i>Parophrys vetulus</i>), Indian catfish (<i>Heteropneustes fossilis</i>) | 88 | Accumulation, Behavior, Biochemistry, Enzyme(s), Histology, Injury, Mortality, Physiology | Not acutely toxic to slightly toxic |
| Nematodes and Flatworks | Flatworm (<i>Dugesia japonica</i>) | 2 | Growth, Mortality | Highly toxic |
| Phytoplankton | Green algae (<i>Chlorella fusca vacuolat</i>), various diatoms (including <i>Cylindrotheca sp.</i> , <i>Hantzschia amphioxys pusilla</i>), Blue-green algae (<i>Anacystis aeruginosa</i>), Cryptomonad (<i>Chilomonas paramecium</i>), Flagellate euglenoid (<i>Entosiphon sulcatum</i>) | 28 | Accumulation, Population | -- |
| Zooplankton | Water flea (<i>Daphnia magna</i>), Ciliate (<i>Tetrahymena pyriformis</i>), Rotifer (<i>Brachionus calyciflorus</i>), Scud (<i>Gammarus pseudolimnaeus</i>) | 21 | Behavior, Growth, Intoxication, Mortality, Physiology, Population | Not acutely toxic to moderately toxic |

BIRDS

There has been relatively little investigation of the effects of CCl₄ exposure on birds. A 1975 study reported that “the chicken was resistant to CCl₄-induced liver necrosis” (Diaz Gomez 1975), making it the lowest in CCl₄-induced lipid peroxidation of the animals tested. The paper reports rat > hamster = guinea pig > chicken = mouse (Diaz Gomez 1975).

MAMMALS

Laboratory experiments have shown a range of toxic effects on mammals, including , carcinogenicity, reproductive toxicity (embryo- and feto-toxicity), as well as growth, and behavioral effects.

For example, in 1974, Schwetz *et al.* found evidence of embryo- and fetotoxicity attributable to inhaled CCl₄ in rats (Schwetz *et al.* 1974, as cited in Gallegos *et al.* 2007). Changes in fetal weight due to exposure were observed (*ibid.*). Based on chemical data, CCl₄ may cause cancer in mammals: “Oral administration to animals produced liver tumors, including hepatocellular carcinomas, in various strains of mice; and in rats caused benign and malignant liver tumors. Administration of CCl₄ to mice resulted in a statistically significant increase in the incidence of neoplastic tumors of the skin” (OxyChem 2010).

Mice exposed acutely via inhalation to 134.3 mg/l CCl₄ were found to have a significantly decreased ability to learn (as judged by a passive-avoidance conditioning task) as compared to controls (Alexeeff & Kilgore 1983). Also, “Prendergast *et al.* (1967) conducted experiments with rats, guinea pigs, rabbits, dogs, and monkeys, using carbon tetrachloride. They reported that all species exhibited a depressed growth curve following a 90-d exposure when compared with controls” (Alexeeff and Kilgore 1983).

Evidence of reproductive impacts appears to be mixed. For instance, “In rats, inhalation exposure during gestation caused maternal weight loss and clear maternal hepatotoxicity, but no effect on conception, number of implants, or number of resorptions. There were no gross anomalies, although fetal size was somewhat decreased. The authors concluded that this response was not treatment related” (OxyChem 2010). However, “In rats, moderate to marked degeneration of testicular germinal epithelium and reduced fertility were seen after inhalation of 200 ppm or higher for up to 192 days,” and “This material has been reported to prolong the estrus cycle and to cause testicular atrophy and to decrease sperm counts in rats, although oral exposure did not adversely affect reproduction. Ovary changes were observed in female mice that were exposed to vapor for 6 hours/day, 5 days/week for 2 years. In addition, absolute and relative testicular weights were elevated in the male mice. Rats exposed twice weekly for five weeks to anesthetizing concentrations exhibited only a small decrease in testes weight” (OxyChem 2010).

There is some evidence that intermittent chronic exposure causes more damage than constant chronic exposure in rats. “Shimizu *et al.* (1973) exposed groups of 4 female

Sprague-Dawley rats to 10, 50 and 100 ppm of CCl₄ vapor for 3 hours a day, 6 days a week for up to 6-8 weeks. The rats were terminated two days after the last inhalation. The intermittent exposure caused a more pronounced and higher number of change indices to occur (34 as opposed to the 17 change indices of the monotonous regimen), indicating a greater intensity of liver damage” (California 2000).

AMPHIBIANS AND REPTILES

There has been relatively little investigation of the effects of CCl₄ exposure on amphibians and reptiles. In a 1980 study titled “Effects of organic compounds on amphibian reproduction,” Fowler’s toad (*Bufo woodhousei fowleri*), bullfrog (*Rana catesbeiana*), and pickerel frog (*Rana palustris*) were studied. Acute high level exposure to CCl₄ caused mortality and had effects on hatching success (Birge *et al.* 1980, as cited in Kegley *et al.* 2010).

EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

There are “low amounts of organic matter and ferrous iron (Fe²⁺) available [in the unconfined aquifer at Hanford]” (Thornton *et al.* 1995, as cited in Patton *et al.* 2007), but the presence of either in higher concentrations would result in dechlorination of CCl₄ (Peterson 2007). In support of this assertion, it has been found that interaction of CCl₄ with clay may reduce its toxicity (Efroymsen 1997).

Increased sensitivity to the toxic effects of CCl₄ exposure can be caused by exposure to or consumption of “alcohols, ketones, phenobarbital, methamphetamine, or other barbituates, other brominated or chlorinated solvents, dichlorodiphenyltrichloroethane (DDT), PBB, chlordecone, nicotine, carbon disulphide and other alkyl disulphides or hypoxia” (OxyChem 2010). A 2007 report from Argonne National Laboratory confirms that interaction with “ketones (e.g. acetone) increases toxicity” of CCl₄ (Peterson 2007). A 1982 study done by Kluwe showed that “20 days oral administration of hexachlorobenzene..., polybrominated biphenyls ... or polychlorinated biphenyls ... increased CCl₄-induced⁹² growth retardation, renal tubular functional impairment, & hepatocellular necrosis in male rats” (Kluwe 1982 as cited in Irwin 1997). Additionally, a 1982 study showed that “[c]oncurrent treatment of mammals with CCl₄ & DDT...[i]ncreases susceptibility (approximately 10 fold) to CCl₄ toxicity” (Booth & McDonald, eds., 1982 as cited in Irwin 1997).

DATA GAPS & CHALLENGES

Most toxicological research has been laboratory-based. Little research has been conducted on the effects of CCl₄ toxicity under field conditions or on wild species. Specifically, there appears to be a paucity of CCl₄ toxicity data on birds, waterfowl, insects, reptiles, amphibians, plants, and wild mammals.

⁹² (0.00, 0.03, 0.25, or 2.00 ml/kg, iontophoretic administration)

- VI. REFERENCES** Abdullah, M.H., J. Sidi, and A.Z. Zaharin Aris. 2007. Heavy metals (Cd, Cu, Cr, Pb and Zn) in *Meretrix meretrix* Roding, water and sediments from estuaries in Sabah, North Borneo. *International Journal of Environmental & Science Education* 2(3):69–74.
- Alexeeff, G.V. and W.W. Kilgore. 1983. Learning impairment in mice following acute exposure to dichloromethane and carbon tetrachloride. *Journal of Toxicology and Environmental Health* 11:569–581.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological Profile for Carbon Tetrachloride. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. August.
- Argonne National Laboratory (ANL) Environmental Assessment Division. 1996. Risk Assessment of Seeps from the 317 Area of Argonne National Laboratory. Work supported by the U.S. Department of Energy, Office of Environmental Management, under contract W-31-109-Eng-38. September.
- Barrows, M.E., S.R. Petrocelli, K.J. Macek, and J.J. Carroll. 1980. Bioconcentration and elimination of selected water pollutants by bluegill sunfish (*Lepomis macrochirus*). *In: Dynamics, Exposure and Hazard Assessment of Toxic Chemicals*, R. Haque (ed.), Chapter 24, Ann Arbor Science Publishers, Ann Arbor, MI.
- California Office of Environmental Health Hazard Assessment (California). 2000. Determination of Noncancer Chronic Reference Exposure Levels Batch 2A. December. Carbon Tetrachloride. CAS Registry Number: 56-23-5. Accessed December 21, 2010 at http://oehha.ca.gov/air/chronic_rels/pdf/56235.pdf
- Davis, M. E., W.O. Berndt, and H.M. Mehendale. 1980. Alterations of xenobiotic excretory function induced by potassium dichromate or carbon tetrachloride pretreatment. *Journal of Toxicology and Environmental Health, Part A: Current Issues*, 1087-2620, 6(2):455 – 465
- Díaz Gómez, M.I., C.R. de Castro, N. D'Acosta, O.M. de Fenos, E.C. de Ferreyra, and J.A. Castro. 1975. Species differences in carbon tetrachloride-induced hepatotoxicity: The role of CCl₄ activation and of lipid peroxidation. *Toxicology and Applied Pharmacology* 34(1):102-114.
- Duffus, O., M. Nordberg, and D.M. Templeton. 2007. IUPAC Glossary of Terms Used in Toxicology, 2nd Ed. *Pure Appl. Chem.* 79(7):1153-1344. Accessed December 8, 2010 at <http://sis.nlm.nih.gov/enviro/iupacglossary/frontmatter.html>
- Duncan, J.P., K.W. Burk, *et al.* 2007. Hanford Site National Environmental Policy Act (NEPA) Characterization. Prepared for the U.S. Department of Energy under Contract DE-AC05-76RL01830. PNNL-6415 Rev. 18, Pacific Northwest National Laboratory. September.
- Efroymsen, R.A., M.E. Will, and G.W. Suter. 1997. Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates

- and Heterotrophic Process: 1997 Revision. Prepared for the U.S. Department of Energy. ES/ER/TM-126/R2. November.
- EuroChlor. 1999. EuroChlor Risk Assessment for the Marine Environment OSPARCOM Region – North Sea: Carbon Tetrachloride. February.
- Gallegos, P., J. Lutz, J. Markwiese, R. Ryti, and R. Miranda. 2007. Wildlife ecological screening levels for inhalation of volatile organic chemicals. *Environmental Toxicology and Chemistry* 26(6):1299-1303.
- Irwin, R.J., M. Van Mouwerik, L. Stevens, M.D. Seese, and W. Basham. 1997. *Environmental Contaminants Encyclopedia*. National Park Service, Water Resources Division, Fort Collins, Colorado. Distributed within the Federal Government as an Electronic Document.
- Kegley, S.E., B.R. Hill, S. Orme, and A.H. Choi. *PAN Pesticide Database*, Pesticide Action Network, North America, San Francisco, CA. Accessed November 11, 2010 at <http://www.pesticideinfo.org>
- Kenaga, E.E. 1980. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. *Ecotoxicology and Environmental Safety* 4(1):26-38.
- Lipton, J. and J. Holmes. 2009. Hanford Site Natural Resource Damage Assessment, Phase I Summary Report. Prepared for Hanford Natural Resource Trustees by Stratus Consulting, Inc., Boulder, Colorado. July 1.
- Manubisan, M.K., M. Odin, and D.A. Eastmond. 2007. Postulated carbon tetrachloride mode of action: A review. *Journal of Environmental Science and Health Part C* 25:185–209.
- Markiwese, J.T., B. Tiller, R.T. Ryti, and R. Bauer. 2008. Using artificial burrows to evaluate inhalation risks to burrowing mammals. *Integrated Environmental Assessment and Management* 4(4):425-430.
- OxyChem. 2010. Safety Data Sheet: Carbon Tetrachloride, Technical Grade. Occidental Chemical Corporation, Dallas, TX.
- Patton, G., D. Dauble, and C. McKinstry. 2007. Evaluation of early life stage fall chinook salmon exposed to *hexavalent chromium* from a contaminated groundwater source. *Environmental Monitoring & Assessment* 133(1-3):285-294.
- Peterson, J., M. MacDonell, L. Haroun, and F. Monette. 2007. Radiological and Chemical Fact Sheets to Support Health Risk Analyses for Contaminated Areas: Carbon Tetrachloride. Argonne National Laboratory Environmental Science Division in collaboration with U.S. Department of Energy Richland Operations Office (R. Douglas Hildebrand) and Chicago Operations Office (Anibal Taboas). March.
- Sample, B.E., M.S. Aplin, R.A. Efroymsen, G.W. Suter, and C.J.E. Welsh. 1997. Methods and Tools for Estimation of the Exposure of Terrestrial Wildlife to

- Contaminants. Oak Ridge National Laboratory Environmental Sciences Division for the U.S. Department of Energy. Publication No. 4650. Contract No. DF-AC05-96OR22464. October.
- Sample, B.E., D.M. Opresko, and G.W. Suter. 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. Risk Assessment Program Health Sciences Division for the U.S. Department of Energy. Contract No. DE-AC05-84OR21400. June.
- U.S. Environmental Protection Agency. 2010. Toxicological Review of Carbon Tetrachloride (CAS No. 56-23-5) in Support of Summary Information on the Integrated Risk Information System (IRIS). Washington, DC. March.
- U.S. Environmental Protection Agency. 2010. An Introduction to Indoor Air Quality (IAQ) Volatile Organic Compounds (VOCs) Technical Overview. Accessed December 8, 2010 at <http://www.epa.gov/iaq/voc2.html>
- U.S. Geological Survey (USGS). 2010. Toxic Substances Hydrology Program Octanol-Water Partition Coefficient (KOW) Definition Page. Accessed December 8, 2010 at <http://toxics.usgs.gov/definitions/kow.html>
- Weber L.J., W.H. Gingerich, and K.F. Pfeifer. 1979. Alterations in rainbow trout liver function and body fluids following treatment with carbon tetrachloride or monochlorobenzene. In: *Pesticide and Xenobiotic Metabolism in Aquatic Organisms*, M.A.Q. Khan, J.J. Lech, and J.J. Menn (eds.). ACS Symposium Series 99, Washington, D.C., pp. 401–413.
- Williams, B.A. and C.J. Chou. 2007. Characterizing vertical contaminant distribution in a thick unconfined aquifer, Hanford Site, Washington, USA. *Environ. Geol* 53:879-890.
- World Health Organization (WHO). 1999. International Programme on Chemical Safety. Environmental Health Criteria 208: Carbon Tetrachloride. Accessed October 2010 at <http://www.inchem.org/documents/ehc/ehc/ehc208.htm#SubSectionNumber:7.5>
- 1

CESIUM (Cs-137) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION

Cesium-137 (Cs-137) is a radionuclide and is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Cesium is a soft metal element with a melting point of only 28.4°C, such that it can be liquid at room temperature. It is one of the alkali metals and in metallic form is extremely reactive (Butterman et al. 2005); however, the chemical forms that occur following the detonation of a weapon or release from a reactor tend to be oxides, salts, or hydroxides.

Natural cesium (Cs-133) is not radioactive; however, there are a number of artificial radioisotopes of cesium, of which Cs-137, a reactor byproduct, is “the most used and well-known” (Butterman et al. 2005). A radionuclide, “Cesium-137 has a half-life of about 30 years and decays by beta decay either to stable Ba-137 or a meta-stable form of barium (Ba-137m). The meta-stable isotope (Ba-137m) is rapidly converted to stable Ba-137 (half-life of about 2 minutes) accompanied by gamma ray emission (ICRP 1983). The first beta decay mode that forms Ba-137m accounts for roughly 95% of the total intensity, while the second mode accounts for about 5%” (WHO 1983).

Exhibit 1 presents the radioactive properties of Cs-137 and its progeny Ba-137m. Because the half-life of Ba-137m is so short, each disintegration of Cs-137 is accompanied shortly thereafter by a disintegration of its progeny, Ba-137m. Hence, it can be assumed that Ba-137m is in equilibrium with Cs-137 whenever Cs-137 is found. Furthermore, each disintegration of Cs-137 results in the emission of a beta particle with an average energy of about 0.6 MeV, followed shortly by a decay of Ba-137m, which emits primarily a photon of about 0.6 MeV, along with an occasional electron (see Exhibit 1).

A large body of information is available about the properties and ecotoxicity of Cs-137. This profile relies in significant part on NCRP Report No. 154 (NCRP 2007), a relatively recent comprehensive review of Cs-137 in the environment, which in turn is an update of NCRP Report No. 52 (NCRP 1977). This profile also draws heavily from ATSDR (2004), the Hazardous Substances Databank (HSDB),⁹³ and reports addressing the operation and remediation of the Hanford facility.⁹⁴

⁹³ The National Institute of Health maintains the Toxicology Data Network (TOXNET), which includes the Hazardous Substance Data Bank (found at <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>).

⁹⁴ At several places in this profile, direct quotes make reference to additional specific source documents. These references are included in the reference section of this profile so that readers can more easily identify and obtain the original source documents cited in the major publications.

EXHIBIT 1 RADIOLOGICAL PROPERTIES OF CS-137 AND ITS SHORT-LIVED PROGENY, Ba-137M (EXCERPTED FROM SHLEIEN *ET AL.* 1998)

| ISOTOPE | HALF-LIFE | PROBABILITY OF DECAY | MAX BETA/ELECTRON ENERGY (MEV) | AVERAGE BETA/ELECTRON ENERGY (MEV) | PHOTON (GAMMA AND X-RAY) PROBABILITY | PHOTON ENERGY (MEV) |
|---------|-----------|----------------------|--------------------------------|------------------------------------|--------------------------------------|---------------------|
| Cs-137 | 30.17 yrs | 94.6% | 0.512 | 0.157 | NA | NA |
| | | 5.4% | 1.173 | 0.415 | NA | NA |
| Ba-137m | 2.55 min | 7.6% | 0.0037 | | 1.04% | 0.004 |
| | | 0.8% | 0.0264 | | 2.07% | 0.032 |
| | | 8.1% | 0.624 | | 3.82% | 0.032 |
| | | 14.6% | 0.656 | | 1.39% | 0.036 |
| | | 0.5% | 0.660 | | 90% | 0.662 |

II. SOURCES NATURAL SOURCES

As discussed in ATSDR (2004), “Naturally-occurring cesium and cesium minerals consist of only one stable isotope, Cs-133. Cesium occurs in the earth's crust at low concentrations. Granites contain an average cesium concentration of about 1 ppm and sedimentary rocks contain about 4 ppm (Burt 1993).” Higher concentrations are found in a number of minerals; however, there are no sources of naturally occurring Cs-137.

ANTHROPOGENIC SOURCES

Cesium-137 is a fission product and was produced in large quantities during above-ground weapons testing in the United States and the former Soviet Union during the 1950s and 1960s and also, to a lesser degree, by China, France, and the United Kingdom (UNSCEAR 2000). It was also produced during below-ground weapons testing, but the fission products were largely confined below ground at the test sites (primarily the Nevada Test Site). Mikhailov (1999) provides a historic account of all nuclear weapons testing by every nation up until 1999.

During testing, Cs-137 was produced at a rate of 0.17 million curies per megaton, and, as a result, fallout from above-ground testing resulted in the widespread distribution of Cs-137 in soil, water, and food. The literature summarizing the concentrations of Cs-137 in air, soil, water, food items, and in aquatic and terrestrial organisms is vast. Summaries of this literature can be found in Eisenbud and Gesell (1997), UNSCEAR (2000 and 2008), and in NCRP (2007). Eisenbud and Gesell (1997) concluded that, over most of the United States, the fallout by the end of above-ground weapons testing in 1965 deposited between 60 and 100 millicuries of Cs-137/km². Table 9.6 in Eisenbud and Gesell (1997) presents the concentrations of Cs-137 in various food items in Chicago in 1968. UNSCEAR (2000) presents a fairly detailed description of the deposition density (Bq/m²) of Cs-137 in the northern and southern hemisphere for

different latitudes and as a function of time up to 2000. Updated information was recently published in UNSCEAR (2008).

The concentration of Cs-137 in the environment from weapons testing is gradually declining due to its 30 year half-life and also due to natural attenuation; i.e., Cs-137 is gradually depleted from soil and sediment by downward migration and erosion, and eventually transported into relatively inaccessible or less accessible environmental compartments, such as the ocean depths.

There have been several occurrences where large quantities of Cs-137 were injected into the atmosphere following the termination of above-ground weapons testing. The most noteworthy is the Chernobyl accident in 1986. Buzulukov and Dobrynin (1993) estimated that 1 to 2 million curies of Cs-137 were released to the atmosphere during the accident. This is a significant fraction of the estimated inventory of Cs-137 in the Chernobyl reactor core at the time of the accident (i.e., about 7 million curies of Cs-137 in the core). Elevated levels of Cs-137 were measured in the atmosphere at many points around the northern hemisphere (reviewed in HDSB). Other accidents involving Cs-137 that resulted in its widespread dispersal in the environment include nuclear waste disposal accidents in the USSR in 1949-1956 and a 1957 accident at the Windscale facility in Great Britain (reviewed in IARC 2000 and ATSDR 1999).

In addition to weapons testing and accidental releases, cesium-137 produced in fission reactors is often present in detectable quantities in the routine gaseous and liquid effluent from operating nuclear reactors and fuel cycle facilities. It is also present in relatively large quantities in low level and high level solid radioactive waste and in spent fuel (Eisenbud and Gesell 1997).

In summary, “Of the roughly 1 EBq (10^{18} Bq) of Cs-137 released to the biosphere, ~90% was produced by atmospheric testing. Approximately 6% was produced by the Chernobyl accident and roughly 4% by nuclear fuel reprocessing facilities. Of the nuclear reactor accidents, the Chernobyl accident on April 26, 1986 in the Ukraine released far more radioactivity, including Cs-137, to the environment than all other nuclear accidents combined” (NCRP 2007).

At Hanford, there are a number of site-specific sources of Cs-137. NCRP (2007) describes these as follows:

“Most historical releases of radiocesium at Hanford were to terrestrial disposal sites and to surface-water impoundments. Approximately 1.6 TBq⁹⁵ of Cs-137 was released to the atmosphere from the separation facilities at Hanford. However, the primary repository of radiocesium at Hanford has been in the 200 Areas where the majority of the Site’s liquid-waste ponds, cribs, trenches, tanks, and solid-waste burial grounds are located. Since operations began in 1944, more than 1 EBq⁹⁶ of Cs-137 have been disposed of and stored in the 200 Areas, most of it in large underground tanks. Leaks from the tanks have released Cs-137 and other radionuclides into subsurface soil. In addition, contaminated process water and

⁹⁵ T stands for tera and means 10^{12} .

⁹⁶ E stands for exa and means 10^{18} .

liquid wastes were historically discharged to cribs, trenches, French drains, ditches, and ponds. Approximately 5×10^8 m³ of liquid waste were percolated into the ground, including more than 1.5 PBq⁹⁷ of Cs-137. Such disposal was designed to allow the liquid wastes to percolate into the ground where adsorption to soil removed most of the Cs-137 and other contaminants before they reached groundwater. The cribs were leach fields covered by soil. The trenches were covered with soil after receiving waste materials. The ditches and ponds were usually left open, providing habitat for plants and animals, including fish. Contaminated solid wastes were disposed of in various burial grounds. The single-pass production reactors generated the irradiated fuel that was sent to the separation facilities to isolate plutonium, but fuel element ruptures during operation of the reactors released fission and activation production to the Columbia River.”

Hanson (2000) provides an overview of the types and inventories of waste at the site and associated historic leakages. More detailed information is provided in many of the citations in that report, such as the paper by Gephart and Lundgren (1998).

III. ENVIRONMENTAL CHEMISTRY

In discussing the environmental chemistry of cesium, it is appropriate to make a distinction between the chemistry of metallic stable cesium in the environment and the various chemical forms of Cs-137 that might be released to air surface water and the subsurface environment at a nuclear facility, such as Hanford. As described in ATSDR (2004),

‘Cesium is a silvery white, soft, ductile metal with only one oxidation state (+1). At slightly above room temperature, cesium exists in the liquid state. Compared to the other stable alkali metals, cesium has the lowest boiling point and melting point, highest vapor pressure, highest density, and lowest ionization potential. These properties make cesium far more reactive than the other members of the alkali metal group. When exposed to air, cesium metal ignites, producing a reddish violet flame, and forms a mixture of cesium oxides. Pure cesium reacts violently with water to form cesium hydroxide, the strongest base known, as well as hydrogen gas. The burning cesium can ignite the liberated hydrogen gas and produce an explosion. Cesium salts and most cesium compounds are generally very water soluble, with the exception of cesium alkyl and aryl compounds, which have low water solubility.’

Tables 4-1 and 4-2 of ATSDR (2004) identify and describe the chemical properties of six forms of cesium, including cesium metal, cesium chloride, cesium carbonate, cesium hydroxide, cesium oxide, and cesium nitrate. When released following the detonation of a weapon, the high temperatures tend to form an oxide. When released in liquid effluent from a reactor it will react with water to form a salt or hydroxide, which are soluble and, due to its positive charge in solution, will tend to bind to negatively charged soil and sediment. In solution, it behaves chemically and biochemically as potassium, and it therefore bioconcentrates and is metabolized in a

⁹⁷ P stands for peta and means 10¹⁵.

manner that it similar to potassium; i.e., it is distributed intracellularly in a manner that is similar to, but not exactly the same as, the essential electrolyte, potassium. (ATSDR 2004).

FATE AND TRANSPORT

Air

As described in ATSDR (2004), when airborne (e.g., due to production from a weapons test, discharges to the atmosphere following an accident, or deliberate releases in gaseous effluent during routine operations at a reactor), “radioactive cesium ... can travel thousands of miles before settling to earth. Wet deposition is considered the most important pathway for the removal of radioactive cesium from the atmosphere. It is a complex process that depends upon meteorological conditions such as temperature, the microphysical structure of the clouds, and the rainfall rate, as well as the physical and chemical properties of the airborne cesium.” Dry deposition can also occur.

Also as described in ATSDR (2004), when deposited onto plants, cesium “is absorbed into the flora through its foliage” (Sawidis *et al.* 1990). The deposited cesium can make its way to soil through decomposition of the contaminated foliage.

Surface Soils

Once deposited onto soil or into the subsurface such as from leaking tanks or from deliberate disposal of radioactive wastes, cesium’s mobility is generally very low. Partition coefficients (K_d values) are a measure of the strength of Cs-137’s binding to soil and sediments, and therefore its potential for movement in soil and the subsurface.⁹⁸

Depending on the characteristics of the soil, Sheppard and Thibault (1990) reported K_d values for cesium ranging from 0.2 to 145,000, with central estimates of 280 for sand, 4,600 for loam, 1,900 for clay, and 270 for organic soils. Included in their reported values of K_d , they report values by Baes and Sharp (1983) of a best estimate of 1,100 and range of 10 to 52,000 for agricultural soils and by Coughtrey *et al.* (1985) of a best estimate of 1,000 and a range of 1,000 to 10,000. The implications of these investigations are that, although the K_d values for cesium at a site can be highly variable, the central estimates are generally quite high, and leaching and migration of cesium out of soil and sediment is expected to be slow. The DOE Biota Dose

⁹⁸ The partition coefficient is expressed as follows: $K_d = C_s/C_l$ where:

K_d = the partition coefficient of a given element in soil,

C_s = the average concentration of a given element in soil in contact with the water for sufficient time to achieve equilibrium, and

C_l = the average concentration of the element in water.

Assessment Committee (BDAC) database contains 19 K_d entries for Cs for sands, clays, and loams that range from 0.2 to 360,000.

Of note, Smith and Amonette (2006) summarize the literature describing the limitations of K_d values, explaining that any measured K_d reflects only the very specific conditions under which those measurements were made. It is for this reason that the reported range of K_d values is so large.

Some site-specific partition coefficient information is available for Hanford. NCRP (2007) reports:

“At Hanford, radiocesium is readily bound to soil by abundant micaceous and other clay minerals (NAS/NRC, 1978). The distribution coefficient (K_d) value for cesium in Hanford soils is $\sim 300 \text{ L kg}^{-1}$ (Napier *et al.*, 1988). The relative high K_d value, which relates to the affinity for a compound to be attached to soil, means that cesium does not move very much with water as it percolates down a soil column. According to Schreckhise *et al.* (1993), water in Hanford soils will percolate through a soil column $\sim 1,000$ times as fast as cesium (*i.e.*, the ratio of the velocity of cesium to water is ~ 0.001). Penetration of small but detectable levels of Cs-137 through soil to the water table beneath high-level waste tanks may be possible through waste-modified soil chemistry, colloid transport, or other mechanisms besides normal solution-phase movement. Klepper *et al.* (1979) reported that Cs-137 was detected in the litter and top 1 cm of soil beneath contaminated plants growing over a leachfield containing high levels of Cs-137. No activity was detected between the soil surface and the contaminated leach field which indicates that the Cs-137 was firmly attached to the soil particles and did not migrate downward in the soil column through percolation. Cline and Rickard (1972) reported on the behavior of Cs-137 applied to soil in two outdoor experimental plots that were located near the 100-F Area. In one plot, the Cs-137 was tilled into the top 13 to 15 cm of soil. In the other plot, the Cs-137 was placed on the soil surface. During the first 3 y, both plots were sprinkled with 30 to 100 cm of water each year and then allowed to lay fallow the last 5 y. The vertical distribution of the Cs-137 in both plots changed very little over the 8 y period. Only relatively small amounts of Cs-137 are located in the accessible surface environment on the Hanford Site.”

NCRP (2007) also notes:

“The soil or sediment is particularly important because it is the primary reservoir of Cs-137 in most ecosystems. ... [T]he strength of its binding to soil or sediment particles ... is mainly dependent on the clay mineral composition and abundance. Other chemical factors that modify its transport include the soil or sediment cation exchange capacity (CEC) and pH, and the soluble potassium levels in the system [cesium is chemically similar to potassium and therefore potassium competes with cesium for binding sites on soil].”

Subsurface Soils

Cesium-137 in the subsurface at Hanford and in the Columbia River has been the subject of intensive investigation. Hanson (2000) explains that the subsurface at Hanford is generally sandy, and, as such, the water-holding capacity of the vadose zone at the site is limited. The implications with respect to leaking underground waste storage tanks is that some contaminants discharged to the subsurface can migrate relatively rapidly through the soil matrix and into the underlying water table. The water table about 200 to 300 feet beneath the surface, and then becomes more shallow as one approaches the Columbia River. Hence, there is a concern that some radionuclides, including Cs-137, can and have reached ground water and also entered the Columbia River.

The speed at which Cs-137 is migrating in the vadose zone very much depends on the chemical and physical properties of the waste and the vadose zone. Because of the complexity of the subsurface environment horizontally, vertically, and over time at the site (especially in the Central Plateau 200 Area where the waste storage tanks are located), predicting the subsurface behavior of most radionuclides, including Cs-137, is a challenging undertaking. Brown and Serne (2008) discuss the history and current status of this issue since the inception of the Vadose Zone Characterization Program (VZCP) in 1997 (DOE 1996). The VZCP is a comprehensive subsurface investigation program in the 200 Areas, where extensive subsurface investigations were implemented in order to better understand the migration of toxicants leaking from the waste storage tanks. Due to the abundance of Cs-137 in the tanks, Cs-137 was used as the benchmark radionuclide for these investigations. In addition, the program resulted in a Cs-137 model that has been used as the basis for ongoing research on the migration pattern and speed of radionuclides and toxic chemicals in the vadose zone in the 200 Areas.

Brown and Serne (2008) report that the waste water migrating beneath the site, along with its dissolved and suspended radionuclides and other contaminants, does not appear to be migrating vertically, as previous believed, due to the geological layering in the vadose zone, which include thin, fine-grained lenses of sediments, which cause horizontal migration. They also report that predicting contaminant migration is complicated by the variable pH in the vadose zone, due to material with a high pH in some tanks, which is buffered by natural minerals in the vadose zone. The uncertainty and variability in pH is important because, in general, an elevated pH tends to cause Cs-137 to precipitate, which could slow migration, while acidic environments could have the opposite effect. In addition, the amount of salts and other ions in solution affect the ion exchange capacity of the soil. Brown and Serne (2008) specifically discuss the effect of these processes on the mobility of Cs-137, citing K_d values ranging from 10 to 1000 ml/g depending on the soil chemistry. Flury *et al.* (2004) confirmed through experiment that ionic strength of the media can have a ten-fold effect on the Cs retardation coefficient.⁹⁹

⁹⁹ The retardation coefficient (R_d) is the velocity of a chemical in the unsaturated and saturated zone relative that of water. It is related to K_d according to the following equation (Codell and Duguid, 1983):

Smith and Amonette (2006) cite studies that reveal that many radionuclides, including Cs-137, can move relatively quickly through the unsaturated zone. The reasons cited include (1) adsorption onto colloids that remain suspended in soil pore water and move at the rate that the water moves through the vadose zone (as opposed to binding to the soil in the vadose zone), (2) pH and oxidative state affect the binding capability of some radionuclides to soil, (3) the presence of organic and inorganic complexing agents, including microbial activity and dissolved carbonates, and (4) chelating agents, such as EDTA. Campbell *et al.* (1994), as cited in Ballou *et al.* (1996), identified several chelating agents in the Hanford tanks. Cherry (2003) isolated colloids from Hanford sediments and demonstrated that they also affected transport through sediment under steady-state unsaturated conditions.

These investigations reveal that the chemistry of cesium in the subsurface at Hanford can be complex, and it is difficult to draw simple conclusions regarding the rate at which it may be moving through the vadose zone at different locations at the site and at different time periods.

Water and Sediment

ATSDR (2004) states: “Since cesium does not volatilize from water, transport of cesium from water to the atmosphere is not considered likely, except by windblown sea sprays. Most of the cesium released to water will adsorb to suspended solids in the water column;” hence, the fate of Cs-137 in the Columbia River is expected to be strongly related to the fate of the river’s sediments. As an alkali metal, it stays in ionic solution (as would potassium and sodium) and tends also to bind to negatively charged sediment particles in suspension in the water column and then deposited in the bottom sediment. It also competes with other alkali (K^+ and Na^+) and alkaline earth metals (Ca^{2+} and Mg^{2+}) metals in solution for binding sites on sediment.

Ritchie (2005) has compiled a bibliography of about 3,000 publications related to Cs-137 erosion and sediment deposition, many of which address Cs-137 at the Hanford reservation and the Columbia River. Cesium-137 is the focus of attention of so many studies primarily due to the widespread contamination of soil, water, and sediment resulting from Cs-137 in fallout. In addition to concern over the potential public health and environment impacts of Cs-137 in the environment, these investigations are using Cs-137 as a tool to better understand the kinetics of soil and sediment erosion.

Biota

At Hanford, biota themselves have been shown to be a transportation mechanism for Cs-137. NCRP (2007) reports that “deep-rooted plants, such as tumbleweed (*Salsola kali*), have transported subsurface ^{137}Cs to the surface. Windblown plants can

$$R_d = n/n_e + \rho_b/n_e K_d$$

Where

n = total porosity

n_e = effective porosity

ρ_b = bulk density (g/cm^3)

transport radionuclides some distance before depositing contamination on the soil surface (Johnson *et al.*, 1994).” In addition, NCRP (2007) states the following:

“Some radionuclides have been taken up by animals from the open disposal systems such as ditches and ponds that received liquid radioactive wastes (Emery and McShane, 1980)... “noteworthy location is the BC Crib area.... Animals removed Cs-137 salts and other radionuclides from an underground disposal facility and, primarily *via* deposition of feces, scattered the radionuclides over a large area (Johnson *et al.*, 1994).” “Another mechanism for contamination of biota is burrowing by small mammals and insects into contaminated subsurface soil. Burrows can then be invaded by other species such as snakes and birds. Contaminated small animals such as rabbits can in turn be consumed by predators such as raptors and coyotes. These phenomena all provide mechanisms for biologically mediated dispersal of radioactive materials well beyond the confines of waste disposal sites.”

BIOACCUMULATION POTENTIAL

Cs-137 can bioconcentrate and has been shown to bioaccumulate in both terrestrial and aquatic food chains (ATSDR 2004). As stated in NCRP (2007), “The passage of radiocesium up through animal food chains, unlike the vast majority of other radionuclides, often increases from one trophic level to the next higher trophic level. For example, predatory animals tend to concentrate Cs-137 in their soft tissues to a higher degree than do the animals upon which they feed” (NCRP 2007).

Of note, however: “The accumulation of cesium varies by orders of magnitude between different biological components within a single environment and also among different ecosystems. Much of this observed behavior can be understood from the chemical properties of cesium and its interactions with soil and sediment particles... The fraction of the Cs-137 in ecosystems that is available for biological uptake and transport is largely determined by the strength of its binding to soil or sediment particles” (NCRP 2007). As stated previously, the strength of Cs-137’s binding to soil (and hence, its bioavailability) depends on a range of location-specific factors that can vary over space and time. In aquatic systems, a large humic content and high levels of potassium cations reduce the bioconcentration and bioaccumulation of Cs-137 (Penttila *et al.* 1993 as cited in ATSDR 2004).

NCRP (2007) cites the following default values for predicting the environmental concentration of Cs-137 at Hanford (from Schreckhise *et al.* 1993):

- Concentration ratios for terrestrial vegetation (Bq kg⁻¹ dry vegetation per Bq kg⁻¹ dry soil):
 - leafy vegetables = 2×10^{-2}
 - root vegetables = 2×10^{-2}
 - fruit = 2×10^{-2}
 - grain = 2×10^{-2}
- Equilibrium transfer coefficient (Bq kg⁻¹ wet food product per Bq d⁻¹ consumed):
 - meat = 3×10^{-2}
 - poultry = 4.4×10^0

- milk = 7×10^{-3}
- eggs = 4.9×10^{-1}
- Concentration ratios for aquatic organisms (Bq kg^{-1} wet tissue per Bq L^{-1} water values):
 - fish = 1.5×10^4
 - crustacea = 5×10^2
 - mollusks = 5×10^2
 - aquatic plants = 1×10^3

A well-researched radioecological characteristic of Cs-137 in freshwater ecosystems is referred to as the trophic level effect. Many investigators have observed a three-fold increase in the concentration of radiocesium as it passes up the food chain from one trophic level to the next in freshwater aquatic biota (Gustafson *et al.* 1966, Kevern, 1966, Nelson et al. 1971, Pendleton, 1962, and others). This effect is attributed to the following. If (1) the assimilation of radiocesium and potassium from food items are the same (McNeill and Trojan, 1960), (2) the concentration of potassium is held constant in the organisms under homeostatic control, and (3) the elimination rate of radiocesium from the organism is one-third that of potassium, the radiocesium to potassium ratio of the food organisms of a fish will be one third that of the fish. Since the potassium concentration is held constant under homeostatic control, the radiocesium concentration triples as one moves up each step in the food chain. This trophic level effect can be impeded if the uptake of radiocesium is reduced if radiocesium uptake is reduced, such as occurs when large amounts of sediment are ingested by bottom feeding organism. Under these circumstances, the presence of sediment in the GI tract of fish binds the radiocesium to the sediment and reduces the assimilation of radiocesium by fish; i.e., the radiocesium is eliminated in the fecal plug as opposed to being absorbed (Mauro 1973).

ACCUMULATION WITHIN TISSUES

As described by Eisenbud and Gesell (1997), cesium is a congener of potassium (K) and is therefore taken up by all organisms in a manner similar to that of potassium. Potassium is an essential element under homeostatic control, is required by all organisms to maintain electrolytic balance, and is biochemically maintained at very narrow intracellular and extracellular and concentration. To a degree, cellular biochemical machinery cannot distinguish between Cs and K. As a result, cells take up cesium using active transport mechanisms in a manner similar to that of potassium (NCRP 1977, Sakhnini and Gilboa, 1998).

Because of the biochemical similarities between cesium and potassium, if soil is low in potassium, there will be a tendency for plants to take up more Cs-137. Extensive evidence of this phenomenon is provided in many studies of the Marshall Islands. Two classic issues of the Journal of the Health Physics Society, Volume 73, No. 1 (1997) and Volume 99, No. 2 (2010), provide a wealth of information on this important radioecological subject.

Overall, however, as described in ATSDR (2004) and NCRP (2007), once assimilated, Cs-137 is metabolized in a manner very similar to that of potassium; i.e., it is

absorbed intracellularly and is relatively uniformly distributed throughout the organism. For example, Cs-137 accumulates in both the shells and soft tissue of the freshwater mussel *Lampsilis radiata* (Harvey 1969 as cited in Havlik 1987).

**IV. TYPICAL
MAJOR
EXPOSURE
ROUTES**

As discussed in other sections of this profile, surface soil at Hanford has been contaminated as a result of the discharge and subsequent deposition of Cs-137 in airborne effluents; the subsurface environment has been contaminated from leaking waste tanks, trenches, and cribs, primarily in the 200 Areas; terrestrial plants have been contaminated from direct deposition of Cs-137 on resuspended soil and from root uptake; and aquatic ecosystems have been contaminated from deliberate and inadvertent discharges of liquid waste to the Columbia River. As described in NCRP (2007), once Cs-137 is in the environment, the primary pathway by which biota are exposed to Cs-137 is through ingestion of contaminated food and water. Once ingested, Cs-137 is readily assimilated¹⁰⁰ and relatively uniformly distributed throughout the organism. Once it is deposited within an organism, the organism experiences internal exposure from the beta and gamma radiation associated with the decay of Cs-137 and its progeny Ba-137m. In addition, once in the environment, biota also experience external exposures from the beta and gamma radiation emitted by Cs-137 and its progeny present in soil, sediment, and, to a lesser extent, in water.

**V. RADIOLOGICAL
ECOTOXICITY**

In theory, biota can be damaged by both the chemical toxicity and radiotoxicity of Cs-137. However, the specific activity of Cs-137 is relatively high and its chemical toxicity is relatively low. For example, ATSDR (2004) explains that, from a chemical toxicity perspective, stable cesium has an LD₅₀ value for rats and mice ranging from 800 to 2,000 mg Cs/kg, and single oral doses of cesium chloride, administered to female mice at dose levels ranging from 125 to 500 mg/kg, have been shown to result in significant increases in chromosomal breaks in bone marrow cells (Ghosh *et al.* 1990 and 1991). It is important to recognize that the specific activity of Cs-137 is 86 Ci/gram or 0.086 Ci per mg. Hence, a single mg of Cs-137 is highly radioactive and extremely radiotoxic. As a result, the chemical toxicity of Cs-137 is of little to no concern relative to its radiotoxicity.

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

There are no beneficial or protective properties associated with exposure to Cs-137. However, as summarized in the HSDB, Cs-137 does have beneficial uses, including radiotherapy; calibration of equipment used to measure correct patient dosages of radioactive pharmaceuticals; measurement and control of the liquid flow in oil pipelines; telling researchers whether oil wells are plugged by sand; ensuring the right fill level for packages of food, drugs, and other products; for the construction of atomic clocks; in process control instruments; and in sewage and sludge sterilization.

¹⁰⁰ An exception to this general rule is Cs-137 that is tightly bound to soil and sediment. When bound to soil and sediment, Cs-137 is not available to be transported across biologic membranes.

MECHANISMS OF ACTION

Radioecological damages to aquatic and terrestrial organisms due to Cs-137 in the environment result from ionization caused by the interaction of its beta particles and photons with living tissue. In particular, upon each disintegration, Cs-137 emits a beta particle with an average energy of about 0.6 MeV and a gamma ray from Ba-137m of 0.662 MeV (Shleien *et al.* 1998).

Internal Beta Exposures

The range of beta particles in matter is given by (Shleien *et al.* 1998, Formula 2a, p. 3-15):

For ($0.01 \leq E \leq 2.5$ MeV):

$$R = 412 * E^{(1.265 - 0.0954 * \ln(E))}$$

Where:

R = range in mg/cm² (range in cm times the density of the absorbing medium in mg/cm³)

E = energy of the beta particle in MeV

Using this equation, the approximate range of Cs-137 beta particles in water (which is similar to tissue) is 1.533 cm.¹⁰¹ Given that the typical energy required to ionize a molecule (i.e., eject an electron from its orbit) is about 34 to 35 eV (see page 17, Casarett 1968), the total number of ion pairs produced by the energy deposited in tissue from the average energy beta particle emitted by Cs-137 is about 17,000 ion pairs (i.e., 0.6 MeV/35 eV).

The pattern of energy deposition for beta particles is described in Morgan and Turner (1973) as follows:

$$\text{Mean linear ion density} = T/R_t \times W$$

Where:

T = average energy of electron liberated

R_t = range or electrons of energy T

W = average energy to form an ion pair

For Cs-137, the equation is $0.6 \text{ MeV} \times 1,000,000 \text{ eV/MeV} \div 1.533 \text{ cm} \times 35 \text{ eV/ion pair} = 1.1 \times 10^4$ ion pairs per cm or about 1 ion pairs per micron. Given that a typical cell is on the order of tens of microns (see page 102 of Curtis and Barnes 1989), a single cell might experience about 10 to 20 ion pairs produced by the passage of an average Cs-137 beta particle. It is this deposited energy in living tissue that results in biological damage.

¹⁰¹ Also see Figure 5.8.1 of Shleien (1998).

External Beta Exposures

Sufficiently energetic beta particles can penetrate the dead layer of the skin of mammals (nominally 70 microns in humans) and deposit energy in underlying tissues. Thus, there is a real potential for exposure to terrestrial organisms from external radiation from beta particles emitted by Cs-137, except for organisms that have a thick outer layer (such as bark of trees, heavy fur, etc.) that can shield the living tissue beneath from the beta emissions. In theory, aquatic organisms can also experience external exposure from beta particles but, due to the limited range of Cs-137 beta particles in water (about 1 cm), only Cs-137 in very close proximity to the organisms can result in exposure to living tissue.

Internal and External Gamma Exposures

Terrestrial and aquatic organisms can also experience internal exposures from the 0.662 MeV photons emitted by Cs-137's short-lived progeny, Ba-137m. However, because only about 10% of the photon energy is deposited per cm of path length in tissue (see Figure 5.4 of Shleien *et al.* 1998), the contribution of gamma exposure from Ba-137m to the internal dose is small compared to the internal dose from the beta emission. However, external exposure to terrestrial organism from the gamma emissions from Cs-137 (and its progeny) in soil must be taken into consideration when assessing the overall dose to terrestrial biota. For aquatic biota, close proximity to sediment containing Cs-137 could contribute to external exposures.

FACTORS AFFECTING TOXICITY

The adverse effects of both external and internal exposure to Cs-137 are due to the deposition of ionizing radiation in living tissue, and the associated disruption of the organism's biochemical machinery primarily through direct ionization of macromolecules and indirect damage from free radicals produced by the ionization of water molecules. There are numerous environmental factors can enhance or reduce the potential for biota to be exposed to Cs-137. As discussed above, the uptake of radiocesium can be enhanced or reduced by the amount of potassium in the environment. If cesium is tenaciously bound to soil and sediment, it is less likely to be available for uptake by biota. Also, as discussed in the papers published in Volume 73 (1) 1997 and in Volume 99(2) 2010 of Health Physics, the addition of potassium to soil (i.e., fertilizer) can reduce the uptake of radiocesium by plants and therefore also protect organisms higher up the food chain.

NCRP (2007) devotes a section (7.3.5) of the report on the extensive research performed in the former Soviet Union following the Chernobyl accident and the contamination of the Pripyat and Dnieper Rivers. The report explains that following contamination of the Pripyat-Dnieper River, the Cs-137 dissolved in surface waters was rapidly bound to suspended sediment and settled to the bottom sediment. The sediment was then transported downstream to the sediment in the Kiev Reservoir. Hence, natural attenuation by sedimentation helps to clear surface waters, but of course, increases exposures to bottom feeding organisms and organisms whose lifecycle includes contact with sediment.

PLANTS

No literature was found that explicitly addresses the radiotoxicity of Cs-137 on plants at Hanford. However, there is an abundance of publications on the effects of radiation in general on plants and plant communities, and some of this is specific to Cs-137. A classic series of investigations on the effects of external gamma ionizing radiation on plant communities was performed at Brookhaven National Laboratory in Upton, New York, in 1962. A large (9,500 Ci) Cs-137 source was placed in a pine forest for 20 hours per day, where the external exposures ranged from several thousand R¹⁰² per day within a few meters of the source to about 1 rad per day at 130 meters from the source. After 6 months of exposure, a total kill zone was observed at dose of >350 R/day. At 10 R/day, there was reduced shoot growth of all tree species, but no trees died (Casarett 1968).

Chapter 13 of Casarett (1968) provides an excellent review of the literature on the effects of radiation on higher plants and plant communities. She provides data showing the percent germination for pollen for a variety of plants, as a function of dose, where the doses ranged from zero to over 6,000 rad. She also summarizes studies on the effects of radiation on the fertilized egg (ovule), where effects on the developing plant were observed at 500 R, and the radiosensitivity of developing embryos (fertilized ovule) varied 100-fold depending on plant species.

Casarett (1968) also presents the results of investigations performed by Sparrow and Woodwell (1962), where the effects of chronic exposure to Co-60 were measured. The effects included growth reduction, failure to set seed, pollen sterility, floral inhibition or abortion, and lethality.

Driver (1994) does not specifically address the toxicity of Cs-137 to plants but does review experiments evaluating the effects of radiation on terrestrial plants (including the Brookhaven experiments). The following is excerpted from Driver (1994):

“Plants are relatively resistant to ionizing radiation. [*It should be noted that experience following the Chernobyl accident found pine trees to be radiosensitive, see below.*] The effects of chronic irradiation (6 months) of a late successional oak-pine forest were studied at Brookhaven National Laboratory (BNL) in New York. Changes in ecosystem structure, diversity, primary production, total respiration, and nutrient-inventory occurred. The most resistant species were the ones commonly found in disturbed places, i.e., generalists capable of surviving a wide range of conditions. Mosses and lichens survived exposures greater than 1000 R/d. No higher plants survived greater than 200 R/d. Sedge (*Carex pennsylvanica*) survived 150 to 200 Rad. Shrubs (*Vaccinium* and *Quercus ilicijolia*) survived 40 to 150 R/d. Oak trees survived up to 40 R/d, whereas pine trees were killed by 16 R/d. No change was noted in the number of species in an oak-pine forest up to 2 R/d, but changes in growth rates were detected at exposures as low as 1 R/d (Woodwell 1970). Severe defects were observed in Tradescatia at an exposure rate of 40 R/d. However, an exposure of 6000 R/d was required to

¹⁰² For simplicity, it can be assumed that one R or Roentgen is equal to 1 rad (or 100 ergs of energy deposited per gram of tissue).

produce the same effect in a hybrid gladiolus (Odum 1956). The sensitivity of various plant species appears to be related to the cross-sectional area of the nucleus in relation to cell size: the larger the nucleus and chromosome volume, the more sensitive the plant (Underbrink and Sparrow 1968, 1974).”

Driver (1994) cites studies by Rickard *et al.* (1981) which found that bulrushes, cattails, and pond weeds were not inhibited from colonizing an industrial pond containing Cs-137 concentrations in the sediment of 28,000 pCi/g dry weight.

Also noteworthy are investigations of the damage done to conifer forests in the vicinity of the 1986 Chernobyl accident. Radiation resulted in the death of many pine stands within approximately 5-10 km of the power plant, resulting in the so-called “red forest.” In addition to mortality, adverse effects observed in the forest included reproduction anomalies, growth reductions, and morphological damage (*ibid.*). The absorbed dose was largely due to beta radiation (90%), with some contribution from gamma radiation (10%), and four distinct zones of damage were identified, with different dose levels associated with different severities and types of injury (see Table 6.3 in IAEA 2006).

AQUATIC BIOTA

There is an abundance of publications on the effects of radiation in general on aquatic organisms, but a limited amount of laboratory and environmental radiotoxicity literature specifically addressing the effects of Cs-137 on aquatic biota. A detailed review of the literature on the effects of radiation on aquatic biota is provided in NCRP (1991). More specifically, NCRP (1991) provides an extensive review on the reproductive effects of radiation on in fish and invertebrates in natural and experimental settings. Tables 3.3 to 3.8 of the report summarize the extensive literature on this subject. Data are available on many life stages of the mosquito fish, roach, pond snail daphnia, Chinook salmon, coho salmon, stickleback, pike, rainbow trout, guppy, and medaka.

This report concludes that:

The discharge of low-level radioactive effluents into the aquatic environment has resulted in chronic, low dose rate exposure aquatic organisms. The fate of individual organisms is, generally, not the major concern but rather the response and maintenance of endemic populations.

Experimental studies to date have shown that fertility and fecundity (gametogenesis) of the organisms and embryonic development are probably the most sensitive components of the radiation response, and it is precisely these attributes which are of importance in determining the fate of the population.

Driver (1994) summarizes the literature on the effects of radiation on aquatic organisms, providing LD₅₀ values for fish (90,000 R), 50% survival doses for male and female germ cells (305 to 500 R), and reduction on population growth rate for

white crappie, largemouth bass, and redhorse (25% reduction at 57 R external exposure).

Driver (1994) also summarizes the literature specifically addressing the effects of Cs-137 on fish. The effects included allergic effects at 2,000 Bq/L or more. Also damage to brain and epithelial cells of renal tubules of carp were observed (Vosniakos *et al.* 1991). Rickard *et al.* (1981) observed no effects on carp monitored in an industrial pond containing sediment levels of Cs-137 of about 28,000 pCi/g dry weight. Also, Kimura and Honda (1977a and 1977b) observed no increase on the mortality rate of rainbow trout embryos exposed for 20 days to up to 10 $\mu\text{Ci/L}$ of Cs-137.

Driver (1994) also references studies on the effects of radiation (not specifically Cs-137) on crustaceans, snails, and daphnia, where effects of exposure to radiation were observed but only at very high dose rates (hundreds to thousands of rad).

A more recent review of this subject is provided by the EPA as part of the “Framework for the assessment of environmental impact project (FASSET at <http://www.fasset.org>). In these studies, zebrafish were exposed to gamma radiation at a dose rate of 30, 100, and 740 mrad/hr. Only the highest dose rate group experienced effects on reproductive output (reduced egg count). IAEA (2006) also summarizes information on chronic effects of ionizing radiation on fish reproduction, taken from the FASSET database.

BIRDS

Driver (1994) summarizes publications addressing the harmful effects of Cs-137 to birds:

“Levels in birds exposed to high levels of radiocesium in the environment have been reported to be in excess of the maximum permissible concentrations for man. However, it was not determined if these levels (average body burden of 5 μCi) were harmful to the birds (Krumholz 1954). Red blood cell abnormalities in mallards that accumulated cesium-137 from an abandoned nuclear reactor cooling tower were observed after 8 months of exposure. Aneuploidy in the blood cells was observed after 9 months of exposure. Such changes only occurred with maximum body burdens of cesium-137 (George *et al.* 1991). Willard (1963) calculated that a chronic dose LD_{50} of 21,700 mGy^{103} would be needed to kill 50% of bluebird (*Sialia sialis*) nestlings over a 16-day period of irradiation with cesium-137. Growth of tree swallows was significantly affected by acute doses of 2700 to 4500 mGy (Zach and Mayoh 1984). Hatching success was reduced by chronic doses of 100 mGy/d (Zach and Mayoh 1984). Birds environmentally exposed to cesium-137 during breeding season received total dose equivalent rates to the whole body of 9.8×10^{-7} Sv/h or 2.8 mSv^{104} for the whole period of 120 days

¹⁰³ mGy refers to a milligray, where “milli” means 0.001, and Gray is a unit of absorbed dose equal to 100 rad. One rad is 100 ergs of energy absorbed per gram of absorbing medium.

¹⁰⁴ mSv refers to millisievert, where “milli” means 0.001, and a Sievert is a unit of dose equivalent equal to 100 rem. For beta and gamma radiation 1 rad equals 1 rem. For alpha radiation, the potential damage per rad is many times higher than that associated with exposure to gamma and beta radiation.

(breeding season). No reproductive or population effects were observed in even the most contaminated individuals and species (Lowe 1991). The number of eggs and chicks produced by American coot (*Fulica americana*) colonizing a cooling pond that received low levels of cesium-137 were similar to the number produced on uncontaminated ponds (Rickard *et al.* 1981). The coots consumed aquatic plants containing about 11,000 pCi of cesium/g dry weight and, inadvertently, sediments containing about 28,000 pCi of cesium/g dry weight (Rickard *et al.* 1981).”

MAMMALS

The literature on the adverse effects of internal and external radiation exposures on mammals is substantial, and a number of studies specific to Cs-137 are available. Most of this work has been laboratory-based, although several field investigations have also been performed following the accidents described above in the section on anthropogenic sources of Cs-137. This profile provides a brief summary of the literature addressing the adverse effects of internal and external Cs-137 exposure on mammals.

ATSDR (2004) summarizes the adverse effects of Cs-137 on humans, drawing heavily from exposure to experimental animals. ATSDR (2004) organizes this information first by route of exposure (inhalation, oral, and external), although it is noted that “it has been proposed that adverse health effects, related to a soluble and readily absorbed compound such as $^{137}\text{CsCl}$, should be similar across the three routes of exposure.” Within exposure route, information is organized by health effect category and exposure duration.

For internal exposure, available information is largely limited to experiments on dogs that used intravenous injections. In particular, dose-related decreased survival was observed in beagle dogs that had received single intravenous injections of $^{137}\text{CsCl}$ in amounts resulting in average initial body burdens of 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula *et al.* 1995 and 1996). Depressed blood cell counts and platelet levels, reduced packed-cell volume, and bone marrow aplasia were observed in dogs that had been administered single intravenous injections of $^{137}\text{CsCl}$, which resulted in average initial body burdens ranging from 36.4 to 141.0 MBq/kg (1.0 to 3.8 mCi/kg) (Nikula *et al.* 1995; Redman *et al.* 1972). Severe bone marrow depression was observed in dogs exposed to $^{137}\text{CsCl}$ by intravenous injection at activity levels resulting in estimated total bone marrow doses of 7–24 Gy (700–2,400 rad) (Nikula *et al.* 1995). Benign and malignant neoplasms were found in a variety of tissues and organs of dogs administered single intravenous doses of $^{137}\text{CsCl}$, which resulted in average initial body burdens ranging from 37 to 147 MBq/kg (1.0 to 4.0 mCi/kg) (Nikula *et al.* 1995 and 1996).

Information is also available on adverse effects of acute external Cs-137 exposure to mammals. Specifically, ATSDR (2004) states that significantly reduced survival was noted in rat fetuses following whole-body irradiation (via a Cs-137 source) of pregnant dams on gestational day 14 at acute radiation doses ≤ 4 Gy (400 rad); an LD₅₀ value was about 5 Gy (500 rad) (Koshimoto *et al.* 1994). In male mice, reduced fertility following external Cs-137 exposure, and sterility, have also been observed, as

has increased total and postimplantation embryo mortality (ATSDR 2004). Gestationally-exposed rats experienced reduced postnatal body weight, impaired motor activity, and decreased thickness within cortical layers of the brain, while gestationally-exposed mice experienced smaller litter sizes, smaller head sizes, retarded odontogenesis, decreased brain weights, and cleft palates (*ibid.*), although ATSDR (2004) notes that “the observed developmental effects were the result of radiation exposure, not the presence of cesium *per se.*” Some exposures to Cs-137 resulted in increased mammary tumors in rats, and ATSDR (2004) similarly notes “[t]hese effects were the result of the gamma radiation, not the presence of cesium *per se.*” Also, the age of the rats at the time of exposure affected their risk of carcinoma, with younger animals having higher risks.

The most comprehensive investigations on the effects of radiation on mammals in a natural setting are those that have been performed and are ongoing in the vicinity of the Chernobyl accident. Though the studies do not specifically focus on Cs-137, Cs-137 was one of the primary long-lived radionuclides released during the accident, resulting in widespread contamination. IAEA (2006) notes that in the fall of 1986, the numbers of small rodents on highly contaminated research plots decreased by two to 10 fold; however, immigration assisted in recovery as early as spring 1987. Other effects in this timeframe include pre-implantation deaths in rodents (*ibid.*). A July 30, 2010 BBC News article¹⁰⁵ reports on recent results of the largest wildlife census of its kind conducted in the vicinity of Chernobyl. The article summarizes investigations performed by Dr. Timothy Mousseau from the University of South Carolina and Dr. Anders Moller from the University of Paris-Sud, France. They found a reduction of biodiversity in the vicinity of Chernobyl, including reptiles and mammals.

VI. EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

ATSDR (2004) states that no data were located regarding interactions of cesium with other chemicals that might influence the toxicity of cesium. Concerns over the possible synergistic effects of exposure to radiation and chemical toxins have been extensively raised and reported in the scientific literature (Burkart *et al.* 1997, Prasad *et al.* 2004), but little consensus has been achieved in quantifying these effects in humans except possibly for radon and smoking (BEIR IV 1988) and certainly in the enhancement of the therapeutic effects of radiotherapy used to treat cancer (e.g., Lew *et al.* 2002). UNSCEAR (2000) Annex H explores the combined effects of radiation and chemical agents, including heavy metals. Only a few data are available from combined exposures of radiation and metals in human populations and no firm evidence of interactions has been observed.

As summarized in ATSDR (2004), there is some literature on observed synergistic adverse effects of radiation and toxic chemicals on organisms other than humans (e.g., salmon (Mothersill *et al.* 2007)). Examples of ionizing radiation and metals producing combined effects in other biological systems include synergistic effects on soil microbial activity from cadmium and zinc in combination with gamma radiation (summarized in UNSCEAR 2000). Also, combined effects of cesium-134/137 and

¹⁰⁵ <http://www.bbc.co.uk/news/science-environment-10819027>

lead found in highly contaminated habitats in the Russian Federation increased the mutation rate in the plant *Arabidopsis thaliana* (summarized in UNSCEAR 2000). However, the authors clearly indicate that the relative importance of different damage-inducing mechanisms of metals for combined exposures in human and non-human populations remains to be elucidated.

ATSDR (2004) states that, overall, there is a clear need for additional research on synergistic effects of multiple stressors in radioecotoxicology (e.g., Salbu and Skipperud 2007; Mothersill and Seymour 2007). In particular, these authors raise the issue of pesticides, organics, and endocrine disruptors and synergistic effects with radioactive materials, particularly with long-term exposure to various biological systems. Manti and D'Arco (2010) summarize the in vitro and animal-model studies and epidemiological surveys with two or more stressors, including radionuclides (DNA-damaging agents). They also emphasize that most research focuses only on the short-term effects of combined single exposures to animal models, and more work is needed to understand chronic exposure to trace contaminants and radioactive elements in the environment, including impacts to long-term genome stability. Specific research is lacking on Cs-137 effects with multiple stressors on biological systems, particularly non-human systems.

VII. DATA GAPS Some literature is available addressing the effects of Cs-137 exposure (and, more generally, radiation exposure) to wild plant species, but little research has been performed on species native to the Hanford site. Species-specific information is of the most use as different plant species have been shown to be differentially sensitive to the effects of radiation.

For aquatic biota, there is an abundance of publications on the effects of radiation in general although a literature specifically addressing the effects of Cs-137 is limited. Also, while some literature is available describing the radiosensitivity of certain aquatic invertebrates (crustaceans, snails, and daphnia), little is available specific to Cs-137, and effects information on unionids appears to be lacking. Specific information on the effects of Cs-137 in amphibians and reptiles is also lacking.

The literature on effects of Cs-137 on mammals is more substantial but, due to human health concerns, has largely focused on domestic or laboratory species, and data on effects under field conditions are fewer. Overall, the majority of research has been laboratory-based, although some field data are available primarily because of accidental releases of Cs-137. In addition, little is known about the combined action of exposure to radiation and other environmental toxicants.

- VIII. REFERENCES** Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for Ionizing Radiation. Atlanta, Georgia: Agency for Toxic Substances and Disease Registry.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Toxicological Profile for Cesium. Atlanta, Georgia: Agency for Toxic Substances and Disease Registry.
- Baes, C.F., III, and R.D. Sharp. 1983. A proposal for estimation of soil leaching and leaching constants for use in assessment models. *Journal of Environmental Quality* 12(1):17-28.
- Ballou, N.E., G.R. Ducatte, C. Quasng, and V.T. Remcho. 1996. Determination of chelating agents in Hanford waste tank stimulant. *Journal of High Resolution Chromatography* 19(4):183–188.
- BEIR IV. 1988. Health Risks of Radon and Other Internally Deposited Alpha-Emitters, BEIR IV, Biological Effects of Ionizing Radiation, National Research Council, National Academy of Sciences, National Academy Press, Washington, DC, 1988.
- Brown, C.F. and R.J. Serne. 2008. Deep vadose zone characterization at the Hanford site: Accomplishments from the last ten years. Waste Management Conference, February 24-28, 2008. Phoenix, AZ. Abstract #8412.
- Burt, R.O. 1993. Cesium and cesium compounds. In: Kroschwitz J.I., Howe-Grant M., eds., *Kirk-Othmer Encyclopedia of Chemical Technology*, 4th ed. Vol. 5. New York: John Wiley & Sons, 749-764.
- Butterman, W.C., W.E. Brooks, and R.G. Reese, Jr. 2005. Mineral Commodity Profiles: Cesium. U.S. Geological Survey Open-File Report 2004-1432.
- Burkart, W., G.L. Finch, and T. Jung. 1997. Quantitative health effects from the combined action of low-level radiation and other environmental agents can new approaches solve the enigma. *Science of the Total Environment* 205(1): 51-70.
- Buzulukov, Y.P. and Y.L. Dobrynin. 1993. Release of radionuclides during the Chernobyl accident. In: S.E. Merwin and M.I. Balonov, eds., *The Chernobyl Papers, Volume I Doses to the Soviet Population and Early Health Effects*. Research Enterprises.
- Campbell, J.A. *et al.* 1994. Analytical methods development: Fiscal year 1993 Progress report, PNL-9062, Pacific Northwest Laboratory, Richland, Washington. January 3, 1994.
- Casarett, A.P. 1968. *Radiation Biology*. Englewood Cliffs, New Jersey: Prentice-Hall, Inc.
- Cherry, K.D. 2003. Nitrate and colloid transport through coarse Hanford sediments under steady state, variable saturated flow. *Water Resources Research* 39(1165): 10PP.

- Cline, J.F. and W.H. Rickard. 1972. Radioactive strontium and cesium in cultivated and abandoned field plots. *Health Phys.* 23(3):317–324.
- Codell and J.D. Duguid 1983. Transport of radionuclides in groundwater. In: John E. Till and H. Robert Meyer, eds., *Radiological Assessment – A Textbook on Environmental Dose Analysis*. Prepared for the Nuclear Regulatory Commission. NUREG/CR-3332, ORNL-5968.
- Coughtrey, P.J., D. Jackson, and M.C. Thorne. 1985. *Radionuclide Distribution and Transport in Terrestrial and Aquatic Ecosystems. A Compendium of Data*. Netherlands: A.A. Balkema.
- Curtis, H. and N.S. Barnes. 1989. *Biology*. Worth Publishers, Inc.
- Driver, C.J. 1994. *Ecotoxicity Literature Review of Selected Hanford Site Contaminants*. PNL-9394, Pacific Northwest Laboratory, Richland, Washington. <http://www.osti.gov/energycitations/servlets/purl/10136486-6sLptZ/native/10136486.pdf>.
- Eisenbud, M. and T. Gesell. 1997. *Environmental Radioactivity from Natural, Industrial, and Military Sources*, Fourth Edition. Academic Press.
- Emery, R.M. and M.C. McShane. 1980. Nuclear waste ponds and streams on the Hanford Site: An ecological search for radiation effects. *Health Phys.* 38(5):787–809.
- Flury, M., S. Czigany, G. Chen, and J.B. Harsh. 2004. Cesium migration in saturated silica sand and Hanford sediments as impacted by ionic strength. *Journal of Contaminant Hydrology* 71:111–126.
- George, L. S., C. E., Dallas, I. L. Brisbin, Jr., and D. L. Evans. 1991. Flow cytometric DNA analysis of ducks accumulating ¹³⁷Cs on a reactor reservoir. *Ecotoxicol. Environ. Safety* 2(1):337-347.
- Gephart, R.E. and R.E. Lundgren. 1998. *Hanford tank cleanup: A guide to understanding the technical issues*. Columbus, Ohio: Battelle Press.
- Ghosh, A., A. Sharma, and G. Talukder. 1990. Clastogenic effects of cesium chloride on mouse marrow cells in vivo. *Mutat Res* 244:295-298.
- Ghosh A., A. Sharma, and G. Talukder. 1991. Cytogenetic damage induced *in vivo* to mice by single exposure to cesium chloride. *Environ Mol Mutagen* 18:87-91.
- Gustafson, P.F., S.S. Brar, and S.E. Muniak, 1966. Cs-137 in edible freshwater fish. *Nature* 211:843.
- Hanson, L.A. 2000. *Radioactive Waste Contamination and Groundwater at the Hanford Site, Principles of Environmental Toxicology*, University of Idaho, November 2000.
- Harvey, R. S. 1969. Uptake and loss of radionuclides by the freshwater clam *Lampsilis radiata* (Gmel.). *Health Phys.* 17:149-154.

- Havlik, M.E. 1987. Effects of contaminants on naiad mollusks (Unionidae): A review. U.S. Department of the Interior, U.S Fish and Wildlife Service. Resource Publication 164.
- International Agency for Research on Cancer (IARC). 2000. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972–PRESENT. (Multivolume work). Available at: <http://monographs.iarc.fr/index.php> p. v75 p. 154-5 (2000)]
- International Atomic Energy Agency (IAEA). 2006. Environmental Consequences of the Chernobyl Accident and their Remediation: Twenty Years of Experience. Report of the Chernobyl Forum Expert Group 'Environment'. Radiological Assessment Reports Series. Vienna.
- International Commission on Radiological Protection (ICRP). 1983. Radionuclide transformations: Energy and intensity of emissions. Oxford: Pergamon Press. ICRP publication 38. 484-503.
- Johnson, A.R., B.M. Markes, J.W. Schmidt, A.N. Shah, S.G. Weiss, and K.J. Wilson. 1994. Historical Records of Radioactive Contamination in Biota at the 200 Areas of the Hanford Site, WHC-MR-0418 (National Technical Information Services, Springfield, Virginia).
- Kevern, N.R. 1966. Feeding rate of carp estimated by a radioisotopic method. Trans. A. Fish. Soc. 95(4):363.
- Kimura, Y. and Y. Honda. 1977a. Uptake and elimination of some radionuclides by eggs and fry of rainbow trout (I). Journal of Radiation Research 18:170-181.
- Kimura, Y. and Y. Honda. 1977b. Uptake and elimination of some radionuclides by eggs and fry of rainbow trout (II). Journal of Radiation Research 18:182-193.
- Klepper, E.L., L.E. Rogers, J.D. Hedlund, and R.G. Schreckhise. 1979. Radioactivity Associated with Biota and Soils of the 216-A-24 Crib, PNL-1948 (National Technical Information Services, Springfield, Virginia).
- Koshimoto, C., S. Takahashi, and Y. Kubota. 1994. Evaluation of the effect of gamma-irradiation on fetal erythropoiesis in rats using blood cell volume as the index. J Radiat Res 35:74-82.
- Krumholz, L.A. 1954. A Summary of Findings of the Ecological Survey of White Oak Creek, Roane, County, Tennessee, 1950–1953, Tennessee Valley Authority, Vol. III. ORO-587, USAEC, Oak Ridge, Tennessee.
- Lew, Y.S., A. Kolozsvary, S.L. Brown, and J.H. Kim. 2002. Synergistic interaction with arsenic trioxide and fractionated radiation in locally advanced murine tumor. American Association for Cancer Research.
- Lowe, V.P.W. 1991. Radionuclides and the Birds at Ravensglass. Environ. Poll. 1-26.

- Manti, L. and A. D'Arco. 2010. Cooperative biological effects between ionizing radiation and other physical and chemical agents. *Mutation Research* 704:115-122.
- Mauro, J. 1973. The accumulation of Cs-137 by *Fundulus heteroclitus* in the Hudson River estuary. PhD Thesis. New York University Medical Center, June 1973.
- McNeill, K.G. and A.O.D. Trojan. 1960. The cesium-potassium discrimination ratio. *Health Physics*, 4:109.
- Mikhailov, V.N. Editor in Chief. 1999. *Catalogue of Worldwide Nuclear Testing*. Begell-atom, LLC.
- Morgan, K.Z. and J.E. Turner. 1973. *Principles of Radiation Protection, A Textbook of Health Physics*. Huntington, New York: Robert E. Krieger Publishing Company.
- Mothersill, C., B. Salbu, L.S. Heier, H.C. Teien, J. Denbeigh, D. Ougton, B.O. Rosseland, and C.B. Seymour. 2007. Multiple stressor effects of radiation and metals in salmon (*Salmo salar*). *Journal of Environmental radioactivity* 96(1-3): 20-31.
- Mothersill, C. and C. Seymour. 2007. Radiation risks in the context of multiple stressors in the environment – issues for consideration. In: Mothersill, C., I. Mosse, and C. Seymour, eds. *Multiple stressors: a challenge for the future*. Book Series: NATO Science for Peace and Security Series C – Environmental Security. pp. 235–246.
- Napier, B.A., R.A. Peloquin, D.L. Strenge, and J.V. Ramsdell. 1988. GENII – The Hanford Environmental Radiation Dosimetry Software System, Volume 1: Conceptual Representation, PNL-6584 (Pacific Northwest Laboratory, Richland, Washington).
- National Academy of Sciences / National Research Council (NAS/NRC). 1978. National Academy of Sciences/National Research Council. *Radioactive Wastes at the Hanford Reservation: A Technical Review* (National Academies Press, Washington).
- National Council on Radiation Protection & Measurements (NCRP). 2007. *Cesium-137 in the Environment: Radioecology and Approaches to Assessment and Management*. NCRP Report No. 154. Recommendation of the National Council on Radiation Protection and Measurements, September 4, 2007.
- National Council on Radiation Protection & Measurements (NCRP). 1991. *Effects of Ionizing Radiation on Aquatic Organisms*. NCRP Report No. 109.
- National Council on Radiation Protection & Measurements (NCRP). 1977. *Cesium-137 from the Environment to Man: Metabolism and Dose*. NCRP Report No. 52. Recommendation of the National Council on Radiation Protection and Measurements, January 15, 1977.

- Nelson, D.J., N.A. Griffith, J.W. Gooch, and S.A. Rucker. 1971. White Oak Lake Studies. ORNL-4634.
- Nikula, K.J., B.A. Muggenburg, and I-Y Chang. 1995. Biological effects of ¹³⁷CsCl injected in beagle dogs. *Radiat Res* 142:347-361.
- Nikula, K.J., B.A. Muggenburg, and W.C. Griffith. 1996. Biological effects of ¹³⁷CsCl injected in beagle dogs of different ages. *Radiat Res* 146:536-547.]
- Odum, E.P. 1956. Ecological Aspects of waste disposal. In: Proceedings from a Conference on Radioactive Isotopes in Agriculture. TID-7512, USAEC, Technical Information Center, Springfield, Virginia.
- Pendleton, R.C. 1962. Accumulation of Cs-137 through the aquatic food web. *Biological Problems in Water Pollution. Third Seminar. August 13-17, 1962.* U.S. department of Health Education and Welfare.
- Penttila, S., T. Kairesalo, and A. Uusi-Rauva. 1993. The occurrence and bioavailability of radioactive ¹³⁷Cs in small forest lakes in southern Finland. *Environ Pollut* 82:47-55.
- Poston, T.M., R.W. Hanf, R.L. Dirkes, and L.F. Morasch, eds. 2003. Hanford Site Environmental Report for Calendar Year 2002. PNNL-14295, Pacific Northwest laboratory, Richland, Washington.
- Prasad, K.N., W.C. Cole, and G.M. Hasse. 2004. Health risks of low dose ionizing radiation in humans: A review. *Experimental Biology and Medicine* 229:378-382.
- Redman, H.C., R.O. McClellan, and R.K. Jones. 1972. Toxicity of ¹³⁷CsCl in the beagle. Early biological effects. *Radiat Res* 50:620-648.
- Rickard, W.H., R.E. Fitzner, and C.E. Cushing 1981. Biological colonization of an industrial pond: Status after two decades. *Environ. Conserv.* 8:241-247.
- Ritchie. 2005. Bibliography of publications of cesium-137 Studies related to erosion and sediment deposition. United States Department of Agriculture, Agricultural Research Service, Hydrology and Remote Sensing Laboratory, BARC-West, Bldg. 007, Beltsville, Maryland.
- Sakhnini, A. and H. Gilboa 1998. Nuclear magnetic resonance studies of cesium-133 in the halophilic halotolerant bacterium Ba1. chemical shift and transport studies. *NMR Biomed* 11(2):80-86.
- Salbu, B. and L. Skipperud 2007. Challenges in radioecotoxicology. In: Mothersill, C., I. Mosse, and C. Seymour, eds. *Multiple Stressors: A Challenge for the Future.* Book Series: NATO Science for Peace and Security Series C – Environmental Security. pp. 3–12.
- Sawidis, T., E. Drossos, and G. Heinrich. 1990. Cesium-137 accumulation in higher plants before and after Chernobyl. *Environ Int* 16:163-169.

- Shleien, B., L.A. Slaback, Jr., and B. K. Birky, 1998. Handbook of Health Physics and Radiological Health (eds.). Third Edition. Williams & Wilkins, A Waverly Company.
- Schreckhise, R.G., K.C. Rhoads, J.S. Davis, B.A. Napier, and J.V. Amsdell. 1993. Recommended Environmental Dose Calculation Methods and Hanford-Specific Parameters, PNL-3777, Rev. 2, Pacific Northwest Laboratory, Richland, Washington.
- Sheppard M.I. and D.H. Thibault. 1990. Default soil to solid/liquid partition coefficients, K_dS, for major soil types: A compendium. Health Physics 59(4):471-482.
- Smith, B. and A. Amonette. 2006. The Environmental Transport of Radium and Plutonium: A Review. Institute for Energy and Environmental Research, June 23, 2006.
- Sparrow, G.M. and G.M. Woodwell. 1962. Prediction of sensitivity of plants to chronic gamma radiation. Radiation Botany 2:9-26.
- Underbrink, A.G., A.H. Sparrow, and V. Pond. 1968. Chromosome and cellular radiosensitivity. II. Use of interrelationships among chromosome volume, nucleotide content and dose of 120 diverse organisms in predicting radiosensitivity. Radiation Botany 8:205-237.
- Underbrink, A.G. and A.H. Sparrow. 1974. The influence of environmental endpoints, dose, dose rate, neutron energy, nitrogen ins, hypoxia, chromosome volume and ploidy on RBE in Tradescantia stamen hairs and pollen. In: biological effects of neutron irradiation, pp. 185–214. International Atomic Energy Agency, Vienna, Austria.
- United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2000. Sources and Effects of Ionizing Radiation. United National Scientific Committee on the Effects of Atomic Radiation, UNSCEAR 2000, Report to the General Assembly, United Nations, New York, 2000.
- United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2008. Sources and Effects of Ionizing Radiation. United National Scientific Committee on the Effects of Atomic Radiation, UNSCEAR 2000, Report to the General Assembly, United Nations, New York, 2008.
- U.S. Department of Energy (DOE). 1996. Vadose zone characterization project at the Hanford tank farms. SX Tank farm Report. DOE/ID/12584-268, GJPO-HAN-4, Department of Energy. September 1976.
- Vosniakos, F. A. Kesidou, A. Kalfa, A. Moumtzis, and P. Karakoltsidis. 1991. Uptake of ¹³⁷Cs in cultured fresh water fish (*Cyprinus carpio*): Physiological and histological effects. Toxicol. Environ. Chem. 31-32:353-356.
- Willard, W.A. 1963. Relative Sensitivity of Nestlings of Wild Passerine Birds to Gamma Radiation. In: V. Shultz and A. W. Clement, eds., Radioecology, pp. 345–349. New York: Van Nostrand Reinhold.

- World Health Organization (WHO). 1983. Selected radionuclides: Tritium, carbon-14, krypton-85, strontium-90, iodine, caesium-137, radon, plutonium. Environmental Health Criteria 25. Geneva: World Health Organization.
- Woodwell, G.M. 1970. Effects of pollution on the structure and physiology of ecosystems. *Science* 168(3930): 429-433.
- Zach, R. and K.R. Mayoh. 1984. Gamma-radiation effects on nestling tree swallows. *Ecology* 65:1641-1647.

CHROMIUM (Cr) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Chromium is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington.

Chromium can exist in oxidation states ranging from -2 to +6 but is most frequently found in the environment in the trivalent (Cr^{+3} or Cr(III)) and hexavalent (Cr^{+6} or Cr(VI)) oxidation states. This profile focuses primarily on Cr(VI) because it is the most toxic form of the metal and because this form is widespread in the Hanford environment.

II. SOURCES **NATURAL SOURCES**

Chromium is a naturally occurring metal. At the Hanford site, the weathering of rocks that contain chromite ore is a natural source of chromium. Specifically, it is found in “basaltic rock fragments within sedimentary formations that overlie the Columbia River Basalt Group” (Dauble *et al.* 2003). This group “forms the main bedrock of the Columbia Basin and Hanford Site” (Duncan *et al.* 2007). In comparison to the amount of chromium generated by anthropogenic activity at the Hanford site, this natural source is minimal (DOE/RL 1997, Potson *et al.* 2000, as cited in Dauble *et al.* 2003).

ANTHROPOGENIC SOURCES

During operations at the Hanford site, reactors in the 100 Areas were cooled with Columbia River water (Patton 2007) to which hexavalent chromium was added to prevent corrosion (AMEC 2008). Diluted coolant solution, which contained a final concentration of approximately 700 $\mu\text{g/L}$ sodium dichromate ($\text{Na}_2\text{Cr}_2\text{O}_7$) (Hope 1996, Petersen 2009), was added to river water used at plutonium reactor systems (Geist 1994, Hazen 2008). At 8 of the 9 Hanford reactors, a “single-pass” design was used (reactors B, D, F, H, DR, C, KE, and KW) and the used cooling water from these single-pass reactors was stored temporarily in nearby basins (Ridolfi 2006) before being returned to the Columbia River (Gephart 2003). The 107-D/DR coolant water retention basins held large volumes of reactor coolant routinely, on the order of 148 to 204 million gallons (561 to 773 million liters) per day (Hope 1996). Contamination occurred through not only direct discharge of spent cooling water into the Columbia River (Gephart 2003), but also through “leakage from coolant water retention basins” (Hope 1996), leeching of waste discharged into trenches and cribs (Geist 1994), and leakage/spillage of $\text{Na}_2\text{Cr}_2\text{O}_7$ stock solution from chemical delivery pipelines (Hope 1996a, Petersen 2009).

In addition to the 100 Areas, chromium is known to have been used or produced at the 200 Area, the 300 Area, and the 1100 Area (Stratus 2009). Chromium was used for decontamination in the 100, 200, and 300 Areas, including decontamination reactors

that were shut down (Hazen 2008) and for oxidation-state control in the Reduction-Oxidation Plant process. “[S]oil column disposal of liquid wastes associated with decontamination activities” (Hope 1996) constitutes another source of chromium contamination.

Chromium has reached the groundwater at Hanford. The annual Hanford Site Groundwater Monitoring Reports demonstrate chromate’s mobility through the vadose zone and upper unconfined aquifer at Hanford (Serne 2007). Groundwater investigations by DOE beginning in 1999 identified a large Cr(VI) plume in the southwest portion of the 100-D Area. Concentrations in the plume have not decreased significantly over the course of the past 10 years, implying that there is a vadose zone source that continues to supply contamination to the aquifer (DOE 2007, as cited in Petersen 2009).

In addition to the plumes found in the 100 Areas near the Columbia River from usage in reactor cooling water, several plumes are found in the “200 Areas from usage in fuel processing to extract plutonium” (Hartman *et al.* 2004, as cited in Serne 2007). Chromium contamination in groundwater at Hanford is also abundant at the S-SX tank farm (Zachara *et al.* 2004), and plumes have extended into the 600 Area.

Thornton *et al.* (1995) concluded that the majority of the chromium found in the Hanford unconfined aquifer of the 100-D and 100-H Areas (where much of the chromium contamination at the Hanford site has originated) is in the hexavalent state. Data from a Technical Report for the Groundwater Protection Project (Characterization of Systems Task) confirms that the dominant chromium species at Hanford is hexavalent chromium (Serne 2007): “Hanford sediments do not appear to reduce and immobilize significant amounts of chromate over time spans of days to a month” (Cantrell *et al.* 2003, as cited in Serne 2007). Serne concludes “it is not clear whether significant amounts of chromate have been reduced in the Hanford vadose zone over the 40 to 50 years of operations and cleanup efforts, but based on observed chromate groundwater plumes it would appear not” (Serne 2007).

III. ENVIRONMENTAL CHEMISTRY Chromium can exist in oxidation states ranging from -2 to +6 but is most frequently found in the environment in the trivalent (Cr^{+3} or Cr(III)) and hexavalent (Cr^{+6} or Cr(VI)) oxidation states. In fact, the chromium in effectively all environmentally important chromium compounds is in one of these two oxidation states (Eisler 2000), and only the trivalent and hexavalent chromium compounds are “biologically significant” (Driver 1994). The trivalent and the hexavalent species are also the most stable, although various other valence states, which are unstable and short-lived, do exist in biological systems (Shanker *et al.* 2005). Cr(VI) is considered the most toxic form of chromium (Eisler 2000; Patton 2001, Mishra 2008, etc.). By comparison, “Cr(III) is less mobile, less toxic and is mainly found bound to organic matter in both soil and aquatic environments” (Becquer *et al.* 2003, as cited in Shanker *et al.* 2005).

In solution, Cr(VI) exists as a component of a complex anion and may take the form of chromate (CrO_4^{2-}), hydrochromate (HCrO_4^{-1}), or dichromate ($\text{Cr}_2\text{O}_7^{2-}$) (Eisler 2000,

EPA 1998). As such, Cr(VI) acts like a divalent anion rather than a hexavalent cation (Kimbrough 1999). At acidic (low) pHs, the dichromate form dominates (Eisler 2000). Under oxygenated conditions, Cr(VI) is the “dominant dissolved stable chromium species in aquatic systems, and it exists as a component of [one of the] complex anion[s]” (Eisler 2000) named above.

Although chromium does not decompose, environmental conditions can lead to changes in oxidation state. Chromate is “relatively soluble over much of the environmental pH range” (Zachara *et al.* 2004) – approximately pH 6.0 – 8.5 (USEPA 1980)). In general, under reducing conditions Cr(VI) converts to Cr(III), but under oxidizing conditions, Cr(VI) forms (Babula 2008): “all stable Cr(VI) anionic compounds strongly oxidize organic matter on contact, yielding oxidized organic matter and Cr(III)” (Eisler 2000). Consequently, within living organisms, Cr(III) is the predominant form of chromium, as Cr(VI) will rapidly oxidize (Eisler 2000).

In contrast to the numerous pathways for the reduction of Cr(VI), there are very few mechanisms for the oxidation of Cr(III) back to Cr(VI) in environmental settings. “Only two constituents in the environment are known to oxidize Cr(III) to Cr(VI): dissolved oxygen and manganese dioxides (MnO₂) (Eary and Rai, 1987 as cited in Palmer *et al.* 1994). “Studies of the reaction between dissolved oxygen and Cr(III) revealed very little (Schroeder and Lee, 1975) or no (Eary and Rai, 1987) oxidation of Cr(III) even for experiments conducted at pH as great as 12.5 for 24 days. Therefore, the transformation of Cr(III) by dissolved oxygen is not likely to be an important mechanism for the oxidation of Cr(III)” (Palmer *et al.* 1994). Experiments have verified that “[t]here is an increase in the rate and amount of Cr(III) oxidation as pH decreases, and the surface area to solution volume increases” (Palmer 1994). Attention to controlling these two parameters might control the process of “re-oxidation” of Cr(III).

FATE AND TRANSPORT

Hexavalent chromium is highly soluble in water and thus tends to be mobile in aquatic systems. In groundwater, Cr(VI) is mobile both due to its solubility and due to its low adsorption to metal oxides in neutral to alkaline waters (Calder 1988, as cited in Eisler 2000). (Cr(VI) adsorption does increase with decreasing pH (Eisler 2000).) Cr(VI)’s tendency to adsorb to particulates is also dependent on other environmental conditions including particle surface area and the density of active sites on the sorbent (Eisler 1986).

As noted previously, the chromium present in the unconfined aquifer at the Hanford Site is predominantly hexavalent (Thornton *et al.* 1995, as cited in Patton *et al.* 2007). At Hanford, it has been estimated that chromium moves at approximately the same velocity as groundwater (Thorne 2004, as cited in Duncan *et al.* 2007). Hexavalent chromium has been measured in near-shore groundwater wells and Hanford Site riverbank springs (Poston *et al.* 2003 as cited in Patton *et al.* 2005).

The water table height under the Hanford site, which ranges from a few feet to 250 feet below the surface (Gerber 2002), is higher than the average elevation of the rest of the river, resulting in a flow of groundwater towards the river. Flows are larger

when the river discharge is low, because that results in the steepest water table gradient towards the river (Hope 1996a, Dauble 2003).

Through analysis of chemical data collected from seep water samples, Thornton *et al.* (1995) also inferred that most of the hexavalent chromium in Hanford groundwater ultimately discharges into the Columbia River without changing its valence state (Thornton *et al.* 1995). As groundwater passes through the riverbank, minor uptake of chromium by sediment occurs, and a small amount of “[c]hromium precipitates as a result of reduction by labile iron and organic matter in sediments. [However, m]ost chromium probably passes through [the] riverbank mixing zone relatively unaltered” (Thornton *et al.* 1995). “Dilution of hexavalent chromium subsequently occurs during the mixing of groundwater and river water, with relatively little change taking place in speciation” (Thornton *et al.* 1995).

Trivalent chromium is less soluble in water, as it tends to form stable complexes with negatively charged inorganic or organic compounds: it is “unlikely to be found uncomplexed in aqueous solution if anionic or particulate compounds (such as decaying plant or animal tissues, or silt or clay particles) are present” – i.e. in oxidizing conditions (Steven *et al.* 1967; Pfeiffer *et al.* 1980; Ecological Analysis 1981, as cited in Eisler 2000).

In soils, Cr(VI) “may be leached, reduced, absorbed, precipitated, or taken up by a[n]... organism” (Bartlett and James 1988, as cited in Eisler 2000). Cr(VI) can also become airborne as sorbent on particulate matter and distributed in that fashion. Because of the high wind force at Hanford, an air exposure pathway cannot be ruled out. Wind-dust storms and high peak winds occur regularly (Hoitink 2005). Chromium’s vapor pressure is negligible, however, and gaseous chromium is not typically encountered.

BIOACCUMULATION POTENTIAL

High accumulations of chromium have been recorded among organisms from the lower trophic levels, but there is little evidence of biomagnification through food chains; in fact, some studies have actually found decreasing concentrations at higher trophic levels (Outridge and Scheuhammer 1993, as cited in Eisler 2000). Results reported in freshwater and marine food webs were similar (Holdway 1988; USPHS 1993, as cited in Eisler 2000).

In general, both living and dead plant tissues accumulate chromium to a substantial extent (Driver 1994, Eisler 2000). However, most plant and invertebrate species die before accumulating levels of chromium that are toxic to potential predators (Outridge and Scheuhammer 1993, as cited in Eisler 2000).

ACCUMULATION WITHIN TISSUES

In 2003, Dauble found that at high environmental concentrations of Cr(VI) (i.e., 2.0 mg/L in water) and at alkaline pH, concentrations in rainbow trout tissues were greatest in gill, liver, kidney, and digestive tract. Even after transfer of fish to chromium-free media, residues tended to remain high in kidney and liver (Van der Putte *et al.* 1981a, as cited in Eisler 2000).

Waterfowl that consumed diets rich in chromium had elevated chromium concentrations in tissues, especially gonads, gallbladder and pancreas (van Eeden and Schoonbee 1992; Table 2.3, as cited in Eisler 2000).

In mammals, although both Cr(III) and Cr(VI) accumulated in the brain, kidney and myocardium of rabbits, the “accumulation of Cr(VI) was highest in brain and that of Cr(III) in kidney; for both valence states there was no correlation between dose and concentration of stored chromium, or extent of tissue damage” (Hatherill 1981 as cited in Eisler 2000). Tissue residues in mice were highest in the heart and spleen (Schroeder *et al.* 1964, as cited in Eisler 2000). In rats, studies have shown that Cr(VI) tends to accumulate in “the reticuloendothelial system, liver, spleen, and bone marrow at high doses; at much lower doses, major accumulation sites were bone marrow, spleen, testes, and epididymis” (Langard and Norseth 1979, as cited in Eisler 2000).

IV. TYPICAL MAJOR EXPOSURE ROUTES

For aquatic biota, ingestion (biotic and abiotic) and/or absorption from Columbia River water or sediment are likely to be the more significant exposure routes. For example, benthic invertebrates can accumulate chromium from sediments or clays (Eisler 2000). Fall chinook salmon embryo development occurs in association with gravel substrates of the Columbia River (Geist 1994), making this species potentially more vulnerable than others (Woodward 1999).

For terrestrial biota, exposure may occur through inhalation of soil or particulate matter containing chromium (Kimbrough 1999), and also through ingestion (biotic or abiotic) or dermal contact (contaminated soil, sediment, water).

Both terrestrial and aquatic plants can be exposed to chromium by absorption through roots from soil or groundwater, or absorption of material deposited on the plant from air (Eisler 2000). Riparian plants near groundwater seeps may be more highly exposed than those not adjacent to seeps.

Human exposure to chromium at Hanford may occur through visits to riverbank areas (e.g., abiotic exposure such as dermal contact) and potentially through the consumption of exposed plants and animals (biotic exposure) (Ridolfi 2006).

V. ECOTOXICITY

In organisms, “trivalent chromium does not readily cross cell membranes, and it forms stable complexes with serum proteins. As a result, it has a low overall toxicity potential and is relatively inactive *in vivo*...[H]exavalent chromium is[, however,] readily taken up by living cells and is highly active in diverse biological systems” (Driver 1994). In fact, Cr(VI) is “the most biologically active chromium chemical species” (Eisler 2000).

Cr(VI) has a variety of lethal and sublethal effects in both aquatic and terrestrial organisms. Consistent with its non-specific mechanism of toxicity, the range of ecotoxicological effects inducible by chromium is broad. These effects can include, but are not limited to: mutagenic effects, teratogenic effects, reduced survival and

fecundity, photosynthesis disruption, growth inhibition, lesions, and abnormal behavior. Early life stages are generally more sensitive to effects than adults (Mishra 2008).

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

Cr(VI) has no known beneficial properties but Cr (III) is an important micronutrient for some plants and animals, including humans, who need it for sugar metabolism (Babula 2008). It is not clear whether Cr(III) is an essential trace element for all organisms (Eisler 2000).

MECHANISM(S) AND LOCI OF TOXICITY

Hexavalent chromium is a strong oxidant. Cr(VI) is rapidly reduced to Cr(III) after penetration of biological membranes. The reduction of Cr(VI) to Cr(III) may be the most important mechanism whereby chromium causes toxicity (USPHS 1993, as cited in Eisler 2000). This reduction leads to oxidative stress, which can lead to cellular toxicity. Reactive oxygen species (ROS) are generated through the suite of reactions that follows Cr(VI) reduction and it is likely that these ROS interact with various tissues, resulting in damage (Mishra 2008).

The formation of metallothionein (an intra-cellular stress-response protein present in animals and plants) is normally increased by the presence of metals. Metallothionein protects cells from metals and reactive oxygen species by scavenging and sequestering the material, but it cannot bind Cr. The presence of Cr(VI) inhibits the formation of metallothionein, which potentially increases the toxicity of other metals (Kimura 2010; Majumder et al. 2003). Inhibition of metallothionein may also contribute to the toxicity of Cr, but additional study is needed to explore this hypothesis.

FACTORS AFFECTING TOXICITY

Biotic factors affecting chromium toxicity include species, age, and developmental stage. For all species, early life stages are generally more sensitive to the toxic effects of chromium than adults (Mishra 2008). Behavioral factors that enhance toxic effects include habitat use and residence time. For example, organisms that have “limited mobility and a small home range (e.g. sculpin) may be at maximum risk for exposure” (Ridolfi 2006). In contrast, fall chinook salmon inhabit the river bottom substrate at Hanford (i.e. gravel nests or redds) from the eyed-egg stage to the swim-up stage. During this stage, the fish may have elevated exposures to chromium, but later when they begin to rear along the shoreline, they are “unlikely” to be exposed to elevated levels (Geist 1994, Patton 2007, Eisler 2000).

Abiotic factors affecting toxicity include water and soil temperature, pH, alkalinity, salinity, hardness of water (Dauble 2003), oxygen content, and organic matter content.

PLANTS

Aquatic plants are among the most sensitive groups of organisms that have been tested (Eisler 2000). Studies report a wide array of potential effects of Cr(VI) exposure to plants including photosynthesis disruption (Eisler 2000). Hexavalent chromium in particular has been shown to be five to ten times more effective at inhibiting growth

than Cr(III) in both freshwater and terrestrial plants (USEPA 1980; Outridge and Scheuhammer 1993 as cited in Eisler 2000). Chromium can also inhibit seed germination (Towhill *et al.* 1978, as cited in Driver 1994).

AQUATIC INVERTEBRATES AND FISH

Invertebrate species are generally more sensitive to hexavalent chromium than fish (USEPA 1980). *Daphnia magna* is known to be very chromium-sensitive, and Cr(VI) exposure has been associated with reduced survival and fecundity (USEPA 1980, as cited in Eisler 2000). Keller and Zam (1991) tested the acute toxicity of chromium and other metals to the juvenile freshwater mussel, *Andonta imbecilis* and reported that this mussel seems to be more sensitive than the insects *Daphnia* and *Chironomos*, although the bluegill was similarly sensitive.

A 1994 review reported that stickleback (*Gasterosteus aculeatus*) may be more sensitive than other freshwater fish (Driver 1994). Data from multiple studies shows that exposure to chromium at concentrations greater than or equal to 1 mg/L are lethal to this species (Anderson 1944, Murdock 1953, Jones 1939, as cited in Driver 1994).

Aqueous exposure to chromium has been shown to reduce the growth of rainbow trout and chinook salmon fingerlings (USEPA 1980, as cited in Eisler 2000). Growth rate of larvae of the fathead minnows was also reduced by Cr(VI) exposure (Eisler 2000). The survival rate of alevins and juveniles of coho salmon was significantly reduced by exposure to chromium (Oson 1958, as cited by Driver 1994).

In 2000, a “USGS Final Report: The Potential for Chromium to Adversely Affect Chinook Salmon (*Oncorhynchus tshawytscha*) in the Hanford Reach of the Columbia River, Washington, USA” (Frag *et al.* 2000) found that aqueous chromium exposure of chinook salmon parr to between 24 and 120 µg Cr/L led to malfunctions associated with reduced growth and survival (Frag *et al.* 2000) This study also found the kidney to be the target organ as evidenced by histological lesions and elevated levels of the “products of lipid peroxidation.” Results of USGS avoidance-preference experiments suggest that there may also be behavioral effects associated with water column exposure to chromium (Delonay *et al.* 2001).

In rainbow trout (aged between 4 and 9 months), “acute chromium poisoning caused morphological changes in gills, kidney, and stomach tissues at higher pH, but only in the gills at lower pH” (Van der Putte *et al.* 1981, as cited in Eisler 2000). Sublethal water concentrations of Cr(VI) were shown to cause avoidance behavior in one-year-old rainbow trout: “The intensity of avoidance response reached a significant level at test concentrations of 0.003 mg Cr/L and higher, and was directly proportional to the Cr(VI) concentration logarithm” (Svecevicus 2007).

BIRDS

Teratogenic effects were documented in chicken embryos after eggs had been injected with Cr(VI). Deformities included short and twisted limbs and growth stunting (Ridgeway and Karnofsky 1952; Giliani and Marano 1979, as cited in Eisler 2000). Overall, however, chickens appear to be more resistant than mammals to the adverse effects of Cr(VI) exposure (Eisler 2000).

MAMMALS

Acute and chronic adverse effects of chromium on warm-blooded organisms are caused mainly by Cr(VI) compounds (Eisler 2000), and nearly all Cr(VI) compounds are potent mammalian mutagens and carcinogens (Eisler 2000). Also in mammals, chromium-containing compounds, especially Cr(VI) compounds, are associated with spermicidal, embryocidal, teratogenic, and other adverse effects on reproduction (Nieboer and Yassi 1988, as cited in Eisler 2000).

AMPHIBIANS AND REPTILES

Hexavalent chromium is known to be toxic to embryos of many species including frogs, as a 2009 study on the anuran *Xenopus laevis* showed (Bosisio *et al.* 2009). This study found evidence of both “embryo lethality and teratogenicity of *Xenopus* embryos exposed to Cr(VI)” (Bosisio *et al.* 2009).

EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

Hexavalent chromium can interact with other metals in solution to produce additive or synergistic effects, “as was the case with nickel salts in acute toxicity to guppies” (Khangarot and Ray 1990, as cited in Eisler 2000). Additionally, effects on rainbow trout (as measured by liver enzyme activity) were intensified by the presence of nickel and cadmium salts in solution (Arillo *et al.* 1982, as cited in Eisler 2000). In another experiment, chromium uptake in rainbow trout increased when ionic cadmium was present (Calamari *et al.* 1982, as cited in Eisler 2000). However, in a 96-hour study of juvenile freshwater mussels, the presence of inorganic mercury reduced acute chromium toxicity (Keller and Zam 1991).

Reducing agents (e.g. trivalent arsenic, divalent iron, vanadium, sulfur dioxide) can convert Cr(VI) to Cr(III), making the contaminant less toxic (Palmer 1994). Conversely, ozone and manganese can convert Cr(III) to Cr(VI), making the contaminant more toxic (Palmer 1994, Eisler 2000). Nitrate and sulfate can also mobilize Cr(VI) (Hazen 2008). It is important to note that the chromate added to the Columbia River water at the Hanford Site was added in conjunction with other substances. Other chemicals known to have been added to Columbia River water in conjunction with Na₂Cr₂O₇ are nitric acid, sulfuric acid, polyacrylamide (Essig 1971, as cited in Ridolfi 2006), chlorine, lime, ferric or aluminum sulfate, and activated silica (Gerber 2002).

DATA GAPS & CHALLENGES

There are a number of data gaps associated with understanding the ecotoxicology of chromium. For one, it can be difficult to quantify chromium in its different ionic states (Eisler 2000), and since hexavalent chromium is distinctly more toxic than trivalent chromium, this can lead to uncertainty in how to interpret measured values. (To this point, Eisler (2000) states “[I]ittle is known about the relationship between concentrations of total chromium in a given environment and biological effects.”)

In addition, there are a number of species groups for which ecotoxicological data is limited or absent. Data on terrestrial invertebrates, reptiles, and amphibians appear to

be particularly limited. Information on the sensitivity of wild bird and wild mammalian species to chromium also appears to be limited: most research has focused on domesticated and/or laboratory species. Extrapolating sensitivity from one species to another may be problematic as even closely related species can vary widely in sensitivity (Eisler 2000).

It is also important to note that most research has been laboratory-based: little research appears to have been conducted on the effects of chromium toxicity under field conditions (Eisler 2000). This is of particular note as many factors have the potential to affect chromium speciation and bioavailability. Furthermore, information on the effects of chromium in the presence of other contaminants appears to be limited.

- VI. REFERENCES** Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Toxicological profile for Chromium (*Draft for Public Comment*). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.
- Amec Geomatrix, Inc. 2008. Technical Evaluation of the Interaction of Groundwater with the Columbia River at the Department of Energy Hanford Site, 100-0 Area. Prepared for the U.S. Department of Energy Assistant Secretary for Environmental Management. Project Hanford Management Contractor for the U.S. Department of Energy under Contract DE-AC06-96RL13200. October.
- Babula, P.V. Adam, R. Opatrilova, J. Zehnalek, L. Havel, and R. Kizek. 2008. Uncommon heavy metals, metalloids and their plant toxicity: A review. *Environmental Chemistry Letters* 6(4):189-213.
- Cadwell, L.L. 1994. Wildlife Studies on the Hanford Site: 1993 Highlights Report. Prepared for the U.S. Department of Energy under Contract DE-AC06-76RLO1830. PNL-9380, Pacific Northwest National Laboratory. April.
- Dauble, D.D., T.M. Poston, G.W. Patton, and R.E. Peterson. 2003. Evaluation of the Effects of Chromium on Fall Chinook Salmon in the Hanford Reach of the Columbia River: Integration of Recent Toxicity Test Results. May. Available at http://www.pnl.gov/main/publications/external/technical_reports/PNNL-14008.pdf
- Delonay, A.J., W.G. Brumbaugh, E.E. Little, and L. Cleveland. 2001. USGS Report: Laboratory Evaluation of the Behavioral Avoidance-Preference Response of Chinook Salmon (*Oncorhynchus tshawytscha*) to Chromium in the Hanford Reach of the Columbia River, Washington, USA. Prepared for the Hanford Natural Resource Trustee Council under Contract 1448-14421-98-N-002. USGS Biological Resources Division, Columbia Environmental Research Center, Columbia, MI. June.
- Driver, C.J. 1994. Ecotoxicity Literature Review of Selected Hanford Site Contaminants. PNL-9394, Pacific Northwest Laboratory, Richland, Washington. Available at <http://www.osti.gov/energycitations/servlets/purl/10136486-6sLptZ/native/10136486.pdf>

- Duncan, J.P. (ed.). 2007. Hanford Site National Environmental Policy Act (NEPA) Characterization. Prepared for the U.S. Department of Energy under Contract DE-AC05-76RL01830. PNNL-6415 Rev. 18, Pacific Northwest National Laboratory. September.
- Eisler, R. 2000. Handbook of Chemical Risk Assessment: Health Hazards to Humans, Plants and Animals, Volume I: Metals. Lewis Publishers, Boca Raton, London, New York, Washington, DC.
- Eisler, R. January. 1986. Chromium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Contaminant Hazard Reviews Report No. 6. *Biological Report* 85 (6.1). U.S. Fish and Wildlife Service, Laurel, MD.
- Evans, J.R., M.P. Lih, and P.W. Dunwiddie. 2003. The Nature Conservancy. Final Report 2003. Biodiversity Studies of the Hanford Site 2002-2003. Prepared for the U.S. Department of Energy and the U.S. Fish and Wildlife Service, Hanford Reach national Monument, in partial fulfillment of federal grant DE-FG-02RL14344.
- Farag, A.M., D.D. Harper, L. Cleveland, W.G. Brumbaugh, and E.E. Little. 2006a. The potential for chromium to affect the fertilization process of Chinook (*Oncorhynchus tshawytscha*) in the Hanford Reach of the Columbia River, Washington, USA. *Archives of Environmental Contamination and Toxicology* 50:575–579.
- Farag, A.M., T. May, G.D. Marty, M. Easton, D.D. Harper, E.E. Little, *et al.* 2006b. The effect of chronic chromium exposure on the health of Chinook salmon (*Oncorhynchus tshawytscha*). *Aquatic Toxicology* 76:246–257.
- Farag, A.M., A.J. DeLonay, W.G. Brumbaugh, E.E. Little, L. Cleveland, and D.F. Woodward. 2000. Final Report: The Potential for Chromium to Adversely Affect Chinook Salmon in the Hanford Reach of the Columbia River, Washington, USA. United States Geological Survey. Submitted to United States Fish and Wildlife Service. October.
- Geist, D. R. 2000. Hyporheic discharge of river water into fall Chinook salmon (*Oncorhynchus tshawytscha*) spawning areas in the Hanford Reach, Columbia River. *Canadian Journal of Fisheries and Aquatic Sciences* 57:1647–1656.
- Geist, D.R., T.M. Poston, and D.D. Dauble. 1994. Assessment of Potential Impacts of Major Groundwater Contaminants to Fall Chinook Salmon (*Oncorhynchus tshawytscha*) in the Hanford Reach, Columbia River. PNL-9990. Prepared by Pacific Northwest Laboratory, operated by Batelle, Richland, WA for the U.S. Department of Energy Under Contract DE-AC06-76RL0 1830.
- Gephart, R.E. 2003. A Short History of Hanford Waste Generation, Storage, and Release. PNNL-13605. Prepared by Pacific Northwest National Laboratory, operated by Batelle, Richland, WA for the U.S. Department of Energy Under Contract DE-AC06-76RL0 1830. October.

- Gerber, M.S. 2002. *On the Home Front: The Cold War Legacy of the Hanford Nuclear Site*, Second Edition. University of Nebraska Press, Lincoln and London, England.
- Hartman, M.J., L.F. Morasch, and W.D. Webber. 2000. Hanford site groundwater report for fiscal year 1999. PNNL-13116. Richland, Washington: Pacific Northwest National Laboratory.
- Hazen, T. 2008. CLU-IN Resources: Hanford Demonstrates Bioimmobilization of Hexavalent Chromium in Ground Water. Available at: <http://www.cluin.org/products/newsletters/tnandt/view.cfm?issue=0108.cfm#1>
- Hoitink, D.J., K.W. Burk, J.V. Ramsdell Jr., and W.J. Shaw. 2005. Hanford Site Climatological Summary 2004 with Historical Data. Prepared by Pacific Northwest National Laboratory, operated by Battelle, Richland, WA for the U.S. Department of Energy Under Contract DE-AC05-76RL01830.
- Hope, S.J. and R.E. Peterson. 1996a. Chromium in River Substrate Pore Water and Adjacent Groundwater: 100-D/DR Area, Hanford Site, Washington. BHI-00778 Rev. 0, Bechtel Hanford Inc., Richland, Washington.
- Keller, A.E. and S.G. Zam. 1991. The acute toxicity of selected metals to the freshwater mussel, *Adonta imbecilis*. *Environ. Toxicol. Chem* 1:539-546.
- Kimbrough, D.E., Y. Cohen, A.M. Winer, L. Creelman, and C. Mabuni. 1999. A Critical Assessment of Chromium in the Environment. *Critical Reviews in Environmental Science and Technology* 29(1):1-46.
- Kimura, T. 2010. Molecular mechanisms of zinc-mediated induction and chromium (VI)-mediated inhibition of mouse Metallothionein-I gene transcription. *Journal of Health Science* 56(2):161-166.
- Larson, K.B., T.M. Poston, and B.L. Tiller. 2008. Evaluation of Using Caged Clams to Monitor Contaminated Groundwater Exposure in the Near-Shore Environment of the Hanford Site 300 Area. Prepared for the U.S. Department of Energy under Contract DE-AC05-76RL01830. PNNL-17270, Pacific Northwest National Laboratory. January.
- Lipton, J. and J. Holmes. 2009. Hanford Site Natural Resource Damage Assessment, Phase I Summary Report. Prepared for Hanford Natural Resource Trustees by Stratus Consulting, Inc., Boulder, Colorado. July 1.
- Majumder, S., K. Ghoshal, D. Summers, B. Shoumei, J. Datta, and S.T. Jacob. 2003. Chromium (IV) Down-regulates heavy metal-induced metallothionein gene transcription by modifying transactivation potential of the key transcription factor, metal-responsive transcription factor 1. *The Journal of Biological Chemistry* 278(28):26216-26226.
- Mishra, A.K. and B. Mohanty. 2008. Histopathological effects of hexavalent chromium in the ovary of a fresh water fish, *Channa punctatus* (Bloch). *Bulletin of Environmental Contamination & Toxicology* 80(6):507-511.

- Newell, R.L. 2003. Biodiversity of Aquatic Macroinvertebrates of the Hanford Reach National Monument, Washington, U.S.A. Report to The Nature Conservancy, Seattle, WA.
- Palmer C.D. and R.W. Puls. 1994. EPA Ground Water Issue: Natural Attenuation of Hexavalent Chromium in Groundwater and Soils. EPA/540/5-94/505 Technology Innovation Office: Office of Solid Waste Emergency Response, US EPA, Washington, DC. October.
- Patton, G., D. Dauble, and C. McKinstry. 2007. Evaluation of early life stage fall chinook salmon exposed to hexavalent chromium from a contaminated groundwater source. *Environmental Monitoring & Assessment* 133(1-3):285-294.
- Patton, G., J. Yokel, D., Delistraty, M. Priddy, and T. Stoops. 2005. Survey of Potential Hanford Site Contaminants in the Upper Sediment for the Reservoirs at McNary, John Day, The Dalles, and Bonneville Dams, 2003.
- Patton, G.W., D.D. Dauble, M.A. Chamness, C.S. Abernethy, and C.A. McKinstry. 2001. Chromium Toxicity Test for Fall Chinook Salmon (*Oncorhynchus tshawytscha*) Using Hanford Site Groundwater: Onsite Early Life Stage Toxicity Evaluation. Prepared for the U.S. Department of Energy and the Hanford Natural Resource Trustee Council under Contract DE-AC06-76RLO1830. PNNL-13471, Pacific Northwest National Laboratory.
- Poston T. M., R.W. Hanf, R.L. Dirkes, and L.F. Morasch. 2001. Hanford Site Environmental Report for Calendar Year 2000. Pacific Northwest National Laboratory, Richland, WA.
- Ridolfi, Inc. December 8, 2006. Preassessment Screen for the Hanford Facility. Prepared for the Confederated Tribes and Bands of the Yakama Nation. Seattle, Washington.
- Serne, R.J. 2007. K_d Values for Agricultural and Surface Soils for Use in Hanford Site Farm, Residential, and River Shoreline Scenarios. Technical Report for Groundwater Protection Project – Characterization of Systems Task. Prepared for Fluor Hanford, Inc. and the U.S. Department of Energy under Contract DE-AC05-76RL01830. PNNL-16531, Pacific Northwest National Laboratory. August.
- Shanker, A.K., C. Cervantes, H. Loza-Tavera, and S. Avudainayagam. 2005. Chromium toxicity in plants. Review Article. *Environmental International* 31:739-753.
- Svecvicius, G. 2007. Avoidance response of rainbow trout *Oncorhynchus mykiss* to hexavalent chromium. *Bulletin of Environmental Contamination and Toxicology* 79:596-600.
- Thornton, E.C., J.E. Amonette, J. Olivier, and D.L. Huang. 1995. Evaluation of Chromium Speciation and Transport Characteristics in the Hanford Site 100D

and 100H Areas. Presented at 1st Symposium, Hydrogeology of Washington State, August 28-30. Prepared for U.S. Department of Energy.

- U.S. Department of Energy (DOE). 2001. Hanford Site Biological Resources Management Plan. DOE/RL 96-32, Revision 0. U.S. DOE, Richland, Washington. August.
- U.S. Environmental Protection Agency (EPA). 1998. Toxicological Review of Hexavalent Chromium (CAS No. 18540-29-9) in Support of Summary Information on the Integrated Risk Information System (IRIS). US EPA: Washington, DC. August.
- U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards. Criterion and Standards Division, Washington, DC. EPA 440/5-80-035. October.
- Venkatramreddy, V., S.S. Vutukuru, and P.B. Tchounwou. 2009. Ecotoxicology of hexavalent chromium in freshwater fish: A critical review. *Rev Environ Health* 24(2):129–145.
- Woodward, D.F., A.M. Farag, A.J. DeLonay, L. Cleveland, W.G. Brumbaugh, and E.E. Little. The Potential for Contaminated Ground Water to Adversely Affect Chinook Salmon (*Oncorhynchus tshawytscha*) under Exposure Conditions Simulating the Hanford Reach of the Columbia River, Washington, USA
- Zachara, J.M., C.C. Ainsworth, G.E. Brown Jr., J.G. Catalano, J.P. McKinley, O. Qafoku, S.C. Smith, J.E. Szecsody, S.J. Traina, and J.A. Warner. 2004. Chromium speciation and mobility in a high level nuclear waste vadose zone plume. *Geochimica et Cosmochimica Acta*. 68(1):13-30.

IODINE (I-129) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Iodine-129 (I-129) is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Iodine is a common element that is widely distributed throughout marine and terrestrial environments (EPA 2002). There are thirty-five known isotopes of iodine, with atomic mass ranging from 108 to 142 (ATSDR 2004). Of these forms, only I-129 and I-127 occur naturally. I-127 is the only stable iodine isotope, and I-129 has the longest half-life of the iodine isotopes, 15.7 million years. The natural ratio of stable I-127 to radioactive I-129 in the environment is more than 10^{14} to 1 (ANL 2001). Considerable attention has been paid to I-129 because its long half-life creates the potential for significant accumulation in the environment from prolonged low-level releases (NCRP 1983).

II. SOURCES **NATURAL SOURCES**
The global inventory of I-129 in 1945, prior to the first nuclear weapons testing, was essentially a constant 40 Ci (NCRP 1983). I-129 in nature is produced predominately by cosmic ray induced spallation of xenon in the upper atmosphere (Schwehr 2004). It is also produced to a much lesser degree by spontaneous fission of U-238 in the lithosphere and the neutron-initiated reactions Te-128(n, γ) and Te-130(n, γ) (Preedy *et al.* 2009; NCRP 1983). Exhibit 1 presents the global inventory of natural I-129.

EXHIBIT 1 MAJOR NATURAL LOCATIONS OF I-129

| SOURCE | KG OF I-129 | TBQ OF I-129 |
|-----------------------------------|-------------|--------------|
| Natural hydrosphere | 100 | 0.65 |
| Natural atmosphere | 0.0005 | 0.000003 |
| Source: Preedy <i>et al.</i> 2009 | | |

ANTHROPOGENIC SOURCES

Since 1945, that atom ratio of I-129 to I-127 has been increasing due to the I-129 added to the environment from anthropogenic sources (NCRP 1983). Most man-made I-129 in the environment comes from two sources: 1) fallout from the detonation of nuclear weapons (especially atmospheric weapons tests) and 2) the nuclear fuel cycle (NCRP 1983). Potential I-129 releases from the nuclear fuel cycle include 1) nuclear

power plant accidents, 2) nuclear fuel reprocessing, and 3) facilities that treat or store radioactive waste (EPA 2002).

I-129 is produced in nuclear explosions of U-235 or Pu-239 at the rate of 30 and 50 μCi per kiloton (KT) TNT equivalent, respectively (NCRP 1983). This equates roughly to a release of 10 Ci from nuclear weapon detonation (NCRP 1983).

Nearly all of the iodine-129 and iodine-131 generated in the United States is present in spent nuclear reactor fuel rods. These fuel rods are currently located at commercial reactor facilities or at DOE facilities across the United States (ATSDR 2004). Iodine-129 is produced in nuclear fission as a decay product of technetium-129 (NCRP 1983). The cumulative yield of iodine-129 is about 1% of all fission products (ANL 2001). Thus, iodine-129 represents only a small fraction of the total fission product inventory in the nuclear fuel cycle. I-129 inventories produced from nuclear power are estimated to be approximately 2,350 Ci (Preedy *et al.* 2009). Exhibit 2 presents the global I-129 inventory from major anthropogenic sources.

EXHIBIT 2 MAJOR ANTHROPOGENIC SOURCES OF I-129 (PREEDY ET AL. 2009)

| SOURCE | KG OF I-129 | TBQ OF I-129 |
|-------------------------------------|-------------|--------------|
| Atmospheric Testing | 50 | 0.32 |
| Chernobyl (1986) | 1-2 | 0.01 |
| Savannah River Site (1953-1990) | 32 | 0.21 |
| Hanford Reservation (1944-1972) | 266 | 1.7 |
| NTS underground nuclear testing | 10 | 0.065 |
| Proposed Yucca Mountain Repository | 13,300 | 87 |
| Spent fuel reprocessing (Europe) | 2,360 | 15 |
| Source: Preedy <i>et al.</i> (2009) | | |

The IAEA estimates that, if the backlog of SNF were to be reprocessed, approximately 7.4 Ci of gaseous I-129 would be released into the atmosphere (NCRP 1983). The remainder would be collected and stored for disposal as high level waste (Preedy *et al.* 2009).

I-129 in the environment surrounding the Hanford site originated from nuclear fuel cycle processes at that site. The Hanford operations included plutonium production and research reactors, chemical separation facilities, and fuel fabrication facilities, all of which involved processing and storing various uranium compounds, resulting in the production of iodine and therefore its subsequent release of some I-129 into the environment.

From 1944 through 1972, the plutonium production operation at the Hanford Site in Washington released about 260 kg of I-129 into the air from its 200-E and W areas (Hu *et al.* 2003; Garland *et al.* 1983). In comparison, the operation of production

reactors from 1953 to about 1990 at the Savannah River Site (SRS) in South Carolina released about 32 kg of I-129 into the air (Hu *et al.* 2003). As iodine-129 is a product of nuclear fission, its contamination is present at numerous areas throughout the Hanford Site especially in groundwater; 2009 levels at and near the site are detailed in the Hanford Site Environmental Report (Poston *et al.* 2010). The largest groundwater plume extends from the 200 Areas to the Columbia River. Very low concentration gradients of iodine-129, less than 100 attocuries/L, have been detected in the river (ANL 2001). According to Poston *et al.* (2010) contaminant plumes totaling approximately 11.3% of the Hanford Site area exceed the drinking water standard for I-129 at Hanford.

A notable release of iodine from Hanford, called the “Green Run,” occurred on December 3, 1949. Hanford workers released a plume of approximately 8,000 Ci of I-131. This was done intentionally and without notifying the public to assess the usefulness of atmospheric sampling for radioisotope indicative of fuel reprocessing (Angelo 2004). I-131 from this release is no longer an environmental concern because of the short half-life of I-131.

The long half-life of I-129 makes anthropogenic additions of I-129 essentially permanent additions to the global inventory of iodine (NCRP 1983). The ratio of I-129 to I-127, in a small number of samples of animal thyroids from locations *remote* from nuclear facilities, was found to range from 10^{-8} to 10^{-7} (Brauer and Ballou 1974; Smith 1977). Ratio values ranging as high as 10^{-4} to 10^{-3} for thyroids and vegetation have been measured *near* some nuclear facilities (Brauer and Ballou 1974). A wide range of ratio values are observed, depending on the geographic location, time of year, and types of materials sampled (NCRP 1983).

III. ENVIRONMENTAL CHEMISTRY

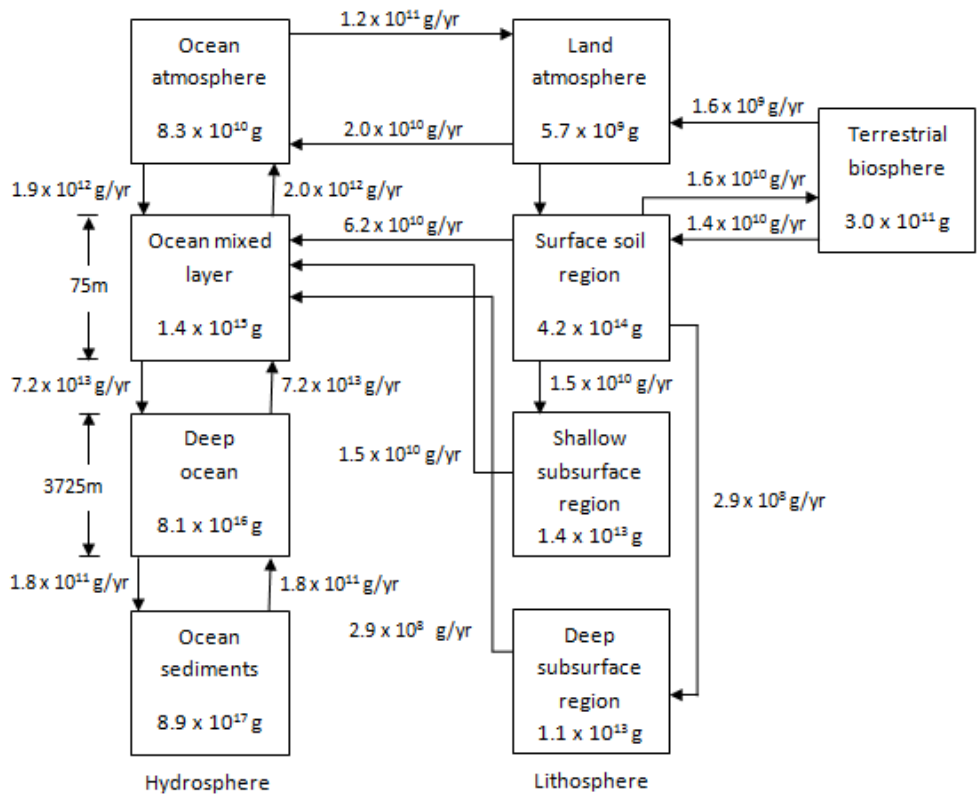
Many of the physical and biological properties of iodine-129 are based on those of other isotopes due to a lack of experimental data (NCRP 1983). Iodine in elemental form exists only as I_2 due to the reactive nature of its valence electrons. It has oxidation states ranging from -1 to +7. In aqueous environments -1 (iodide, I^-) and +5 (iodate, IO_3^-) are its dominant states (ATSDR 2004). In reducing environments, aqueous iodine usually occurs as the very mobile iodide anion. Iodide in this state is readily metabolized by the body. Under oxidizing conditions, aqueous iodine is present in the more reactive, iodate anion form (Hu 2003). As iodate, the mobility of iodine is slowed through interactions with clay and organic compounds in the soil.

Iodine coexists in various proportions of inorganic and organic iodine in different environments. A significant fraction of iodine in aqueous environments and in the atmosphere exists as organically bound iodine. The transfer of iodine among the various portions of the environment depends on its chemical and physical form (Holland 1963, Perkins 1963). Inorganic vapor is the most chemically reactive form of iodine, but iodine associated with particles and organic compounds, such as methyl iodide (CH_3I), is readily metabolized (Morgan *et al.* 1967).

FATE AND TRANSPORT

The environmental transport, distribution, and transformation of iodine is driven by a complex series of physical, chemical, and biological processes that are collectively known as the global iodine cycle (WHO 2006). The cycle, represented below from Kocher (1981), involves the transfer of iodine between the ocean, land, and terrestrial biosphere (see Exhibit 3).

EXHIBIT 3 THE GLOBAL IODINE CYCLE (RECREATED FROM KOCHER 1981 AS CITED BY WHO 2006)



As seen above, the main driver for the iodine cycle is the exchange of iodine between ocean water and its atmosphere. Some aquatic biological aspects of this cycle involve the reduction of iodate to iodide and then its conversion to organic iodine compounds by algae at the ocean surface water (Vogt *et al.* 1999). The volatility of these compounds combined with direct evaporative losses results in the transfer of a range of iodine compounds to the ocean atmosphere (WHO 2006).

Numerous studies of the sorption of iodine on sediments, soils, pure minerals, oxide phases, and rock materials have been conducted. An extensive review of these studies is presented in Selinus (2005) and WHO (2006).

Soils/Sediments/Rocks

In the lithosphere, natural iodine is an ultra-trace element. Its crustal abundance is estimated to be 0.3 mg/kg. Iodine content in most rock forming minerals is fairly uniform. Sedimentary rocks show a greater range of iodine content with clay rich rocks more enriched than sand-rich rocks. The highest concentrations of iodine have been found in organic-rich shales, with concentrations as high as 44 mg/kg. Sediments of marine origin can also be extremely enriched with iodine concentrations as high as 20,000 mg/kg recorded from some samples. Soils near oceans also have elevated iodine concentrations, though the ocean influence does not extend very far inland (Selinus 2005).

Most I-129 present in soil around the world comes from fallout from atmospheric nuclear weapons tests. I-131 produced in these tests has decayed away. Iodine may also be found as a contaminant where spent nuclear fuel was processed (ANL 2001).

Iodine concentrations in sandy soil are about the same as in interstitial water (in the pore spaces between soil particles). It binds preferentially to loam, where the concentration in soil is estimated to be 5 times higher than in interstitial water. I-129 is one of the more mobile radionuclides in soil due to its water solubility in several chemical forms (NEI 2006). Iodine travels with infiltrating water to groundwater (ANL 2001).

The geochemistry of soil iodine reflects not only the input of iodine but also the soil's ability to retain iodine. Iodine retention in soil is dependent on many factors. It is likely that organic matter is the most influential component in soil iodine retention. Organic-rich sediments can be and frequently are strongly enriched in iodine levels, correlated to their content of organic matter (Selinus 2005).

There is also evidence showing iron and aluminum oxides play an important role in soil retention of iodine. The sorption of iodine by aluminum and iron oxides is strongly dependent on the soil pH. Sorption is greatest in acidic conditions (Whitehead 1984).

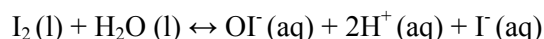
Overall, "Retention of iodine in the soil is influenced by a number of factors, including soil pH, soil moistness, porosity of soil, and composition of organic and inorganic (e.g., aluminum and iron oxides) components (Sheppard and Evenden 1995; Whitehead 1984). Approximately 1% of iodine received through atmosphere-to-soil deposition is returned through volatilization of molecular iodine and methyl iodide; the remaining iodine is eventually returned to the oceans through surface water and groundwater (USNRC 1979; Whitehead 1984). The average residency time of iodine in the soil at 0.3- and 1-meter depths has been suggested to be 80 and 800 years, with only 1–3% of deposited iodine migrating to the 1-meter depth (DOE 1986)" (ATSDR 2004).¹⁰⁶

¹⁰⁶ At several places in this profile, direct quotes make reference to additional specific source documents. These references are included in the reference section of this profile so that readers can more easily identify and obtain the original source documents cited in the major publications.

Water

Most forms of iodine are readily soluble in water. Iodine enters surface waters and ground waters primarily through rainwater for non-coastal land regions and the combination of rain and ocean spray in coastal areas (WHO 2006). Most I-129 is discharged in gaseous effluents, with some also present in liquid effluents. Iodine-129 deposited on land from effluents and natural I-129 production may eventually percolate through soil to groundwater and, together with I-129 in liquid effluent discharged directly to water, ultimately reach ocean waters. Most of the earth's stable iodine is located in the oceans (ATSDR 2004); therefore the marine environment will eventually constitute the primary reservoir of most anthropogenic I-129 (NCRP 1983). The average concentration in seawater is about 60 ppb, but it varies from place to place. This concentration is thought to be relatively uniform with depth. Rivers usually contain about 5 ppb of iodine, and in mineral sources some ppm concentrations can even be found.

Iodine is strongly reactive, although less than other halogens. Iodine cannot be found as an element, but rather as I_2 molecules, as I^- ions, or as iodate. "When iodine is added to water, the following reaction results:



I_2 molecules and water molecules react to substances such as hypiodite (OI^-). The reaction can move both ways of the equilibrium, depending on the pH of the solution" (Lenntech 2009).

The iodine in groundwater can be present in the forms I^- , I_2 , IO^- , or IO_3^- . If the ground water has a high reduction potential or certain bacteria are present, the iodine also may be present as CH_3I (USNRC 1979).

Air

Iodine enters the atmosphere mainly through volatilization of methyl iodide and, to a lesser extent, molecular iodine from the ocean surface (ATSDR 2004). I-129 is introduced naturally through the iodine cycle as well as through gaseous effluents. In air, some iodine compounds photochemically decompose to iodine and its radicals. These products then go on to react with atmospheric gasses to produce a range of additional reactive iodine species. The iodine species can also react with aerosols or water droplets to form iodine anions. Concentrations of I-129 in the atmosphere range from 2-14 ng/m³ in air and 17-52 ng/m³ over land (WHO 2006). The gaseous inorganic and particulate forms of iodine are precipitated from the atmosphere through wet (meteorological events) and dry (gravitational settling) deposition processes (Whitehead 1984). The deposition of iodine will depend on particle size and concentration, wind turbulence, and the chemical form of iodine. If precipitation occurs over land, iodine will be deposited onto plant surfaces or soil surfaces, or into surface waters.

Because the atmospheric residence time of iodine ranges from 10 to 18 days, it easily travels around the globe (Schwehr 2004, Whitehead 1984). In fact, anthropogenic I-129 from reprocessing emissions has been found in river water (Schink *et al.* 1995)

and in rainwater of the northern hemisphere (Moran *et al.* 1999). These measurably high anthropogenic I-129 emissions are found not only in their source area in western Europe, but also in the United States, where known atmospheric releases currently are negligible, as well as in the southern hemisphere (Fehn and Snyder 2000).

BIOACCUMULATION POTENTIAL

Terrestrial Systems

The global iodine cycle is essential to terrestrial life, especially considering the majority of iodine in the earth's surface is inaccessible and only liberated in small amounts from weathering and dissolution. The transfer of iodine to land and the terrestrial environment decreases by distance from the ocean (WHO 2006).

When iodine is strongly sorbed in most soils, it will not be readily bio-available. Therefore, high concentrations of iodine in the soil does not necessarily mean that plants growing in that soil will incorporate large amounts of iodine. In fact, a 1985 study by Al-Ajely found no correlation between the iodine concentrations in soils and the plants growing in them.

Iodine content in plants is generally low. Grass and herbage from around the world have an iodine content of roughly 0.2 mg/kg (Selinus 2005). A 1994 study of Japanese plants by Yuita found the mean iodine content of different plant parts to be green leaves 0.46 mg/kg, fruit 0.14, edible roots 0.055, and seeds 0.0039.

In most cases the major pathway for elements into a plant is through the root system, followed by translocation into the upper parts of the plant. The iodide ion has been shown experimentally to be taken up through the root system and more readily incorporated into the plant than iodate. However, there is little translocation from the roots to the upper plant (Selinus 2005). It has been demonstrated that rice grown in areas flooded with water high in iodine only have slightly elevated levels of iodine compared to rice grown in drained soil (Preedy *et al.* 2009) Notably though, when leaves are submerged in the plant, a dramatic increase in iodine content is seen. From these considerations, it is likely that root uptake of iodine is only a small part of iodine content in plants (Selinus 2005).

It is likely that the most important pathway of iodine into plants is through direct absorption from the atmosphere. Several studies have shown that plant leaves can absorb iodine. Increased humidity increases the absorption of gaseous iodine through the leaf stomata. Iodine taken in through the leaf slowly incorporates into the rest of the plant (Selinus 2005).

Aquatic Systems

Iodine has been shown to bioaccumulate in many seawater and freshwater aquatic plants (Poston 1986). Freshwater plants such as algae contain 10^{-5} % by weight of iodine, whereas marine plants (algae) contain 10^{-3} % by weight (NCRP 1983). In freshwater fish, iodine concentrations in tissues range from 0.003 to 0.81 ppm, which gives concentration ratios (fish/water) of 0.9–810. In marine fish, the iodine

concentrations range between 0.023 and 0.11 ppm, yielding concentration ratios of between 10 and 20 (Poston 1986).

ACCUMULATION WITHIN TISSUES

Iodine is unique in that, unlike other organs in the body, thyroidal cells are able to absorb iodine. The average human body contains 10-20 mg of iodine, more than 90% of which concentrates in the thyroid (NCRP 1983). All vertebrates have thyroids that absorb iodine; however, the quantity of iodine absorbed is dependent on a number of factors. Invertebrates do not have thyroid tissue but have an organ known as an endostyle that, among other functions, absorbs iodine. The endostyle is thought to be an evolutionary precursor to the thyroid (Evans and Claiborne 2006).

Under normal conditions, the thyroid maintains a nearly static amount of iodine. Large changes in iodine intake affect this concentration. In addition to the thyroid, iodine also concentrates in much lesser quantities in the kidney, mammary glands, salivary glands, gastric mucosa, placenta, ovary, skin and hair of mammals (ATSDR 2004). Iodine is readily taken into the bloodstream from both the lungs and through the gastrointestinal tract (nearly 100%) after inhalation and ingestion. In a simplified model that does not reflect intermediate redistribution of iodine, once in the bloodstream, 20% of iodine is quickly excreted in feces, 30% is deposited in the thyroid, and the remainder is eliminated from the body within a short time. In humans, clearance time from the thyroid varies with age, with biological half-lives ranging from 11 days in infants to 23 days in a five-year-old child, and 80 days in adults (ANL 2001). The whole-body effective biological half-life of iodine-129 is 140 days in humans (Multi-Agency 2004). Biological half-lives in non-human species are not well documented.

Iodine also concentrates in animal products, specifically milk and eggs. Iodine found in these products is directly influenced by iodine intake from feed and water. Lactating animals excrete 10% or more iodine taken in through milk, depending on the rate of excretion (Committee 2005).

IV. TYPICAL MAJOR EXPOSURE ROUTES

Although literature on non-humans is sparse, a great deal of literature is dedicated to the exposure routes through which humans are exposed to iodine. Iodine enters the body through inhalation, ingestion, and absorption. Dietary intake is the main source of iodine to the general population. Marine seafoods typically contain the highest amounts of iodine (160-3200 µg/kg). Kelp, seaweeds and sea salt also are known to have a high iodine content (NCRP 1983). In most industrialized nations, the most important sources of iodides are dairy products, eggs, grain, and cereal products. Other sources include meat and poultry, fruits, and legumes (WHO 2006). Additionally, iodine is added to salts in many countries to reduce iodine deficiency disorders. NCRP (1983) estimates that meat, milk, and milk products contribute the most to dietary iodine intake of Americans over the course of a lifetime. Cultures that consume more seafood and seaweed gain a larger percentage of their iodine intake from these sources.

Inhalation and absorption of iodine occurs to a much lesser extent. It is estimated that 5 µg/day and inhaled in coastal areas, assuming time is spent both indoors and outdoors. Iodine vapor have also been shown to penetrate the skin. Experiments show iodine absorbed dermally is only 1-2% of that absorbed through the lungs for inhalation. Consequently, dermal absorption is not considered a main contributor to iodine exposure from the air.

V. CHEMICAL ECOTOXICITY

Chemical toxicity of iodine is of larger concern than radiotoxicity from I-129 (e.g., Sheppard and Evenden 1995, Laverlock *et al.* 1995). Iodine is an essential component of all animal life. Iodine's chemical effects are most completely documented for humans but due to the function the thyroid serves in all species, much of the information is applicable to other vertebrates. Overall, "Several reviews are available on iodine toxicity in mammals *in NRC* (1980), *SCF* (2002), *McDowell* (2003) and *ATSDR* (2004); however, reports on iodine toxicity to fish from diet and aquatic environments are sparse... Significant species differences exist in the tolerance levels iodine because of the differences in basal metabolic rate and iodine metabolism. All species appear to have a wide margin of safety for this iodine" (Committee 2005).

Iodine deficiency is the major cause of mental retardation, endemic goiter, and cretinism worldwide. These effects are also seen in animals but minimal research has been done investigating these effects. The thyroid responds to a shortage of dietary iodine by enlarging and more actively transporting iodine from the blood, thereby concentrating sufficient iodine to maintain normal function. In contrast, when iodine ingestion is excessive, the thyroid decreases the transport of iodine. This mechanism, known as the Wolff-Chaikoff effect, leads to a transient decrease in thyroid hormone synthesis for about 48 hours. Normal thyroid hormone synthesis resumes shortly after despite continued ingestion of excess iodine (BRER 2004).

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

Iodine is an essential element to virtually all living organisms (other than plants), and there is a vast body of literature documenting its beneficial effects on humans. In vertebrates, iodine is used by the thyroid to produce multiple essential hormones.

The major use of iodine, iodine radionuclides, and iodine compounds is in medical diagnosis and treatment. Iodine-123, I-125, and I-131 are used for diagnostic imaging of the thyroid gland and the kidneys. Iodine-131 is used to treat hyperthyroidism and thyroid cancer. Stable iodine in the form of potassium iodide is added to commercial salt to prevent iodine deficiency disorders. Iodine in the form of the hormone thyroxine is also used for thyroid and cardiac treatment and hormone replacement therapy in iodine deficiency. Iodine radionuclides are used as a tracer in the laboratory and industry to study chemistry mechanisms and processes and to study biological activities and processes. Iodine is a bactericide and is used as an antiseptic and sterilization of drinking water (USNRC 1979). Iodine can also be used to treat syndrome X, infertility, growth retardation, and polycystic ovarian syndrome (Preedy *et al.* 2009).

In instances when large quantities of radioiodine are thought to be released, such as in a nuclear accident, potassium iodine is distributed to populations to decrease their uptake of radioiodine. Effectively, this treatment lowers the radioiodine concentration by providing a huge influx of stable iodine. This lowers the probability that the radioiodine will be taken up by the thyroid.

I-129, specifically, is not used in medical treatments (EPA 2002). Its long half-life and low energy beta prevents it from being useful in medical applications. I-129 in some instances has been used as a radiotracer.

MECHANISM(S) AND LOCI OF CHEMICAL TOXICITY

Aqueous iodine ($I_{2(aq)}$) acts as a biotic killing agent under acidic conditions. Since I_2 is nonpolar, it can pass through the membrane lipid bilayer and form N-iododerivatives from amino acids, oxidize the thiol group in cysteine, react with phenolic alcohol groups, and interfere with unsaturated fatty acid carbon double bonds (Gottardi 1991). Aqueous I_2 is most stable at lower acidic pHs. Under acidic conditions, biocidal $I_{2(aq)}$ levels can be maintained for longer periods (Gottardi 1991) and, as solution pH increases, iodine equilibria shift toward the non-bactericidal I^- species.

As noted previously, iodine is unique in that, unlike other organs in the body, thyroidal cells are able to absorb iodine, and many of iodine's effects in animals occur through its effects on the thyroid and on thyroidal processes. For example, excess iodine may result in hyperthyroidism or hypothyroidism, although the mechanisms involved in these responses are not entirely understood. Many studies conducted on the mechanisms of iodine toxicity show the following direct effects on the thyroid gland: 1) inhibition of iodide transport and uptake by the thyroid, 2) accumulation of iodotyrosines, 3) inflammation and degradation of follicular cells, and 4) damage to follicular cell DNA (Committee 2005).

FACTORS AFFECTING TOXICITY

The toxicity of iodine depends largely on its chemical form. The observation of toxicity of iodine is predominantly focused on the iodide and iodate species of iodine. These forms are found in iodized salt, milk and water. However, the toxicity of other species can be much higher. Iodine is often used as a means of water disinfectant. Iodine doses as low as 1 mg/l in water kill bacteria within minutes. Elemental iodine that remains in the water can be toxic to humans (Preedy *et al.* 2009).

Iodine toxicity is also species dependent. Laverlock *et al.* (1995) found *Daphnia magna* to be more sensitive than rainbow trout fry. In particular, *Daphnia* were equally sensitive to I_2 ($LD_{50} \geq 0.16$ mg/l) and I^- ($LD_{50} \geq 0.17$ mg/l) but were less sensitive to IO_3^- ($LD_{50} \geq 10.3$ mg/l). In contrast, rainbow trout fry were relatively sensitive to I_2 ($LD_{50} \geq 0.53$ mg/l) and less sensitive to I^- ($LD_{50} \geq 860$ mg/l) and IO_3^- ($LD_{50} \geq 220$ mg/l). The effect of water hardness and total organic carbon on lethality was not uniform but depended on the chemical form of iodine used and on the test species. A follow-up study on the single cell organism, *Tetrahymena pyriformis*, found iodine toxicity to increase as follows: $CaI_2 < KI < KIO_3 < I_2 < KIO_4$ (Preedy *et al.* 2009).

PLANTS

High concentrations of iodine have been shown to be toxic to most plants (Sheppard and Evenden 1995). Unlike mammals, plants do not require iodine, and they can be adversely affected by low (micromolar) concentrations. The degree of phytotoxicity is dependent on the species of iodine that exists in the soil solution. Typically, Γ is more phytotoxic than IO^{-3} (Mackowiak and Grossl 1999, Umaly and Poel 1971), which is potentially due to the greater ability of plant roots to absorb the reduced form (Böszörményi and Cseh 1960). Once in the plant, Γ may oxidize to I_2 , which iodinate photosystem II components (Takahashi and Satoh 1989).

High concentrations of iodine in soil have been shown to inhibit growth in pak choi, spinach, and rice. This indicates that uptake of iodine in certain plants has the potential for toxic effects (Preedy *et al.* 2009). In a study by Mackowiak *et al.*, rice receiving the highest dose of I_2 (20 μM) under neutral conditions experienced the least growth and the greatest iodine biomass concentrations (2004). Few studies appear to be available on wild plant species.

AQUATIC INVERTEBRATES AND FISH

Most of the planet's stable iodine is located in the oceans because of the solubility of most forms of iodine; therefore, the marine environment will eventually be the primary resting place for much of anthropogenic I-129 (NCRP 1983). Elevated aquatic concentrations of iodine have led to elevated iodine levels in marine animals. Certain tropical sponges can contain up to 14% iodine by weight (Mellor 1946).

Oral toxicity of iodine to fish has not been studied. High levels of iodine in water can be toxic to aquatic animals (Committee 2005); although "[i]nformation is sparse regarding the acute or chronic toxicity of iodine to freshwater biota" (Laverlock *et al.* 1995). Channel catfish populations experienced 100% mortality from concentrations as low as 0.73 mg I/L over 24-hour exposure periods. This same result was also achieved with 7.2 mg I/L over one hour (LeValley 1982). An exposure of 8.0 mg/L was lethal to some species of mullet. Mortality in both cases was caused by gill damage and asphyxiation (LeValley 1982). As noted previously, Laverlock *et al.* (1995) determined median lethal concentrations of elemental iodine, iodide, and iodate to rainbow trout fry and to *Daphnia magna* under several different water quality regimes.

MAMMALS

Iodine toxicosis in humans and in laboratory surrogates has been widely studied, driven largely by human health concerns. Studies in wild species appear to be few.

Physiological responses are dependent on the dose and the duration of the iodine intake. There are also significant differences between species and their tolerance to high dietary concentrations of iodine (ASDR 2004).

The chronic administration of large doses of iodine to many domesticated and experimental animals has been shown to reduce iodine uptake by the thyroid, which in many cases leads to antithyroidal or goitrogenic effects. High levels of iodide inhibit

organic iodine formation and saturate the active transport mechanism of this ion causing iodide goiter (Committee 2005).

Excess iodine may result in hyperthyroidism or hypothyroidism, but the mechanisms involved in these responses are not entirely understood. Many studies conducted on the mechanisms of iodine toxicity show the following direct effects on the thyroid gland: 1) inhibition of iodide transport and uptake by the thyroid, 2) accumulation of iodotyrosines, 3) inflammation and degradation of follicular cells, and 4) damage to follicular cell DNA (Committee 2005).

Other indirect effects of iodine toxicosis include 1) poor absorption of thyroid hormones resulting in greater excretion in feces, 2) changes in thyroid hormone transport, 3) elevated hepatic microsomal enzyme activities which cause increases in iodotyrosine excretion, and 4) interference in transthyretin metabolism (Committee 2005; ATSDR 2004).

Large acute doses of iodine produce different effects. The LD₅₀ of mice fed iodate ranged from 483-698 mg/kg (Webster *et al.* 1966). Webster *et al.* (1966) also found three doses of 100 mg I/kg body weight to cause anorexia and occasional vomiting in dogs, while larger doses (200-250 mg I/kg body weight) caused death preceded by anorexia or coma. Severe retinal changes were seen in laboratory animals administered sodium iodate intravenously above 10 mg/kg (Burgi *et al.* 2001).

As noted previously, inhalation is generally considered to be a minor exposure route. However, if I₂ or methyl iodide vapors are inhaled they can be absorbed and would be expected to exert effects that are similar to that of iodide absorbed after ingestion, including effects on the thyroid gland. Furthermore, iodine (I₂) is a strong oxidizing agent; therefore, exposure to high air concentrations of I₂ vapor could potentially produce upper respiratory tract irritation and possibly oxidative injury (ATSDR 2004).

BIRDS

Information on iodine's effects on birds is very limited. Domesticated birds' diets are typically supplemented with iodine and in some cases higher concentrations are used to enhance the iodine content in eggs. A study by the University of Natal found excess iodine in poultry diets prevented sexual maturation in fowl, decreased rate of lay, decreased egg weight, and increased body weight (Lewis 2004). Similar studies in chickens and turkeys by Perdomo *et al.* (1966), Arrington *et al.* (1967), Marcilese *et al.* (1968) and Christensen *et al.* (1991) found reduced fertility, decreased egg production, egg size and hatchability.

AMPHIBIANS AND REPTILES

Information on the effect of iodine on amphibians and reptiles is limited. Both reptiles and amphibians have a dietary need for iodine and are sensitive to elevated and depressed concentrations in their diet. Iodine dietary supplements are often necessary in domesticated reptiles and amphibians (Swingle 1923). Higher quantities of iodine are known to be toxic but the toxic concentration is not documented.

Iodine is known to be an essential component in the amino acid and protein molecule responsible for the metamorphosis of amphibians. Without iodine amphibians are unable to go through metamorphosis. Non-thyroidal iodine is also effective in inducing metamorphosis in urodele and anuran larvae that lack thyroids (Swingle 1923).

VI. RADIOLOGICAL MECHANISM OF ACTION ECOTOXICITY

Iodine-129 decays by emitting a low energy beta and gamma particle to produce xenon-129. I-129's very long half-life (1.57×10^7 yr) and low specific activity limit its radioactive hazards, as does the low energy of its beta and gamma emissions. In fact, accurate detection and measurement of the isotope is difficult and tedious (NCRP 1983) because of its weak emissions.

Radioecological damages to aquatic and terrestrial organisms due to I-129 in the environment result from ionization caused by the interaction of its beta and gamma particles with living tissue. In particular, upon each disintegration, I-129 emits a beta particle with an average energy of 50.3 keV and a maximum energy of 154.4 keV (Shultis and Faw 2008) The range of beta particles in matter is given by Shleien *et al.* (1998), Formula 2a, p. 3-15:

for ($0.01 \leq E \leq 2.5$ MeV):

$$R = 412 * E^{(1.265 - 0.0954 * \ln(E))}$$

Where:

R = range in mg/cm^2 (range in cm times the density of the absorbing medium in mg/cm^3)

E = energy of the beta particle in MeV

Using this equation, the average approximate range of I-129 beta particles in tissue is about $0.004 \text{g}/\text{cm}^2$ or 0.004 cm. Given that the typical energy required to ionize a molecule (i.e., eject an electron from its orbit) is about 34 to 35 eV (see page 17, Casarett 1968), the total number of ion pairs produced by the energy deposited in tissue from the average energy beta particle emitted by I-129 is about 1400 ion pairs (i.e., $0.05 \text{ MeV}/35 \text{ eV}$).

The pattern of energy deposition for beta particles is described in Morgan and Turner (1973) as follows:

$$\text{mean linear ion density} = T/R_t \times W$$

Where:

T = average energy of electron liberated

R_t = range or electrons of energy T

W = average energy to form an ion pair

For I-129, the equation is $50.3 \text{ keV} \times 1000 \text{ eV}/\text{keV} \div 0.003 \text{ cm} \times 35 \text{ eV}/\text{ion pair} = 4.79 \times 10^5$ ion pairs per cm or about 48 ion pairs per micron. Given that a typical cell

is on the order of tens of microns (see page 102 of Curtis and Barnes, 1989), a single cell might experience about 500 to 600 ion pairs produced by the passage of an average I-129 beta particle. It is this deposited energy in living tissue that results in biological damage. Radioiodine toxicity is most likely in tissues that can transport and accumulate iodide (ATSDR 2004).

Sufficiently energetic beta particles can penetrate the dead layer of the skin of mammals (nominally 70 microns in humans) and deposit energy in underlying tissues. I-129 emits beta particles with an average energy of 50.3 keV and that have a range of about 3.3 cm in air and 0.04 cm in tissue (Shleien *et al.* 1998). Thus, there is some potential for exposure from external beta radiation from I-129, including all aquatic and terrestrial organisms and all stages of their life cycle, except for organisms that have a thick outer layer (such as bark of trees, heavy fur, scales, etc.) that can shield the living tissue beneath from the beta emissions.

RADIOECOTOXICOLOGICAL EFFECTS

Terrestrial and aquatic organisms can also experience internal exposures from the approximate 0.04 MeV photons emitted by I-129. However, because only about 10% of the photon energy is deposited per cm of path length in tissue (see Figure 5.4 of Shleien *et al.* 1998), the contribution of gamma exposure from I-129 to the internal dose is small as compared to that from the internal dose from the beta emissions of I-129. However, external exposure to terrestrial organism from the gamma emissions from I-129 in soil must be taken into consideration when assessing the dose to terrestrial biota. For aquatic biota, close proximity to sediment containing I-129 could contribute to external exposures.

In most animal and human life, iodine concentrations are highest in the thyroid gland. The metabolic constraints that control the quantity of stable iodine in the thyroid gland also restrict the amount of I-129 that can be absorbed and thus also restrict the potential for radiotoxicological effects from I-129.

For example, the ICRP reference man¹⁰⁷ (ICRP 1975) has 13 mg of iodine, 12 mg (>90%) of which is located in the thyroid. Using this reference, combined with an assumption of constant exposure only to pure I-129 (which has a specific activity of 0.17 mCi/g), the theoretical maximum I-129 activity in the thyroid is limited to roughly 2 μ Ci in an adult human. This is an overly conservative assumption considering the highest ratios of I-129 to I-127 seen in animals living near nuclear facilities is in the range of 0.001 (Brauer and Ballou 1974). Encountering iodine only in the form I-129 over a lifetime to achieve this maximum activity is impossible but is presented for comparison with reasonable intake.

Under considerably more realistic conditions, NCRP (1983) states “A steady dietary intake of one pCi I-129 ingested daily would lead to an equilibrium burden of 8.7 pCi in the thyroids of 1 to 4 year old children and 22 and 43 pCi [10^5 times smaller than

¹⁰⁷ In order to standardize the radiation doses to humans per unit intake of a given radionuclide, the International Commission on Radiological Protection defines a reference man, which standardizes the size, weight and function of every organ and structure in the human body.

the above theoretical maximum possible] in those of 14-year olds and adults, respectively, resulting in dose equivalent rates of 4.9, 2.1, 1.7 and 2.6 mrem/y in 1-, 4-, and 14-year olds, and adult, respectively.” NCRP (1983) also estimates a dose to the thyroid to 7 mrem/nCi intake. The thyroid gland in adults is considered to be radioresistant in terms of cell death and failure of function. It has the capacity to actively concentrate iodine. Radioiodine can, therefore, deliver considerable doses to the gland without causing the thyroid to fail. A dose of at least 300 Gy (30,000 rem)¹⁰⁸ is required to cause total ablation of the thyroid within a period of two weeks (WHO 2001). This is exponentially larger than any expected environmental exposure and would only be expected in a medical setting where killing the thyroid is the intended consequence.

In a 1983 study conducted by Book, rats were fed I-129 over a lifetime. These rats experienced no significant increase in tumor occurrence nor were any difference seen in longevity between exposed and control rats.

No literature was found that explicitly addresses the radiotoxicity of I-129 on plants. However, there are a number of publications on the effects of radiation in general on plants and plant communities. Chapter 13 of Casarett (1968) provides an excellent review of the literature on the effects of radiation on higher plants and plant communities. She provides data showing the percent germination for pollen for a variety of plants, as a function dose, where the doses ranged from zero to over 6,000 rad. She also summarizes studies on the effects of radiation on the fertilized egg (ovule), where effects on the developing plant were observed at 500 R,¹⁰⁹ and the radiosensitivity of developing embryos (fertilized ovule) varied 100-fold depending on plant species. That said, the low energy of radiation from I-129 and its low specific activity suggest radiological damage to plants from I-129 to be unlikely.

VII. EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

Natural and synthetic chemicals distributed in plant and plant products have the potential to alter the uptake of iodine in the thyroid of vertebrates. Although the effects of goitrogens on iodine toxicity are not fully understood, changes in thyroid metabolism associated with high intakes of goitrogens may influence iodine toxicity and the concentration of iodine in tissue and animal products (Committee 2005). Some agricultural species (broccoli, kale, spinach, cabbage, soy products, and turnips) have naturally high concentrations of goitrogens (ATSDR 2004); it is not known whether native plants at Hanford may also have high concentrations of these compounds.

There are two types of goitrogens: thiocyanates and goitrin. Thiocyanates inhibit the uptake of iodine in the thyroid, but their action is reversible by additional iodine supplementation. Goitrin inhibits the synthesis of thyroid hormone through the inhibition of thyroid peroxidase. Goitrin effects are not reversible by iodine

¹⁰⁸ 1 Gy= 100 rad. Units of absorbed radiation dose. Although, technically different, by convention, 1 rad is loosely considered equivalent to 1 rem.

¹⁰⁹ For simplicity, it can be assumed that one R or Roentgen is equal to 100 rad (or 100 ergs of energy deposited per gram of tissue).

supplement (Committee 2005). Normal iodine uptake may also be inhibited by bromine, fluoride, cobalt, manganese, and nitrate (ATSRD 2004).

Various substituted phenols with hydroxyl groups in the meta positions have been shown to increase thyroid iodide accumulation and to inhibit iodothyronine production in the thyroid (ATSDR 2004).

VIII. DATA GAPS

The health impacts of I-129 on humans are well understood (ATSDR 2004): effects of acute exposures to radioiodine (predominately I-131¹¹⁰) have been extensively studied in humans and in mammalian laboratory surrogates. An enormous amount of epidemiological and case literature derives from the clinical use of I-131 in diagnostic procedures and in treatment of thyroid gland enlargement and thyrotoxicosis. In addition, epidemiology studies have examined health effects resulting from accidental environmental exposures due to nuclear detonations and releases from nuclear power plants. These studies collectively and credibly identify the thyroid gland as the primary target of radioiodine. Other tissues that are either in close proximity to the thyroid gland, such as the parathyroid gland, or that accumulate iodine, such as the salivary gland, also are affected by exposures to radioiodine; however, these effects occur at absorbed radiation doses that are clearly cytotoxic to the thyroid gland. In addition, the toxicokinetics of iodine in humans has been substantially explored and characterized in both experimental studies and clinical cases.

Although the effects of stable and radioiodine are well understood in humans, the literature on the effects of I-129 on wild species is extremely limited if not absent. Literature on the effects of non-radioactive iodine on wild species has relevance as most of the effects of I-129 are expected to be chemical rather than radiological. Information on mammalian laboratory species is plentiful; however, information appears to be quite limited for aquatic species and may be absent for wild birds, wild mammals, amphibians, and reptiles.

IX. REFERENCES

- Al-Ajely, K.O. 1985. Biological prospecting as an effective tool in the search for mineral deposits. Ph.D. Thesis, University of Wales, Aberystwyth.
- Angelo, J.A. 2004. Nuclear Technology. Greenwood Publishing Group. Westport, Connecticut: 398-399
- Argonne National Laboratory (ANL). 2001. Human Health Fact Sheet. <http://www.stoller-eser.com/factsheet/iodine.pdf>. Accessed 5 January, 2011.

¹¹⁰ I-131 metabolically behaves the same as I-127 and I-129; however, its radiological properties are considerably different than I-129. I-131 decays by 364 keV gamma emission (81% abundance) and beta emission with a mean energy of 190 keV (89% abundance) with a half-life of 8 days. I-131 properties allow it to be used as a surrogate to easily trace where iodine goes in the body. I-131 decays away in a matter of days and thus its risks are very different than I-129 which essentially never decays.

- Arrington, L.R., R.A. Santa Cruz, R.H. Harms, and H.R. Wilson. 1967. Effects of excess dietary iodine upon pullets and laying hens. *Journal of Nutrition* 92:325-330.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Toxicological Profile for Iodine.
- Board on Radiation Effects Research (BRER), Division on Earth and Life Studies. 2004. National Research Council of the National Academies. Distribution and Administration of Potassium Iodide in the Event of a Nuclear Incident. National Academies Press. Washington D.C.
- Book S.A. 1983. I-129 uptake and effects of lifetime feeding in rats. *Health Physics* 45.I:61-66.
- Böszörményi Z. and E. Cseh. 1960. The uptake and reduction of iodate by wheat-roots. *Curr. Sci.* 29:340–341.
- Burgi, H., T. Schafner, and J.P. Sciler. 2001. The toxicology of iodate review of literature. *Thyroid* 11:449-546 (as cited by Committee 2005).
- Brauer, F.P. and N.E. Ballou. 1974. Detection systems form the low-level radiochemical analysis of iodine-131, iodine-129 and natural iodine in environmental samples. *IEEE Trans. Nucl. Sci.* 21:445.
- Casarett, A.P. 1968. *Radiation Biology*. Englewood Cliffs, New Jersey: Prentice-Hall, Inc.
- Christensen, V.L., W.E. Donaldson, J.F. Ort, and Grimes. 1991. Influence of diet mediated maternal thyroid alterations on hatchability and thyroid metabolism of turkey embryos. Iodine toxicity in large turkey hens. *Poultry Science* 70:1594-1601.
- Committee on Minerals and Toxic Substances in Diets and Water for Animals, National Research Council (Committee). 2005. Mineral Tolerance of Animals. National Academies Press; 2 ed:182-190.
- Curtis, H. and N.S. Barnes. 1989. *Biology*. Worth Publishers, Inc.
- Evans, D.H. and J.B. Claiborne. 2006. *The Physiology of Fishes*, 3rd Edition. Boca Raton, FL: CRC Press, p. 282
- Fehn U. and G. Snyder. 2000. ¹²⁹I in the southern hemisphere: global redistribution of an anthropogenic isotope. *Nuclear Instrument Methods B* 172:366-71.
- Garland, T.R., D.A. Cataldo, K.M. McFadden, R.G. Schreckhise, and R.E. Wildung. 1983. Comparative Behavior of ⁹⁹Tc, ¹²⁹I, ¹²⁷I and ¹³⁷Cs in the Environment Adjacent to a Fuels Reprocessing Facility. *Health Physics* 44(6):658-662.
- Gottardi, W. 1991. Iodine and iodine compounds. In: Block, S.S. (ed.), *Disinfection, Sterilization and Preservation*. Philadelphia, PA: Lea & Febiger, pp. 152–382.

- Holland, J.Z. 1963. Physical origin and dispersion of radioiodine. *Health Physics* 9:109.
- Hu, Q., P. Zhao, and J.E. Moran. 2003. Transport of iodine Species in the Terrestrial Environment. American Geophysical Union Fall Meeting, San Francisco, CA.
- International Commission of Radiation Protection (ICRP). 1975. Report of the Task Group on Reference Man. Oxford: Pergamon Press (ICRP Publication No. 71).
- Kocher, D.C. 1981. On the long-term behavior of I-129 in the terrestrial environment. International symposium on migration in the terrestrial environment of long-term radionuclides in the nuclear fuel cycle, Knoxville, TN 27-31 July. Symposium organized by the International Atomic Energy Agency, the Commission of European communities, and the nuclear energy Agency of the Organization for Economic Co-operation and Development (as cited in WHO 2006).
- Laverlock, M.J., M. Stephenson, and C.R. Macdonald. 1995. Toxicity of iodine, iodide, and iodate to *Daphnia magna* and rainbow trout (*Oncorhynchus mykiss*). *Archives of Environmental Contamination and Toxicology* 29(3):344-350.
- Le Valley, M.J. 1982. Acute toxicity of iodine to channel catfish. *Bull. Environ. Contam. Toxicol.* 29:7-11.
- Lewis, P.D. 2004. Responses of domestic fowl to excess iodine: A review. *British Journal of Nutrition* 91:29-39.
- Lenntech Water Treatment. 2009. Iodine (I) and Water. Available from, as of January 9, 2011: <http://www.lenntech.com/periodic/water/iodine/iodine-and-water.htm>
- Mackowiak, C.L. and R.P. Grossl. 1999. Iodate and iodide effects on iodine uptake and partitioning in rice (*Oryza sativa* L.) grown in solution culture. *Plant Soil* 212:135-143.
- Mackowiak, C.L. P.R. Grossl, and K.L. Cook. 2005. Iodine toxicity in a plant-solution system with and without humic. *Plant and Soil* 269:141-150.
- Marcilese, N.A., R.H. Harms, R.M. Valsecchi, and L.R. Arrington. 1968. Iodine uptake by ova of hens given excess iodine and effect upon ova development. *Journal of Nutrition* 94:117-120.
- McDowell, L.R. 2003. *Minerals in Animal and Human Nutrition*, 2nd ed. Amsterdam: Elsevier (as cited by Committee 2005).
- Mellor, J.W. 1946. *Comprehensive Treatise on inorganic and Theoretical Chemistry* Vol. II, P, Cl, Br, I, Li, Na, K, Rb, Cs. London: Longmans, Green and Co., Ltd.
- Moran, J.E., S. Oktay, P.H. Santschi, D.R. Schink, U. Fehn, and G. Snyder. 1999. World-wide redistribution of Iodine-129 from nuclear fuel reprocessing facilities: Results from meteoric, river, and seawater tracer studies. *Environmental Science & Technology* 33(15): 2536-2542.

- Morgan, A., Morgan, D.J., Evans, J.C. and Lister, B.A.J. 1967. Studies on the retention and metabolism of inhaled methyl iodide II: Metabolism of methyl iodide. *Health Physics*.13:1067 (as cited by NCRP 1983).
- Morgan, K.Z. and J.E. Turner. 1973. *Principles of Radiation Protection*. Huntington, NY: Robert E. Krieger Publishing Company.
- Multi-Agency Radiological Laboratory Analytical Protocols Manual Volume II. 2004. NUREG-1576, EPA 402-B-04-001B, NTIS PB2004-105421. Chapter 14-165. July 2004. Available from, as of January 7, 2011: <http://www.nrc.gov/reading-rm/doc-collections/nuregs/staff/sr1576/sr1576v2.pdf>
- National Council on Radiation Protection and Measurements (NCRP). 1983. Iodine-129 evaluation of releases from nuclear Power generation. NCRP Report No. 75.
- Nuclear Energy Institute (NEI). 2006. *Nuclear Waste Disposal for the Future: The Potential of Reprocessing and Recycling*.
- Nuclear Regulatory Commission (NRC). 1980. *Mineral Tolerance of Domestic Animals*. Washington, D.C.: National Academy Press (as cited by Committee 2005).
- Perdomo, J.T., R.H. Harms, and L.R. Arrington. 1966. Effect of dietary iodine upon egg production fertility and hatchability. *Proc Soc Exp Biol Med* 122(3):758-60.
- Perkins, R.W. 1963. Physical and chemical form of I-131 in fallout. *Health Physics* 9:1113.
- Poston, T.M. 1986. Literature review of the concentration ratios of selected radioisotopes in freshwater and marine fish. Richland, WA, Battelle Pacific Northwest Laboratories, pp. 1-21(as cited by WHO 2006)
- Poston, T.M., J.P. Duncan, and R.L. Dirkes, eds. 2010. *Hanford Site Environmental Report for Calendar Year 2009*. PNNL-19455. Pacific Northwest National Laboratory, Richland, WA.
- Preedy, V.R., G.N. Burrow, and R. Watson. 2009. *Comprehensive Handbook of Iodine: Nutritional, Biochemical, Pathological and Therapeutic Aspects*. Academic Press; ed. 1.
- Selinus, O. 2005. *Essentials of Medical Geology: Impacts of the natural environment on Public Health*.
- Schink, D.R., P.H. Santschi, O. Corapcioglu, P. Sharma, and U. Fehn. 1995. 129I in Gulf of Mexico waters. *Earth Planet. Sci. Lett.* 135:131-138.
- Schwehr, K.A. 2004. *Specification and transport of Anthropogenic 129 Iodine and Natural 127 Iodine on Surface and Subsurface Environments*. Texas A&M University.

- Scientific Committee on Food (SCF). 2002. Opinion of the Scientific Committee on Food on the Tolerable Intake Level of Iodine. European Commission. Brussels. (as cited by Committee 2005).
- Shleien, B., L.A. Slaback, Jr., and B.K. Birky. 1998. Handbook of Health Physics and Radiological health, Third Edition. Williams & Williams, A Waverly Company, pp. 2-12.
- Smith, D.D. 1977. 129I in Animal Thyroids from Nevada and Other Western States. Report No. EPA-600/3-77-067. U.S. Environmental Protection Agency.
- Sheppard, S.C. and W.G. Evenden. 1995. Toxicity of soil iodine to terrestrial biota, with implications for 129I. *Journal of Environmental Radioactivity* 27(2): 99-116.
- Shultis, J.K. and Faw, R.E. 2008. Fundamentals of Nuclear Engineering, Second Edition. CRC Press, Boca Raton, FL: pp. 566
- Swingle, W.W. 1923. Iodine and amphibian metamorphosis. *The Biological Bulletin* 45(5):229.
- Takahashi Y. and K. Satoh. 1989. Identification of the photochemically iodinated amino-acid residue on D1-protein in the photosystem II core complex by peptide mapping analysis. *Biochim. Biosphys. Acta.* 973:138–146.
- Umaly R.C. and L.W. Poel. 1971. Effects of iodine in various formulations on the growth of barley and pea plants in nutrient solution culture. *Ann. Bot.* 35:127–131.
- U.S. Environmental Protection Agency (EPA). 2002. EPA Facts about Iodine.
- U.S. Nuclear Regulatory Commission (USNRC). 1979. A Dynamic Model of the Global Iodine Cycle for the Estimation of Dose to the World Population from Releases of Iodine-129 to the Environment. Division of Safeguards, Fuel Cycle, and Environmental Research. NUREG/CR-0717.
- Vogt, R., R. Sander, R. Van Glasgow, and P.H. Crutzen. 1999. Iodine chemistry and its role in halogen activation and ozone loss in the marine boundary layers: A model study. *Journal of Atmospheric Chemistry* 32:375-95.
- Webster, S.H., Stohlman, and B. Highman. 1966. The toxicology of potassium and sodium iodates III. Acute and subacute oral toxicology of potassium iodate in dogs. *Toxicology of Applied Pharmacology* 8:185-192 (as cited in Committee 2005).
- Whitehead, D.C. 1984. The sorption of iodide by soil components. *Journal of the Science of Food and Agriculture* 25:73-79.
- World Health Organization (WHO), International Agency for Research on Cancer. 2001. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to

Man. Geneva:(Multivolume work). Available at:
<http://monographs.iarc.fr/index.php>. V78 404 (2001).

World Health Organization (WHO). 2006. Iodine and Inorganic Iodines: Human Health Aspects (Concise International Chemical Assessment Document 72).

World Health Organization, United Nations Children's Fund and International Council for the Control of Iodine Deficiency Disorder (WHO/UNICEF/ICCD). 2001. Assessment of Iodine Deficiency Disorders and Monitoring Their Eliminations: A Guide for Programme Managers, 2nd ed. Geneva.

Yuita, K. 1994. Overview and dynamics of iodine and bromine in the environment, 2: Iodine and bromine toxicity and environmental hazards. Japan Agricultural Research Quarterly 28(2):100-111.

MERCURY (Hg) ECOTOXICOLOGICAL PROFILE¹¹¹

I. INTRODUCTION Mercury (Hg) is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Heavy and silver-colored, mercury is the only metal that is liquid at standard conditions of temperature and pressure (25°C and 1 atm).

Assessing the potential effects of mercury exposure, particularly to methylmercury, has been an active area of research for over thirty years (Scheuhammer and Sandheinrich 2008) and has resulted in the generation of an extremely large body of information. Mercury fate, transport, and ecotoxicology have been the focus of special issues in journals and of conferences/workshops, and have been the subject of several books as well as a tremendous number of journal articles. This profile does not attempt to comprehensively review all available ecotoxicological information on mercury but rather aims to provide a broad overview of its known characteristics and properties, including its better-studied ecological effects with a particular focus on those of the most relevance to natural resource damage assessment.

II. SOURCES Mercury is a toxic element found ubiquitously throughout the environment. The sources of mercury to the biosphere can be grouped as follows (UNEP 2002):

- Natural sources, such as volcanic activity, forest fires, and weathering of rocks;
- Current/ongoing anthropogenic activities, such as fossil fuel combustion, leaks from industrial activities, and the disposal or incineration of wastes; and
- Re-mobilization of past anthropogenic releases from environmental media such as soils, sediments, waterbodies, landfills, and waste piles.

Estimates of the increase in atmospheric deposition of mercury since preindustrial times range from 1.5 to 4, excluding industrial areas where deposition rates are higher (Swain *et al.* 1992, UNEP 2002). North American anthropogenic sources on average contribute roughly 20 to 30 percent of total mercury deposition within the continental United States (Seigneur *et al.* 2004, Selin *et al.* 2007). The remainder comes from anthropogenic emissions of other countries and natural sources. There is uncertainty with respect to how much of anthropogenic emissions is attributable to new releases as distinct from remobilization; however, several researchers have estimated these to be approximately equal or at least within a factor of two of each other (Seigneur *et al.* 2004).

At Hanford, use and spillage of chemicals, including mercury, in the 100 Area resulted in the contamination of facilities and soil (EPA 1999). One specific example

¹¹¹ Selected portions of this document are derived from IEC (2010).

of such releases relates to a five-year project termed “Project P-10-X” that commenced in 1949 (Kincaid *et al.* 2006). This project, the purpose of which was tritium production, took place in the 100-B Area and released both tritium and mercury to the environment. Indeed, the “second largest source of contamination emitted from Project P-10-X operations was the mercury used in the Toepler pumps and pressure gauges. It is estimated that hundreds of liters of contaminated mercury was disposed to the 100-B crib, with subsequent diffusion through surrounding soil and groundwater” (*ibid.*).

III. ENVIRONMENTAL CHEMISTRY

Mercury’s chemical forms include elemental mercury, or Hg(0), oxidized inorganic mercury, as Hg²⁺ and Hg₂²⁺, and organic forms, principally methylmercury. As an element, mercury does not break down although it does change among these chemical forms, and its form determines not only its environmental fate but also its potency as a toxicant. In particular, elemental mercury can be oxidized to Hg²⁺ which can in turn be methylated by sulfate-reducing bacteria. Elemental mercury is volatile and can be lost to the atmosphere and transported long distances before being deposited. In contrast, Hg²⁺ tends to bind to sediment particles (EPA 2006). The dominant factors controlling mercury speciation in solution include dissolved ions, pH, and redox potential (Gabriel and Williamson 2004).

From a biological perspective, the most hazardous form of mercury is methylmercury, both because of its bioaccumulation and biomagnification potentials and also because organic forms are the most toxic (Wolfe *et al.* 1998, Boening 2000).

The main mechanism through which mercury becomes methylated is thought to be through the action of sulfate-reducing bacteria, particularly in freshwater sediments and wetlands (Wiener *et al.* 2003, Evers *et al.* 2005). Many factors affect the rate of mercury methylation in waterbodies, including pH, acid neutralizing capacity, sulfate content, dissolved organic matter, waterbody morphometry, and temperature (Wiener *et al.* 2003, EPRI 2004, EPA 2005). Wetlands tend to be areas of higher methylmercury production and may contribute methylmercury to associated waterbodies (Wiener *et al.* 2003, EPA 2005b). In general, mercury methylation rates are higher in lower alkalinity, low pH waterbodies, in surface waters with large upstream or adjoining wetlands, in waters with adjoining or upstream terrestrial areas subject to flooding, and in dark-water lakes and streams (Scheuhammer *et al.* 2007). Demethylation of methylmercury is possible, but this process is less well understood. There is some evidence that at least in some predatory aquatic species, a portion of the methylmercury burden may become demethylated, especially in the liver and kidney, and perhaps brain (Scheuhammer *et al.* 2007).

In contrast to aquatic ecosystems, concentrations of methylmercury in soils are generally low (EPA 2005, Gabriel and Williamson 2004); indeed, the concentration of methylmercury is generally less than 2% of the total mercury concentration in soil (Schluter 2000 as cited in Gabriel and Williamson 2004). Methylation can occur in the terrestrial environment, but this process is less well understood (Gabriel and Williamson 2004).

FATE AND TRANSPORT

Mercury is primarily released to the environment in its elemental and inorganic forms. When released to the atmosphere, it can be transported around the globe, and through wet and dry depositional processes, may be deposited in areas far from its point of release.

Atmospheric mercury can deposit directly to surface waters and to soils. Although in most watersheds, the primary source of total mercury to surface waters is atmospheric deposition, erosion and runoff can also be large sources (EPA 2006, Gabriel and Williamson 2004). Groundwater can also be a source of mercury to surface waters, and the interface between ground water and streams can be an important methylation site (EPA 2006). Inorganic mercury, as Hg^{2+} in aquatic systems, is generally bound to dissolved organic carbon (EPA 2006).

Gabriel and Williamson (2004) review biogeochemical factors affecting the speciation, ligand formation, and transportation of mercury in terrestrial environments. These authors note that adsorption in soils is one of the most widely researched areas in the terrestrial biogeochemistry of mercury. A number of factors affect the element's chemical form in soils. These factors include the presence of certain dissolved species (particularly S^- , Cl^- and dissolved organic carbon), pH, and redox potential. Factors affecting mercury's adsorption to soils include the soil particles' surface area, organic content, cation exchange capacity, and grain size. Overall, soils have a high affinity for mercury in its elemental, inorganic, and organic forms, such that even the elemental form can be fairly resistant to volatilization loss, and soils can sequester a large percentage of atmospherically deposited mercury (*ibid.*).

BIOACCUMULATION POTENTIAL

Methylmercury is the only form of mercury that biomagnifies through food chains (Chan *et al.* 2003, EPRI 2004), with higher trophic level organisms acquiring increasingly large body burdens (EPA 1997, EPA 2005). Nearly all of the mercury in fish is in the form of methylmercury (Wiener and Spry 1996, EPA 1997, Eisler 2000), and even in predatory insects, methylmercury comprises much of the body burden (Mason *et al.* 2000, Cristol 2008). Overall, the proportion of methylmercury in organisms is a function of food chain length.

Methylmercury has repeatedly been shown to accumulate in aquatic food webs. Uptake and bioaccumulation in terrestrial ecosystems has been less well studied, although it has been shown that plants can take up elemental mercury directly from the atmosphere and can accumulate mercury in their leaves (EPA 2006). Uptake from soils via roots is also possible, and this mercury may be translocated to the leaves (Suszycynsky and Shann 1995 as cited in Boening 2000). Mosses are key vegetative species in that they tend to accumulate more mercury than other plants (Boening 2000).

Cristol *et al.* (2008) have reported methylmercury biomagnification in a terrestrial habitat immediately adjacent to a mercury-contaminated river in Virginia; in this food web, spiders provided a substantial exposure pathway to songbirds. Rimmer *et al.*

(2010) found increasing mercury concentrations at higher trophic levels in a montane forest.

ACCUMULATION WITHIN TISSUES

Methylmercury does not partition based on lipophilicity but does preferentially bind with sulfhydryl groups in proteins (Webb *et al.* 2006). In vertebrates, mercury has long been known to accumulate to high levels in the kidneys and liver (Tan *et al.* 2009). The pituitary gland, thyroid and gonads have also been shown to accumulate mercury concentrations similar to, or modestly lower than, those in kidneys and livers (*ibid.*).

In mammals, where neurotoxic effects are a common focus, effects thresholds are frequently expressed as concentrations in brain tissue (e.g., see Scheuhammer *et al.* 2007). In bats, fur and blood have also been evaluated (Wada *et al.* 2010).

In fish, accumulation of mercury in the brains appears to occur to a lesser extent than in mammals (Boening 2000). Muscle tissue, however, is frequently evaluated, as this tissue is of particular interest when human exposure is of concern. When the purpose of the research is to estimate effects to fish themselves, concentrations in whole fish (especially if small) are commonly targeted (e.g., Friedman *et al.* 2006, Hammerschmidt *et al.* 2002, Drevnik and Sandheinrich 2003).

In birds, both brain and egg concentrations have commonly been measured (Scheuhammer *et al.* 2007, Tan *et al.* 2009). Blood has also been a preferred tissue to characterize exposure in birds (e.g., Burgess and Meyer 2008, Brasso and Cristol 2008). Bergeron *et al.* (2007) report that blood concentrations can also be used as an of mercury exposure in turtles.

IV. TYPICAL MAJOR EXPOSURE ROUTES

Fish and wildlife are primarily exposed to methylmercury rather than to other chemical forms; furthermore, exposure occurs predominately through the diet (Scheuhammer *et al.* 2007). Because of the biomagnification of methylmercury through food webs, top predators, particularly in aquatic food chains, are likely to be the most exposed, while terrestrial non-piscivorous species typically experience relatively low mercury exposures (*ibid.*)

For fish embryos, maternal transfer of dietary methylmercury is the primary method of exposure (Hammerschmidt and Sandheinrich 2005 as cited in Scheuhammer *et al.* 2007). Maternal transfer also occurs in mammalian, reptilian, amphibian, and avian species (Tan *et al.* 2009, Bergeron *et al.* 2010).

V. ECOTOXICITY

Mercury is a known mutagen, teratogen, and carcinogen (Eisler 2000). In higher organisms, inorganic mercury is primarily nephrotoxic (Khan and Wang 2009), while methylmercury's effects in wildlife include neurotoxicity, affecting endpoints such as behavioral alterations and sensory impacts (e.g., to vision and hearing), and developmental effects (EPA 1997, Eisler 2000). Mercury can cross both the blood-brain barrier and the placental barrier, making it "one of a few known developmental

neurotoxins” (Khan and Wang 2009). Species sensitivity to mercury varies, and within a species the early life stages are generally the most sensitive to these types of effects (Wiener and Spry 1996, Eisler 2000, Boening 2000).

Endocrine-related effects, including reproductive impairment, have also been observed in fish, birds, and mammals (Tan *et al.* 2009). The “estrogenic” properties of mercury compounds may be responsible for these effects, and some evidence suggests that endocrine effects “may be observed at lower doses or before onset of the extensively studied neurological symptoms” (*ibid.*). Endocrine effects are not limited to the hypothalamic-pituitary-gonadal axis but also include impacts to the hypothalamic-pituitary-thyroid axis and the hypothalamic-pituitary-adrenal axis (*ibid.*).

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

Mercury has no known beneficial effects or protective properties.

MECHANISM(S) AND LOCI OF TOXICITY

Mercury is cytotoxic (Tan *et al.* 2009). At the molecular level, it interacts with reduced sulfhydryl groups (Chan *et al.* 2003). Sulfhydryl groups are part of many proteins and enzymes; thus, methylmercury may interfere with the actions of these structures, directly or indirectly altering cellular metabolism. Cellular processes affected by mercury can include ionic homeostasis, synaptic function, oxidative stress, and protein synthesis (Khan and Wang 2009). The literature has documented impacts of methylmercury on the activity of certain enzymes, including several enzymes present in the brain (Hoffman and Heinz 1998, Wolfe *et al.* 1998).

PLANTS

Less is known about the effects of mercury on plants, although several laboratory studies of aquatic and terrestrial species have been conducted. These have found evidence of impacts to a variety of endpoints including photosynthesis, root/shoot weight and length, chlorophyll content, enzyme activities, mitotic activity, transpiration, water uptake, and chlorophyll synthesis (Boening 2000).

FISH AND AQUATIC INVERTEBRATES

Through laboratory dosing studies, researchers have investigated the sensitivity to mercury of a wide range of fish species, including Atlantic salmon, rainbow trout, brook trout, catfish, amphipods, mummichog, fathead minnows, walleye, golden shiner, and others (Eisler 2000, Scheuhammer *et al.* 2007). These studies have found that mercury reduces fish growth, increases tissue histopathology, and impairs olfactory receptor function (Eisler 2000). Symptoms in fish potentially related to neurotoxicity include changes in activity level, coordination, ability to capture prey, predator avoidance, emaciation, brain lesions, and death (Wiener and Spry 1996, Eisler 2000, Weis 2009).

Reproductive impacts represent another key set of endpoints for fish. Laboratory studies have demonstrated impacts of mercury on a wide range of reproductive health metrics including gonadal development, production of sex hormones, gametogenesis,

sperm morphology and motility, vitellogenesis, fecundity, fertilization, hatching success, and embryo development/malformations among other reproductive endpoints (Scheuhammer *et al.* 2007, Tan *et al.* 2009, Weis 2009, Crump and Trudeau 2009). Exposures during sensitive periods can produce delayed effects in later life phases (Weis 2009).

Some effects observed in laboratory studies (e.g., growth and survival) are relatively unlikely to occur in most wild fish, inasmuch as ambient mercury concentrations are generally lower than those found to cause these effects (EPA 2005). However, fish reproduction may be a more sensitive endpoint, and it is plausible that reproductive effects from methylmercury may occur under field conditions (Scheuhammer *et al.* 2007, Crump and Trudeau 2009). Also, more recent laboratory studies have specifically focused on environmentally realistic concentrations (e.g., Friedman *et al.* 1996, Hammerschmidt *et al.* 2002, Drevnick and Sandhenrich 2003).

Field studies of effects of mercury on fish are more limited than laboratory studies (Scheuhammer *et al.* 2007). Friedmann *et al.* (2002) compared the reproductive health of wild largemouth bass from three New Jersey lakes with varying degrees of mercury contamination. The authors evaluated organosomatic indices, condition factor, serum cortisol, testosterone, and 11-ketotestosterone and found a significant difference for this latter metric, and a (non-statistically significant) 50% decrease in testosterone among fish from the most heavily contaminated lake.

Webb *et al.* (2006) investigated white sturgeon from the lower Columbia River and found several significant correlations between tissue methylmercury concentrations and measures of reproductive health in these fish. These include negative correlations between: (a) plasma androgens and Hg in muscle tissue, (b) plasma estrogens and Hg in liver tissue, (c) condition factor and both gonad/liver Hg concentrations, (d) relative weight and both gonad/liver Hg concentrations, and (e) in immature males, gonadosomatic index and gonad Hg concentrations. The authors hypothesize that “[t]he physiologic result of decreased circulating sex steroids... may be altered gametogenesis, a delay in sexual maturation, and/or decreased reproductive success” but note that “further studies with older and larger fish would need to be conducted to determine if mercury is negatively impacting the onset of maturation and reproductive potential.” Webb *et al.* (2006) further note that the gonadosomatic index has been found to be inversely related to tissue mercury in several teleosts.

In aquatic invertebrates, many researchers have investigated mercury concentrations; however, data on effects are fewer. Skinner and Bennett (2007) evaluated gill deformities in macroinvertebrates (mayflies, caddisflies, and stoneflies) from stream areas in New York State. The authors found the highest rate of gill deformities (28%) among animals with the highest mercury concentrations; however, the authors did not report mercury concentrations in macroinvertebrates from the reference area (instead noting that water column levels were non-detectable).

BIRDS

Dosing studies of bird species have found evidence of toxicity, ranging from blood and tissue chemistry changes to brain lesions, reduced growth, developmental

alterations, behavioral alterations, reproductive impairment, and death (Frederick 2000, Eisler 2000). Reproductive effects include not only embryomortality and development but also appear to extend to juvenile survival (Wolfe *et al.* 1998). Mercury may also be associated with immunotoxic effects including a higher potential for infection by disease organisms (Scheuhammer *et al.* 2007). Avian species investigated include mallards, quail, ring-necked pheasants, chickens, house sparrows, northern bobwhite, goshawks, red-tailed hawks, tree swallows, and others (Thompson 1996, Eisler 2000, Wada *et al.* 2009, Heinz *et al.* 2009). Egg injection experiments have demonstrated a range in LC50 concentrations among different species of avian embryos exposed to mercury (Heinz *et al.* 2009).

Although a number of correlative studies of mercury and various avian reproductive endpoints under field conditions did not find effects or were subject to confounding factors (Thompson 1996), there are several examples that strongly suggest mercury is adversely impacting at least some species at some locations. Of these, the common loon is probably the best studied. Effects associated with field exposure to mercury in this species include elevated corticosterone hormone levels, reduced foraging behavior, reduced incubation activity, and reduced fledgling production (Evers 2004, Burgess and Meyer 2008).

In addition, available data suggest that sublethal impacts to birds in the Florida Everglades are likely. (The Florida Everglades is amongst the better-studied sites with respect to mercury.) In particular, field studies suggest that mercury may predispose juvenile great white herons to disease (Spalding *et al.* 1994 as cited in Frederick 2000). Dosing of great egrets at environmentally realistic levels resulted in impaired immunological responses, reduced appetite, and altered behavior (Frederick 2000). Altogether, "it is strongly suspected that exposure of nestlings to Everglades diets is likely to result in increased juvenile mortality" (*ibid.*). Population-level impacts are possible, as modeling suggests populations of great egrets are sensitive to changes in juvenile survival (*ibid.*).

Bald eagles and osprey are high trophic-level predators that have experienced elevated mercury levels in some environments; however, available data indicate a lack of association between mercury exposure and productivity of these species (Scheuhammer *et al.* 2007).

Fewer field studies are available for non-piscivorous birds, although several recent studies have examined tree swallows near the mercury-contaminated South River in Virginia. In particular, Brasso and Cristol (2008) found "subtle" reproductive effects—i.e., reduced productivity for young females in the contaminated area that were breeding for the first time in one of the years of the study. Wada *et al.* (2009) found nestlings from the contaminated area to have suppressed adrenocortical responses, plasma triiodothyronine and thyroxin concentrations relative to reference levels, suggesting endocrine disruption in these organisms. Hawley *et al.* (2009) found evidence of sublethal immunosuppressive effects in female tree swallows associated with the South River relative to reference birds. In addition, Edmonds *et al.* (2010) identified elevated mercury levels in some North American populations of the wetland obligate and rapidly declining rusty blackbird and found concentrations

that are “among the highest reported for wild populations of passerines at sites without a known local source of mercury.” The authors suggest that mercury should be considered as a potential contributor to the species’ decline at certain locations.

MAMMALS

Methylmercury causes neurotoxic effects in mammals, including brain lesions, ataxia, anorexia, disorientation, tremors/convulsions, lethargy, paralysis, and death (Wolfe *et al.* 1998, Frederick 2000, Scheuhammer *et al.* 2007). Effects on reproduction have also been examined: for instance, Dansereau *et al.* (1999) found a tentative link between methylmercury in the diet of mink and whelping rates.

Most research on the effects of mercury on mammals has been in the form of laboratory studies, and the most commonly evaluated non-domestic species are mink and otter (Wolfe *et al.* 1998, Eisler 2000, Scheuhammer *et al.* 2007). Effects thresholds in these species are often expressed as dietary exposures or achieved concentrations in brain tissues (e.g., Scheuhammer *et al.* 2007).

There have been a few scattered incidents of apparent mercury toxicity to wild mammals, including the death of a Florida panther (Roelka *et al.* 1991, as cited in Thompson 1996). This same research also suggested a potential impact of mercury exposure on Florida panther kitten survival (*ibid.*). Sleeman *et al.* (2010) report the discovery of a moribund river otter next to the mercury-contaminated South River, Virginia, and identify mercury poisoning as the cause. Scheuhammer *et al.* (2007) states that current levels of methylmercury in mink and piscivorous mammals may be sufficiently high in certain mercury-sensitive environments to cause “subtle neurotoxic and other consequences.”

Wada *et al.* (2010) evaluated adrenocortical responses in female big brown bats near a mercury-contaminated river in Virginia. Although bats captured at the contaminated site had 2.6 times higher mercury concentrations in blood and fur, no differences were observed in adrenocortical responses.

AMPHIBIANS AND REPTILES

Less is known about the effects of mercury on amphibians and reptiles than other species groups. Amphibians and reptiles can have increased tissue concentrations of methylmercury, varying with geographic region and diet (Tan *et al.* 2009). A significant number of investigations of the effects of mercury exposure to amphibians used exposure conditions have not been “representative of conditions in natural ecosystems” (Unrine *et al.* 2004); however, in a mesocosm experiment, the southern leopard frog larvae experienced increased mortality and malformations, and reduced metamorphic success when exposed to dietary mercury expected to reflect the highest concentrations associated with atmospheric deposition (Unrine *et al.* 2004). The authors conclude that “dietary Hg concentrations in sites with little or no source contamination may be sufficient to disrupt normal development and key life history characteristics of amphibians.”

Salamanders from a mercury-contaminated site with “among the highest documented [mercury concentrations] in amphibians” caught half as much prey as reference

animals (Burke *et al.* 2010). Furthermore, in one of two tests of locomotion two tests of locomotion, there appeared to be a significant effect on speed and responsiveness (*ibid.*).

EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

Selenium (Se), which is both an essential and a toxic element, has been shown to act in an antagonistic fashion towards mercury in many studies including those on bacteria, zooplankton, mayflies, amphipods, perch, walleye, quails, mallards, rats, mice, and pigs (Khan and Wang 2009, Yang *et al.* 2008). Indeed, Khan and Wang (2009) characterize their interaction as “one of the best known examples of biological antagonism” while noting that both additive and synergistic effects of mercury and selenium have at times been observed. Whether antagonism or other interactions predominate is a function on the elements’ relative concentration, bioavailabilities, and the species’ (and organ’s) sensitivity (Khan and Wang 2009). The fundamental mechanism for mercury-selenium antagonism is not yet understood (Khan and Wang 2009, Yang *et al.* 2008).

In addition, in some cases, the interaction of selenium and methylmercury appears to be complex. Mallard diets supplemented with selenium ameliorated the neurotoxic effects of methylmercury but increased reproductive impairment (Heinz and Hoffman 1998).

DATA GAPS

Most studies of mercury's effects have been laboratory dosing studies. Until recently, many of these studies have used mercury concentrations that are much higher than those typically encountered in the environment (Crump and Trudeau 2009, Unrine *et al.* 2004), rendering it difficult to extrapolate laboratory results into field conditions.

Field studies of any species group are fewer, and the interpretation of results can be complicated by confounding factors such as the presence of multiple contaminants or other environmental factors (Friedman *et al.* 2002, Crump and Trudeau 2009). The common loon is the probably the best-studied species, having been studied repeatedly in the field and also having corroboration from laboratory studies (Scheuhammer *et al.* 2007). Field studies have also been conducted on a limited number of other avian and fish species, and for one salamander species.

Overall, most studies have focused on aquatic or aquatically-linked organisms, especially fish species and piscivores, presumably because of the higher rates of methylation in aquatic ecosystems and consequent potential for higher bioavailability of methylmercury to these organisms. Considerably less research has been devoted to effects on invertebrates (aquatic and terrestrial), plants, amphibians, reptiles, and non-aquatically linked birds and wild mammals.

- VI. REFERENCES** Bergeron, C.M., C.M. Bodinof, J.M. Unrine, and W.A. Hopkins. 2010. Bioaccumulation and maternal transfer of mercury and selenium in amphibians. *Environmental Toxicology and Chemistry* 29(4):989-997.
- Bergeron, C.M., J.F. Husak, J.M. Unrine, C.S. Romanek, and W.A. Hopkins. 2007. Influence of feeding ecology on blood mercury concentrations in four species of turtles. *Environmental Toxicology and Chemistry* 26(8):1733–1741.
- Boening, D.W. 2000. Ecological effects, transport, and fate of mercury: a general review. *Chemosphere* 40:1335-1351.
- Burgess, N.M. and M.W. Meyer. 2008. Methylmercury exposure associated with reduced productivity in common loons. *Ecotoxicology* 17:83-91.
- Burke, J.N., C.M. Bergeron, B.D. Todd, and W.A. Hopkins. 2010. Effects of mercury on behavior and performance of northern two-lined salamanders (*Eurycea bislineata*). *Environmental Pollution* 158:3546-3551.
- Chan, H.M., A.M. Scheuhammer, A. Ferran, C. Loupelle, J. Holloway, and S. Weech. 2003. Impacts of mercury on freshwater fish-eating wildlife and humans. *Human and Ecological Risk Assessment* 9(4):867-883.
- Cristol, D.A., R.L. Brasso, A.M. Condon, R.E. Fovargue, S.L. Friedman, K.K. Hallinger, A.P. Monroe, and A.E. White. 2008. The movement of aquatic mercury through terrestrial food webs. *Science* 320:335.
- Crump, K.L. and V.L. Trudeau. 2009. Critical review: Mercury-induced reproductive impairment in fish. *Environ Toxicol Chem* 28(5):895-907.
- Dansereau, M., N. Larivere, D. DuTremblay, and D. Belanger. 1999. Reproductive performance of two generations of female semidomesticated mink fed diets containing organic mercury contaminated freshwater fish. *Archiv. Environ. Contam. & Toxicol.* 36(2):221-226.
- Drevnick, P.E. and M.B. Sandheinrich. 2003. Effects of dietary methylmercury on reproductive endocrinology of fathead minnows. *Environ. Sci. Technol.* 37(19):4390-4396.
- Edmonds, S.T., D.C. Evers, D.A. Cristol, C. Mettke-Hofmann, L.L. Powell, A.J. McGann, J.W. Armiger, O.P. Lane, D.F. Tessler, P. Newell, K. Heyden, and N.J. O’Driscoll. 2010. Geographic and seasonal variation in mercury exposure of the declining rusty blackbird. *The Condor* 112:(4):789-799.
- Eisler, R. 2000. Handbook of chemical risk assessment: Health hazards to humans, plants, and animals. Volume 1: Metals. Lewis Publishers, Boca Raton, FL.
- Electric Power Research Institute (EPRI). 2004. Atmospheric Mercury Research Update. Palo Alto, CA. 1005500.
- Evers, D.C. 2004. Status assessment and conservation plan for the common loon (*Gavia immer*) in North America. U.S. Fish and Wildlife Service, Hadley, MA.

- Evers, D.C., N.M. Burgess, L. Champoux, B. Hoskins, A. Major, W.M. Goodale, R.J. Taylor, R. Poppenga, and T. Daigle. 2005. Patterns and interpretations of mercury exposure in freshwater avian communities in northeastern North America. *Ecotoxicology* 14:193-221.
- Frederick, P.C. 2000. Mercury contamination and its effects in the Everglades ecosystem. *Reviews in Toxicology* 3:213-255.
- Friedman, A.S., M.C. Watzin, T. Brinck-Johnsen, and J.C. Leiter. 1996. Low levels of dietary methylmercury inhibit growth and gonadal development in juvenile walleye (*Stizostedion vitreum*). *Aquatic Toxicology* 35:265-278.
- Hammerschmidt, C.R., M.B. Sandheinrich, J.G. Wiener, and R.G. Rada. 2002. Effects of dietary methylmercury on reproduction of fathead minnows. *Environ. Sci. Technol.* 35(5):877-883.
- Hawley, D.M., K.K. Hallinger, and D.A. Cristol. 2009. Compromised immune competence in free-living tree swallows exposed to mercury. *Ecotoxicology* 18:499-503.
- Heinz, G.H. and D.J. Hoffman. 1998. Methylmercury chloride and selenomethionine interactions on health and reproduction in mallards. *Environ. Tox. & Chem.* 17(2):139-145.
- Heinz, G.H., D.J. Hoffman, J.D. Klimstra, K.R. Stebbins, S.L. Kondrad, and C.A. Erwin. 2009. Species differences in the sensitivity of avian embryos to methylmercury. *Arch. Environ. Contam. Toxicol.* 56:129-138.
- Hoffman, D.J. and G.H. Heinz. 1998. Effects of mercury and selenium on glutathione metabolism and oxidative stress in mallard ducks. *Environ. Tox. & Chem.* 17(2):161-166.
- Industrial Economics, Inc. (IEc). 2010. Effects of Air Pollutants on Ecological Resources: Literature Review and Case Studies. Draft Report. February. Available 10 November 2010 at <<http://www.epa.gov/air/sect812/prospective2.html>>.
- Khan, M.A. and F. Wang. 2009. Mercury-selenium compounds and their toxicological significance: Toward a molecular understanding of the mercury-selenium antagonism. *Environ. Toxicol. Chem.* 28(8):1567-1577.
- Mason, R.P., J.-M. Laporte, and S. Andres. 1999. Factors controlling the bioaccumulation of mercury, methylmercury, arsenic, selenium, and cadmium by freshwater invertebrates and fish. *Arch Environ Contam Toxicol* 38:283-297.
- Rimmer, C.C., K.P. McFarland, D.C. Evers, E.K. Miller, Y. Aubry, D. Busby, and R.J. Taylor. 2005. Mercury concentrations in Bicknell's thrush and other insectivorous passerines in montane forests of northeastern North America. *Ecotoxicology* 14:223-240.

- Rimmer, C.C., E.K. Miller, K.P. McFarland, R.J. Taylor, and S.D. Faccio. 2010. Mercury bioaccumulation and trophic transfer in the terrestrial food web of a montane forest. *Ecotoxicology* 19:697-709.
- Seigneur, C., K. Vijayaraghavan, K. Lohman, P. Karamchandani, and C. Scott. 2004. Global source attribution for mercury deposition in the United States. *Env. Sci. & Tech.* 38(2):555-569.
- Selin, N.E., D.J. Jacob, R.J. Park, R.M. Yantosca, S. Strode, L. Jaegle, and D. Jaffe. 2007. Chemical cycling of atmospheric mercury. *Journal of Geophysical Research.* 112, D02308, doi:10.1029/2006JD007450
- Scheuhammer, A.M., M.W. Meyer, M.B. Sandheinrich, and M.W. Murray. 2007. Effects of environmental methylmercury on the health of wild birds, mammals, and fish. *Ambio* 36(1):12-18.
- Scheuhammer, A.M. and M.B. Sandheinrich. 2008. Recent advances in the toxicology of methylmercury in wildlife. *Ecotoxicology* 17(2):67-68.
- Skinner, K.M. and J.D. Bennett. 2007. Altered gill morphology in benthic macroinvertebrates from mercury enriched streams in the Neversink Reservoir Watershed, New York. *Ecotoxicology* 16:311-316.
- Sleeman, J.M., D.A. Cristol, A.E. White, D.C. Evers, R.W. Gerhold, and M.K. Keel. 2010. Mercury poisoning in a free-living northern river otter (*Lontra Canadensis*). *Journal of Wildlife Diseases* 46(3):1035-1039.
- Thompson, D.R. 1996. Mercury in birds and terrestrial mammals. In: W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds.). *Environmental Contaminants in Wildlife*. Lewis Publishers, Boca Raton, FL, pp. 297-339.
- United Nations Environment Programme (UNEP). 2002. Global mercury assessment. Inter-Organization Programme for the Sound Management of Chemicals.
- Unrine, J.M., C.H. Jagoe, W.A. Hopkins, and H.A. Brant. 2004. Adverse effects of ecologically relevant dietary mercury exposure in southern leopard frog (*Rana sphenoccephala*) larvae. *Environmental Toxicology and Chemistry* 23(12):2964-2970.
- U.S. Environmental Protection Agency (EPA). 1999. EPA Superfund Record of Decision: Hanford 20-Area (USDOE) and Hanford 100 Area (USDOE). EPA ID: WA1890090078 and WA3890090076, OU(s) 15 & 27. Benton County, WA. July 15.
- U.S. Environmental Protection Agency (EPA). 1997. Mercury study report to Congress. Volume I: Executive Summary. Office of Air Quality Planning and Standards, Office of Research and Development. EPA-452/R-97-003. December.
- U.S. Environmental Protection Agency (EPA). 2005. Regulatory impact analysis of the Clean Air Mercury Rule. EPA-425/R-05-003. March.

- U.S. Environmental Protection Agency (EPA). 2006. Mercury transport and fate through a watershed. Synthesis Report of Research from EPA's Science to Achieve Results (STAR) Grant Program. January.
- Wada, H., D.A. Cristol, F.M.A. McNabb, and W.A. Hopkins. 2009. Suppressed adrenocortical responses and thyroid hormone levels in birds near a mercury-contaminated river. *Environ. Sci. Technol.* 43:6031-6038.
- Wada, H., D.E. Yates, D.C. Evers, R.J. Taylor, and W.A. Hopkins. 2010. Tissue mercury concentrations and adrenocortical responses of female big brown bats (*Eptesicus fuscus*) near a contaminated river. *Ecotoxicology* 19:1277-1284.
- Webb, M.A.H., G.W. Feist, M.S. Fitzpatrick, E.P. Foster, C.B. Schreck, M. Plumlee, C. Wong, and D.T. Gundersen. 2006. Mercury concentrations in gonad, liver, and muscle of white sturgeon *Acipenser transmontanus* in the Lower Columbia River. *Arch. Environ. Contam. Toxicol.* 50:443-451.
- Wiener, J.G. and D.J. Spry. 1996. Toxicological significance of mercury in freshwater fish. In: W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds.). *Environmental Contaminants in Wildlife*. Lewis Publishers, Boca Raton, FL.
- Wiener, J.G., D.P. Krabbenhoft, G.H. Heinz, and A.M. Scheuhammer. 2003. Ecotoxicology of mercury. In: Hoffman, D.J., B.A. Rattner, G.A. Burton, Jr., and J. Cairns, Jr. (eds.). *Handbook of Ecotoxicology*. Lewis Publishers, Boca Raton, FL.
- Weis, J.S. 2009. Reproductive, developmental and neurobehavioral effects of methylmercury in fishes. *Journal of Environmental Science and Health Part C* 27:212-225.
- Wolfe, M.F., S. Schwarzbach, and R.A. Sulaiman. 1998. Effects of mercury on wildlife: A comprehensive review. *Environ. Tox. & Chem.* 17(2):146-160.
- Yang, D.-Y., Y.-W. Chen, J.M. Gunn, and N. Belzile. 2008. Selenium and mercury in organisms: Interactions and mechanisms. *Environ. Rev.* 16:71-92.

POLYCHLORINATED BIPHENYLS (PCBs) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Polychlorinated biphenyls (PCBs) are hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington.

PCBs are a group of synthetic chemicals, of which there are 209 individual compounds (or congeners) possessing similar chemical structures. This structure consists a biphenyl core with between 1 and 10 chlorine atoms attached. PCBs have the generic formula $C_{12}H_{(10-x)}Cl_x$, where x is an integer from 1 to 10. Congeners differ in their chemical properties and in their ecotoxicological effects (Kannan *et al.* 1989 as cited in Eisler 2000).

II. SOURCES There are no natural sources of PCBs. First manufactured in the United States in 1929 by the Monsanto Chemical Company (Eisler 2000), PCBs mixtures were manufactured and sold under a variety of trade names including Aroclor. PCBs were used in a wide range of commercial applications including use as coolants and lubricants in electrical equipment, in carbonless copy paper, as flame retardants, and for a variety of other purposes (ATSDR 2000). Due to increasing concerns about the human health and environmental impacts of these compounds, U.S. production of PCBs was halted in 1977 (ATSDR 2000).

PCBs' long history of use and their chemical properties have made their presence nearly ubiquitous in the global environment. At Hanford, PCBs were not manufactured onsite, nor were they used as an input to other manufacturing activities. However, PCBs were intentionally released at parts of the site: "PCB waste oils were mixed with non-PCB oils and disposed of on roadways during a period of unregulated disposal. A limited number of roads were identified as likely to have had PCB-containing waste oil applied" (WCH 2007; in particular, see Figure 3 and Table 6 in that document). In addition, "Materials and equipment known to contain PCBs have been used across the Hanford Site ... [for example] sites and equipment such as electrical substations, transformers, capacitors, roofing material, and caulking can be found at various locations across the Hanford Site indiscriminant of operable unit," and potential on-site exposure sources therefore include "releases from past disposal, leaks, or spills" (*ibid.*). The presence of PCBs has been confirmed in nine waste sites listed in the Hanford Waste Information Data System, and in 67 waste sites in the 100 and 300 Areas (*ibid.*). Elevated levels of PCBs were also detected in the bottom sludge of spent nuclear fuel storage basins at 100-N, 100-K, and 100-KE (*ibid.*). PCBs have been detected in the site's underground storage tanks; they may have been used in 200 Area operations, and they have been detected at elevated concentrations in area pond sediments (*ibid.*).

III. ENVIRONMENTAL CHEMISTRY PCBs are highly lipophilic (hydrophobic) pollutants, meaning that they have a strong affinity for organic material such as fat as opposed to water, although there are considerable differences in hydrophobicity among the various congeners.

PCBs are also highly stable under most environmental conditions and can persist in the environment for decades. That said, under anaerobic conditions, some reductive dechlorination of PCBs can occur (e.g., Karcher *et al.* 2007). In addition, some transformation (hydroxylation) of PCBs occurs within living organisms, primarily in the liver (Sipes and Schnellmann 1987 as cited in Eisler 2000), although this may not be beneficial to the organism given the toxicity of the resulting metabolites (Parkinson and Safe 1987 as cited in Eisler 2000). Altogether, however, as a result of environmental degradation/transformation processes, the proportions of individual PCB congeners present in environmental media may differ from those present in the original source material.

FATE AND TRANSPORT

PCBs have a low solubility in water and adhere strongly to organic materials in soils and sediments. The more highly chlorinated congeners sorb more strongly. If present at high levels in groundwater, PCBs will tend to form dense non-aqueous phase liquids (DNAPLs), the fate and transport of which can be complex to model. Leaching of PCBs from soil is slow, particularly for the more highly chlorinated congeners

In surface waters, PCBs also adhere strongly to sediments and are transported downstream; they can also be resuspended and/or deposited. The transportation of sediments is considered to be a dominant PCB transportation mechanism (Bush and Kadlec 1995). Although they have a low solubility in water, PCBs can also associate and disassociate from sediments, potentially contributing to the toxicity of pore waters to benthic biota.

PCBs are volatile, allowing for their evaporation and airborne transportation over very long distances. PCBs have been detected in the snow and sea water of remote polar regions as well as in associated organisms (Eisler 2000). However, “volatilization from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column” (HSDB undated).

BIOACCUMULATION POTENTIAL

Due to their lipophilicity, PCBs are highly bioaccumulative, increasing in concentration at higher trophic levels within the food web. PCBs in higher trophic level organisms may reach levels that are many thousands of times greater than in water (ATSDR 2000). The precise extent of accumulation, however, depends on a variety of factors in addition to trophic position, including feeding strategy, longevity, fat content, sex, and reproductive status (HRTC 2002, Eisler 2000).

As for other lipophilic compounds, the tendency for PCBs to bioaccumulate in food webs is sometimes measured through the calculation of bioconcentration factors (BCFs), bioaccumulation factors (BAFs) and/or biota-sediment accumulation factors

(BSAFs). BCFs represent ratios of the contaminant concentration in the organism divided by the concentration in the water. BCFs in “various fresh water and marine species are generally in the range of 5×10^2 – 4×10^4 for lower chlorinated PCB congeners and about 1×10^3 – 3×10^5 for tetra- to hexa-PCBs [while]... coplanar PCBs and the more highly chlorinated congeners can have aquatic organism BCFs as high as 2×10^6 (ATSDR 2000).

BAFs are defined as a ratio between the concentration in a tissue of the organism of interest and the combined concentration of PCBs in sediment, food, and water (ATSDR 2000). ATSDR (2000) reports that “[t]ypical field- measured BAFs range from 2.1×10^3 to 3.9×10^6 for total PCBs (*ibid.*).

BSAFs represent ratios of the contaminant concentration in the organism, normalized to the percent lipid, divided by the concentration in the organic carbon fraction of the sediment. BSAFs also vary by congener and organism, and can also vary between sites. Some of this variability may be due to differing food web structures, while some may be due to differences in black carbon levels (Cornelissen and Gustafsson 2005).

ACCUMULATION WITHIN TISSUES

The PCBs lipophilicity results in their tendency to be associated with fat within tissues. PCB measurements are commonly reported on both fresh weight and lipid weight bases. The choice of which tissue to analyze to characterize PCB concentrations within organisms depends on the organism and the purpose of the measurement.

In general, for smaller organisms (e.g., insects, soft parts of bivalves, tadpoles), whole bodies may be analyzed, and it may sometimes be necessary to combine multiple organisms into composite samples to garner a sufficient sample mass for analysis.

For fish, if the purpose of the sampling is to characterize potential exposure of piscivorous mammals or birds, the entire fish is typically analyzed. For fish health concerns, fish eggs or reproductive organs may be the focus (e.g., if reproductive endpoints are of interest); fish livers are also frequently used as indicators of PCB exposure.

If the concern is human consumption, measurements are most frequently made of muscle tissue (fillets). To reflect the eating habits of some Native American tribal members, it is also important to evaluate other sample types that reflect traditional methods of preparing fish for consumption.

For birds, eggs and livers are the most frequently sampled, although some have evaluated PCB concentrations in plasma (especially in chicks), muscle, or in adipose tissue (Eisler 2000 – Table 24.11). In reptiles, eggs are also sampled (Eisler 2000 – Table 21.10). Body burdens in amphibians have been evaluated as concentrations in egg masses, larvae, ovaries, and whole adults (e.g., FEL 2002a,b).

In mammals, the liver is probably the most frequently analyzed tissue, although muscle, fat, and brain have also been measured (Eisler 2000 – Table 24.12).

IV. TYPICAL MAJOR EXPOSURE ROUTES

Major routes of exposure for aquatic organisms can include direct absorption from the water column (bioconcentration) as well as the ingestion of contaminated food or prey items (Bush and Kadlek 1995, Hoffman *et al.* 1996). Sediment ingestion can be a major uptake route in benthic invertebrates (Kaag *et al.* 1997).

Maternal transfer represents a significant route of exposure for many young organisms. Birds, reptiles, and fish deposit PCBs into their eggs (e.g., Kelly *et al.* 2008, Cook *et al.* 2003), and mammals can transfer PCBs to their young as developing fetuses (e.g., Grieg *et al.* 2007). Human exposure to PCBs typically occurs through the consumption of PCB-contaminated food or from breathing contaminated air; maternal transfer and transfer through breast milk also occur (ATSDR 2000).

V. ECOTOXICITY

PCBs are harmful to fish and wildlife. While acute mortality at environmentally relevant concentrations is uncommon, PCBs can cause a range of serious sublethal effects. Reproductive effects—including reduced numbers, growth, survival, and development of offspring—are among the most sensitive endpoints for animals exposed to PCBs (EPA 2000). Additional effects include behavioral changes, lesions, immune system dysfunction, neurotoxicity, and hormone imbalances. PCBs are also probable human carcinogens (ATSDR 2000).

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

PCBs have no known beneficial effects or protective properties.

MECHANISM(S) AND LOCI OF TOXICITY

Many of the adverse ecotoxicological effects of PCBs are thought to occur through the ability of certain PCB congeners to bind with the aryl hydrocarbon receptor (AhR). In particular, a subset of the PCB congeners have a chemical structure that is similar to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), the most toxic of all halogenated aromatic hydrocarbons (Van den Berg *et al.* 2006). When one of these “dioxin-like” (or coplanar) PCB molecules enters a cell and binds to the AhR receptor in the cytoplasm, a series of inter-cellular events takes place resulting in the AhR receptor complex becoming translocated to the nucleus and stimulating transcription of certain genes that code for the production of monooxygenase enzymes with aryl hydrocarbon hydroxylase (AHH) activity and ethoxyresorufin-O-deethylase (EROD) activity (Landers and Bunce 1991, ATSDR 2000, Denison and Nagy 2003). Toxic responses occur via subsequent mechanisms that have not been fully elucidated (ATSDR 2000).

In addition to dioxin-like toxic effects, some of PCBs’ effects are mediated through mechanisms that are independent of the Ah receptor (ATSDR 2000), although these mechanisms and associated effects have not been studied to the same extent as the dioxin-like effects. Even so, research has demonstrated that non-coplanar congeners can interfere with intracellular calcium-based signaling pathways, causing cellular, organ-level and organismal effects including neurotoxicity (Fischer *et al.* 1998). Additional effects that have been investigated include estrogenicity, insulin release,

neutrophil (a type of white blood cell) function, and behavioral changes (Fischer *et al.* 1998).

FACTORS AFFECTING TOXICITY

The exact nature of these effects depends on the level and duration of exposure, the mixture of congeners to which the organism is exposed, the sensitivity of the organism to PCBs, lipid content, and the presence of other contaminants (e.g., dioxins and furans) (Eisler 2000).

Biotic factors affecting toxicity include species, age, and developmental stage. With respect to dioxin-like effects, embryos and juveniles are generally the most sensitive life stages (Eisler 2000).

PLANTS

Although a few studies have shown that PCBs in soils can reduce plant growth, these effects occurred at concentrations higher than those observed in the environment; overall, information on the toxicological effects of PCBs on plants is limited (Nagpal 1992).

FISH AND AQUATIC INVERTEBRATES

In general, acute toxicity of PCBs to fish is low; in addition, resistance to bacterial diseases and behavioral responses also appear to be relatively insensitive endpoints (Niimi 1996). Egg viability, fry mortality, hepatic lesion/tumor frequency, and enzyme activity are more sensitive (Niimi 1996, Eisler 2000). Many studies have evaluated these endpoints in a laboratory context, using waterborne or food-based exposures, or even injections into fish eggs (Niimi 1996, Walker and Peterson 1991, Wright and Tillitt 1999).

Of fish, lake trout are the most sensitive for early life stage mortality associated with dioxin-like compounds (Cook *et al.* 2003). Historically, PCBs were associated with reproductive failures of lake trout in Green Bay and Lake Michigan, but since 1980, other factors appear to be substantially more important to the survival of fry in these areas (Stratus 1999). In chinook salmon, PCBs have been associated with reduced hatching success in some studies (Ankley *et al.* 1991 as cited in Eisler 2000) but not others (Williams and Giesy 1992 as cited in Niimi 1996). In Columbia River white sturgeon sampled downstream of the Hanford reach, researchers found negative correlations between a number of health metrics—including condition factor, as well as plasma androgens and gonad size in males—and tissue burdens of contaminants, including (but not limited to) PCBs (Feist *et al.* 2005).

Walleye from Fox River, Wisconsin with average tissue burdens of 4.6 to 8.6 ppm, had significantly increased hepatic lesions and tumors compared to reference area fish (26% versus 7%), although no clear differences between sites were evident for other health metrics evaluated (immunological system effects, biochemical changes, disease, or endocrine system) (Barron *et al.* 1999).

Biochemical responses are generally more sensitive with changes in activity levels occurring at lower concentrations than other endpoints (Niimi 1996), and liver in

particular is the primary target organ for induction of monooxygenase enzymes in fish (Eisler 2000).

PCBs may also affect benthic invertebrates. Fuchsman *et al.* (2006) summarizes acute mortality results from water-based laboratory toxicity testing of marine and freshwater invertebrate species. Some spiked sediment PCB studies have also been performed for selected marine benthic invertebrate species (DiPinto *et al.* 1993, Swartz *et al.* 1998). However, few studies appear to have examined benthic community health in freshwater areas where PCBs are the predominant contaminant of concern. Similarly, few sediment toxicity studies are available that use field-collected materials from PCB-contaminated sites. Also of note, invertebrate AhRs do not strongly bind dioxins and related chemicals (Hahn 2002).

BIRDS

In birds, endpoints commonly associated with sufficient PCB exposure include embroethality, chick mortality, and developmental abnormalities including beak deformities. These effects have been observed in a variety of field studies on fish-eating birds in the Great Lakes especially during the 1950s and 1960s (reviewed in Hoffman *et al.* 1996). PCBs have also been associated with impaired bald eagle reproduction in the Great Lakes region in the period 1986-2000 (Best *et al.* 2010). A study of nesting bald eagles along the Lower Columbia River did not find a statistically significant correlation between contaminant concentrations (including DDE and PCBs) and productivity, but the authors did conclude that at the older territories egg contaminant levels remained high enough to impair reproduction (Buck *et al.* 2005).

PCBs may also cause endocrine disruption in birds, although these effects have been difficult to characterize in the field (Ottinger *et al.* 2009). Field studies also suggest that PCBs can cause reduced retinoid (Vitamin A) levels and histological abnormalities of the thyroid gland, although some laboratory studies have not found a consistent relationship between contaminants and thyroid hormone alterations (reviewed in Rolland 2000).

Avian species vary in their sensitivity to PCBs by over 1000-fold, with domestic chickens being uniquely sensitive (Head *et al.* 2008). Research suggests that part of the chicken's particular sensitivity to dioxin-like compounds is attributable to the presence of two amino acids, Ile324 and Ser380, at key points in the ligand binding domain of the chicken's AhR (Karchner *et al.* 2006). Other species whose AhR genes were sequenced had a Val/Ala genotype and were the least sensitive, whereas those with an Ile/Ala genotype had intermediate sensitivity (Head *et al.* 2008). Of course, variability in avian sensitivity to PCBs is also affected by other factors, as suggested by the observation that American kestrels are more sensitive to PCB 77 than herring gulls even though their AhR genes are identical at the amino acid level (*ibid.*).

MAMMALS

PCBs can affect growth, survival, reproduction, and metabolism of mammals (Eisler 2000). Mink (*Mustela vison*) are among the most sensitive mammals to the effects of

PCBs (Eisler 2000). Laboratory studies have shown that these effects include, but are not limited to, a variety of reproductive impairments such as reduced whelping, reduced kit growth and survival, as well as causing jaw lesions (e.g., Hornshaw *et al.* 1983, Restum *et al.* 1998, Bursian *et al.* 2006). PCBs also may be estrogenic in mammals, although the evidence is not conclusive (Eisler 2000). Otters are also sensitive to PCBs (Smit *et al.* 1996).

Studies have also suggested that coplanar PCBs alter thyroid hormones and vitamin A status in wildlife, decreasing circulating levels of these compounds (Rolland 2000). Vitamin A (retinoids) and thyroid hormones play critical roles in mammal development and throughout life (*ibid.*).

AMPHIBIANS AND REPTILES

The reproduction and survival of amphibians and reptiles can be adversely affected by PCBs at environmentally relevant concentrations. Wood frogs (*Rana sylvatica*) from vernal pools with higher levels of PCB contamination displayed skewed sex ratios and higher rates of abnormalities in metamorph specimens (FEL 2002b). Furthermore, adult female leopard frogs (*Rana pipiens*) collected from PCB-contaminated sites near the Housatonic River, Massachusetts, were not as reproductively fit as external reference specimens: only traces of mature oocytes were identified in these animals, and the proportion of mature oocytes was significantly negatively correlated with ovary tissue total PCB concentrations (FEL 2002a).

Snapping turtles are also affected by PCB exposure. For instance, Hudson River (NY) females pass on PCBs to their eggs, with the juveniles suffering from high rates of mortality eight or more months after hatching (Eisenreich *et al.* 2009).

EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

With respect to dioxin-like toxicity, PCBs are generally considered to act in an additive fashion with dioxins/furans and other contaminants that act through the Ah receptor, and a “toxic equivalency” (TEQ) method has been established to quantify this relationship (e.g., Van Den Berg *et al.* 2005). Because PCBs also act through mechanisms that are independent of the Ah-receptor, however, the accuracy of this approach may be limited (ATSDR 2000). For example, mixtures of planar PCBs and dioxins produced synergism of AHH activity in fish liver at low doses but antagonistic effects at high doses, potentially due to the contributions of nonplanar compounds (Janz at Metcalfe 1991b as cited in Eisler 2000).

DATA GAPS

Relatively little information is available about potential impacts of PCBs to plants. Among animals, species differ in their sensitivities to PCBs, and while this seems to be at least partly attributable to the Ah receptor genotype, additional unidentified factors also likely influence this parameter. The degree to which PCBs affect benthic invertebrates under field conditions is unclear.

The most dramatic effects of PCBs on fish under field conditions are those associated with lake trout in the Great Lakes. These appear to have attenuated over time with

reductions in concentrations of PCBs and other contaminants that act through the Ah receptor; whether more subtle effects persist in these or other populations of wild fish, affecting their fitness, and whether these effects are attributable to PCBs as distinct from other contaminants present at sites, is not always clear.

Among mammals, with the exception of mink, relatively little information is available about the sensitivity to PCBs of most species likely to be present in the Hanford area.

Finally, non-dioxin like effects of PCBs are less well understood than dioxin-like effects, and the mechanisms through which these effects occur are also not clearly established. In part because of these factors, the use of TEQs to characterize dioxin-like toxicity has limitations.

- VI. REFERENCES** Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for polychlorinated biphenyls (PCBs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.
- Barron, M.C., M. Anderson, D. Beltman, T. Podrabsky, W. Walsh, D. Cacela, and J. Lipton. 1999. Association between PCBs, liver lesions and biomarker responses in adult walleye (*Stizostedium vitreum vitreum*) collected from Green Bay, Wisconsin. April 13. Available at <<http://www.fws.gov/midwest/FoxRiverNRDA/documents/walleye.pdf>>.
- Bush, B. and M.J. Kadlec. 1995. Dynamics of PCBs in the aquatic environment. Great Lakes Research Review 1(2):24-30.
- Bursian, S.J., C. Sharma, R.J. Aulerich, B. Yamini, R.R. Mitchel, K.J. Beckett, C.E. Orazio, D. Moore, S. Svirsky, and D.E. Tillitt. 2006. Dietary exposure of mink (*Mustela vison*) to fish from the Housatonic River, Berkshire County, Massachusetts, USA: Effects on organ weights and histology and hepatic concentrations of polychlorinated biphenyls and 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic equivalence. Environmental Toxicology and Chemistry 25 (6):1541–1550.
- Cook, P.M., J.A. Robbins, D.D. Endicott, K.B. Lodge, P.D. Guiney, M.K. Walker, E.W. Zabel, and R.E. Peterson. 2003. Effects of aryl hydrocarbon receptor-mediated early life stage toxicity on lake trout populations in Lake Ontario during the 20th century. Environ. Sci. Technol 37(17):3864-3877.
- Cornelissen, G. and O. Gustafsson. 2005. Prediction of large variation in biota to sediment accumulation factors due to concentration-dependent black carbon adsorption of planar hydrophobic organic compounds. Environmental Toxicology & Chemistry 24(3):495-498.
- Denison, M.S. and S.R. Nagy. 2003. Activation of the aryl hydrocarbon receptor by structurally diverse exogenous and endogenous chemicals. Ann. Rev. Pharmacol. Toxicol. 43:309-334.

- Di Pinto, L.M., B.C. Coull, and G.T. Chandler. 1993. Lethal and sublethal effects of the sediment-associated PCG Aroclor® 1254 on a meiobenthic copepod. *Environmental Toxicology and Chemistry* 12:1909-1918.
- Eisenreich, K.M., S.M. Kelly, and C.L. Rowe. 2009. Latent mortality of juvenile snapping turtles from the Upper Hudson River, New York, exposed maternally and via diet to polychlorinated biphenyls (PCBs). *Environ. Sci. Technol* 43(15):652-6057.
- Eisler, Ronald. 2000. *Handbook of Chemical Risk Assessment. Health Hazards to Humans, Plants, and Animals. Volume 2: Organics.* CRC Press, Boca Raton, Florida.
- Feist, G.W., M.A. Webb, D.T. Gundersen, E.P. Foster, C.B. Schreck, A.G. Maule, and M.S. Fitzpatrick. 2005. Evidence of detrimental effects of environmental contaminants on growth and reproductive physiology of white sturgeon in impounded areas of the Columbia River. *Environmental Health Perspectives* 113(12):1675-1682.
- Fischer, L.J., R.F. Seegal, P.E. Ganey, I.N. Pessah, and P.R.S. Kodavanti. 1998. Symposium overview: Toxicity of non-coplanar PCBs. *Toxicological Sciences* 41:49-61.
- Fort Environmental Laboratories, Inc. (FEL). 2002a. Final Report – Frog Reproduction and Development Study. 2000 *Rana pipiens* Reproduction and Development Study. Prepared for Weston Solutions, Inc.
- Fort Environmental Laboratories, Inc. (FEL). 2002b. Final Report – Frog Reproduction and Development Study. 2000 *Rana sylvatica* Vernal Pool Study. Prepared for Weston Solutions, Inc.
- Grieg, D.J., G.M. Ylitalo, A. Hall, D.A. Fauquier, and F.M.D. Gulland. 2007. Transplacental transfer of organochlorines in California sea lions (*Zalophus californianus*). *Environmental Toxicology & Chemistry* 26(1):37-44.
- Head, J.A., M.E. Hahn, and S.W. Kennedy. 2008. Key amino acids in the aryl hydrocarbon receptor predict dioxin sensitivity in avian species. *Environ. Sci. Technol* 42(19):7535-7541.
- Hoffman, D.J., C.P. Rice, and T.J. Kubiak. 1996. PCBs and dioxins in birds. In: W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds.). *Environmental Contaminants in Wildlife – Interpreting Tissue Concentrations.* SETAC, Special Publications Series, CRC, Boca Raton, FL, USA, pp. 165-207.
- Hornshaw, T.C., R.J. Aulerich, and H.E. Johnson. 1983. Feeding Great Lakes fish to mink: Effects on mink and accumulation and elimination of PCBs by mink. *J. Toxicol. Environ. Health* 11(4):933-946.
- Hazardous Substances Data Bank (HSDB). Undated. Polychlorinated biphenyls. National Institutes of Health, U.S. Department of Health and Human Services, Bethesda, MD. Available at <http://toxnet.nlm.nih.gov/> (accessed December 14, 2010).

- Hudson River Trustee Council (HRTC). 2002. Hudson River Natural Resource Damage Assessment Plan. September.
- Kaag, N.H.B.M., E.M. Foekema, M.C.Th. Scholten, and N.M. van Straalen. 1997. Comparison of contaminant accumulation in three species of marine invertebrates with different feeding habits. *Environmental Toxicology and Chemistry* 16(5): 837–842.
- Karcher, S.C., J.M. VanBriesen, and M.J. Small. 2007. Numerical method to elucidate likely target positions of chlorine removal in anaerobic sediments undergoing polychlorinated biphenyl dechlorination. *Journal of Environmental Engineering* 133(3):278-286.
- Karchner, S. I., D.G. Franks, S.W. Kennedy, and M.E. Hahn. 2006. The molecular basis for differential dioxin sensitivity in birds: Role of the aryl hydrocarbon receptor. *Proc. Natl. Acad. Sci. U.S.A.* 103:6252–6257.
- Kelly, S.M., K.M. Eisenreich, J.E. Baker, and C.L. Rowe. 2008. Accumulation and maternal transfer of polychlorinated biphenyls in snapping turtles of the Upper Hudson River, New York, USA. *Environ. Contam. Toxicol.* 27(12):2565-2574.
- Landers, J.P. and N.J. Bunce. 1991. Review article: The Ah receptor and mechanism of dioxin toxicity. *Biochem J.* 276:273-287.
- Nagpal, N.K. 1992. Water quality criteria for polychlorinated biphenyls (PCBs). Technical Appendix. Ministry of Environment, Lands and Parks, Province of British Columbia. Available at <<http://www.env.gov.bc.ca/wat/wq/BCguidelines/pcbs/index.html>>.
- Niimi, A.J. 1996. PCBs in aquatic organisms. In: W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds.). *Environmental Contaminants in Wildlife – Interpreting Tissue Concentrations*. SETAC, Special Publications Series, CRC, Boca Raton, FL, USA, pp. 117-152.
- Ottinger, M.A., E.T. Lavoie, M. Abdelnabi, M.J. Quinn, A. Marcell, and K. Dean. 2009. An overview of dioxin-like compounds, PCB, and pesticide exposures associated with sexual differentiation of neuroendocrine systems, fluctuating asymmetry, and behavioral effects in birds. *J. Environ. Science and Health Part C* 27(4):286-300.
- Restum, J.C., S.J. Bursian, J.P. Giesy, J.A. Render, W.G. Helferich, E.B. Shipp, and D.A. Verbrugge. 1998. Multigenerational study of the effects of consumption of PCB-contaminated carp from Saginaw Bay, Lake Huron, on mink. 1. Effects on mink reproduction, kit growth, and survival, and selected biological parameters. *J. Toxicol. Environ. Health* 54():343-375.
- Smit M.D., P.E.G. Leonards, A.J. Murk, A.W.J.J. de Jongh, and B. van Hattum. 1996. Development of otter-based quality objectives for PCBs. Amsterdam: Institute for Environmental Studies, Vrije Universiteit. 129 pp.
- Stratus Consulting, Inc. 1999. Injuries to fishery resources, Lower Fox River/Green Bay natural resource damage assessment. Final report. Prepared for the U.S. Fish

and Wildlife Service, U.S. Department of the Interior, and U.S. Department of Justice. November 8.

Swartz, R.C., P.F. Kemp, D.W. Schults, and J.O. Lamberson. 1988. Effects of mixtures of sediment contaminants on the marine infaunal amphipod, *Rhepoxynius abronius*. *Environmental Toxicology and Chemistry* 7:1013-1020.

U.S. Environmental Protection Agency (EPA). 2000. Phase 2 Report. Further Site Characterization and Analysis. Volume 2E – Revised Baseline Ecological Risk Assessment, Hudson River PCBs Reassessment. November.

Walker, M.K. and R.E. Peterson. 1991. Potencies of polychlorinated dibenzo-p-dioxin, dibenzofuran, and biphenyl congeners, relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin, for producing early life stage mortality in rainbow trout (*Oncorhynchus mykiss*). *Aquat Toxicol* 21:219–38.

Washington Closure Hanford (WCH). 2007. Polychlorinated Biphenyl Presence in the Columbia River Corridor. River Corridor Closure Contract. Prepared for the U.S. Department of Energy, Richland Operations Office, Office of Assistant Manager for River Corridor. September.

Wright P.J. and D.E. Tillitt. 1999. Embryotoxicity of Great Lakes lake trout extracts to developing rainbow trout, *Aquatic Toxicol* 47(2):77-92.

van den Berg, M., L.S. Birnbaum, M. Denison, M. De Vito, W. Farland, M. Feeley, H. Fielder, H. Hakansson, A. Hanberg, L. Haws, M. Rose, S. Safe, D. Schrenk, C. Tohyama, A. Tritscher, J. Tuomisto, M. Tysklind, N. Walker, and R.E. Peterson. 2006. The 2005 World Health Organization re-evaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. *Toxicological Sciences* 93:223-241.

PLUTONIUM (Pu)

ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Plutonium (Pu) is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Plutonium is primarily a man-made radioactive element, which consists of 20 isotopes (Pu-228 to Pu-277), the most important of which from an environmental toxicological perspective are Pu-238, Pu-239, Pu-240, and Pu-241. The primary radiological properties of the most common isotopes of plutonium are shown in Exhibit 1.

EXHIBIT 1 PLUTONIUM ISOTOPES AND HALF-LIVES (ATSDR 2008)*

| RADIOISOTOPE AND CAS NUMBER | DECAY MODE(S)/ENERGY (MEV) | RADIOACTIVE HALF LIFE (YEARS)** | INITIAL DECAY PRODUCT(S) | SPECIFIC ACTIVITY (CI/G) |
|---|--|---------------------------------|--------------------------|--------------------------|
| Pu-238 13981-16-3 | Alpha/5.559 Spontaneous fission | 88 4.75x10 ¹⁰ | U-234 | 17 |
| Pu-239 15117-48-3 | Alpha/5.244 Spontaneous fission | 21,110 8x10 ¹⁵ | U-235 | 0.063 |
| Pu-240 14119-33-6 | Alpha/5.255 Spontaneous fission | 6,560 1.14x10 ¹¹ | U-236 | 0.23 |
| Pu-241 14119-32-5 | Beta/0.02 (99+%) alpha/5.138 (0.002%) | 14.3 | Am-241 U-237 | 100 |
| Notes: * Originally from Baum <i>et al.</i> 2002, ChemIDplus 2009, Clark et al 2006; Lide 2008 * Two half lives are provided because each isotope has two modes of decay, alpha decay and spontaneous fission, each with its own half life. Note that spontaneous fission is a very rare event relative to alpha decay. | | | | |

II. SOURCES As ATSDR (2008) states:

“Plutonium is primarily a human-made radioactive element of the actinide series and was the first human-made element to be synthesized in weighable amounts. Plutonium was first synthesized by the bombardment of uranium with deuterons (²H) by Seaborg and co-workers in 1940. Although 20 isotopes of plutonium (²²⁸⁻²⁴⁷Pu) have been identified, the alpha-emitting ²³⁸Pu and ²³⁹Pu isotopes are the ones most commonly encountered and widely studied for potential adverse health effects. The main sources

of plutonium in the environment are releases from research facilities, nuclear weapons testing, waste disposal, nuclear weapons production facilities, and accidents. Atmospheric testing of nuclear weapons, which ended in 1980, is the source of most of the plutonium in the environment worldwide, which released approximately 10,000 kilograms of plutonium. Plutonium released to the atmosphere reaches the earth's surface through wet and dry deposition to the soil and surface water. Once in these media, soluble plutonium can sorb to soil and sediment particles or bioaccumulate in terrestrial and aquatic food chains.”

In addition, small quantities of plutonium are ubiquitous in the environment from global fallout from the reentry and burn up of satellites that used Pu-238 as a heat source in nuclear batteries used to produce electricity in devices, such as unmanned spacecraft and interplanetary probes (ATSDR 2008).

Plutonium also occurs naturally in the environment as a result of the interaction of neutrons, primarily from spontaneous fission of uranium, with U-238 (ATSDR 2008). Trace amounts of naturally occurring Pu-239 are also found in naturally occurring uranium ores, although in such small amounts that extraction is not practical. Small amounts of ^{244}Pu also exist in nature from remnants of primordial stellar nucleosynthesis and from “natural” reactors such as the Oklo natural reactor in the African nation of Gabon, which existed about 2 billion years ago (DOE 2005a as cited in ATSDR 2008).

Taylor (2001) provides a detailed description of the natural processes that are responsible for naturally occurring plutonium in the environment, and estimates a concentration of about 100 amole/kg of the earth’s crust, where “a” stands for atto, which is a prefix meaning 10^{-18} , or about 50 $\mu\text{Bq/kg}$ of earth’s crust. Taylor (2001) also estimates that the rate of production of Pu-239 in the earth’s crust through natural processes (i.e., interaction of neutrons with U-238 to produce Np-239, which produces Pu-239 through beta decay) is about 28 kg of Pu-239 per year. Taylor (2001) also provides estimates of the concentration of the various isotopes of plutonium in the earth’s crust from weapons testing, SNAP-9A satellite fallout, and releases from nuclear industry, along with the chronic daily inhalation and ingestion rate of plutonium by humans in Japan, New York, and Poland at different time periods from 1962 to 1986.

Essien (1994) provides a tabulation of the concentration of plutonium in the environment primarily from global fallout. He reports bimonthly average concentrations of Pu-239 in rain and snow samples in Fayetteville, Arkansas between March 1980 and April 1983 ranging from 2.06 to 18.20 fCi/L. (Note: “f” refers to “femto,” which is 10^{-15} of a curie.) Table 6-6 of Eisenbud and Gesell (1997) report the range of U-238 in different types of soil and rock, where the reported values range from 7 to 60 Bq/kg (or 0.2 to 1.6 pCi/g, where “p” stands for pico or 10^{-12}).

Man-made sources of plutonium are also present in the environment at many sites throughout the United States that were involved in weapons production. Hanford served as a plutonium production facility starting in 1945. Relatively large quantities of plutonium and other transuranic elements (relative to the ubiquitous levels in the

environment from natural and other anthropogenic sources) were discharged to the subsurface, primarily in the 200 Area (the Hanford Central Plateau) associated primarily with activities at the Z-Plant (Plutonium Finishing Plant) complex and unlined ditches and ponds, high level waste tanks, and landfills. In the past, plutonium and americium (Am) migrated deep into the subsurface at certain locations at Hanford, although plutonium and other transuranics are not currently being detected in significant concentrations in any associated groundwaters (Felmy *et al.* 2010).

Plutonium-238, plutonium-239/240, plutonium-241, and other isotopes were released into the air from the 100, 200, 300, 400, and 600 Areas of the Hanford Site. The Z plant (in the 200 West Area) also discharged plutonium in its gaseous effluents (ORAUT-TKBS-0006-4, 2010), which resulted in plutonium deposited in soil on and in the vicinity of the Hanford Reservation.

The annual Hanford Site Environmental Reports provide information on the quantities of plutonium released to the Columbia River each year.¹¹² For example, for calendar year 2009, Poston *et al.* (2010) provides information regarding waterborne releases of plutonium isotopes and measurements taken in various aquatic environmental media. Poston *et al.* (2010) estimate that approximately 3.6e-6 curies (Ci) of Pu-238 and 3.0e-5 Ci of Pu-239/240 were contained in liquid effluent discharged in the 100 Areas in 2009. Generally, this effluent consists of secondary cooling water discharged from the 100-K Area to the Columbia River via the NPDES-permitted 1908-K Outfall.

III. ENVIRONMENTAL CHEMISTRY

As a preface to this section, it is appropriate to point out that the subject of environmental chemistry is vast, and can include both the physical and chemical behavior of plutonium in the environment and its transport through aquatic and terrestrial ecosystems. Many compendia have been published that address this broad subject. Till and Meyer (1983) is one of the earliest compilations and descriptions of models and parameters addressing the environmental behavior of radionuclides, including plutonium. A recent addition to the literature on this subject is IAEA (2010). In addition, the U.S. Department of Energy Richland Operations Office (DOE-RL) periodically updates the System Assessment Capability (SAC), which is an environmental assessment tool that can be used to assist in the analysis of the movement of contaminants from waste sites at the Hanford Site into and through the vadose zone, groundwater, atmosphere, and Columbia River. These reports can serve as a convenient starting point for compiling generic and site-specific models and modeling parameters for assessing the behavior of plutonium in the environment at Hanford.

Plutonium can exist in various oxidation states, including valence III, IV, V, VI, and VII (although, as discussed below, once exposed to the environment, plutonium oxidizes rapidly). As indicated in Exhibit 1, certain plutonium isotopes have long half-lives and will not rapidly decay. In addition, as discussed in greater detail below,

¹¹² The quantity of plutonium “released” to the Columbia River is based on concentrations of plutonium in liquid effluents. The extent to which plutonium might be entering the Columbia River from groundwater discharges is a subject of active research and concern.

plutonium generally binds strongly to soil and sediment, but there are physical, chemical, and biological processes that can increase its mobility in soil. The conventional wisdom is that natural attenuation of plutonium is generally slow, but there are exceptions (see Smith and Amonette 2006).

FATE AND TRANSPORT

Once released to the environment, plutonium reacts readily with oxygen to form plutonium oxide, which is highly insoluble, binds strongly to soil and sediment, and generally does not migrate rapidly in the environment. However, as discussed in ATSDR (2008), the chemistry of plutonium is complex, and its mobility in the environment will depend on its oxidation state, which is affected by soil and water chemistry and microbial action. Its mobility also depends on the presence of complexing agents, such as chelating agents, colloids, and organic material. However, in general, plutonium is not very mobile in soil and sediment. Smith and Amonette (2006) describe experience at Idaho National Laboratories and at the Nevada Test Site where plutonium moved in the vadose zone much more quickly than anticipated. They also cite studies at Hanford where many radionuclides moved relatively quickly through the 90 meter thick unsaturated zone below the Hanford waste tanks. The reasons cited for this unexpected behavior include (1) adsorption of plutonium onto colloids¹¹³ that remain suspended in soil pore water and move at the rate that the water moves through the vadose zone (as opposed to binding to the soil in the vadose zone), (2) pH and oxidative state affect the binding capability of plutonium to soil, (3) the presence of organic and inorganic complexing agents, including microbial activity and dissolved carbonates, and (4) chelating agents, such as EDTA.

The fate of radionuclides, including plutonium in soils and in the subsurface, is understood in a general sense, and models are available to predict the behavior of plutonium in the environment. Fundamental to the movement of radionuclides, including plutonium, in soil and the subsurface are the partition coefficients, or K_d values. Partition coefficients describe the strength of plutonium's binding to soil and sediments.

Sheppard and Thibault (1990) compiled and reviewed the literature on the partition coefficients of numerous elements, including plutonium, where the partition coefficient is expressed as follows:

$$K_d = C_s/C_1$$

where:

K_d = the partition coefficient of a given element in soil,

C_s = the average concentration of a given element in soil in contact with the water for sufficient time to achieve equilibrium, and

C_1 = the average concentration of the element in water.

¹¹³ Zhao (1997) presents a detailed description of actinide colloid chemistry.

Depending on the characteristics of the soil, Sheppard and Thibault (1990) reported K_d values for plutonium ranging from 11 to 300,000, with central estimates of 550 for sand, 1,200 for loam, 5,100 for clay, and 1,900 for organic soils. Included in their reported values of K_d , are values by Baes and Sharp (1983) of a best estimate of 1,800 and range of 11 to 300,000 for agricultural soils and by Coughtrey (1985) of a best estimate of 5,000 and a range of 18 to 10,000. The implications of these investigations are that, although the K_d values for plutonium at a site can be highly variable, the central estimates are generally quite high, and leaching and migration of plutonium out of soil and sediment is expected to be extremely slow. The U.S. Department of Energy (DOE) Biota Dose Assessment Committee (BDAC) database contains 15 K_d entries for Pu for sands, clays, and loams that range from 10 to 330,000 for clays and 18 to 16,000 for sands.

Notwithstanding these general characteristics of plutonium, Felmy *et al.* (2010) reports that the chemical form of transuranics, including plutonium, in the deep subsurface sediments and the past mechanism of vertical migration at the Hanford reservation remain largely unknown. Initial studies performed as part of research performed by Felmy *et al.* (2010) indicate that the chemical form of Pu can vary from disposal site to disposal site, depending upon the waste type, and the chemical form can also differ between surface sediments and deep subsurface sediments at the same site. The implications of these investigations are that there is uncertainty regarding the chemical forms of plutonium in the subsurface in the vicinity of Hanford and also uncertainty in its mobility.

Smith and Amonette (2006) summarize the literature describing the limitations of K_d values, explaining that any measured K_d reflects only the very specific conditions under which those measurements were made. It is for this reason that the reported range of K_d values is so large.

A review of transuranic contamination, including plutonium, in sediment and groundwater at Hanford is provided by Cantrell (2009). The review summarizes the types, quantities, and sources of liquid waste containing plutonium and other transuranics that was disposed of at various locations at the Hanford site. They emphasize that, notwithstanding the large quantities of plutonium that have been disposed of and entered the near surface vadose zone, only miniscule amounts have entered the groundwater. Cantrell (2009) explains that the reasons for the slow movement of plutonium in the vadose zone at Hanford are the typical oxidizing and neutral to slightly alkaline pH conditions in the vadose zone. However, he also explains that transuranics disposed of with acidic waste moved much more quickly in the vadose zone (e.g., 36 meters below ground surface).

These investigations reveal that the chemistry of plutonium in the subsurface at Hanford is complex, and it is difficult to draw simple conclusions regarding the rate at which plutonium may be moving through the vadose zone at different location at the site and at different time periods.

BIOACCUMULATION POTENTIAL

ATSDR (2008) reports “Plutonium has been shown to bioconcentrate in aquatic organisms at the lower end of the food chain. However, data do not indicate that plutonium is bioconcentrated in plants, higher aquatic organisms, or animals. In addition, there is no indication that plutonium is biomagnified in terrestrial or aquatic food chains.” Hinton and Pinder (2000) studied Pu in the environment in the vicinity of the Savannah River Plant. These authors found that: (1) regardless of the crop type, plutonium contamination of plants was dominated by retention of plutonium bearing particles on plant surfaces from direct airborne deposition and resuspension, rather than root uptake, and, as such, it is the surface characteristics of the plant that determined the degree of contamination. Furthermore, over 99% of the plutonium inventory in the aquatic ecosystem was in the sediment. Hinton and Pinder (2000) also generally describe the factors that affected the cycling of plutonium in aquatic ecosystems.

Aquatic Food Webs

Notwithstanding the relatively high K_{ds} reported for plutonium and the relatively low bioaccumulation factors for plutonium that have generally been observed in higher organisms, even small amounts of plutonium absorbed by higher organisms following intake by ingestion are of concern. In addition plutonium adsorbed to the cell membrane of microorganisms, the surface of fish eggs, or the root hairs of higher plants are also of potential concern. As discussed below, some studies have observed high concentrations of plutonium in aquatic organisms relative to the concentration of plutonium in the water in which they reside. Hence, it is difficult to make broad generalizations regarding the transport and re-concentration of plutonium in the aquatic environment.

A convenient method for relating the concentration of a given radionuclide in water to that in aquatic organisms is the use of empirically determined bioaccumulation factors (BFs). The radioecological literature is filled with estimates of BFs for plutonium in fish and other aquatic organisms, expressed as follows:

$$BF = C_{\text{biota}}/C_{\text{water}}$$

where:

BF is the bioaccumulation factor

C_{water} is the measured or estimated average concentration of a given radionuclide in water, and

C_{biota} is the measured or estimated average concentration of a given radionuclide in aquatic organisms living in the water and achieving a quasi-steady state equilibrium with the radionuclide in the water.

BFs are useful because actual measurements of the concentration of a given radionuclide in specific aquatic organisms at a site might be lacking. Without such information, it is difficult to estimate the potential ecological or public health damage due to the presence of the radionuclide in the environment. One method that can be

used to estimate these concentrations in aquatic biota is by multiplying the observed or estimated concentration of a given radionuclide in water by an appropriate BF for the aquatic biota of concern.

NCRP (1996) recommends a default BF for plutonium for freshwater fish of 30. However, caution must be used when selecting BFs because many site-specific factors affect the BF, including the chemical form of the plutonium, water chemistry, the composition of the food chain, the role of sediment in contributing to or limiting the uptake of plutonium by the organism, and uncertainty regarding the degree to which plutonium concentrations have reached equilibrium in organisms and the environment. For example, Cummins (1994) summarizes plutonium BFs for fresh water organisms as observed at the Savannah River Site (SRS) and as reported in the literature as ranging from 0.4 to 840,000. The BF for freshwater fish muscle ranged from 0.4 to 5600, the range for macroinvertebrate larvae ranged from 587 to 840,000, and the range for macrophytes ranged from 6,600 to 100,000.

Emery and Klopfer (1974) present a Hanford-specific study of the ecological behavior of plutonium and americium in a freshwater system at Hanford. They studied a shallow 14 acre pond that received waste waters from the waste trenches that were used for the disposal of waste water containing plutonium and americium. The pond contained macrophytes, algae, benthic invertebrates, and goldfish. The report explains that the majority of the plutonium and americium was in the sediment (390 pCi/g dry weight of Pu in the sediment and 0.01 pCi/L of Pu in the water). The report goes on to describes the levels of Pu and Am found in the various organisms, providing some site-specific information regarding the behavior of Pu in one aquatic ecosystem at the site.

Terrestrial Food Webs

Fundamental to understanding the movement of radionuclides from soil to biota in terrestrial ecosystems are the environmental transfer coefficients, including soil-to-plant transfer factors and plant-to-animal transfer factors. These transfer factors are used to predict the concentrations of radionuclides in plants given the radionuclide concentrations in soil, and the concentrations of radionuclides in animals given the concentrations of radionuclides in the diet of the animals of concern. (Note that transfer factors are based on an assumption of equilibrium concentration in soil, plants, and animals.)

For plants, these transfer factors are also referred to as concentration ratios (CRs) and can be expressed as the concentration of a given radionuclide in a plant (dry or wet weight) per unit concentration of a given radionuclide in soil (dry weight). Care must be used when interpreting CRs reported in the literature since some CRs include only the radionuclides that have been absorbed into the organism, while other CRs also include radionuclide contaminants that have adsorbed onto the surface of the organisms. This distinction is important because, whether one is concerned with chemical or radiological toxicity, the potential for injury is generally greater if the plutonium is absorbed within the cell, where it can directly interfere with the cell's biochemistry. As shown below, the differences between CRs for plutonium that do

and do not include surface contamination are large because plutonium is generally not very biologically mobile (i.e., it does not readily cross biological membranes).

ANRCP (1998) presents a literature review of chromium, uranium, and plutonium in plant systems. Some of the key points made in that review are: (1) up to 1000-fold increases in tumbleweed uptake of plutonium in the presence of diethylenetriamine-pentaacetate added to soil,¹¹⁴ (2) soil-to-plant transfer factors have an enormous range (from 10^{-9} to 10^{-3}), (3) the Pu concentration in roots exceeded those in shoots by factors of 3 to 8, and (4) direct deposition by dust is an important mechanism by which plants can be contaminated by plutonium.

A large body of literature has been compiled on the transfer factors for many elements. For example, Sheppard and Evenden (1997) state that over 7,000 CRs have been compiled for 22 elements. Since the publication of this article, the CR database has expanded considerably. More recent compendia on environmental transfer factors are provided in IAEA (1994), IAEA (2010), and numerous articles in the *Journal of Health Physics* and many other publications dealing with ecology and radioecology.

Exhibit 2 shows data from Peterson (1983), which provides a number of dry weight soil-to-plant CRs for Pu-239. Of note, these values represent a compendium published in 1983. The numbers of reported values of CRs have increased substantially since then.

Most of the published CR values for plutonium are for pasture and food crops, and the dry weight CRs are consistently a small fraction of 1.0 (e.g., see the review by Napier, et al. 2007, where the reported dry weight CR ratios for all plants ranged from 0.00000048 to 0.39). Complicating the interpretation of CR ratios for plants is the contribution to plant contamination by raindrop splash, where soil particles with relatively high concentrations of plutonium are splashed onto the surface of plants (Dreicer 1983).

Some information is available on plutonium concentration ratios for local Hanford species. Price (1972) found only “slight” uptake of plutonium by tumbleweed and cheatgrass (concentration ratios of 46×10^{-6} and 17×10^{-6} , respectively). Similarly, Price (1973) found plant/soil concentration ratios for tumbleweed and cheatgrass to be between 14×10^{-6} and 310×10^{-6} , depending on which acid was also added to the soil.

¹¹⁴ Ballou, et al. (1996) reports that many chelating agents, including EDTA, were found in the waste tanks at Hanford.

EXHIBIT 2 PLUTONIUM CONCENTRATION RATIOS (CRS) IN PLANTS (FROM PETERSON 1983)

| TYPE OF PLANT | NUMBER OF MEASUREMENTS | PU-239 DRY WEIGHT CR* |
|--|------------------------|-----------------------|
| Forage: Legumes: alfalfa, clover, sorghum | 24 | 2.3e-4 |
| Forage: Legumes: alfalfa, clover, sorghum (includes external contamination) | 6 | 0.066 |
| Forage: Grasses | 35 | 9.2e-5 |
| Forage: Grasses (includes external contamination) | 9 | 0.014 |
| Grains (kernels): wheat, oat, barley | 20 | 1.5e-6 |
| Grains (kernels): wheat, oat, barley (includes external contamination) | 8 | 0.018 |
| Grains: corn, rice (dry) (includes surface contamination) | 4 | 0.014 |
| Leafy: cabbage, lettuce, spinach | 3 | 1.75e-4 |
| Leafy: cabbage, lettuce, spinach (includes surface contamination) | 1 | 1e-3 |
| Root: radish, carrot, turnip, beet | 5 | 3.7e-4 |
| Root: radish, carrot, turnip, beet (includes surface contamination) | 1 | 4.6e-3 |
| Root: potato, sweet potato | 1 | 1.4e-3 |
| Root: potato, sweet potato (includes surface contamination) | 5 | 1e-3 |
| Legumes: bean, pea, soybean | 19 | 8.1e-6 |
| Legumes: bean, pea, soybean (includes surface contamination) | 5 | 1.0e-3 |
| Fruits: tomatoes, cucumbers, apples etc. | 2 | 1.0e-4 |
| * pCi/kg dry weight plant of the edible part of the plant per pCi/kg dry weight soil. The values represent the 84th percentile confidence level on the mean. | | |

Sheppard and Evenden (1997) investigated the uncertainty and variability of soil-to-plant transfer factors and found that, in general, for a given element, the 95th percentile confidence interval for transfer factors encompasses a range of 1,300-fold. In addition, they report that, for a given crop of interest at a given site for a given radionuclide, the uncertainty/variability is much smaller; i.e., the 95% confidence interval is about a factor of 5. Peterson (1983) explains that a significant portion of the variability in the transfer factors is due to the variability in K_d , i.e., if the K_d is high, the amount of the radionuclide dissolved in water and available for root uptake is small, as is often (but not always) the case for plutonium. Peterson (1983) also reports that, if chelating agents are present in the soil, the transfer factors can increase dramatically (i.e., up to a factor of over 800 has been observed). The transfer factor also depends on the depth of the contamination and the root depth. If the contamination is not located in the root zone, uptake is minimized.

Published plutonium transfer factors for animals are quite limited and emphasize beef, since the primary concern in the literature is to develop methods to assess impacts on humans. These transfer factors are expressed in units of radionuclide concentration in beef per unit intake of the radionuclide ingested by the animal (i.e., pCi/kg of beef per pCi/day ingested, which reduces to units of days/kg). For beef, Ng *et al.* (1979) reports a value of $1e-6$ d/kg for plutonium. Till and Moore (1988) report a value of $1.4e-5$ d/kg. DOE (2002) and (2004) provide methods to predict uptake of radionuclides by animals other than beef cows. IAEA (1994 and 2010) also provide transfer factors for many radionuclides and elements, including plutonium.

The most important point of this discussion is that there is considerable variability and uncertainty in the partition and transfer factors of plutonium in aquatic and terrestrial ecosystems between sites.

ACCUMULATION WITHIN TISSUES

The primary concern associated with exposure of higher organisms to plutonium is inhalation of airborne particles of plutonium oxide bound to respirable aerosols and deposition in the lungs, where it has a generally very slow clearance rate. In addition, as discussed above, as a general rule, plutonium does not readily cross biological membranes and enter cells or systemic circulation. However, when it does, it is transported primarily to bone and the liver, where it has a relatively low clearance rate; i.e., a half life on the order of years (Taylor 1989). A complete description of the biokinetics of plutonium in humans is provided in the publications of the International Commission on Radiation Protection, in particular ICRP (1995 and 1996).

The biochemical mechanism for the transport of plutonium that crosses biological membranes is believed to be transport by transferrin (Turner and Taylor 1968), a protein in blood plasma and cytoplasm that binds the ferric ion, and transfers it for utilization (Stryer 1988). Welch (1992) summarizes the literature that confirms that plutonium binds to transferrin and follows a similar metabolic pathway as iron in mammalian systems, and eventually deposited on bone surfaces or incorporated into the iron-storage protein ferritin.

In fish, plutonium is concentrated in bones rather than in muscle tissues (NCRP 1984 as cited in ATSDR 2008), while in lobsters, plutonium accumulated primarily in the gills and exoskeleton (Swift 1992 as cited in ATSDR 2008). In birds and in mammals in general, it can be assumed that plutonium also tends to accumulate in bone and the liver.

IV. TYPICAL MAJOR EXPOSURE ROUTES

Plutonium in water and sediment is accumulated to varying degrees by aquatic organisms through direct adsorption and absorption for the lower trophic levels and through ingestion of food, water, and sediment by organisms higher up the food chain. As discussed above, in aquatic organisms, accumulation is expressed in terms of bioaccumulation factors. In plants, accumulation is expressed in terms of concentration ratios, also referred to as soil-to-plant transfer factors. In mammals, accumulation in muscle is expressed in terms of food-to-meat transfer factors (see also IAEA 1994).

An attempt to build models that can be used to quantitatively analyze the accumulation, fate, and effects of a large number of radionuclides, including plutonium, in many organisms other than man is provided in DOE (2002) and (2004) and in ICRP (2008). These publications and models extrapolate from a limited amount of radioecological data to develop methods to predict the fate and effects of a large number of radionuclides, including plutonium, in organisms other than man.

Plutonium can enter the various trophic levels of a terrestrial food chain by deposition onto plant surfaces and, when deposited on or in soil, by adsorption to the surface of microorganisms and plant roots. It can also be ingested or inhaled by higher organisms and, to a limited degree, be absorbed by the gastrointestinal (GI) tract, where it can have toxic effects on the organisms either through direct exposure of the GI tract to radiation emitted by the radionuclide as it passes through the GI tract, or to both chemical and radiological systemic effects from plutonium that is absorbed from the GI tract and transferred to other organs in the body, primarily to bone and the liver. (Till and Meyer 1983 present a comprehensive description of the behavior of radionuclides in the environment, including plutonium.)

V. CHEMICAL ECOTOXICITY The chemical ecotoxicity of plutonium is not well-studied because the radiological ecotoxicity of plutonium is believed to be limiting for most organisms; chemical ecotoxicity, thus, has not been directly investigated. For example, a literature review of the ecotoxicity of plutonium at the Hanford Reservation states that “the toxicity of plutonium is related to the radioactive properties of the radionuclide rather than its chemical properties” (Driver 1994). Wilding and Garland (1982), who studied the effects of plutonium on soil microorganisms (organisms that are known to be highly radio-resistant), concluded that the toxicological effects they observed were due to the radiological and not the chemical toxicity of plutonium. Others have likened plutonium’s chemical toxicity to that of other heavy metals such as lead (Craig 2010).

Of note, care must be taken in interpreting the potential chemical and radiological toxicity of the observed concentrations of plutonium in organisms. For example, as noted previously, plutonium often adsorbs to the surface of microorganisms, plants, and plant roots rather than being absorbed by them. In addition, plutonium observed in fish could be associated with plutonium in sediment in the organism’s gastrointestinal tract. If not actually assimilated into the plasma, tissue, and cytoplasm of an organism, there is some question regarding the extent of damage that the observed concentrations can cause. The potential for chemical toxicological effects as a heavy metal is questionable because, if not absorbed, it would not have access to the intracellular or systemic biochemical machinery. As a radionuclide, those isotopes of plutonium that emit beta and gamma radiation could cause tissue damage if adsorbed to external surfaces of the organisms. However, because of the low penetrating power of alpha particles, radiation damage associated with external alpha exposure would be limited, except for perhaps plant root hairs and fish eggs, which have very thin cell walls protecting the cytoplasm from external alpha radiation.

VI. RADIOLOGICAL ECOTOXICITY A large body of literature exists on the observed radiological toxicity of the various isotopes of plutonium. The following presents a limited review of the literature on this subject, drawing heavily from Driver (1994). Note that many of these experimental outcomes are expressed in terms of effects observed at a given concentration of plutonium in water, soil, and sediment. No attempt is made here to convert these exposure settings to the dose (in rads) experienced by the organisms.

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

There are no known beneficial or protective properties of plutonium.

MECHANISM(S) AND LOCI OF TOXICITY

Considerable literature exists which characterizes and quantifies the potential damages to terrestrial and aquatic organisms due to radionuclides in the environment.

However, the literature specifically addressing the radioecological impacts of isotopes of plutonium in the environment is limited. Driver (1994) summarizes the literature, stating that the damages to aquatic and terrestrial organisms due to plutonium in the environment are due to ionization caused by the interaction of highly energetic alpha particles with living tissue.¹¹⁵

In particular, typical alpha particles, which have an energy of about 5 MeV, have a range in air of only about 4 cm (see Figure 6.7 of Shleien, *et al.* 1998). In addition, a layer of tissue of 0.07 mm will stop a 7.5 MeV alpha particle (see Table 3-1 of Shleien, *et al.* 1999). Hence, unless the plutonium alpha particle is in intimate contact with living tissue or is intracellular, it has a limited potential for biological damage. However, if it is in close proximity to living tissue and/or inside the cytoplasm or the nucleus of a cell, it deposits its 5 MeV alpha energy over a relatively short distance. For example, the typical binding energy of a hydrogen electron is about 13.6 eV and the typical energy required to ionize a molecule (i.e., eject an electron from its orbit) is about 34 to 35 eV (see page 17, Casarett, 1968). Hence, in a relatively short distance, a large number of ion pairs are produced by the passage of a 5 MeV alpha particle through tissue. For example, the total number of ion pairs produced by the energy deposited in tissue from a 5 MeV alpha particle is about 140,000 ion pairs (i.e., 5 MeV/35 eV). The pattern of energy deposition for a 5 MeV alpha particle is about 110 keV/micron, which is also referred to as the linear energy transfer (see page 28 of Casarett, 1968). Hence, about 3000 ion pairs are produced per micron. Given that a typical cell is on the order of tens of microns (see page 102 of Curtis and Barnes, 1989), a single cell might experience 30,000 ion pairs produced by the passage of a single 5 MeV alpha particle. It is this deposited energy in living tissue that results in biological damage.

FACTORS AFFECTING TOXICITY

As discussed in more detail in the following sections, a variety of factors can affect the ecotoxicological effects of plutonium, including dose, exposure period, species, and route of exposure.

¹¹⁵ Among the various isotopes of plutonium, the primary concern is exposure to alpha particles. However, the isotopes of plutonium emit x-rays and some emit beta particles.

PLANTS

Literature explicitly addressing the effects of plutonium on plants and plant communities is sparse. However, there is an abundance of publications on the effects of radiation in general on plants and plant communities. A classic series of investigations on the effects of external gamma ionizing radiation on plant communities was performed at Brookhaven National Laboratory in Upton, New York in 1962. A large (9500 Ci) Cs-137 source was placed in a pine forest for 20 hours per day, where the external exposures ranged from several thousand R¹¹⁶ per day within a few meters of the source to about 1 rad per day at 130 meters from the source. After 6 months of exposure, a total kill zone was observed at dose of >350 R/day. At 10 R/day there was reduced shoot growth of all tree species but no trees died (Casarett, 1994).

Chapter 13 of Casarett (1968) provides an excellent review of the literature on the effects of radiation on higher plants and plant communities. She provides data showing the percent germination for pollen germination for a variety of plants, as a function dose, where the doses ranged from zero to over 6000 rad. She also summarizes studies on the effects of radiation on the fertilized egg, where effects on the developing plant were observed at 500 R, and the radiosensitivity of developing embryos varied 100-fold depending on plant species.

Casarett (1968) also presents the results of investigations performed by Sparrow and Woodard (1962) where the effects of chronic exposure to Co-60 were measured. The effects included 10% growth reduction, failure to set seed, 50% growth reduction, pollen sterility, floral inhibition or abortion, severe growth inhibition, and measures of LD50 and LD100 levels.

Driver (1994) also does not specifically address experiments on the toxicity of plutonium in plants, but does summarize the general literature at that time regarding the effects of radiation on terrestrial plants (including the Brookhaven experiments). The following is excerpted from Driver (1994):

Plants are relatively resistant to ionizing radiation. The effects of chronic irradiation (6 months) of a late successional oak-pine forest were studied at Brookhaven National Laboratory (BNL) in New York. Changes in ecosystem structure, diversity, primary production, total respiration, and nutrient-inventory occurred. The most resistant species were the ones commonly found in disturbed places, i.e., generalists capable of surviving a wide range of conditions. Mosses and lichens survived exposures greater than 1000 R/d. No higher plants survived greater than 200 R/d. Sedge (*Carex pennsylvanica*) survived 150 to 200 Rad. Shrubs (*Vaccinium* and *Quercus ilicijolia*) survived 40 to 150 R/d. Oak trees survived up to 40 R/d, whereas pine trees were killed by 16 R/d. No change was noted in the number of species in an oak-pine forest up to 2 R/d, but changes in growth rates were detected at exposures as low as 1 R/d (Woodwell 1970). Severe defects were observed in *Tradescatia* at an exposure rate of 40 R/d. However, an

¹¹⁶ For simplicity, we can assume that one R or Roentgen is equal to 1 rad (or 100 ergs of energy deposited per gram of tissue).

exposure of 6000 R/d was required to produce the same effect in a hybrid gladiolus (Odum 1956). The sensitivity of various plant species appears to be related to the cross-sectional area of the nucleus in relation to cell size: the larger the nucleus and chromosome volume, the more sensitive the plant (Underbrink and Sparrow 1968, 1974).

Driver (1994) goes on to provide a tabulation of the effects of chronic exposure to external radiation on a variety of plants.

One line of research of particular note is investigations of the damage done to pine forest in the vicinity of the Chernobyl accident. From this work, it became clear that pine trees are among the most radiosensitive organisms. "According to reports from Soviet scientists at the First International Conference on the Biological and Radiological Aspects of the Chernobyl Accident (September 1990), fallout levels in the 10 km zone around the plant were as high as 4.81 GBq/m². The so-called "red forest" of pine trees killed by heavy radioactive fallout lies immediately behind the reactor complex within the 10 km zone. The "red forest" covered some 4 km² and only pine trees died while birch and aspen survived. The "red forest" is so named because it was reported by evacuees that in the days following the accident the trees glowed red, apparently due to heavy radioactive fallout."¹¹⁷

It is noteworthy that the above general discussion of the effects of radiation on trees emphasizes external exposure to gamma emitters and direct deposition of fallout. Specific research on the radiotoxic effects on trees of plutonium mixed in soil is sparse. The National Aeronautics and Space Administration (NASA 1995) investigated this matter in its Final Environmental Statement (FEIS) for the Cassini Mission. Appendix C of the FEIS investigates the impacts of plutonium dioxide fuel (mainly Pu-238), used as a radioisotope thermoelectric generator (RTG), if soil were contaminated following an accident. The appendix explores the chemical behavior of PuO₂ in soil, water, and sediment. However, it concluded that, due to the very low soil-to-plant uptake factors, there was little potential for uptake by trees. However, the report did not explore the external exposure of the root hairs of trees to Pu-238's alpha emissions.

AQUATIC INVERTEBRATES AND FISH

Literature explicitly addressing the effects of plutonium on aquatic biota is sparse. However, there is an abundance of publications on the effects of radiation in general on aquatic organisms. Driver (1994) summarizes the literature on the effects of radiation on aquatic organisms, providing LD50 values fish (90,000 R), 50% survival doses for male and female germ cells (305 to 500 R), and reduction on population growth rate for white crappie, largemouth bass, and redhorse (25% reduction at 57 R external exposure).

EA (2002) compares the effects of alpha and gamma radiation on the reproductive output of a freshwater fish. In particular, zebrafish were exposed to gamma radiation

¹¹⁷ See http://www.wordiq.com/definition/Chernobyl_accident#Impact_on_the_natural_world

at a dose rate of 30, 100, and 740 mrad/hr. Only the highest dose rate group experienced effects on reproductive output (reduced egg count). Polonium (Po)-210 spiked meals were designed to deliver internal dose rates 0.8, 2.5, 18.5, and 74 mrad/hr (of note, polonium is an alpha-emitter). After the experiments, it was determined that the doses to the testes were actually 0.96, 1.9, 8.4, and 21.4 mrad/hr. Among these fish, no adverse effects were observed. The study also concluded that alpha relative biological effectiveness (RBE¹¹⁸) was <7 to <20, and that the use of an RBE of 35 for internal exposure to alpha emitters is considered an upper bound for egg production as a biological endpoint.

Till *et al.* (1976) evaluated the radiological and chemical toxicity of plutonium and uranium on the developing embryos of fish. Eggs were used from carp and fathead minnows. Fertilized eggs were developed in a high specific activity solution of Pu-238 and low specific activity solution of Pu-244. The penetration of the plutonium was evaluated, and results indicated that it accumulated in the carp embryos and was evenly distributed in the egg volume. Overall, abnormalities was the most sensitive endpoint evaluated, followed by larval survival and then egg hatchability.

Till *et al.* (1976) also found that although concentration of 7.5 $\mu\text{Ci/ml}$ of Pu-238 did not entirely inhibit carp eggs from hatching, a significant number of abnormalities resulted, and most of the larvae died within several hours of hatching. At 3.9 pCi/ml normal hatching occurred, although there were a significant number of abnormalities in comparison to the control group, and the larvae died within 48 hours. Concentrations of 1.6 pCi/ml and 0.16 pCi/ml did not affect hatching, and the authors did not find a significant effect on larval survival at these levels.

Till *et al.* (1976) also found fathead minnow eggs to be more sensitive than carp eggs: at a concentration of 1.3 $\mu\text{Ci/ml}$ of Pu-238, hatching was severely inhibited. At 0.85 pCi/ml of Pu-238, many eggs hatched prematurely. A concentration of 0.26 pCi/ml resulted in a significant number of abnormal larvae, and one-third of the eggs hatched prematurely. No obvious effects on the fathead minnow eggs were found at 0.0056 $\mu\text{Ci/ml}$ of Pu-238 (*ibid.*).

Till *et al.* (1976)'s toxicity experiment using Pu-244 found that an alpha activity of 0.02 $\mu\text{Ci/ml}$ (equivalent to a concentration of 20 ppm), prevented both carp and fathead minnow eggs from hatching. A concentration of 9 ppm delayed hatching by approximately 6 hours compared to the control group, and by the fourth day, the fathead minnow eggs had died. The authors suggest that the absence of hatching in the 20 ppm exposure may have been the result of plutonium's chemical rather than radiological toxicity (*ibid.*).

Till *et al.* (1976) evaluated the dosimetry of the concentrations used in the above experiments and conclude that the doses from Pu-238 that affected the survival of the larvae were estimated to be about 8,200 rads for carp eggs and 1,900 rads for fathead minnows. A larger number of abnormal larvae than in the control groups were

¹¹⁸ For a given biological endpoint, such as egg production, different types of radiation, such as gamma, beta, and alpha emitters, can have different levels of toxicity for the same dose in rads. Relative biological effectiveness expresses the potential toxicity of a given dose of radiation relative to gamma radiation.

produced by Pu-238 doses of approximately 4,300 rads to carp and 570 rads to fathead minnows.

SOIL MICROORGANISMS AND INVERTEBRATES

Wilding and Garland (1982) studied the effects of plutonium in soil on microorganisms. Significant effects on bacteria were observed at Pu-239 concentrations as low as 1 µg/g of soil in the form of plutonium nitrate. Fungi were not affected until exposed to much higher levels (180 µg/g).

Driver (1994) cites literature that observed that 1780 Ci/m² of Pu-239/240 in soil mixed to a depth of 25-30 cm decreased the population density of earthworms and insect larvae by 50% over three years.

BIRDS

Driver (1994) states that no information is available on plutonium effects in wild birds. A limited search of the published literature did not identify any experimental or environmental studies that investigated the potential toxic effects of plutonium on birds.

MAMMALS

The literature on the effects of plutonium on mammals is extensive, primarily because of concern over the toxicity of plutonium on humans. ARSDR (2008) provides a detailed discussion of the toxicity of plutonium on man and a variety of mammals organized “first by route of exposure (inhalation, oral, and dermal) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects). These data are discussed in terms of three exposure periods: acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more). ATSDR (2008) cites numerous studies conducted in nonhuman primates, dogs, and rodents. The ATSDR discussions primarily focused on the wealth of information that has been developed on the toxicology of plutonium in beagle dogs exposed by inhalation. A detailed account is provided of the levels of exposure where adverse effects were observed under acute and chronic exposures.

With respect to ingestion, ATSDR (2008) states:

“No studies were located regarding death or lifespan shortening in humans after oral exposure to plutonium. In neonatal rats, given a single 1.2×10^4 kBq ²³⁸Pu/kg dose (as plutonium citrate) by gavage, 45% mortality was observed by 2 weeks post exposure; no deaths were reported following dosing at 3.7 kBq/kg (Fritsch *et al.* 1987). No studies were located regarding respiratory, cardiovascular, hematological, musculoskeletal, hepatic, renal, or dermal/ocular effects in humans or animals after oral exposure to plutonium.

No studies were located regarding gastrointestinal effects in humans after oral exposure to plutonium. Gastrointestinal effects were observed in neonatal rats following oral administration of ²³⁸Pu/kg (as plutonium citrate) by gavage (Fritsch *et al.* 1987). Mild hypertrophy of the crypts of the small intestine, which form the secretions of the small intestine, was observed in the rats receiving a 5,300 kBq

²³⁸Pu/kg dose. Total disappearance of epithelial cells and crypts combined with intestinal hemorrhaging, was observed in rats that received 17,400 kBq ²³⁸Pu /kg (Fritsch *et al.* 1987). Increased neutrophils were noted on the surface epithelium and superficial cellular layers of the large intestine in adult rats given 155 µCi ²³⁸PuO₂/kg (5,740 kBq/kg) (Sullivan *et al.* 1960). This effect was noted at 3 (but not 6) days post exposure. No studies were located regarding the following health effects in humans or animals after oral exposure to plutonium: immunological and lymphoreticular effects, neurological effects, reproductive effects, developmental effects, or cancer.”

It is clear that there is a vast body of laboratory-based literature on the effects of plutonium on non-human mammals. However, field studies on the toxicity of plutonium on mammals were not identified, nor were any laboratory studies of wild species.

VII. EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

In theory, one would expect additive and synergistic effects of exposure to plutonium with other environmental toxicants, similar to those observed between other heavy metals. For example, Schubert *et al.* (1978), Tabata *et al.* (2003), Traore *et al.* (1999), and Sanchez *et al.* (2001) address the synergistic cytotoxic and nephrotoxic effects of a number of heavy metals. In addition, there may also be synergistic effects associated with the combined action of exposure to plutonium as a chemical and radiological toxin. Concerns over the possible synergistic effects of exposure to radiation and chemical toxins have been extensively raised and reported in the scientific literature (Burkart *et al.* 1997, Prasad *et al.* 2004) but little consensus has been achieved in quantifying these effects in humans, except possibly for radon and smoking (BEIR IV, 1988) and certainly in the enhancement of the therapeutic effects of radiotherapy used to treat cancer (e.g., Lew *et al.* 2002). Animal models (rats) have been used to demonstrate synergistic effects of plutonium and cigarette smoke (Mauderley *et al.* 2010). UNSCEAR (2000) Annex H explores the combined effects of radiation and chemical agents, including heavy metals. Only a few data are available from combined exposures of radiation and metals in human populations and no firm evidence of interactions has been observed.

There is some literature on observed synergistic adverse effects of radiation and toxic chemicals on organisms other than humans (e.g., salmon, Mothersill *et al.* 2007). Examples of ionizing radiation and metals producing combined effects in other biological systems include synergistic effects on soil microbial activity from cadmium and zinc in combination with gamma radiation (summarized in UNSCEAR 2010). Also combined effects of cesium-134/137 and lead found in highly contaminated habitats in the Russian Federation increased the mutation rate in the plant *Arabidopsis thaliana* (summarized in UNSCEAR 2010). However, the authors clearly indicate that the relative importance of different damage-inducing mechanisms of metals for combined exposures in human and non-human populations remains to be elucidated.

Overall, there is a clear need for additional research on synergistic effects of multiple stressors in radioecotoxicology (e.g., Salbu and Skipperud 2007, Mothersill and

Seymour 2007). In particular, these authors raise the issue of pesticides, organics, and endocrine disruptors and synergistic effects with radioactive materials, particularly with long-term exposure to various biological systems. Manti and D'Arco (2010) summarize the in vitro and animal-model studies and epidemiological surveys with two or more stressors, including radionuclides (DNA-damaging agents). They also emphasize that most research focuses only on the short-term effects of combined single exposures to animal models, and more work is needed to understand chronic exposure to trace contaminants and radioactive elements in the environment, including impacts to long-term genome stability. Specific research is lacking on plutonium effects with multiple stressors on biological systems, particularly non-human systems.

VIII. DATA GAPS

As described in ATSDR (2008), though the environmental fate and transport of plutonium is understood to a degree, it is also acknowledged that, due to its complex chemistry, “information on environmental compartments, such as flux rates, and the mechanisms and rates of several processes involved in biogeochemical cycling of plutonium are still undefined.” Also, “the data available on the uptake of plutonium by plants is limited.” In fact, though there is an abundance of literature citing plutonium bioaccumulation factors in aquatic organisms and soil-to-plant transfer factors in terrestrial plants, species specific bioaccumulation factors and soil-to-plant transfer factors for plutonium at Hanford appear to be limited.

There is limited literature addressing the chemical toxicity of plutonium because it is widely acknowledged (see discussion above) that at all trophic levels the radiotoxicity of plutonium is limiting. In addition, there appears to be a need to better understand the degree to which the combined chemical and radiological toxicity of plutonium might be additive and/or synergistic.

There appears to be some literature on the radiotoxicity of plutonium on aquatic and terrestrial organisms based on laboratory experiments. However, field studies of effects are limited, as are experimental data on the effects of plutonium on wild organisms.

In addition, there are data gaps related to the sensitivity of plant root hairs and fish eggs to external exposure to alpha emitters, such as Pu-239, in soil and sediment, respectively. With respect to fish eggs, this gap is based on calculations of the range of alpha particles in tissue as compared to the thickness of the egg chorion, which does not appear to be thick enough to protect the egg cytoplasm from the potential harmful effects of external alpha emitters in river sediment (at least for some species of fish). With regard to plant root hairs, they consist of a filament of cells without a protective membrane, other than the cell membrane, which is not thick enough to protect the cell interior from the potential harmful effects of exposure to external sources of alpha emitters in soil. The literature reviewed here is silent on these subjects and appears to be worthy of further consideration.

- IX. REFERENCES** Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Toxicological Profile for Plutonium. Draft. U.S. Department of Health and Human Services.
- Amarillo National Resource Center for Plutonium (ANRCP). 1998. Literature Review: Phytoaccumulation of Chromium, Uranium, and Plutonium in Plant Systems, ANRCP-1998-3. May.
- Baes, C.F. III and R.D. Sharp. 1983. A proposal for estimation of soil leaching and leaching constants for use in assessment models. *J. Environ. Qual.* 12(1):17-28.
- Ballou, N.E., G.R. Ducatte, C. Quasng, and V.T. Remcho. 1996. Determination of chelating agents in Hanford waste tank stimulant. *Journal of High Resolution Chromatography* 19(4):183-188.
- Baum, E.M., H.D. Knox, and T.R. Miller. 2002. Nuclides and Isotopes. Chart of the Nuclides. 16th ed. KAPL, Inc. Lockheed Martin, pp. 70-71.
- Burkart, W., G.L. Finch, and T. Jung. 1997. Quantitative health effects from the combined action of low-level radiation and other environmental agents can new approaches solve the enigma” *Science of the Total Environment*, 205(1):51-70.
- Cantrell, K.J. 2009. Transuranic Contamination in Sediment and Groundwater at the U.S. DOE Hanford Site, PNNL-18640. August.
- Casarett, A.P. 1968. Radiation Biology. Englewood Cliffs, New Jersey: Prentice-Hall, Inc.
- ChemIDplus. 2009. Plutonium and selected isotopes. ChemIDplus. Bethesda, MD. U.S. Library of Medicine. Available at <http://chem.sis.nlm.nih.gov/chemidplus/chemidlite.jsp>. Accessed March 16, 2009.
- Clark, D.L., S.S. Hecker, D.D. Jarvinen, and M.P. Neu. 2006. Plutonium and plutonium compounds. In: Kirk-Othmer Encyclopedia of Chemical Toxicology. John Wiley & Sons, Inc., pp 667-712.
- Committee on the Biological Effects of Ionizing Radiation (BEIR). 1988. Health Risks of Radon and Other Internally Deposited Alpha-Emitters, BEIR IV, Biological Effects of Ionizing Radiation, National Research Council, National Academy of Sciences, National Academy Press, Washington, D.C. 1988.
- Coughtrey, P.J., D. Jackson, and M.C. Thorne. 1985. Radionuclide Distribution and Transport in Terrestrial and Aquatic Ecosystems. A Compendium of Data. Netherlands: A.A. Balkema.
- Craig, D.K. 2010. Toxicological versus Radiological Hazards of Pu-239. Westinghouse Savannah River Laboratory, WSMS-SAE-01-0204, Contract No. DE-AC09-96SR18500.
- Cummins, C.L. 1994. Radiological Bioconcentration Factors for Aquatic, Terrestrial, and Wetland Ecosystems at the Savannah River Site (U). WSRC-TR-94-0391, Rev 0, September 1994. Prepared for the U.S. Department of Energy under Contract No. DE-AC09-89SR18035.

- Curtis, H. and N.S. Barnes. 1989. *Biology*. Worth Publishers, Inc.
- Dreicer, M., T.E. Hakonson, F.W. Whicker, and G.C. White. 1983. Investigation of Contaminated Soil to Plant Surfaces by Raindrop Splash, 1983. UCRL-88540, October 21.
- Driver, C.J. 1994. Ecotoxicity Literature Review of Selected Hanford Site Contaminants, PNL-9394. Prepared for the U.S. Department of Energy under Contract DE-AC06-76RLO 1830. Pacific Northwest Laboratory, Richland Washington 99352. March.
- Eisenbud, M. and T. Gesell. 1997. *Environmental Radioactivity from Natural, Industrial, and Military Sources*, Fourth Edition. Academic Press.
- Eney, R.M., D.C. Klopfer, and W.C. Weimer. 1974. The Ecological Behavior of Plutonium and Americium in a Freshwater Ecosystem: Phase I limnological Characterization and Isotopic Distribution. BNWL-1867, Battelle Pacific Northwest Laboratories. September.
- Essien, I.O. 1994. Alpha recoil atoms of plutonium in the environment. *Journal of Radiological and Nuclear Chemistry, Articles*, 178(1):165-172.
- Environmental Agency (EA). 2002. An Investigation into the Effects of Chronic Radiation on Fish. R&D Technical Report P3-053/TR. Environmental Agency, Department for the Environment, Food and Rural Affairs and the Scottish and Northern Ireland Forum for Environmental Research.
- Felmy, A.R., K.J. Cantrell, and S.D. Conradson. 2010. Plutonium Contamination Issues in Hanford Soils and Sediments: Discharges from the Z-Plant (PFP) Complex. *Physics and Chemistry of the Earth, Parts A/B/C* 35(6-8):292-297. Migration 2009. 12th International Conference on the Chemistry and Migration of Actinides and Fission Products in the Geosphere.
- Hinton, T.G. and J.E. Pinder III. 2000. A review of plutonium releases from the Savannah River site, subsequent behavior within terrestrial and aquatic environments and resulting doses to humans. In: A. Kudo (ed.). *Plutonium in the Environment*. Elsevier Science Ltd. Pp. 413-436.
- International Atomic Energy Agency (IAEA). 1992. Effects of Ionizing Radiation on Plants and Animals at Levels Implied by Current Radiation Protection Standards. Technical Report Series No. 332. IAEA, Vienna.
- International Atomic Energy Agency (IAEA). 1994. Handbook of Parameter Values for the Prediction of Radionuclide Transfer Factors in Temperate Environments, International Atomic Energy Agency, Technical Report Series No. 364. Vienna, 1994.
- International Atomic Energy Agency (IAEA). 2010. Handbook of Parameter Values for the Prediction of Radionuclide transfer in Terrestrial and freshwater Environments, Technical Report Series No. 472, International Atomic Energy Agency, Vienna, 2010.

- International Commission on Radiological Protection (ICRP). 2008. ICRP Publication 108: Environmental protection: The concept and use of reference animals and plants. *Annals of the International Commission on Radiation Protection* 38(4-6).
- International Commission on Radiological Protection (ICRP). 1995. ICRP Publication 68: Dose coefficients for intakes of radionuclides by workers. *Annals of the International Commission on Radiation Protection* 24(4).
- International Commission on Radiological Protection (ICRP). 1996. ICRP Publication 72: Age dependent doses to the members of the public from intake of radionuclides part 5, compilation of ingestion and inhalation coefficients. *Annals of the International Commission on Radiation Protection* 26(1).
- Kevern, N.R. and S.A. Spigarelli. 1971. Effects of Selected Limnological Factors on the Accumulation of ¹³⁷Cs Fallout by Largemouth Bass, C00-1795-4. Presented at the 3rd National Symposium on Radioecology, Oak Ridge, TN, May 10-12.
- Lew, Y.S., A. Kolozsvary, S.L. Brown, and J.H. Kim. 2002. Synergistic interaction with arsenic trioxide and fractionated radiation in locally advanced murine tumor. *Cancer Res* 62: 4202-4205.
- Lide, D.R. 2008. *CRC Handbook of Chemistry and Physics*. 2007-2008. 88th ed. Boca Raton, FL.: CRC Press. Taylor & Francis Group, pp. 4-26, 4-27, 4-81, 11-197, 11-198.
- Manti, L. and A. D'Arco. 2010. Cooperative biological effects between ionizing radiation and other physical and chemical agents. *Mutation Research* 704: 115-122.
- Mauderly, J.L., S.K. Seilkop, E.B. Barr, A.P. Gigliotti, F.F. Hahn, C.H. Hobbs, and G.L. Finch. 2010. Carcinogenic interactions between a single inhalation of (PuO₂)-Pu-239 and chronic exposure to cigarette smoke in rats. *Radiation Research* 173: 665-676.
- Mothersill, C., B., Salbu, L.S. Heier, H.C. Teien, J. Denbeigh, D. Oughton, B.O. Rosseland, and C.B. Seymour. 2007. Multiple stressor effects of radiation and metals in salmon (*Salmo salar*). *Journal of Environmental Radioactivity*. 96(1-3):20-31.
- Mothersill, C. and C. Seymour. 2007. Radiation risks in the context of multiple stressors in the environment – issues for consideration. In: Mothersill, C., I. Mosse, and C. Seymour, eds. *Multiple stressors: a challenge for the future*. Book Series: Nato Science for Peace and Security Series C – Environmental Security. Pp. 235-246.
- Napier, B.A., R.J. Fellows, and K.M. Krupka. 2007. Soil-to-Plant Concentration Ratios for Assessing Food-Chain Pathways in Biosphere Models. NUREG/CR-6941, PNNL-16741.
- National Aeronautic and Space Administration (NASA). 1995. Final Environmental Impact Statement for the Cassini Mission. Solar System Exploration Division,

Office of Space Science, National Aeronautics and Space Administration,
Washington, D.C. June.

- National Council on Radiation Protection & Measurements (NCRP). 1984. Radiological Assessment. Predicting the Transport, Bioaccumulation and Uptake by Man of Radionuclides Released to the Environment. NCRP Report No. 76. National Council on Radiation Protection and Measurement, Bethesda, MD, pp. 73-85, 127-152.
- National Council on Radiation Protection & Measurements (NCRP). 1991. Effects of Ionising Radiation on Aquatic Organisms. Report No. 109. National Council on Radiation Protection and Measurements, Bethesda, MD.
- National Council on Radiation Protection & Measurements (NCRP). 1996. Report No. 123. National Council on Radiation Protection and Measurement, Screening Models for Releases of Radionuclides to Atmosphere, Surface Water, and Ground. January 22.
- Ng, Y.C., C.S. Colsher, and S.E. Thompson. 1979. Transfer factors for assessing the dose from radionuclides in agricultural products. In: Biological Implications of Radionuclides Released from Nuclear Industries, Proc. Symp. Vienna 26-30 March 1979, vol. 2, pp. 295-318.
- Odum, E.P. 1976. Ecological aspects of waste disposal. In: Proceedings from a Conference on Radioactive Isotopes in Agriculture. TID-7512, USAEC, Technical Information Center, Springfield, Virginia.
- ORAUT-TKBS-0006-4. 2010. Oak Ridge Associated Universities Team, Hanford Site-Occupational Environmental dose, Revision 3, January 7.
- ORAUT-TKBS-0006-2. 2010. Oak Ridge Associated Universities Team, Hanford Site Description, Revision 1, February 22.
- Peterson Jr., H.T. 1983. Terrestrial and aquatic food chain pathways. In: Till and Meyer (1983), Radiological Assessment – A Textbook on Environmental dose Assessment, edited by John E. Till and H. Robert Meyer, NUREG/CR-3332, ORNL-5968.
- Poston, T.M., J. P. Duncan, and R.L. Dirkes. 2010. Hanford Site Environmental Report for Calendar Year 2009. Prepared for the U.S. Department of Energy by Pacific Northwest Laboratory under contract DE-AC05-76RL01830, PNL, Richland, Washington. September.
- Prasad, K.N., W.C. Cole, and G. M. Hasse. 2004. Health Risks of Low Dose Ionizing radiation in Humans: A Review. *Exp Biol Med* 229: 378-382.
- Price, K.R. 1972. Uptake of ²³⁷Np, ²³⁹Pu, ²⁴¹Am, and ²⁴⁴Cm from soil by tumbleweed and cheatgrass. AEC Research and Development Report. Battelle, Pacific Northwest Laboratories. BNWL-1688.

- Price, K.R. 1973. Tumbleweed and cheatgrass uptake of transuranium elements applied to soil as organic acid complexes. Battelle, Pacific Northwest Laboratories. BNWL-1755.
- Salbu, B. and L. Skipperud. 2007. Challenges in radioecotoxicology. In: Mothersill, C., I. Mosse, and C. Seymour, eds. Multiple stressors: a challenge for the future. Book Series: Nato Science for Peace and Security Series C – Environmental Security. Pp. 3-12.
- Sanchez D.J., M. Belles, M.L. Albina, J.J. Sirvent, and J.L. Domingo. 2001. Nephrotoxicity of simultaneous exposure to mercury and uranium in comparison to individual effects of these metals in rats. *Biol Trace Elem Res* 84(1-3):139-54.
- Schubert, J., E.J. Riley and S.A. Tyler. 1978. Combined effects in toxicology. A rapid systematic testing procedure: cadmium, mercury, and lead. *Toxicol Environ Health* 4(5/6):763-776.
- Sheppard S.C. and W.G. Evenden. 1997. Variations in transfer factors for stochastic models: Soil-to-plant transfer. *Health Physics* 72(5):727-733.
- Sheppard M.I. and D.H. Thibault. 1990. Default soil to solid/liquid partition coefficients, K_d s, for major soil types: A compendium. *Health Physics* 59(4):471-482.
- Shleien, B., L.A. Slaback, Jr., and B.K. Birky. 1998. Handbook of Health Physics and Radiological Health, Third Edition. Williams & Williams, A Waverly Company.
- Smith, B. and A. Amonette. 2006. The Environmental Transport of Radium and Plutonium: A Review. Institute for Energy and Environmental Research. June 23.
- Sparrow, G.M. and G.M. Woodwell. 1962. Prediction of sensitivity of plants to chronic gamma radiation. *Radiation Botany* 2:9-26.
- Stryer, L. 1988. Biochemistry, 3rd Edition. New York: W.H. Freeman and Company.
- Swift, D.J. 1992. The accumulation of plutonium by the European lobster (*Homarus gammarus* L). *J. Environ Radioact.* 16(1):1-24.
- Tabata M., S.A. Kumar, and E. Nyarko. 2003. Enhanced conformational changes in DNA in the presence of mercury(II), cadmium(II) and lead(II) porphyrins. *J Inorg Biochem* 94(1-2):50-8.
- Taylor, D.M. 1989. The biodistribution and toxicity of plutonium, americium, and neptunium. *Science of the Total Environment* 83(3):217-225.
- Traore A, M. Bonini, S.D. Dano, and E.E. Creppy. 1999. Synergistic effects of some metals contaminating mussels on the cytotoxicity of the marine toxin okadaic acid. *Arch Toxicol* 73(6):289-95.
- Underbrink, G. and A.H. Sparrow. 1974. The influence of environmental endpoints, dose, dose rate, neutron energy, nitrogen ions, hypoxia, chromosome volume and

- ploidy on RBE in *Tradescantia* stamen hairs and pollen. In: Biological Effects of Neutron Irradiation, International Atomic Energy Agency, Vienna, Austria, pp 185-214.
- United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2000. Sources and Effects of Ionizing Radiation.
- Woodwell, G.M. 1970. Effects of pollution on the structure and physiology of ecosystems. *Science* 168(3930):429-433.
- Taylor, D.M. 2001. Environmental plutonium – Creation of the universe to twenty-first century mankind. In: Plutonium in the Environment, Edited Proceedings of the Second Invited International Symposium, November 9-12, 1999, Osaka, Japan, Elsevier.
- Thompson, P. 1988. Environmental monitoring for radionuclides in marine ecosystems: Are species other than man protected adequately? Viewpoint Article, *J. Environ. Radioactivity* 7:275-283.
- Till, J.E., S.V. Kaye, and J.R. Trabalka. 1976. The Toxicity of Uranium and Plutonium to the Developing Embryos of Fish. Oak Ridge National Laboratory. July.
- Till, J.E. and H.R. Meyer. 1983. Radiological Assessment – A Textbook on Environmental Dose Assessment. J.E. Till and H.R. Meyer (ed.), NUREG/CR-3332, ORNL-5968.
- Till J.E. and R.E. Moore. 1988. A pathway analysis approach for determining acceptability levels of contamination of radionuclides in soil. *Health Physics* 55(3):541-548.
- Turner, G.A. and D.M. Taylor. 1968. The transport of plutonium, americium and curium in the blood of rats. *Phys. Med. Biol.* 13(4):535-546.
- U.S. Department of Energy (DOE). 1995. Closing the Circle on the Splitting of the Atom, The Environmental Legacy of Nuclear Weapons Production in the United States and What the Department of Energy is Doing About It. The U.S. Department of Energy, Office of Environmental Management. January.
- U.S. Department of Energy (DOE). 2002. A Graded Approach for Evaluating Radiation Doses to Terrestrial and Aquatic Biota. DOE-STD-1153-2002.
- U.S. Department of Energy (DOE). 2004. Interagency Steering Committee on Radiation Standards, User's Guide, Version 1, RESRAD-BIOTA: A tool for implementing a graded approach to biota dose evaluation. DOE/EH-0676, ISCORS Technical Report 2004-02, January.
- U.S. Department of Energy (DOE). 2005a. Plutonium. Radiological and chemical fact sheet to support health risk analysis for contaminated areas. Available at: http://www.evs.anl.gov/pub/doc/ANL_Contaminant_Fact_Sheets_All_070418.pdf. Accessed May 21, 2007.
- Welch, S. 1992. The Ion Carrier. Boca Raton, FL: CRC Press.

- Wildung, R.E. and T.R. Garland. 1982. Effects of plutonium on soil microorganisms. *Applied and Environmental Microbiology* 43(2):418-423.
- Zhao, P. 1997. Literature Review of Intrinsic Actinide Colloids Related to Spent Fuel Waste Package Release Rates. UCRL-ID-126039, January.

STRONTIUM (SR-90) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Strontium-90 (Sr-90) is a radionuclide and is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Strontium is a hard, white-colored metal that in its natural form is found in the minerals celestite (SrSO₄) and strontianite (SrCO₃). Its concentrations in most mineral materials are on the order of a few parts per million (ppm) (ATSDR 2004). Strontium, which is chemically similar to calcium, exists as four stable isotopes in nature: Sr-84, Sr-86, Sr-87, and Sr-88, while Sr-90 is an artificial isotope formed in nuclear reactors or during the explosion of nuclear weapons (*ibid.*).

As a radionuclide, Sr-90 has a half-life of about 29 years and decays by beta decay to yttrium-90 (Y-90), which is also radioactive. Yttrium-90 decays by beta decay to zirconium-90 (Zr-90), which is stable (ATSDR 2004).

Exhibit 1 summarizes the radiological properties of Sr-90 and its progeny, Y-90. Of note, Y-90 has a half-life of about 64 hours. This means that the Y-90 will grow in and achieve equilibrium with its parent in about 10 half-lives or 641 hours. Once equilibrated, every disintegration of Sr-90 is associated with a disintegration of Y-90. This is important because Y-90 has much more energetic beta particles, and will be responsible for most of the beta energy deposited in an organism per disintegration of Sr-90, once the Sr-90 is absorbed.

EXHIBIT 1 RADIOLICAL PROPERTIES OF SR-90 AND IT'S PROGENY, Y-90 (EXCERPTED FROM SHLEIEN *ET AL.* 1998)

| ISOTOPE | HALF-LIFE | PROBABILITY OF DECAY | MAX BETA/ELECTRON ENERGY (MEV) | AVERAGE BETA/ELECTRON ENERGY (MEV) |
|---------|-----------|----------------------|--------------------------------|------------------------------------|
| Sr-90 | 28.6 yrs | 1.0 | 0.546 | 0.1958 |
| Y-90 | 64.1 hrs | 1.0 | 2.2839 | 0.9348 |

This profile draws heavily from a number of authoritative literature summaries on Sr-90, including, UNSCEAR (2000 and 2008), ATSDR (2004), the Hazardous

Substance Databank (HSDB),¹¹⁹ and reports addressing the operation and remediation of the Hanford facility.¹²⁰

II. SOURCES NATURAL SOURCES

There are no naturally occurring sources of Sr-90.

ANTHROPOGENIC SOURCES

Sr-90 is a fission product and was produced in large quantities during above-ground weapons testing in the United States and the former Soviet Union during the 1950s and 1960s and also, to a lesser degree, by China, France, and the United Kingdom (UNSCEAR 2000). It was also produced during below-ground weapons testing, but the fission products were largely confined below ground at the test sites (primarily the Nevada Test Site). An historic account of all nuclear weapons testing by every nation through 1999 is provided in Mikhailov (1999).

During testing, Sr-90 was produced at a rate of 30 to 40 atoms of Sr-90 for every 1,000 fissions (or about 0.10 million curies per megaton), and, as a result, fallout from above-ground testing resulted in the widespread distribution of Sr-90 in soil, water, and food (see Glasstone and Dolan 1977). The literature summarizing the concentrations of Sr-90 in air, soil, water, food items, and in aquatic and terrestrial organisms is vast. Summaries of this literature can be found in Eisenbud and Gesell (1997) and UNSCEAR (2000 and 2008). Eisenbud and Gesell (1997) provides a detailed summary of global fallout in the northern and southern hemispheres from 1963 through 1981, with the atmospheric inventory peaking at about 5 million curies in 1963, the time at which above-ground testing ceased. The atmospheric inventory declined to about 10 million curies in 1980, with periodic spikes associated with above-ground testing performed by other nations.

Figure 9-20 in Eisenbud and Gesell (1997) presents isocontour lines of global Sr-90 deposition as estimated based on soil samples collected from 1965 to 1967 (i.e., shortly after the conclusion of above-ground weapons testing by the U.S. and the former Soviet Union). The middle latitudes of the northern hemisphere (including the U.S.) experienced a total Sr-90 deposition of about 80 millicuries per square kilometer. UNSCEAR (2000) also presents a fairly detailed description of the deposition density (Bq/m²) of Sr-90 in the northern and southern hemispheres for different latitudes and as a function of time up to 2000. Updated information was recently published in UNSCEAR (2008).

The concentration of Sr-90 in the environment from weapons testing is gradually declining due to its 29 year half-life, and also due to natural attenuation; i.e., Sr-90 is

¹¹⁹The National Institute of Health maintains the Toxnet database that includes the Hazardous Substance Database, which can be found at: <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/f?./temp/-4x0j5B:1>

¹²⁰ At several places in this profile, direct quotes make reference to additional specific source documents. These references are included in the reference section of this profile so that readers can more easily identify and obtain the original source documents cited in the major publications.

gradually depleted from soil and sediment by downward migration and erosion, and eventually transported into relatively inaccessible or less accessible environmental compartments, such as the ocean depths.

In addition to weapons testing, Sr-90 has been and continues to be released in the routine liquid and gaseous effluents of nuclear facilities. The concentrations of Sr-90 in effluents of nuclear facilities are monitored as part of routine effluent monitoring programs. In addition, the environmental radiological surveillance programs at these facilities also look for Sr-90 in environmental samples collected in the vicinity of these facilities. However, the levels of Sr-90 in the effluent of these facilities are relatively low compared to that of Cs-137. Tichler *et al.* (1988) show similar results. As a result, Sr-90 is not routinely detected in the environment at levels above those resulting from fallout. The reason is that, although Sr-90 is produced at about the same rate as Cs-137 in the fission process and its inventory in reactor cores is about the same as that for Cs-137, its ability to escape the fuel cladding, enter the coolant, and be released from these facilities is much less. However, like Cs-137, Sr-90 is present in large quantities in high-level radioactive waste and spent fuel.

At Hanford, local sources include activities associated with the reactors at the 100 Area North and leakage of the high-level and low-level radioactive wastes that have been stored onsite in the 200 Area tank farms. Vermeul *et al.* (2009) provides background information on the origin of Sr-90 contamination in the subsurface environment in the 100 Area and describes concerns that it is migrating to the nearby Columbia River. Reactor operation in 100-N Area required the disposal of bleed and feed cooling water, waste water from the spent fuel storage basis, and other reactor-related sources. This waste water was disposed of in cribs and trenches, and the water was allowed to percolate downward through the soil. This water contained fission and activation products, including Sr-90, which has migrated to the Columbia River via the groundwater. The migration and control of this source of contamination is the subject of extensive research cited in Vermeul *et al.* (2009). A detailed description of the efforts being employed to reduce the flux of Sr-90 to the Columbia River by this pathway is provided in DOE (2005).

OTA (1991), DOE (1995a), and DOE (1997) provide a general overview of waste tank use and associated leakages from these. Detailed information is provided in Hanson (2000) and its citations such as Gephart and Lundgren (1998). In summary, 0.6 to 1.4 million gallons have leaked from the single-shell high-level waste tanks containing a total of 1 to 2 million curies, primarily Sr-90 and Cs-137. As a result of the wastewater leakage from the single shell tanks in the 200 areas and also seepage from cribs and tanks in the 100 Area, there are ground water plumes containing Sr-90 beneath the site and migrating toward the Columbia River.

The behavior of this leakage in the subsurface environment is monitored (e.g., monitoring is being performed by the Defense Nuclear Facilities Safety Board, Linzau and Quirk 2010) and is also the subject of numerous publications.

III. ENVIRONMENTAL CHEMISTRY

Strontium can exist in two oxidation states: 0 and +2 (written as Sr^0 and Sr^{2+}), although under normal environmental conditions, the +2 oxidation state dominates (ATSDR 2004). Because strontium and calcium are both alkaline earth metals in group IIa of the periodic table of the elements, their chemical properties are similar and, as one would expect, their metabolism is similar (see Eisenbud and Gesell 1997). The metabolism of Sr-90 has been extensively studied. As described in NCRP (1991):

“... after radiostrontium is ingested, a fairly substantial part is absorbed from the gastrointestinal tract... That which is absorbed is (a) deposited in the bone volume; (b) distributed in the exchangeable pool which can be considered to be comprised on the plasma, extracellular fluid, soft tissue and bone surfaces; or (c) removed from the body by urinary and fecal excretion.”

Because of the similarity in the chemistries of the two elements, their biokinetics are qualitatively similar and the concentrations of Sr-90 in tissues have been reported as a Sr/Ca ratio (i.e., pCi of Sr-90 per gram of Ca), referred to as the “sunshine unit” in the early years of the weapons testing program (NCRP 1991).

FATE AND TRANSPORT

Air

Strontium released to the atmosphere can be transported and redeposited by either dry or wet deposition. ATSDR (2004) states: “Dry deposition results from gravitational settling, impact, and sorption on surfaces (NCRP 1984). Experimental data on dry deposition of strontium, present in the ambient atmosphere, is limited. Rain, sleet, snow, or other forms of moisture can wash airborne particles containing strontium from the atmosphere by the process of wet deposition. Wet deposition depends on conditions such as particle solubility, air concentration, rain drop size distribution, and rain fall rate (NCRP 1984). Hirose *et al.* (1993) examined the mechanism of aerial deposition of ^{90}Sr derived from the Chernobyl accident, and found that 96% of atmospheric ^{90}Sr returned to earth as wet deposition.”

Water

In water, most forms of strontium are dissolved (ATSDR 2004), but as discussed below, strontium will tend to bind to suspended and deposited sediment and organic detritus.

Soil and Sediment

Strontium “has moderate mobility in soils and sediments, and sorbs moderately to metal oxides and clays (Hayes and Traina 1998)” (ATSDR 2004). Partition coefficients (K_d values) are a measure of the strength of Sr-90’s binding to soil and sediments, and therefore its potential for movement in soil and the subsurface.¹²¹

ATSDR (2004) summarizes literature that altogether has reported a wide range of K_d values for Sr^{2+} , ranging from 4.7 to 496 L/kg. At Hanford, K_d values of 15-40 L/kg were measured for Sr^{2+} -90 in aquifer sediments near Liquid Waste Disposal Facilities (DOE 1996 as cited in ATSDR 2004).

ATSDR (2004) notes that the range in these K_d values reflects “differences in soil and sediment conditions as well as the analytical techniques used (Bunde *et al.* 1997).” In particular, factors such as soil type, organic matter content, the presence of calcium (Ca^{2+}) and magnesium (Mg^{2+}) ions, can affect the tendency of strontium to precipitate (e.g., as strontium carbonate or as Sr^{2+} -organic matter complexes) and can affect its overall mobility (ATSDR 2004). It is also important to recognize limitations of K_d values, as summarized by Smith and Amonette (2006), who explain that any measured K_d reflects only the very specific conditions under which those measurements were made.

In addition to the literature cited in ATSDR (2004), there are many publications on the behavior of Sr-90 in terrestrial ecosystems. For example, Schultz and Riedel (1961) describe investigations that showed that, as Sr-90 in soil ages, it becomes less available for uptake by plants. They also cite studies that show availability for uptake remains essentially the same with time. Hence, it is difficult to make general statements about the mobility of Sr-90 in terrestrial ecosystems.

Animals can also play a role in terrestrial transportation of Sr-90: “Subsurface ^{90}Sr can be transported from soil to top soil by burrowing animals, and is spread to the surrounding environment via animal tissues and fecal deposits. At the Subsurface Disposal Area at the INEL [Idaho National Laboratory], deer mice had the highest contamination of all animals from ingestion of ^{90}Sr -contaminated low level nuclear waste. In addition, the biotic intrusion of soils covering the waste site brings water infiltration into buried LLW (Arthur and Janke 1986)” (ATSDR 2004).

BIOACCUMULATION POTENTIAL

Since strontium is chemically and biochemically similar to calcium, strontium is readily taken up by biota and transported up the food chain. As discussed below, higher terrestrial and aquatic organisms (i.e., vertebrates) have a relatively high potential to accumulate Sr-90 because strontium is a chemical congener of calcium and is bioconcentrated in bone. However, also as discussed below, many plants do not

¹²¹ The partition coefficient is expressed as follows: $K_d = C_s/C_l$ where:

K_d = the partition coefficient of a given element in soil,

C_s = the average concentration of a given element in soil in contact with the water for sufficient time to achieve equilibrium, and

C_l = the average concentration of the element in water.

have high bioaccumulation factors because of their lesser dependence on calcium as an essential nutrient as compared, for example, to vertebrates.

Table 6-2 of ATSDR (2004) summarizes bioconcentration factors for Sr-90 measured at the Savannah River Site. These range from less than one to 13 for terrestrial plants but for aquatic plants range from 2,100 to 9,400. In other aquatic fish and invertebrates, values of approximately 60,000 have been measured. Overall, “The study illustrates that the organisms with the highest uptake are aquatic organisms such as fish (large-mouthed bass), macroinvertebrates (insects), macrophytes (white-water lilies and bladderwort), and zooplankton. Because of the similarity of strontium to calcium, boney fish had a very high BCF, with a value >50,000 measured in the boney tissue (Friday 1996). In the muscle tissue of boney fish, BCF values for ⁹⁰Sr ranged from high (benthic invertebrate and fish feeders; 610) to very high (piscivores; 3,400). Because strontium and calcium are chemically similar, the concentration of calcium in water can influence the bioaccumulation of strontium in biota. Organisms such as fish bioaccumulate strontium with an inverse correlation to levels of calcium in water. However, this correlation is not universal and does not apply to other organisms such as algae and plants (NCRP 1984).”

For plants, as presented in ATSDR (2004):

“Strontium is not necessary for growth or reproduction for most plants, but is typically absorbed to satisfy the plant’s metabolic requirements for calcium (NCRP 1984). Soil to plant concentration ratios for strontium (the ratio of the concentration of strontium in wet vegetation to the concentration of strontium in dry soil) are 0.017–1.0 (NCRP 1984), and indicate that strontium can be easily absorbed into plants from soil. The uptake of strontium by plants is greatest in sandy soils having low clay and organic matter content (Baes *et al.* 1986). The concentration of nutritive mineral elements in soil such as calcium lower the intake of strontium to the aboveground phytomass. The average reduction of the soil-to-plant concentration ratios for ⁹⁰Sr caused by amendment with Ca or K is around 50–60% (Lembrechts 1993). Strontium may be deposited on plant surfaces from the atmosphere, remain on the plant, be washed off, or be absorbed directly into the plant through leaves. Contamination by direct deposition on foliage surfaces is predominantly a short-term mechanism with a weathering half-life of approximately 14 days (Lassey 1979).”

ACCUMULATION WITHIN TISSUES

Strontium is a metabolic analog of calcium (Eisenbud and Gesell 1997) and therefore tends to accumulate in tissues rich in that mineral. In bony vertebrates, strontium-90, whether absorbed from the lung, gastrointestinal tract, or bloodstream (dermal exposure), in large part becomes deposited in the bone (Driver 1994). Any tissue where calcium is deposited will have relatively high concentrations of Sr-90 (egg shells, milk, exoskeletons, etc.). In fact, as described in Eisenbud and Gesell (1997), it was common practice to express the concentration of Sr-90 in a given sample in terms of pCi of Sr-90 per gram of calcium in the sample. In addition, the radioecological literature often derived what is referred to as the “observed ratio”

(OR). The OR is the concentration of Sr-90 in a given biotic sample relative to the concentration of calcium in that sample (i.e., pCi/g of Sr-90 per gram of calcium) divided by that same ratio as measured in the organism's precursor food item. An example is the OR for plants as compared to soil was reported at about 0.7; for human bone to diet, it was reported to be about 0.15; from diet to cow milk it was reported as 0.15 (Eisenbud and Gesell 1997). The OR is a useful metric because it allows one to predict the concentration of Sr-90 in a given organism if there is knowledge of the concentration of Sr-90 and calcium in its diet.

ATSDR (2004) also discusses the transport and accumulation of strontium in plants:

“Carini *et al.* (1999) examined the mechanism of translocation in three species of fruit-bearing plants exposed to aerial deposition of ⁸⁵Sr and found that translocation of ⁸⁵Sr is localized to the area of contamination on the plant. However, uptake of strontium through the leaves is minor compared to root uptake. Once absorbed in the plant, strontium translocates to other parts of the plant, such as the leaves or fruit. Translocation of strontium in plants is affected by the particular species and stage of organism growth, and the most metabolically active parts (growing) will accumulate higher concentrations of strontium (Kodaira *et al.* 1973).

Strontium, taken up by plants and translocated to the above-ground plant compartments, has been observed for deep-rooted plants, such as chasima (*Chrysothamnus nauseosus*), mulberry vegetation (*Morus alba*), quaking aspen (*Populus tremuloides*), and red maple (*Acer rubrum*) growing on top of low-level waste burial sites or contaminated soils (Cooper and Rahman 1994; DOE 1995; Fresquez *et al.* [1996]).”

IV. TYPICAL MAJOR EXPOSURE ROUTES

As discussed above, terrestrial plants take up Sr-90 primarily through their roots. Higher terrestrial and aquatic organisms accumulate Sr-90 primarily via the ingestion of contaminated food (NCRP 1991; Eisenbud and Gesell 1997).

The following was excerpt from ATSDR (2004) describes the major exposure routes of strontium for humans, which includes inhalation and diet. This general observation is also applicable to other terrestrial vertebrates. For fish, as described above, uptake is primarily from its diet.

“If a person breathes in vapors or dust containing a chemical form of strontium that is soluble in water, then the chemical will dissolve in the moist surface inside the lungs and strontium will enter the bloodstream relatively quickly. If the chemical form of strontium does not dissolve in water easily, then particles may remain in the lung for a time. When you eat food or drink water that contains strontium, only a small portion leaves the intestines and enters the bloodstream. Studies in animals suggest that infants may absorb more strontium from the intestines than adults. If a fluid mixture of a strontium salt is placed on the skin, the strontium will pass through the skin very slowly and then enter the bloodstream. If the skin has scratches or cuts, strontium will pass through the skin much more quickly. Once

strontium enters the bloodstream, it is distributed throughout the body, where it can enter and leave cells quite easily.”

V. RADIOLOGICAL ECOTOXICITY

In theory, biota can be damaged by both the chemical toxicity and radiotoxicity of Sr-90. However, the specific activity of Sr-90 is relatively high and its chemical toxicity is relatively low. For example, with respect to stable strontium, Driver (1994) states “strontium toxicity to copepods is low. The 48-h LC₅₀ of strontium in the copepods (*Cyclops abyssorum* and *Eudiaptomus padanus*) is 300 mg/L and 180 mg/L respectively. Cladoceran sensitivity to strontium is also moderate (75 mg/L, 48-h LC₅₀) (Baudouin and Scoppa 1974).” ATSDR (2004) explains that, “In acute exposure studies in mice, the oral LD₅₀ for strontium nitrate was reported to be 2,350 mg strontium/kg in males (Llobet *et al.* 1991a)... No studies were located regarding death in animals following chronic-duration oral administration of stable strontium.”

It is important to recognize that the specific activity of Sr-90 is 139 Ci/gram or 0.139 Ci per mg.¹²² Hence, a single mg of Sr-90 is highly radioactive and extremely radiotoxic. As a result, the chemical toxicity of Sr-90 is of little concern relative to its radiotoxicity.

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

There are no beneficial or protective properties associated with exposure to Sr-90.

MECHANISMS OF ACTION

Radioecological damages to aquatic and terrestrial organisms due to Sr-90 in the environment result from ionization caused by the interaction of its beta particles with living tissue. As noted previously, upon each disintegration, Sr-90 and its progeny, Y-90, emit a beta particle with an average energy of about 0.196 and 0.935 MeV, respectively (Shleien *et al.* 1998).

In higher organisms, Sr-90 will be metabolized as if it were calcium. In bony animals, therefore, Sr-90 is deposited in large part in bone (ATSDR 2004), and many of its effects are associated with that tissue. However, depending on the organism and its diet and ecological niche, exposure to the beta emissions from Sr-90 and its progeny, Y-90, can be to any tissue in any stage of its life cycle. Hence, the mechanism of action is very much dependent on a multitude of factors which are highly site- and organism-specific. For example, in addition to damage to bone, the mechanism of action could also be effects on fish eggs sitting in sediment, damage to fish gills, and damage to plant root hairs, to name a few potential mechanisms of action.

Internal Beta Exposures

Figure 5.8.1 of Shleien *et al.* (1998) indicates that beta particles of this energy have a range in water (which is equivalent to tissue in terms of stopping power) of about 0.5 cm²/g, which is about 1 cm in tissue. Given that the typical energy required to

¹²² The equation used to derive specific activity is provided on page 3-17 of Schleien *et al.* (1998).

ionize a molecule (i.e., eject an electron from its orbit) is about 34 to 35 eV (see Casarett 1968, page 17), the total number of ion pairs produced by the energy deposited in tissue from the typical energy beta particle emitted by Sr-90/Y-90 is about 29,000 ion pairs (i.e., ~1 MeV/35 eV).

The pattern of energy deposition for beta particles is described in Morgan and Turner (1973) as follows:

$$\text{Mean linear ion density} = T/R_t \times W$$

Where:

T = average energy of electron liberated

R_t = range or electrons of energy T

W = average energy to form an ion pair

For Sr-90, the equation is $1 \text{ MeV} \times 1,000,000 \text{ eV/MeV} \div 0.5 \text{ cm} \times 35 \text{ eV/ion pair} = 5.7 \times 10^4$ ion pairs per cm or about 5.7 ion pairs per micron. Given that a typical cell is on the order of tens of microns (see page 102 of Curtis and Barnes 1989), a single cell might experience about 50 to 60 ion pairs produced by the passage of a typical Sr-90 beta particle. It is this deposited energy in living tissue that results in biological damage.

External Beta Exposures

Sufficiently energetic beta particles can penetrate the dead layer of the skin of mammals (nominally 70 microns in humans) and deposit energy in underlying tissues. Thus, there is a real potential for exposure to terrestrial organisms from external radiation from beta particles emitted by Sr-90, except for organisms that have a thick outer layer (such as bark of trees, heavy fur, etc.) that can shield the living tissue beneath from the beta emissions. In theory, aquatic organisms can also experience external exposure from beta particles but, due to the limited range of Sr-90 beta particles in water (about 1 cm), only Sr-90 in very close proximity to the organisms can result in exposure to living tissue.

FACTORS AFFECTING TOXICITY

There are numerous environmental factors can enhance or reduce the potential for biota to be exposed to Sr-90. As discussed above, the uptake of radiostrontium can be enhanced or reduced by the amount of calcium in the environment or in an organism's diet. Also, if strontium is tenaciously bound to soil and sediment, it is less likely to be available for uptake by biota.

PLANTS

No studies were found on the effects of Sr-90 on plants, but there is an abundance of literature on the effects of radiation in general on plants and plant communities. Although most of these studies address external exposure to gamma radiation, if the exposure is expressed in units of dose (i.e., rad and for both internal and external exposures), these studies have applicability to internal exposure to Sr-90 (expressed in units of rad) because both gamma and beta radiation have a similar linear energy

transfer. In other words, whether the exposure is to external gamma or internal beta, the effects of comparable exposures, expressed in units of rad, should be similar.

A classic series of investigations on the effects of external gamma ionizing radiation on plant communities was performed at Brookhaven National Laboratory in Upton, New York, in 1962. A large (9,500 Ci) Cs-137 source was placed in a pine forest for 20 hours per day, where the external exposures ranged from several thousand R¹²³ per day within a few meters of the source to about 1 rad per day at 130 meters from the source. After 6 months of exposure, a total kill zone was observed at a dose of >350 R/day. At 10 R/day, there was reduced shoot growth of all tree species, but no trees died (Casarett 1968).

Chapter 13 of Casarett (1968) provides an excellent review of the literature on the effects of radiation on higher plants and plant communities. She provides data showing the percent germination for pollen for a variety of plants, as a function of dose, where the doses ranged from zero to over 6,000 rad. She also summarizes studies on the effects of radiation on the fertilized egg (ovule), where effects on the developing plant were observed at 500 R, and the radiosensitivity of developing embryos (fertilized ovule) varied 100-fold depending on plant species.

Casarett (1968) also presents the results of investigations performed by Sparrow and Woodwell (1962), where the effects of chronic exposure to Co-60 were measured. The effects included 10% growth reduction, failure to set seed, 50% growth reduction, pollen sterility, floral inhibition or abortion, severe growth inhibition, and LD₅₀ and LD₁₀₀.

Driver (1994) summarizes the general literature at that time regarding the effects of radiation on terrestrial plants (including the Brookhaven experiments):

“Plants are relatively resistant to ionizing radiation. [*It should be noted that experience following the Chernobyl accident found pine trees to be radiosensitive, see below.*] The effects of chronic irradiation (6 months) of a late successional oak-pine forest were studied at Brookhaven National Laboratory (BNL) in New York. Changes in ecosystem structure, diversity, primary production, total respiration, and nutrient-inventory occurred. The most resistant species were the ones commonly found in disturbed places, i.e., generalists capable of surviving a wide range of conditions. Mosses and lichens survived exposures greater than 1000 R/d. No higher plants survived greater than 200 R/d. Sedge (*Carex pennsylvanica*) survived 150 to 200 Rad. Shrubs (*Vaccinium* and *Quercus ilicijolia*) survived 40 to 150 R/d. Oak trees survived up to 40 R/d, whereas pine trees were killed by 16 R/d. No change was noted in the number of species in an oak-pine forest up to 2 R/d, but changes in growth rates were detected at exposures as low as 1 R/d (Woodwell 1970). Severe defects were observed in *Tradescatia* at an exposure rate of 40 R/d. However, an exposure of 6000 R/d was required to produce the same effect in a hybrid gladiolus (Odum 1956). The sensitivity of various plant species appears to be related to the cross-sectional area of the nucleus in relation to cell

¹²³ For simplicity, it can be assumed that one R or Roentgen is equal to 1 rad (or 100 ergs of energy deposited per gram of tissue).

size: the larger the nucleus and chromosome volume, the more sensitive the plant (Underbrink and Sparrow 1968, 1974).”

Table 2.2 of Driver (1994) provides a tabulation of the effects of chronic exposure to external radiation on a variety of plants. The exposure levels at which no effects were observed ranged from 10 R/day for the lily to 1720 R/day for the wood rush. These exposures were performed under controlled experimental conditions.

Also noteworthy are investigations of the damage done to conifer forests in the vicinity of the 1986 Chernobyl accident. Radiation resulted in the death of many pine stands within approximately 5-10 km of the power plant, resulting in the so-called “red forest.” In addition to mortality, adverse effects observed in the forest included reproduction anomalies, growth reductions, and morphological damage (*ibid.*). The absorbed dose was largely due to beta radiation (90%), with some contribution from gamma radiation (10%), and four distinct zones of damage were identified, with different dose levels associated with different severities and types of injury (see Table 6.3 in IAEA 2006).

AQUATIC INVERTEBRATES AND FISH

Very little literature was found that specifically addresses the radiotoxicity of Sr-90 on aquatic organisms. There is, however, an abundance of publications on the effects of radiation in general on aquatic organisms. Of note, most of these studies involved external exposures to gamma radiation or internal exposure to internal gamma and beta radiation. The internal exposure investigations are probably more applicable to this profile, because Sr-90/Y-90 is a pure beta emitter and the vast majority of exposure would be internal exposure to beta emissions. However, most of the experimental work addresses uniform whole-body exposure to beta and gamma (both internal and external), while the concern with Sr-90 for most higher organisms is the localized deposition of beta particle energy in bone.

For organisms where Sr-90 does not concentrate in calcium rich tissue but is distributed more uniformly in soft tissue, the effects would be more like those observed for external or internal uniform exposures. Hence, the literature on the effects of radiation exposure in general, when expressed in units of rad, has some applicability to internal exposure of biota to Sr-90.

NCRP (1991) provides an extensive review on the reproductive effects of radiation on in fish and invertebrates in natural and experimental settings. Tables 3.3 to 3.8 of the report summarize the extensive literature on this subject. Data are available on many life stages of the mosquito fish, roach, pond snail daphnia, Chinook salmon, coho salmon, stickleback, pike, rainbow trout, guppy, and medaka. This report concludes that:

The discharge of low-level radioactive effluents into the aquatic environment has resulted in chronic, low dose rate exposure aquatic organisms. The fate of individual organisms is, generally, not the major concern but rather the response and maintenance of endemic populations.

Experimental studies to date have shown that fertility and fecundity (gametogenesis) of the organisms and embryonic development are probably the most sensitive components of the radiation response, and it is precisely these attributes which are of importance in determining the fate of the population.

Driver (1994) summarizes the literature on the effects of radiation on aquatic organisms, providing LD₅₀ values fish (90,000 R), 50% survival doses for male and female germ cells (305 to 500 R), and reduction on population growth rate for white crappie, largemouth bass, and redhorse (25% reduction at 57 R external exposure).

Driver (1994) also references studies on the effects of radiation (not specifically Sr-90) on crustaceans, snails, and daphnia, where effects of exposure to radiation were observed but only at very high dose rates (hundreds to thousands of rad). Casarett (1968)¹²⁴ similarly states that “Most invertebrates have been shown to be more resistant than vertebrates [to the effects of ionizing radiation].”

BIRDS

Driver (1994) provides a review of literature specifically citing studies on the effects of Sr-90 on birds at Hanford, as follows:

“Radiostrontium levels of up to 1700 and 560 pCi/kg ash of the eggshells and inner egg contents, respectively, have been found in Canada goose eggs on the Hanford Site (Rickard and Sweany 1977). No impacts on clutch size, hatching success, viability of the young, or population parameters have been associated with these levels of contamination when compared to uncontaminated goose populations.”

Although few studies were found on the effects of Sr-90 on birds, some information is available on the effects of radiation in general on birds and avian communities, and this has some applicability to Sr-90.

For example, Casarett (1968) cites an LD₅₀ of 800 rad for birds, which is higher than that for mammals but lower than that for fish. Møller and Mousseau (2007a) report on a census of the bird populations in the vicinity of Chernobyl and correlated the varying levels of “background” radiation with bird abundance. They found a decreasing bird population density with increasing background radiation, where the background radiation in the vicinity of Chernobyl varied from 0.04 to 135.89 mR/hr. (To place these values into perspective, typical background radiation is about 0.01 mR/hr.) Raptor abundance was also reduced in contaminated areas (Møller and Mousseau 2009b). Also of note, Møller and Mousseau (2009a) found that the abundance of insects and spiders (prey items for some birds) to be negatively correlated to radiation exposure around Chernobyl.

Reasons for differential interspecific responses to radiation in birds have also been explored. Møller *et al.* (2010) found species that responded more strongly to the impact of the Chernobyl radiation to be those that in the past were more susceptible to

¹²⁴ Although this publication is highly authoritative, it is also quite dated. There is certainly more information now available, especially after the Chernobyl accident. Hence this table should be used only to obtain a general sense of the sensitivity of different types of organisms to the acute effects of radiation.

factors causing mutations in mitochondrial DNA. Møller and Mousseau (2007b) investigated the role of antioxidants (which are used by the body to neutralize free radicals and reduce the negative effects of radiation). The authors identified associations between increased use of antioxidants in certain species (e.g., those that migrate, have greater dispersal distances, larger relative egg sizes, and carotenoid-based plumage) and radiation sensitivity as indicated by relative abundance around Chernobyl. Galvan *et al.* (2010) found that highly pigmented birds were more adversely affected (i.e., reduced population density) by the radiation in the vicinity of Chernobyl than less pigmented birds. The article hypothesizes that this may be because ionizing radiation depletes antioxidants, which are required in greater quantities in more highly pigmented birds. Møller *et al.* (2011) further explored the issue of radiation levels and the abundance of birds and eight other taxa (spiders, dragonflies, grasshoppers, bumblebees, butterflies, amphibians, reptiles, and mammals) and found statistically significant effects of radiation on all groups, with effect sizes being larger in taxa with longer natal dispersal distances and higher population densities.

MAMMALS

Driver (1994), HSDB, and ATSDR (2004) all provide reviews of large numbers of studies on the effects of Sr-90 on mammalian species. Information specific to wild species and to field conditions is, however, extremely limited. The vast majority of studies represent laboratory studies on domestic animals such as dogs, mice, rats, mice, swine, and cows. Exposure routes used in these studies include inhalation (to aerosol and particulate-bound forms), dermal exposure, and ingestion through food or drinking water. Endpoints evaluated include death, carcinogenic effects, and also immunological, neurological, reproductive, and developmental effects.

Exposure through ingestion and aerosol-based inhalation resulted in the rapid passage of Sr-90 through the body and into the skeleton, where the radionuclide becomes associated with bone (ATSDR 2004), and many of the observed adverse effects are associated with this partitioning within the body and consequent skeletal radiation exposure. In many studies, younger animals were more sensitive to these effects than were older animals, and some studies noted age-related differences in incorporation of Sr-90 into skeletal tissues, with juveniles having higher skeletal activities than adults (ATSDR 2004).

The only identified study on a non-domestic or laboratory species is described in Driver (1994): “Tumorigenicity has also been observed in wild rodents. A muskrat from White Oak Lake that had more than 1 Jlc of strontium per gram of bone, a total body burden of nearly 100~Ci (Krumholz and Rust 1954), displayed advanced osteogenic sarcoma with metastasized cells to both kidneys and lungs.”

VI. EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

ATSDR (2004) does not cite interactions of Sr-90 with other chemicals that might influence the toxicity of Sr-90. However, concerns over the possible synergistic effects of exposure to radiation and chemical toxins have been extensively raised and reported in the scientific literature (Burkart *et al.* 1997; Prasad *et al.* 2004), but little

consensus has been achieved in quantifying these effects in humans, except possibly for radon and smoking (BEIR IV 1988) and certainly in the enhancement of the therapeutic effects of radiotherapy used to treat cancer (e.g., Lew *et al.* 2002). UNSCEAR (2000) Annex H explores the combined effects of radiation and chemical agents, including heavy metals. Only a few data are available from combined exposures of radiation and metals in human populations and no firm evidence of interactions has been observed.

As summarized in ATSDR (2004), there is some literature on observed synergistic adverse effects of radiation and toxic chemicals on organisms other than humans [e.g., salmon (Mothersill *et al.* 2007)]. Examples of ionizing radiation and metals producing combined effects in other biological systems include synergistic effects on soil microbial activity from cadmium and zinc in combination with gamma radiation (summarized in UNSCEAR 2000). Furthermore, although not specific to Sr-90, combined effects of cesium-134/137 (another beta emitter) and lead found in highly contaminated habitats in the Russian Federation increased the mutation rate in the plant *Arabidopsis thaliana* (summarized in UNSCEAR 2000). However, the authors clearly indicate that the relative importance of different damage-inducing mechanisms of metals for combined exposures in human and non-human populations remains to be elucidated.

ATSDR (2004) states that, overall, there is a clear need for additional research on synergistic effects of multiple stressors in radioecotoxicology (e.g., Salbu and Skipperud 2007; Mothersill and Seymour 2007). In particular, these authors raise the issue of pesticides, organics, and endocrine disruptors and synergistic effects with radioactive materials, particularly with long-term exposure to various biological systems. Manti and D'Arco (2010) summarize the *in vitro* and animal-model studies and epidemiological surveys with two or more stressors, including radionuclides (DNA-damaging agents). They also emphasize that most research focuses only on the short-term effects of combined single exposures to animal models, and more work is needed to understand chronic exposure to trace contaminants and radioactive elements in the environment, including impacts to long-term genome stability. Specific research is lacking on Sr-90's effects with multiple stressors on biological systems, particularly non-human systems.

VII. DATA GAPS Information on the effects of Sr-90 in domestic/laboratory mammals is relatively extensive; however, effects data for other taxa are far fewer. For example, this profile identified no Sr-90 specific effects information for plants, aquatic organisms, reptiles, or amphibians, and it identified very little Sr-90 effects information for birds and wild mammals. Also, as described above, little is known about the combined action of exposure to radiation and other environmental toxicants.

That said, some research on the effects of radiation has applicability to Sr-90, especially studies of exposure from internal or external beta or gamma emitters, if the exposure is expressed in units of dose (i.e., rad). The research on the effects beta and gamma radiation in general on terrestrial and aquatic organisms is more substantial.

VIII. REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Toxicological Profile for Strontium. Agency for Toxic Substances and Disease Registry. April 2004.
- Arthur, W.J. and D.H. Janke. 1986. Radionuclide concentrations in wildlife occurring at a solid radioactive waste disposal area. *Northwest Sci* 60(3):154-165.
- Baes, C.F., C.T. Garten, and F.G. Taylor. 1986. Long-term environmental problems of radioactively contaminated land. *Environ Int* 12:543-553.
- Baudouin, M.F. and P. Scoppa. 1974. Toxicity of heavy metals for freshwater zooplankton: Influence of some environmental factors. *Boll. Zool.* 41(4):457.
- BEIR IV. 1988. Biological Effects of Ionizing Radiation. Health Risks of Radon and Other Internally Deposited Alpha-Emitters. National Research Council. National Academy of Sciences. Washington, D.C. 1988.
- Bunde, R.L., J.J. Rosentreter, and M.J. Liszewski. 1997. Effects of calcium and magnesium on strontium distribution coefficients. *Environ Geol* 32(3):219-229.
- Burkart, W., G.L. Finch, and T. Jung. 1997. Quantitative health effects from the combined action of low-level radiation and other environmental agents can new approaches solve the enigma. *Science of the Total Environment* 205(1): 51-70.
- Carini, F., I.A. Scotti, and, P.G. D'Alessandro. 1999. ^{134}Cs and ^{85}Sr in fruit plants following wet aerial deposition. *Health Phys* 77(5):520-529.
- Casarett, A.P. 1968. *Radiation Biology*. Englewood Cliffs, New Jersey: Prentice-Hall, Inc.
- Cooper, E.L., and M.M. Rahman. 1994. A study of cycling of ^{90}Sr in a natural forest on the Canadian Shield. *Sci Total Environ* 157:107-113.
- Curtis, H. and N.S. Barnes. 1989. *Biology*. Fifth Edition. Worth Publishers, Inc.
- Downie, E.D., S. Macpherson, and E.N. Ramsden. 1959. The effect of daily feeding of ^{90}Sr to rabbits. *Br J Cancer* 13:408-423.
- Driver, C.J. 1994. Ecotoxicity Literature Review of Selected Hanford Site Contaminants. PNL-9394, Pacific Northwest Laboratory, Richland, Washington. <http://www.osti.gov/energycitations/servlets/purl/10136486-6sLptZ/native/10136486.pdf>.
- Eisenbud, M. and T. Gesell. 1997. *Environmental Radioactivity from Natural, Industrial, and Military Sources*, Fourth Edition, Academic Press.
- Fresquez, P.R., T.S. Foxx, and L. Naranjo. 1996. Uptake of Strontium by Chamisa (*Chrysothamnus nauseosus*) Shrub Plants Growing over a Former Liquid Waste Disposal Site at Los Alamos National Laboratory: Proceedings of the HSRC/WERC Joint Conference on the Environment. Los Alamos, New Mexico: Los Alamos National Laboratory.

- Friday, G.P. 1996. Radiological Bioconcentration Factors for Aquatic Terrestrial and Wetland Ecosystems at the Savannah River Site. Aiken, South Carolina: U.S. Department of Energy. DE-AC09-89SR18035. WSRC-TR- 96-0231.
- Galvan, I., T.A. Mousseau, and A.P. Møller. 2010. Bird population declines due to radiation exposure at Chernobyl are stronger in species with pheomelanin-based coloration. *Physiological Ecology*, 165(3): 579-851, December 2010.
- Gephart, R.E. and R.E. Lundgren. 1998. Hanford tank cleanup: a guide to understanding the technical issues. Columbus, Ohio: Battelle Press.
- Glasstone, S. and P.J. Dolan. 1977. The Effects of Nuclear Weapons, Third Edition. Prepared and Published by the United States Department of Defense and the Energy Research and Development Administration. U.S. Government Printing Office, Washington, DC.
- Hanson, L.A. 2000. Radioactive Waste Contamination and Groundwater at the Hanford Site, Principles of Environmental Toxicology, University of Idaho, November 2000.
- Hayes, K.F. and S.J. Traina. 1998. Metal ion speciation and its significance in ecosystem health. In: *Soil Chemistry and Ecosystem Health*. Soil Science Society of America, Special Publication No. 52. Madison, Wisconsin: Soil Science Society of America, 46-83.
- Hirose, K., S. Takatani, and M. Aoyama. 1993. Wet deposition of radionuclides derives from the Chernobyl accident. *J Atmos Chem* 17:16-71.
- International Atomic Energy Agency (IAEA). 2006. Environmental Consequences of the Chernobyl Accident and their Remediation: Twenty Years of Experience. Report of the Chernobyl Forum Expert Group 'Environment'. Radiological Assessment Reports Series. Vienna.
- Kodaira, K., A. Tsumura, and H. Kobayashi. 1973. Uptake of radioactive strontium and cesium in rice plants: (1) Accumulation of Sr and Cs in rice grains through roots. *J Radiat Res* 14:31-39.
- Krumholz, L.A., and J.H. Rust. 1954. Osteogenic sarcoma in the muskrat from an area of high environmental radiostrontium. *AMA Arch. Path.*11:270.
- Lassey, K.R. 1979. The transfer of radiostrontium and radiocesium from soil to diet: Models consistent with fallout analyses. *Health Phys* 37:557-573.
- Lembrechts, J. 1993. A review of literature on the effectiveness of chemical amendments in reducing the soil-to-plant transfer of radiostrontium and radiocesium. *Sci Total Environ* 137:81-98.
- Lew, Y.S., A. Kolozsvary, S.L. Brown, and J.H. Kim. 2002. Synergistic Interaction with Arsenic Trioxide and Fractionated Radiation in Locally Advanced Murine Tumor. American Association for Cancer Research.
- Linza, W. and R. Quirk. 2010. Memo to T.J. Dwter, Technical Director, Hanford activity report for the week ending November 19, 2010. November 19.

- Llobet, J.M., M.T. Colomina, and J.L. Domingo. 1991a. Effect of chelating agents on tissue distribution and excretion of strontium following semichronic strontium ingestion. *Res Commun Chem Pathol Pharmacol* 71(2):243-246.
- Manti, L. and A. D'Arco. 2010. Cooperative biological effects between ionizing radiation and other physical and chemical agents. *Mutation Research* 704:115-122.
- Mikhailov, V.N. editor in Chief. 1999. *Catalogue of Worldwide Nuclear Testing*. Begell-atom, LLC.
- Møller, A.P. and T.A. Mousseau. 2011. Efficiency of bio-indicators for low-level radiation under field conditions. *Ecological Indicators* 11(2):424-430.
- Møller, A.P., J. Erritzoe, F. Karadas, and T.A. Mousseau. 2010. Historical mutation rates predict susceptibility to radiation in Chernobyl birds. *Journal of Evolutionary Biology* 23(10):2132-2142.
- Møller, A.P. and T.A. Mousseau. 2007a. Species richness and abundance of forest birds in relation to radiation at Chernobyl. *Biology Letters of the Royal Society* 3: 483-486.
- Møller, A.P. and T.A. Mousseau. 2007b. Determinants of interspecific variation in population declines of birds after exposure to radiation at Chernobyl. *Journal of Applied Ecology* 44: 909-919.
- Møller, A.P. and T.A. Mousseau. 2009a. Reduced abundance of insects and spiders linked to radiation at Chernobyl 20 years after the accident. *Biology Letters of the Royal Society* 5(3): 356-359.
- Møller, A.P. and T.A. Mousseau. 2009b. Reduced abundance of raptors in radioactively contaminated areas near Chernobyl. *Journal of Ornithology* 150(1):239-246.
- Morgan, K.Z. and J.E. Turner. 1973. *Principles of Radiation Protection, A Textbook of Health Physics*. Huntington, New York: Robert E. Krieger Publishing Company.
- Mothersill, C., B. Salbu, L.S. Heier, H.C. Teien, J. Denbeigh, D. Ougton, B.O. Rosseland, and C.B. Seymour. 2007. Multiple stressor effects of radiation and metals in salmon (*Salmo salar*). *Journal of Environmental Radioactivity* 96(1-3): 20-31.
- Mothersill, C. and C. Seymour. 2007. Radiation risks in the context of multiple stressors in the environment – issues for consideration. In: Mothersill, C., I. Mosse, and C. Seymour, eds. *Multiple stressors: a challenge for the future*. Book Series: NATO Science for Peace and Security Series C – Environmental Security, pp. 235–246.
- National Council on Radiation Protection and Measurements (NCRP). 1984. *Radiological assessment: Predicting the transport, bioaccumulation, and uptake by man of radionuclides released to the environment*. NCRP Report No. 76.

Bethesda, Maryland: National Council on Radiation Protection and Measurements.

- National Council on Radiation Protection and Measurements (NCRP). 1991. Recommendations of the National Council on Radiation Protection and Measurements, Some Aspects of Strontium Radiobiology. NCRP Report No. 110, August 31, 1991.
- Odum, E.P. 1956. Ecological aspects of waste disposal. In: Proceedings from a Conference on Radioactive Isotopes in Agriculture. TID-7512, USAEC, Technical Information Center, Springfield, Virginia.
- Office of Technology Assessment (OTA). 1991. Complex Cleanup. The Environmental Legacy of Nuclear Weapons Production. Congress of the United States.
- Prasad, K.N., W.C. Cole, and G.M. Hasse. 2004. Health risks of low dose ionizing radiation in humans: A review. *Experimental Biology and Medicine* 229: 378-382.
- Rickard, W.H. and H.A. Sweany. 1977. Radionuclides in Canada goose eggs. In: *Biological Implications of Metals in the Environment*, pp. 623–627. Proceedings of the Fifteenth Annual Hanford Life Sciences Symposium at Richland, Washington, September 29–October 1, 1975. CONF-750929, U.S. Department of Energy, Washington, DC.
- Salbu, B. and L. Skipperud. 2007. Challenges in radioecotoxicology. In: Mothersill, C., I. Mosse, and C. Seymour, eds. *Multiple Stressors: A Challenge for the Future*. Book Series: NATO Science for Peace and Security Series C – Environmental Security, pp. 3–12.
- Shleien, B., L.A. Slaback, Jr., and B. K. Birky. 1998. *Handbook of Health Physics and Radiological Health* (Eds). Third Edition. Williams & Wilkins, A Waverly Company.
- Schultz, R.K, and H.H. Riedel. 1961. Effect of aging on fixation of strontium-90 by soils. *Soil Science* 91(4):262-264.
- Smith, B. and A. Amonette. 2006. *The Environmental Transport of Radium and Plutonium: A Review*. Institute for Energy and Environmental Research, June 23, 2006.
- Sparrow, G.M. and G.M. Woodwell. 1962. Prediction of sensitivity of plants to chronic gamma radiation. *Radiation Botany* 2:9-26.
- Tichler, J., N. Norden, and J. Congemi. 1988. *Radioactive Material Released from Nuclear Power Plants, Annual Report 1985*. Brookhaven National Laboratory, Prepared for the U.S. Nuclear Regulatory Commission, NUREG/CR-2907, BNL-NUREG-51528, Vol. 6.
- Underbrink, A.G. and A.H. Sparrow. 1974. The influence of environmental endpoints, dose, dose rate, neutron energy, nitrogen ins, hypoxia, chromosome

- volume and ploidy on RBE in *Tradescantia* stamen hairs and pollen. In: biological effects of neutron irradiation, pp. 185–214. International Atomic Energy Agency, Vienna, Austria.
- Underbrink, A.G., A.H. Sparrow, and V. Pond. 1968. Chromosome and cellular radiosensitivity. II. Use of interrelationships among chromosome volume, nucleotide content and dose of 120 diverse organisms in predicting radiosensitivity. *Radiation Botany* 8:205-237.
- United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2000. Sources and Effects of Ionizing Radiation. Report to the General Assembly, United Nations, New York.
- United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2008. Sources and Effects of Ionizing Radiation. Report to the General Assembly, United Nations, New York.
- U.S. Department of Energy (DOE). 1995. Radionuclide Concentrations in Terrestrial Vegetation and Soil on and Around the Hanford Site, 1983 Through 1993. Richland, Washington: U.S. Department of Energy. DE-AC06-76RLO1830.
- U.S. Department of Energy (DOE). 1995a. Department of Energy. Closing the Circle on the Splitting of the Atom. Office of Environmental Management. January 1995.
- U.S. Department of Energy (DOE). 1996. Strontium-90 Adsorption-desorption Properties and Sediment Characterization at the 100-Area. Richland, Washington: U.S. Department of Energy. DE-AC06-76RLO 1830.
- U.S. Department of Energy (DOE). 2005. Strontium-90 Treatability Test Plan for 100-R-2 Groundwater Operable Unit. DOE/RL-2005-96.
- U.S. Department of Energy (DOE). 1997. Department of Energy. Linking Legacies. DOE/EM-0319.
- Vermeul, V.R., B.G. Fritz, J.S. Fruchter, J.E. Szecsody, and M.D. Williams. 2009. 100-NR-2 Apatite Treatability Test FY09 Status: High Concentration Calcium-Citrate-Phosphate Solution Injection for In-Situ Strontium-90 Immobilization. PNNL-SA-70033, December 2009.
- Woodwell, G.M. 1970. Effects of pollution of the structure and physiology of ecosystems. *Science* 168(3930):429-433.

TECHNETIUM (Tc-99) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Technetium-99 (Tc-99) is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Technetium is primarily a man-made element that only occurs in the earth's crust in minute quantities (EPA 2002). All forms of technetium are radioactive; it is the lowest atomic number (43) element with no stable isotopes. Forty-three isotopes and isomers are thought to exist, with atomic mass ranging from 86 to 113 (HSDB 2008); according to Garcia-Leon (2005), 24 isotopes with 9 isomers are known (it appears that there is some ambiguity in the literature with regard to the number of isotopes). Technetium-99 (Tc-99) is the most common and readily available of all the isotopes, as it is generated in the fission of uranium (^{235}U) and plutonium (^{239}Pu) at a rate of about 6%. Tc-99 is a beta-emitter, $E_{\text{max}} = 294 \text{ keV}$, with a very long half-life of $T_{1/2} = 2.11 \times 10^5 \text{ yr}$ (Garcia-Leon 2005). Its short-lived gamma ray-emitting nuclear precursor, technetium-99m, is used in nuclear medicine for a wide variety of diagnostic tests due to its short half-life of a little over six hours (Krupke and Serne 2002).

II. SOURCES **NATURAL SOURCES**

No primordial Tc-99 is thought to exist because it would have decayed long ago, but there is non-primordial Tc-99 in minute quantities in the earth's crust from spontaneous fission, mainly of uranium-238 (EPA 2002, Schwochau 2000). In 1937, it was artificially produced and called technetium, then considered the first man-made chemical element. It is not naturally present in significant amounts (i.e., relative to the amounts in the environment due to nuclear programs activities), but was found in African rock samples in the early 1960s in trace amounts, confirming the presence of a natural nuclear reactor (Scerri 2009).

ANTHROPOGENIC SOURCES

Most Tc-99 in the environment comes from a few sources: 1) the detonation of nuclear weapons (especially atmospheric weapons tests), 2) nuclear reactor airborne emissions, 3) nuclear fuel reprocessing plant airborne emissions, and 4) facilities that treat or store radioactive waste (EPA 2002). The production and use of technetium compounds in nuclear medicine and as superconducting materials also may result in their release to the environment from various waste streams, although the medical industry uses 99-m isomer that decays in a matter of hours (HSDB 2008). Rough estimates indicate that, including all possible sources, at least 49,000-64,000 TBq had been produced worldwide by the mid-1990s (Garcia-Leon 2005, Yoshihara 1996).

In the absence of a local source, due to its low specific activity, estimated concentrations in surface soil tend to be very low—on the order of 0.0001 picocuries per gram (pCi/g) (ANL 2005).

Technetium in the environment surrounding the Hanford site originated from nuclear fuel cycle processes at that site. The Hanford operations included plutonium production and research reactors, chemical separation facilities, and fuel fabrication facilities, all of which involved processing and storing various uranium compounds, resulting in the production of technetium.

As technetium-99 is a product of nuclear fission, it is present at numerous areas throughout the Hanford Site including in groundwater; 2009 levels at and near the site are detailed in the Hanford Site Environmental Report (Poston *et al.* 2010). According to Poston *et al.* (2010) contaminant plumes totaling approximately 2.4 km² in area exceed the drinking water standard for Tc-99 at Hanford. The only wild plant tested for Tc-99, as reported in the most recent Hanford Site Environmental report (Poston *et al.* 2010) was an edible wild chive (*Allium schoenoprasum*) growing in riparian habitats along the Hanford Reach of the Columbia River. The samples did not contain Tc-99 above the minimum detectable limit. However, because Tc-99 is a low energy beta-emitter, it is more difficult to detect than gamma-emitting radionuclides, so some surveys for radionuclides are not designed to detect Tc-99 (e.g., most vegetation monitoring, Hanford Site Environmental Report, Poston *et al.* 2010). Exhibit 1 presents selected data on Tc-99 concentrations in environmental media.

EXHIBIT 1 SELECTED TECHNETIUM-99 CONCENTRATIONS IN WATER, SEDIMENTS, AND BIOTA

| | LOCATION | AMOUNT | SOURCE |
|---|-----------------------|--|-----------------------------------|
| Groundwater | DOE Idaho NL | 2000-2840 pCi/L | Idaho DEQ (2003) |
| | Savannah River Site | 200-1100 pCi/L | Carleton <i>et al.</i> (1993) |
| Surface Water | Savannah River Site | 0.42-0.58 pCi/L | Carleton <i>et al.</i> (1993) |
| Soil | Chernobyl 30 km zone | 1.1-14.1 Bq/kg dry wt org. soil 0.13-0.83 Bq/kg dry wt mineral layers | Uchida <i>et al.</i> (1999) |
| Biota | | | |
| <i>Potamogeton lucens</i> (aquatic plant) | Yenisei River, Russia | 82 Bq/kg biomass | Bolsunovskii <i>et al.</i> (2010) |
| Farmed salmon | Scotland | 0.11 Bq/kg max | Scottish EPA (2003) |

III. ENVIRONMENTAL CHEMISTRY

Krupke and Serne (2002 and 2003) summarize the extensive literature on technetium environmental chemistry. Technetium exists in valence states from +7 to -1. In natural environments, the most stable valence states of technetium are +7 and +4 (i.e., Tc(VII) and Tc(IV)) under oxidizing and reducing conditions, respectively. Dissolved technetium is present in aerobic systems as the aqueous Tc(VII) oxyanion species pertechnetate (TcO⁴⁻), which is essentially nonadsorptive (i.e., K_d values are ≈0

ml/g) at circumneutral and basic pH values and is highly soluble. Therefore, the concentration of Tc(VII) in the vadose zone and groundwater is not limited by adsorption or solubility processes, and it will be highly mobile in aerobic environments.

Under reducing conditions, technetium is present in the +4 oxidation state, which is not very soluble and is highly sorbed, usually existing in lower oxidative forms such as TcO_2 , $\text{TcO}(\text{OH})_2$, TcS_2 or in complexes with humic material (HSDB 2008, Multi-agency NUREG 2004). Technetium (IV) is considered to be essentially immobile in reducing subsurface environments. It is highly sorbed to iron and aluminum oxides and clays in this state (Krupke and Serne 2002).

The results of studies of waste samples from underground storage tanks at the Hanford Site indicate that a significant fraction of the technetium in the waste is present in the +4 oxidation state. According to Krupke and Serne (2002), “future conceptual models for the release of technetium from the Hanford underground storage tanks will need to consider the potential mobility of Tc(IV) in the near-field, vadose sediments and potential interactions of organics present in the tanks with respect to complexing and stabilizing Tc(IV) and possibly other intermediate valence states of dissolved technetium.”

Immobilization of the high-level nuclear waste stored at the Hanford Reservation has been complicated by the presence of soluble, lower-valent (<+4) technetium species. These species cannot be removed by ion-exchange and are difficult to oxidize. Lukens *et al.* (2004) found that soluble complexes of the Tc(I)-carbonyl species exist, especially $\text{fac-Tc}(\text{CO})_3(\text{gluconate})(2-)$, with implications for storage and clean-up. Extensive work has been done on clean-up techniques, utilizing both microbial and chemical means (e.g., iron-sulfides, Watson and Ellwood 2003).

FATE AND TRANSPORT

As described above, anaerobic vs. aerobic conditions influence the chemical behavior of technetium, and the transport potential of the +7 and +4 oxidation states differ dramatically. Because the pertechnetate anion (TcO_4^-) is highly soluble and is not strongly sorbed at neutral and basic pH conditions, it is highly mobile in most oxidizing systems. In the +4 valence state, technetium exists as the tetravalent cation and is relatively immobile in the absence of strongly complexing ligands. Numerous studies of the sorption of technetium on sediments, soils, pure minerals, oxide phases, and crushed rock materials have been conducted. An extensive review of these studies is presented in Krupke and Serne (2003) and K_d values for Tc-99 at the Hanford site are summarized in Krupke and Serne (2002).

Soils/Sediments

The behavior of Tc-99 in soils depends on many factors, such as texture and organic content. In soils rich in organic matter, Tc-99 is retained and does not have high mobility. Under aerobic conditions, technetium compounds in soils dominated by pertechnetate (TcO_4^-) are readily taken up by plants (EPA 2002; see “Bioaccumulation Potential”). Under both aerobic and anoxic conditions, little adsorption is observed for

technetium in lake sediment with low organic matter content (Tagami and Uchida 1999a).

Keith-Roach *et al.* (2003) investigated the relative uptake of Tc-99 among sites in coastal environments in Europe with reducing waters and/or sediments that represent potential sinks. Uptake varied greatly between the sites, with the highest occurring at an almost permanently anoxic fjord, followed by a brackish, seasonally eutrophic fjord, then a sub-oxic salt marsh, and finally sulfidic and iron-reducing muddy sandy sediments. Relatively high uptake of Tc into sediments at particular fjords occurred due to the fact that TcO^{4-} was exposed to both reducing sediments and high mixing of in the water column, or an oxic/anoxic water boundary. The lowest sediment uptake site could be explained by the speciation of technetium at this site in the carbonate phase, which is largely soluble. The other three sites showed that organic matter, in conjunction with reducing conditions, was very important for binding and retaining Tc-99 in sediments.

The Argonne National Laboratory's Human Health Fact Sheet (ANL 2005) states: "From the surface [technetium] can move rapidly downward with percolating water because most technetium compounds do not bind well to soil particles. The concentration associated with sandy soil particles is estimated at 0.1 of that in interstitial water (in the pore spaces between the soil particles), although technetium binds more tightly to clay soils (with concentration ratios 10 times higher). For this reason, technetium-99 has been found in groundwater at several DOE sites." IAEA (2010) summarizes concentration ratios and K_d values for technetium.

Groundwater

In groundwater, technetium's behavior is highly dependent on its oxidative state, and as noted previously, the predominant species under oxidizing conditions, pertechnetate (TcO^{4-}), tends to be highly mobile, nonadsorptive, and soluble. The pertechnetate ion is stable over the complete pH range of natural waters, and is not known to form any strong aqueous complexes (Krupke and Serne 2002). Due to these properties, it has high dissemination potential in natural systems.

Under reducing (anaerobic) conditions, technetium precipitates mainly as technetium dioxide (TcO_2), which is very insoluble and relatively immobile (ANL 2005).

Water

The reactions of technetium in water are similar to those that occur in soils. In natural waters, the reduced form of technetium may also form highly stable complexes with humic and fulvic acids (Schulte and Scoppa 1987). However, additional studies are needed to determine the stability constants and potential roles of important complexing ligands, such as carbonate, phosphate, sulfide, and others, on the adsorption and solubility of Tc(IV). Technetium is primarily released to and present in the vadose zone and groundwater at the Hanford Site as oxidized Tc(VII), pertechnetate (Krupke and Serne 2002).

Air

Because technetium compounds are ionic, volatilization from moist or dry soil surfaces or surface water will not occur (HSDB 2008). Ionic technetium compounds only exist in the particulate phase in air. Particulate phase technetium compounds may be removed from the air by wet and dry deposition (HSDB 2008). Tagami and Uchida (1999b) developed a sensitive analytical method of determining concentration of Tc-99 in rain and dry fallout by inductively coupled plasma mass spectroscopy (ICP-MS).

BIOACCUMULATION POTENTIAL

Terrestrial Systems

Plant affinity for pertechnetate (TcO^{4-}) applied to soil is high and generally exceeds that reported for other non-nutrient nuclides resulting from the nuclear fuel cycle (Menzel 1965, cited in Schwochau 2000).

Driver (1994) notes that “Plants readily concentrate technetium in their tissues and play an important role in technetium cycling in the environment. Plants are able to effectively accumulate technetium at soil levels as low as $0.01\mu\text{g/g}$ In general, between 47% and 74% of the technetium applied to soil in water is assimilated by plants.” Eudicots appear to bioaccumulate Tc-99 to a greater extent than do monocots (Driver *et al.* 1994, Willey *et al.* 2010).

Tagami and Uchida (2005a) studied the uptake of Tc-99 and rhenium (a chemical analogue of Tc) in three crop species (*Cucumis sativus*, *Raphanus sativus*, and *Brassica chinensis*). The results showed that Tc and Re uptake occurred not only with water mass flow or active nutrient uptake, but also with uptake of nutrient cations such as K^+ . It is likely that the stable chemical form under aerobic conditions, pertechnetate, is used in cation transport as a substitute ion, such as Cl^- . After TcO^{4-} passes through a root surface, it moves through the xylem together with cations. Due to these uptake mechanisms, Tc is highly accumulated in plants, and chemical effects are likely important (Tagami and Uchida 2005a). However, when Tagami and Uchida (2005b) collected plant samples in the Chernobyl area to obtain transfer factors of Tc-99 in the soil-plant system under environmental conditions, their experimental results indicated that Tc-99 released from the Chernobyl Nuclear Power Plant showed low transfer rates to plants.

Other forms of technetium such as TcO_2 are not as readily taken up by plants (Sheppard and Evendon 1991, in Driver 1994, Yoshihara 1996). Furthermore, Yoshihara (1996) states that “even under aerobic conditions, the transfer rate to plants decreases with time, indicating that soluble pertechnetate changes to insoluble forms by action of microorganisms which produce a local anaerobic condition around themselves” (e.g., studies by Landa *et al.* 1977, Hoffman *et al.* 1980, Van Loon *et al.* 1989, Tagami and Uchida 1996). In contrast, experiments with ryegrass showed that mobility of Tc-99 was not changed by aerobic microbial activity, and bioavailability of Tc-99 decreased with biomass production and not with time (Echevarria *et al.* 1997).

Mean soil to plant transfer factors (F_v) range from 9.4×10^{-2} for tubers in loamy soil to 2.5×10^2 for leafy vegetables growing in loamy soil (IAEA 2010). Van Loon *et al.* (1989) describe a general soil-to-plant transfer function for technetium. Concentration factors for technetium from the upper 15 cm of soil at field sites have been reported to range from 3 to 370 (Garland *et al.* 1983) and from 2 to 200 (Hoffman *et al.* 1980). Laboratory studies produced concentration factors of 10 to 1200 (Landa *et al.* 1977, Wildung *et al.* 1977, Mousney and Myttenaere 1981). For native plants at the Hanford Site, Rouston and Cataldo (1977) suggest a concentration factor of 76 to 390 for tumbleweed and 54 to 421 for cheat grass from five Hanford project soils.

Factors that affect the extent of uptake by plants include soil type (e.g., sand versus peat), alkalinity, root depth, nutrients (manganese, sulfate, phosphate, and molybdenate), and the presence of actinides (Sheppard *et al.* 1983, Garten *et al.* 1986a, Sheppard and Evenden 1985, Landa *et al.* 1977, Cataldo *et al.* 1989, Masson *et al.* 1989).

Field studies near an old radioactive waste disposal site at Oak Ridge, TN, indicated that following root uptake, metabolism by deciduous trees rendered Tc-99 less biogeochemically mobile than expected, based on chemistry of the pertechnetate TcO_4^- anion (Garten and Lomax 1989). In the leaf, TcO_4^- is converted to less soluble forms apparently associated with structural components of leaf cell walls. This conversion explains why Tc-99 is not easily leached by rainfall from tree foliage and why Tc-99 appears to accumulate in forest floor leaf litter layers at the Oak Ridge study site (Garten and Lomax 1989).

Technetium compounds do not appear to be readily bioaccumulated by animals, although data are somewhat mixed. Driver (1994) states: “although assimilation of ingested technetium compounds can be high, retention of the radionuclide is low in animals. Transfer of technetium incorporated in plant tissue to animals and its retention in their tissues are even lower than for unincorporated technetium, indicating a low potential for food chain magnification.” However, Garten, *et al.* (1986b) presents research that indicated a substantial increase of technetium-99 (20 and 16-fold, respectively) above levels found in contaminated soil samples from a radioactive waste storage site for snails and millipedes.

Based on a review of the literature, Thorne (2003) summarized biokinetic models for the uptake and retention of iodine, technetium, selenium and uranium for agricultural animals, reindeer, and humans.¹²⁵ This article includes a summary of transfer factors for technetium-95m in birds; in particular, results have been reported for Tc-95m in hens and Japanese quail. Thomas *et al.* (1984) found that although as much as 8.4% of ionic technetium was transferred to quail eggs, only 2% was transferred when the radionuclide was ingested in plant material. Of the technetium deposited in the egg, 80% appeared in the yolk and 20% in the albumin. In hens, technetium concentrations are much higher in eggs than in meat, but this is not the case in Japanese quail. Using Tc-95m in feed, Thomas *et al.* (1986) found transfer factors of 3.5×10^{-1} d/kg for meat

¹²⁵ Of note, however, most studies of the transfer of technetium to animals have employed the short-lived gamma-emitting radioisotopes Tc-95m and Tc-99m, not Tc-99.

and 9×10^{-2} d/kg for eggs for quail. It is unclear how applicable these numbers are to Tc-99 in the environment because the chemical form used in the experiments may differ from that in various environments.

Aquatic Systems

The freshwater microalgae *Chorella emersonii*, *Chlamydomonas reinhardtii* and *Scenedesmus obliquus* were all found to adsorb Tc-99 in the form of TcO^{4-} (Garnham *et al.* 1992). That adsorption was concentration-dependent and increased with decreased external pH.

Hattink *et al.* (2003) confirmed that aquatic plants show a strong accumulation and retention of Tc-99, even after they have died. Accumulation experiments in duckweed suggested that reduction of TcO^{4-} and subsequent complexation are responsible for the accumulation of Tc-99 in the plant. A steady state concentration of TcO^{4-} in duckweed was reached within 24 hours, but the total concentration of Tc-99 increased continuously. Only a small part ($\leq 5\%$) of Tc-99 was present as TcO^{4-} and elimination experiments showed TcO^{4-} to be the mobile species. Other Tc-99 species are responsible for the retention of Tc-99 in duckweed. It is known that these species are not bio-available and only slowly re-oxidize to pertechnetate, resulting in a longer residence time in ecosystems.

Wolterbeek (2001) assessed whether the transfer factor or bioconcentration factor is an important parameter in the environmental distribution of Tc-99. This study evaluated the transfer factor concept for Tc-99 in duckweed by evaluating Tc-99 steady-state concentrations in duckweed against growth rate and nutrient concentration. They concluded that Tc-99 accumulation is not homostatically controlled and the transfer factor is inversely proportional to the growth rate in aquatic biota. To date, few measurements on the uptake of Tc-99 by aquatic plants (or other organisms) have been made under field conditions.

Some laboratory studies have indicated that technetium is not highly bioconcentrated in aquatic organisms. Driver (1994) states: “The Commission of the European Communities (1979) suggests a technetium concentration factor for freshwater fish of 30 L/kg. This value is then multiplied by the concentration of technetium in the water to obtain the concentration of technetium in the organism (Zeevaert *et al.* 1989). In the absence of site-specific data, recommended default values for the water-based bioconcentration factor for technetium in the flesh of freshwater fish are 15 (NRCC 1982), 30 (CSA 1987), and 15 (Poston and Klopfer 1985, Myers *et al.* 1989).”

Blaylock *et al.* (1982) obtained concentration factors for fish and snails by spiking a small experimental freshwater pond with Tc-95m (most freshwater studies have not used the longer-lasting Tc-99). A model using the pond data was developed to calculate steady-state body burdens for freshwater biota. The concentration factors based on the calculated body burden for carp (*Caprinus carpio*), mosquitofish (*Gambusia affinis*), and snails (*Helisoma sp.*) were 11, 75 and 121, respectively.

Masson *et al.* (1989) summarizes the bioaccumulation potential of Tc-99 as follows: “Concentration factors (CF) from water to organisms are generally very low (1 to 10);

however, CF greater than 1000 have been observed for some biota such as macrophytic brown algae, worms and lobsters. Biochemical analysis has shown that Tc-99 was essentially free and partially bonded to proteins. The transfer factors between sediments and species are very low (TF less than 0.5).”

ACCUMULATION WITHIN TISSUES

In mammals, absorption of inorganic pertechnetate from the gastrointestinal tract is about 90%, although when technetium has been incorporated in plant tissue, the absorption rate is greatly reduced (Sullivan *et al.* 1979). Hunt *et al.* (2001) investigated the uptake of Tc-99 in the human gut after subjects consumed contaminated lobster. A low value of the gut transfer factor (f₁ value) was determined by comparing intake and fecal measurements: up to 0.1 with a maximum (two standard deviations) level of about 0.3.

Polygastric animals appear to absorb less technetium than monogastric animals (Gerber *et al.* 1989). This reduced absorption may be due to the reduction of TcO⁴⁻ in the rumen of polygastric animals that interferes with its reabsorption from the intestine (Jones 1989).

Once absorbed, technetium’s biological half-life in mammals appears to be short; however, very little research has been done on Tc-99, with most studies focused on the medically-used Tc-95m.

In rats and guinea pigs, 75-80% of ingested technetium-95m had been excreted within two days (Sullivan *et al.* 1979). According to an EPA fact sheet (primary source unknown), the human body excretes half the ingested Tc-99 within 60 hours, continuing to excrete half of the remaining Tc-99 every 60 hours that follow. After 120 hours, one-fourth of the ingested Tc-99 remains in the body, and nearly all of the ingested Tc-99 will be excreted from the body within a month (EPA 2010).

Once absorbed, various organs and tissues can accumulate technetium, especially the kidneys, which are responsible for excretion of about 50% of intravenously administered technetium (Thorne 2003). Other organs and tissues that take up technetium are the stomach, salivary glands, thyroid, choroid plexus, mucus membranes, small and large intestines, sweat glands (Thorne 2003), and the thyroid and parathyroid (McGill *et al.* 1971) as well as bones and skin (Gerber *et al.* 1989). Hair also accumulates technetium and may be useful as a bioindicator of technetium exposure (Gerber *et al.* 1989). However, limited data exist on the relationship between tissue activity and exposure or dose-response.

According to Sharp *et al.* (1998), technetium is generally found in body fluids as pertechnetate, regardless of the form in which it was administered. About 70–80% of the pertechnetate ions become bound to serum proteins, but unbound technetium rapidly diffuses into interstitial fluids and protein-bound technetium is released to compensate for this diffusion (Sharp *et al.* 1998).

Studies are lacking on accumulation of Tc-99 in the tissue of non-mammalian species. A recent summary includes estimates of concentration ratios (CR) in reptiles in

terrestrial environments (Wood *et al.* 2010). Only Tc-95m has been studied in Japanese quail and hens (summarized in Thorne 2003).

IV. TYPICAL MAJOR EXPOSURE ROUTES As discussed in other sections of this profile, surface soil can and has been contaminated as a result of the discharge of Tc-99 in airborne effluents from nuclear facilities, contamination of the subsurface environment at many sites from leaking tanks, and aquatic ecosystems have been contaminated from deliberate and inadvertent discharges of liquid waste to the environment. Though its beta energy is weak, Tc-99 in the environment can result in external exposure from Tc-99 that comes into close contact with biota. However, the principle pathway of exposure to Tc-99 is from internal exposures resulting from root uptake by plants and ingestion by aquatic and terrestrial animals

V. CHEMICAL ECOTOXICITY The chemical versus radiological toxicity of technetium is debated in the literature. Driver (1994) summarizes its toxicity: “Although technetium has a long half-life and is distributed more readily in the environment than most other radionuclides with long half-lives, technetium-99 as a beta-emitter is much less toxic than the alpha-emitting actinides. The toxicity of technetium in animals is low and appears to be related to the radioactive properties of the radionuclide rather than its chemical properties. However, a chemical toxicity has been associated with reduced fertility. Technetium is very toxic to plants. Its chemical properties affect the distribution, and biological half-life in plants, and may influence the retention of plutonium in target tissues (Roucoux and Colle 1986).”

According to Schwochau (2000), “the radiological toxicity of Tc-99 might be even less than its chemical toxicity.” He states that technetium-99 is often thought to be a significant long-term risk to humans, spreading more readily in the environment than many other radionuclides (as pertechnetate), but presents limited evidence for this risk. The literature on the topic of radiological toxicity is lacking, although most recent studies point to effects of chemical toxicity, mainly in plants.

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

There are no known beneficial effects associated with exposure to Tc-99. The gamma-emitting precursor, Tc-99m is used in medical therapy in brain, bone, liver, spleen, kidney, and thyroid scanning and for blood flow studies. Tc-99m is the radioisotope most widely used as a tracer for medical diagnosis (EPA 2002).

MECHANISM(S) AND LOCI OF CHEMICAL TOXICITY

In plants, it is likely that Tc-99 exhibits a role in nutrient competition and/or substitution in uptake or metabolism (Berlyn *et al.* 1980). More specifically, it appears that incorporation of technetium results in technetium-cysteine, which is unable to form disulfide-like bridges. Formation of technetium-cysteine leads to nonfunctional proteins that accumulate and to increased production proteins (which end up defective) that, in turn, lead to metabolic dysfunction, especially in young tissue

where protein synthesis is critical (Cataldo *et al.* 1989). Cellular effects of technetium have also been attributed to alteration of membrane permeability (Neel and Onasch 1989) and cellular energetics (microorganisms, Gearing *et al.* 1975; aquatic plants, Hattink and Wolterbeek 2004). Limited information on mechanisms of toxicity on animals is available.

PLANTS

Research on the toxicity of technetium to plants has focused largely on agricultural species and aquatic plants. In most studies, adverse effects have been observed in the early stages of development (e.g., germination) and on plant growth.

For example, laboratory-grown soybeans exhibited stunted growth when exposed to Tc-99: at the 0.1 µg/g level, growth did not occur beyond cotyledon expansion; at the 5 µg/g level growth ceased three days after emergence (Berlyn *et al.* 1980).

Observations of mitotic figures in soybean plants exposed to Tc-99 did not reveal any chromosome aberrations, micronuclei, or chromosomal bridges (Wildung *et al.* 1977). Schwochau (2000) states that this study suggests that growth effects were due to chemical toxicity, possibly due to nutrient competition and/or substitution in uptake or metabolism, but that a radiological effect cannot be ruled out.

Driver (1994)'s literature review states: "Growth anomalies only occur in plants germinated in the presence of technetium, indicating that the toxicity of this radionuclide is probably associated with early stages of plant growth such as embryonic cell division. Adverse effects on germinating wheat seedlings were first observed at shoot tissue concentrations of 0.68 to 2.8 µCi/g (a specific activity of 17 mCi/g corresponds to technetium levels in tissue of 40 to 165 ppm). The threshold dose rate that induced depression of shoot-tissue yield occurred at 2 rad/d. This low-dose rate suggests technetium toxicity is chemical rather than radiological.

Technetium-treated plants display similar symptomology to plants suffering from 2,4-Dichlorophenoxyacetic acid (2,4-D) poisoning (an artificial plant hormone, causing unsustainable, rapid growth, leaf withering, and eventual plant death [Landa *et al.* 1977]). In lettuce, the chemical toxicity threshold (growth reduction) was observed at concentrations of 0.2 ng/g dry weight of soil (Masson *et al.* 1989). The lethal concentration for Swiss chard was 0.05 µg/g technetium/g dry soil. Even at low concentrations of 0.1 µg/g technetium/g dry soil, technetium has been shown to inhibit plant growth and development in soybeans (Cataldo *et al.* 1978). Toxic effects were largely observed in buds and young leaves rather than in mature tissues (Finch 1983)."

The specific mechanism of toxic effects in plants is not well-established. Hattink and Wolterbeek (2004) concluded that Tc-99 continuously accumulates in the biomass of duckweed, eventually leading to toxic effects that are a result of oxidative stress rather than damage from radiation. Woodard-Blankenship (1995) showed that in low light conditions, Tc-99 significantly decreased growth and the concentration of chlorophylls in soybean leaves, presumably by inducing damage in chloroplasts by peroxidation of membrane lipids. As exposure to Tc-99 increased, growth and development abnormalities become more evident. It was not clear, however, whether

the observed effects resulted from radiological damage, chemical toxicity, or some combination thereof.

Another study (Hattink and Wolterbeek 2002) suggested interference with Tc-99 accumulation was the result of three mechanisms. These mechanisms are: 1) changes in the plant physiological status, 2) competitive effects with nutrient uptake, and 3) electrostatic cell wall interactions. This study focused on whether these mechanisms are relevant for *aquatic* plants grown under natural conditions. Tc-99 accumulation in five *aquatic* plant species correlated strongly with the calcium concentration in the water. Growth rate or possible competition with Cl^- , NO_3^- , PO_4^{3-} or SO_4^{2-} did not significantly affect, if at all, the Tc-99 accumulation in submerged *aquatic* plants. The study also suggested that water hardness and electrostatic cell wall interactions are the dominant factors interfering with the Tc-99 accumulation in submerged *aquatic* plants (Hattink and Wolterbeek 2002).

Vázquez *et al.* (1990) treated beans with and without cotyledons with technetium, determining that treated plants without cotyledons displayed increased autophagic vacuole activity, accumulation of protein bodies in roots, and decreased starch content and severe ultrastructural alterations in chloroplasts. Their results support the hypothesis that toxicity is mainly associated with anabolic processes in developing tissues.

AQUATIC INVERTEBRATES AND FISH

Research is limited on the toxicological effects (either chemical or radiological) of technetium on aquatic biota. The limited available information, including Hanford-specific information focuses on exposures and potential risks rather than direct measurements of effects (e.g., Smith *et al.* 2001, Poston *et al.* 2003). Durand *et al.* (1994) investigated the biochemical affinity and metabolic behavior of technetium in marine invertebrates (lobster).

MAMMALS

Limited information exists on the chemical toxicity of technetium to mammals. In one study, Gerber *et al.* (1989) gave rats large amounts of Tc-99 in either a normal or an iodine-deficient diet for several months starting 2 weeks before mating. Newborns were continued on these diets after weaning. Administration of very high concentrations of technetium ($\geq 10 \mu\text{g/g}$) in food was required to produce deleterious effects to thyroid function, fertility, and postnatal development in mammals (rats, Gerber *et al.* 1989). Van Bruwaene *et al.* (1986) found similar results in sheep. Iodine deficiency only slightly influenced the Tc-99 toxicity in the rats (Gerber *et al.* 1989). Gerber *et al.* (1989) opined that the fertility and fetal development impacts were likely caused by the chemical rather than radiation toxicity of technetium.

Because effects were found only at high concentrations in this study, Schwochau (2000) states, “it seems unlikely that contamination levels in the environment would ever reach levels that could lead to serious non-stochastic effects, even in the developing organism.” Consistent with this view, Driver (1994) notes, “[although] there are few available data on the chemical toxicity of technetium in animals... it is

chemically similar to rhenium, and its toxicity is probably between manganese and rhenium. The toxicity of common manganese compounds varies from 90 to 934 mg/kg (Bowen 1979; NIOSH 1987). Rhenium toxicity is low. Intraperitoneal injection of rhenium trichloride results in an LD50 of 280 mg/kg (Lewis 1992)."

BIRDS

Information on technetium's effects on birds is very limited. Driver (1994) summarizes that although technetium is concentrated in avian oocytes (Roche *et al.* 1957, Thomas *et al.* 1984), no impacts to developing embryos have been noted. No more recent studies examining Tc-99 effects on birds were found.

VI. RADIOLOGICAL ECOTOXICITY

The literature specifically addressing the radioecological impacts of technetium-99 in the environment is limited. Technetium-99 decays by emitting a beta particle to produce the stable isotope ruthenium-99. Tc-99's very long half-life (2.11×10^5 yr) and low specific activity limit its radioactive hazards.

Radioecological damages to aquatic and terrestrial organisms due to Tc-99 in the environment result from ionization caused by the interaction of its beta particles with living tissue. In particular, upon each disintegration, Tc-99 emits a beta particle with an average energy of 84.6 keV and a maximum energy of 294 keV (Shleien *et al.* 1998). The range of beta particles in matter is given by (Shleien *et al.* 1998, Formula 2a, p 3-15):

for $(0.01 \leq E \leq 2.5 \text{ MeV})$:

$$R = 412 * E^{(1.265 - 0.0954 * \ln(E))}$$

where R = range in mg/cm^2 (range in cm times the density of the absorbing medium in mg/cm^3)

E = energy of the beta particle in MeV

Using this equation, the approximate range of Tc-99 beta particles in tissue is about 0.010 cm. Given that the typical energy required to ionize a molecule (i.e., eject an electron from its orbit) is about 34 to 35 eV (see page 17, Casarett 1968), the total number of ion pairs produced by the energy deposited in tissue from the average energy beta particle emitted by Tc-99 is about 2400 ion pairs (i.e., 84.6 keV/35 eV).

The pattern of energy deposition for beta particles is described in Morgan and Turner (1973) as follows:

$$\text{mean linear ion density} = T/R_t \times W$$

where:

T = average energy of electron liberated

R_t = range or electrons of energy T

W = average energy to form an ion pair

For Tc-99, the equation is $84.6 \text{ keV} \times 1000 \text{ eV/keV} \div 0.010 \text{ cm} \times 35 \text{ eV/ion pair} = 2.39 \times 10^5$ ion pairs per cm or about 23.9 ion pairs per micron. Given that a typical cell is on the order of tens of microns (see page 102 of Curtis and Barnes, 1989), a single cell might experience about 240 ion pairs produced by the passage of an average Tc-99 beta particle. It is this deposited energy in living tissue that results in biological damage.

Sufficiently energetic beta particles can penetrate the dead layer of the skin of mammals (nominally 70 microns in humans) and deposit energy in underlying tissues. Tc-99 emits beta particles with an average energy of 86.4 keV and that have a range of about 9 cm in air and 0.010 cm (100 microns) in tissue (Shleien et al. 1998). Thus, there is a real potential for exposure from external radiation from Tc-99, including all aquatic and terrestrial organisms and all stages of their life cycle, except for organisms that have a thick outer layer (such as bark of trees, heavy fur, scales, etc.) that can shield the living tissue beneath from the beta emissions.

As discussed above, most studies have interpreted toxic effects on plants as mainly from chemical toxicity, possibly due to nutrient competition and/or substitution in uptake or metabolism. Several studies have suggested possible technetium impacts on cellular energetics, possibly by elevating levels of ATP (e.g., soybeans, Woodard-Blakenship 1995; blue-green algae, Gearing *et al.* 1975). It is likely that the mechanism for these effects is chemical rather than radiological, but more research is needed.

Barnaby and Boeker (1999) call for a reassessment of the radiological risks of Tc-99 to human health, with concerns about the large discharges of Tc-99 into the ocean at Sellafield, in particular. Tc-99 is known to concentrate in seaweed, periwinkles, lobster, and mussels; these authors call on researchers to investigate the possibility of radiation induced genomic instability, as well as cancer risk from Tc-99.

VII. EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

The presence of actinides has been reported to enhance technetium uptake in plants in some soil types (Masson et al. 1989). Technetium uptake in leaves of radish plants grown in calcareous soils increased 4 times in the presence of uranium and 4.5 times in plutonium-amended soils. Plutonium also appeared to increase technetium uptake 1.5 times in the leaves and 3 times in roots of plants grown in acid soils. The presence of americium in organic soils resulted in a six-fold increase in leaf uptake of technetium (Roucoux and Colle 1986, Masson et al. 1989). However, because no soil plant concentrations or statistical information is presented, the validity of the reported technetium actinide relationship is unclear. Incorporation of technetium in plant tissue alters the absorption and retention of the radionuclide in animal tissues.

Overall, additional studies are needed to determine the stability constants and potential roles of important complexing ligands, such as carbonate, phosphate, sulfide, and others, on the adsorption and solubility of Tc(IV) (EPA 2010). Synergistic effects with other toxins have not been studied extensively, and there may also be synergistic effects associated with technetium's role as a chemical and radiological toxin.

VIII. DATA GAPS Partially because of the difficulty in detecting Tc-99 as a beta-emitter, the fact that it will usually be present in the environment with other radionuclides (especially actinides), and lack of stable isotopes of this element, chemical toxicity to biota is difficult to determine. Overall, there is a paucity of information on Tc-99 toxicity to humans (Tc-99 is not addressed in the ATSDR toxicity profiles) and animals, even though this is the decisive criterion for assessing the consequences of the occurrence of Tc-99 in the environment (Schwochau 2000). Driver (1994) and this review found no studies on the toxicity of Tc-99 to amphibians and reptiles, potentially important classes of biota on which to focus in the Columbia River watershed. Similarly, toxicity of Tc-99 on birds has not been extensively studied; most (limited) research on accumulation in tissues has focused on Tc-99m in domestic fowl and has not investigated physiological effects. Information on radiological toxicity is limited, although chemical toxicity appears to be more biologically significant for a wide range of taxa than is radiological toxicity. Beresford (2010) and other papers within volume 49 of *Radiation and Environmental Biophysics* summarize the status of radiological data used in environmental assessments to date, including methods to model wildlife impacts when data are lacking; however, data on technetium-99 and effects on wildlife are particularly scarce.

- IX. REFERENCES**
- Argonne National Laboratory (ANL). Human Health Fact Sheet. 2005.
<http://www.ead.anl.gov/pub/doc/technetium.pdf>. Accessed 9 December, 2010.
- Barnaby, F. and E. Boeker. 1999. Is technetium (Tc-99) radiologically significant? *Medical Conflict Survival* 15: 57-70.
- Beresford, N.A. 2010. The transfer of radionuclides to wildlife. *Radiation and Environmental Biophysics* 49: 505-508.
- Berlyn, G.P., S.S. Dhillon, and E.E. Koslow. 1980. Technetium: a toxic waste product of the nuclear fuel cycle: effects on soybean growth and development. *Environmental Management* 4: 149-156.
- Blaylock, B.G., M.L. Frank, and D.L. DeAngelis. 1982. Bioaccumulation and of ^{95m}Tc in fish and snails. *Health Physics* 42: 257-66.
- Bolsunovskii, A.Y., Y.V. Aleksandrova, and A.G. Degermendzhi. 2010. First data on technetium-99 content in the ecosystem of the Yenisei River. *Doklady Earth Sciences* 434: 1219-1221.
- Bowen, H. 1979. *Environmental Chemistry of the Elements*. Academic Press, London.
- Canadian Standards Association (CSA) 1987. *Guidelines for Calculating Derived Release Limits for Radioactive Material in Airborne and Liquid Effluents for Normal Operation of Nuclear Facilities*. National Standard of Canada. CAM/CSA-N288.1-M8, Canadian Standards Association, Rexdale, Toronto, Canada. (as cited in Driver 1994)

- Carleton, W.H., M. Denham, and A.G. Evans. 1993. Assessment of Technetium at the Savannah River Site Environment. WSRC-TR-93-217 July 1993.
- Casarett, A.P. 1968, Radiation Biology, Prentice-Hall, Inc. Englewood Cliffs, New Jersey.
- Cataldo, D.A., R.E. Wildung, and T.R. Garland. 1978. Technetium accumulation, fate, and behavior in Plants. In: Environmental Chemistry and Cycling Processes, eds., D.C. Adriano, T.L. Brisbin, Jr. Department of Energy Symposium Series No. 45, CONF-760429, pp. 538-549. National Technical Information Service, Springfield, Virginia.
- Cataldo, D.A., T.R. Garland, R.E. Wildung, and R.I. Fellows. 1989. Behavior and effects of technetium in plants. Health Physics 2: 281-287.
- Cataldo, D.A., and R.E. Wildung. 1983. The role of soil and plant metabolic processes in controlling trace element behavior and bioavailability to animals. Science of the Total Environment 2: 159-168.
- Commission of the European Communities. 1979. Methodology for Evaluating the Radiological Consequences of Radioactive Effluents Released in Normal Operations. Joint Report by the National Radiological Protection Board and the Commissariat a l'Energie Atomique; CEC; V/3865n9-EN, Luxembourg. (as cited in Driver 1994)
- Curtis, H. and N.S. Barnes, Biology, Worth Publishers, Inc. 1989.
- Driver C.J. 1994. Ecotoxicity Literature Review of Selected Hanford Site Contaminants. PNL-9394, Pacific Northwest Laboratory, Richland, Washington. <http://www.osti.gov/energycitations/servlets/purl/10136486-6sLptZ/native/10136486.pdf>
- Durand, J.P., M.C. Milcent, F. Goudard, F. Paquet, P. Germain, T. Nafissi, and J. Pieri. 1994. Chemical behavior of three radionuclides (cesium, americium, and technetium) and their uptake at the cytosolic level in aquatic organisms. Biochemistry and Molecular Biology International 33: 521-534.
- Echevarria, G., P.C. Vong, E. Leclerc-Cessac, and J.L. Morel. 1997. Bioavailability of Technetium-99 as affected by plant species and growth, application form, and soil incubation. Journal of Environmental Quality 26: 947-956.
- Finch, R.R. 1983. Metabolic Effects and Distribution Studies of Tc99 in Soybeans. San Diego State Univ. Thesis (as cited in Driver 1994).
- García-León, M. 2005. 99-Tc in the environment: sources, distribution and methods. Journal of Nuclear and Radiochemical Sciences 6: 253-259.
- Garland, T.R, D.A. Cataldo, K.M. McFadden, R.G. Schreckhise, and R.E. Wildung. 1983. Comparative Behavior of 99Tc, 129I, 127I and 137Cs in the Environment Adjacent to a Fuels Reprocessing Facility. Health Physics 44: 658-662.

- Garnham G.W., G.A. Codd, and G.M. Gadd. 1992. Uptake of technetium by freshwater green microalgae. *Applied Microbiology and Biotechnology* 37: 679-684.
- Garten, Jr., C.T., C.S. Tucker, and T.G. Scott. 1986a. Plant uptake of Np-237 and Tc-99 under field conditions. *Journal of Environmental Radioactivity* 4: 91-99.
- Garten, Jr., C.T., C.S. Tucker, and B.T. Walton. 1986b. Environmental fate and distribution of technetium-99 in a deciduous forest ecosystem. *Journal of Environmental Radioactivity* 3: 163-188.
- Garten, Jr., C.T. and R.D. Lomax. 1989. Tc-99 cycling in maple trees – characterization of changes in chemical form. *Health Physics* 57: 299-307.
- Gearing, P., C. VanBaalén, and P.L. Parker. 1971. Response of blue-green algae to technetium. In: *Radionuclides in Ecosystems*, ed. D. J. Nelson. Oak Ridge National Laboratory, Oak Ridge, Tennessee.
- Gearing P., C. VanBaalén, and P.L. Parker. 1975. Biochemical effects of pertechnetate (Tc-99) on microorganisms. *Plant Physiology* 55: 240-246.
- Gerber, G., B.M. Van Hees, C.T. Garten, Jr., C.M. Vandecasteele, J.K. Vankerkom, R. Van Bruwaene, R. Kirchmann, J. Colard, and M. Cogneau. 1989. Technetium absorption and turnover in monogastric and polygastric animals. *Health Physics* II(2): 315-319.
- Hattink, J. and H. Wolterbeek. 2002. Accumulation of technetium in floating aquatic plants. *Radioprotection* 37: 633-638.
- Hattink, J., A.V. Harms, and J.J.M. de Goeij. 2003. Uptake, biotransformation, and elimination of Tc-99 in duckweed. *Science of the Total Environment* 312: 59-65.
- Hattink, J., L. Weltje, H.Th. Wolterbeek, and J.J.M. de Goeij. 2004. Accumulation, elimination and retention of ⁹⁹Tc by duckweed. *Journal of Radioanalytical and Nuclear Chemistry* 259: 135-139.
- Hazardous Substances Data Bank (HSDB). 2008. 7543, last revision 20080425. <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/r?dbs+hsdb:@term+@DOCNO+7543>. Accessed 4 November, 2010.
- Hoffman, F.O., J.W. Huckabee, D.M. Lucas, C.T. Garten, T.G. Scott, R.L. Walker, P.S. Gouge, and C.V. Holmes. 1980. Sampling of Technetium-99 in Vegetation and Soils in the Vicinity of Operating Gaseous Diffusion Facilities. ORNL-7386, Oak Ridge National Laboratory, Oak Ridge, Tennessee.
- Hunt, G.J., A.K. Young, and R.A. Bonfied. 2001. Transfer across the human gut of environmental technetium in lobsters (*Homarus gammarus*) from the Irish Sea. *Journal of Radiological Protection* 21: 21-29.
- Idaho Department of Environmental Quality: Contamination of Groundwater at the INL Sept 26, 2003. available at:

http://www.deq.idaho.gov/inl_oversight/contamination/tank_farm.cfm as of October 22, 2007. Accessed 5 November, 2010.

- International Atomic Energy Agency (IAEA). 2010. Handbook of parameter values for the prediction of radionuclide transfer in terrestrial and freshwater environments. IAEA-TRS 472. IAEA, Vienna. 208 pp.
- Jones, B.E.V. 1989. Technetium metabolism in goats and swine. *Health Physics* 57: 331-336.
- Keith-Roach, M.J., K. Morris, and H. Dahlgaard. 2003. An investigation into technetium binding in sediments. *Marine Chemistry* 81: 149-162.
- Krupke, K.M. and R.J. Serne. 2002. Geochemical Factors Affecting the Behavior of Antimony, Cobalt, Europium, Technetium, and Uranium in Vadose Sediments. PNNL-14126. Richland, WA: Pacific Northwest National Laboratory, U.S. Department of Energy. December, 2002.
- Krupke, K.M. and R.J. Serne. 2003. Understanding Variation in Partition Coefficient, K_d , Values: Volume III. Review of Geochemistry and Available K_d Values for Americium, Arsenic, Curium, Iodine, Neptunium, Radium, and Technetium. EPA 402-R-99-004C, U.S. Environmental Protection Agency, Washington, D.C. Pacific Northwest National Laboratory, Richland, Washington.
- Landa, E.R., L.J. Hart Thorvig, and R.G. Gast. 1977. Uptake and Distribution of Technetium-99 in Higher Plants. In: *Biological Implications of Metals in the Environment*, pp. 390-401. Proceedings of the Fifteenth Annual Hanford Life Sciences Symposium at Richland, Washington. September 29-October 1, 1975. CONF-750959, U.S. Department of Energy, Washington, D.C.
- Lewis, R.J., Sr. 1992. *SZX's Dangerous Properties of Industrial Materials*, Eighth Edition. Van Nostrand Reinhold, New York (as cited in Driver 1994).
- Lukens, W.W., D.K. Shuh, N.C. Schroeder, and K.R. Ashley. 2004. Identification of the non-pertechnetate species in Hanford waste tanks, Tc(I) – carbonyl complexes. *Environmental Science Technology* 38: 229-233.
- Masson, M., F. Patti, C. Colle, P. Roucoux, A. Grauby, and A. Saas. 1989. Synopsis of French experimental and in situ research on the terrestrial and marine behavior of Tc. *Health Physics* 57: 269-279.
- McGill, P.E., M.G. Harden, J.W.K. Robertson and J. Shimmins. 1971. A comparison between the uptake of technetium-99m and iodine-131 by the thyroid gland. *Journal of Endocrinology* 49: 531-536.
- Morgan, K.Z. and J.E. Turner, 1973. *Principles of Radiation Protection*, Robert E. Krieger Publishing Company, Huntington, New York.
- Mousney, I.M. and C. Myttenaere. 1981. Absorption of technetium by plants in relation to soil type, contamination and time. *Plant Soil Quality* 1: 403-412.
- Multi-Agency Radiological Laboratory Analytical Protocols Manual Volume II. 2004. NUREG-1576, EPA 402-B-04-001B, NTIS PB2004-105421. Chapter 14-165.

July 2004. Available from, as of October 12, 2006: <http://www.nrc.gov/reading-rm/doc-collections/nuregs/staff/sr1576/sr1576v2.pdf>

- Myers, D.K. 1989. The General Principles and Consequences of Environmental Radiation Exposure in Relation to Canada's Nuclear Fuel Waste Management Concept. ABeL 9917, Atomic Energy of Canada Limited, Chalk River Nuclear Laboratories, Chalk River, Ontario, Canada.
- National Institute for Occupational Safety and Health (NIOSH). 1987. Registry of Toxic Effects of Chemical Substances. U. S. Department of Health and Human Services, Public Health Service, Washington D.C.
- National Research Council of Canada (NRCC). 1982. Data Sheets on Selected Toxic Elements. 19252, National Research Council, Ottawa, Canada.
- Neel, J.W. and M.A. Onasch. 1989. Cytological effects of Tc on young soybean plants. *Health Physics* 57: 289-298.
- Poston, T.M., and D.C. Klopfer. 1985. A literature review of the concentration factors of selected radionuclides in freshwater and marine fish. PNL-5484, Pacific Northwest Laboratory, Richland, Washington.
- Poston, T.M., E.J. Antonia, and R.E. Peterson. 2003. Application of biota dose assessment committee methodology to assess radiological risk to salmonids in the Hanford reach of the Columbia River. Conference Information: 3rd International Symposium on the Protection of the Environment from Ionising Radiation (SPEIR 3), July 22-26, 2002 Darwin, Australia. In: Book Series 17: 397-405.
- Poston, T.M., J.P. Duncan, and R.L. Dirkes, eds. 2010. Hanford Site Environmental Report for Calendar Year 2009. PNNL-19455. Pacific Northwest National Laboratory, Richland, WA. 393 pp.
- Roche, J., R. Michel, E. Volpen, and B. Sanzo 1957. Sur la fixation du manganese et du rhenium radioactifs par le corps thyroïde et les oocytes de la poule pondeuse. *C.R. Soc. Biol.* 151: 1098-1100. (as cited in Driver 1994)
- Roucoux, P., and C. Colle. 1986. Retention par les Vegetaux du Technetium Relache dans l'Environnement en Association avec d'Autres Radiopolluants. In: *Technetium in the Environment*. (CEA-IPSN, Saint Paul-lez-Durance, France), Elsevier Applied Science Publ., London and New York. (as cited in Driver 1994)
- Routson, R.C. and D.A. Cataldo. 1977. Tumbleweed and cheatgrass uptake of Tc from five Hanford soils. BNWL-2183. Battelle, Pacific Northwest Laboratories, Richland, WA.
- Scerri, E. 2009. Tales of technetium. *Nature Chemistry* 1: 332.
- Schulte, E.H. and P. Scoppa. 1987. Sources and behavior of technetium in the environment. *Science of the Total Environment* 54: 163-181.
- Schwochau, K.S. 2000. *Technetium: Chemistry and radiopharmacological applications*. Wiley-Vch, Germany.

- Scottish Environmental Protection Agency. 2003. Analysis of farmed salmon for technetium-99 and other radionuclides. July 2003. Available at <http://www.food.gov.uk/multimedia/pdfs/contact.pdf>
- Sharp, P.F., H.G. Gemmell, and F.W. Smith. Eds. 1998. Practical Nuclear Medicine, second ed. Oxford University Press, Oxford, UK.
- Sheppard, M.I., T.T. Vandergraaf, D.H. Thibault, J. Reid, and A. Keith. 1983. Technetium and uranium: sorption by and plant uptake from peat and sand. Health Physics 44: 635-643.
- Sheppard, S.C. and W.G. Evenden. 1985. Mobility and uptake by plants placed near a shallow water table interface. Journal of Environmental Quality 14: 554-560.
- Sheppard, S.C. and W.G. Evenden. 1991. Heavy metals in the environment: can aquatic macrophytes mobilize technetium by oxidizing their rhizosphere? Journal of Environmental Quality 20: 738-744.
- Shleien, B., L.A. Slaback, Jr., and B.K. Birky, 1998, Handbook of Health Physics and Radiological health, Third Edition, Williams & Williams, A Waverly Company, 1998.
- Smith, V., M. Fegan, D. Pollard, S. Long, E. Hayden, and T.P. Ryan. 2001. Technetium-99 in the Irish marine environment. Journal of Environmental Radioactivity 56:269-284.
- Sullivan, M.F., T.R. Garland, D.A. Cataldo, and R.G. Schreckhise. 1979. Absorption of plant incorporated nuclear fuel cycle elements from the gastrointestinal tract. In: Biological Implications of Radionuclides Released from Nuclear Industries, Vol. II., pp. 447-457. IAEA-SM-237/58, International Atomic Energy Agency, Vienna, Austria.
- Tagami K. and S. Uchida. 1996. Microbial role in immobilization of technetium in soil under waterlogged conditions. Chemosphere 33: 217-225.
- Tagami, K. and S. Uchida. 1999a. Chemical transformation of technetium in soil during the change of soil water conditions. Chemosphere 38: 963-971.
- Tagami, K. and S. Uchida. 1999b. Determination of Tc-99 in rain and dry fallout by ICP-MS. Journal of Radioanalytical and Nuclear Chemistry 197: 409-416.
- Tagami, K. and S. Uchida. 2005a. A comparison of concentration ratios for technetium and nutrient uptake by three plant species. Chemosphere 60: 714-717.
- Tagami, K. and S. Uchida. 2005b. Soil-to-plant transfer factors of technetium-99 for various plants collected in the Chernobyl area. Journal of Nuclear and Radiochemical Sciences 6: 216-264.
<http://wwwsoc.nii.ac.jp/jnrs/paper/JN63/jn6327.pdf>
- Thomas, J.M., L.L. Cadwell, D.A. Cataldo, T.R. Garland, and R.E. Wildung. 1984. Concentration of orally administered and chronically fed ^{95m}Tc in Japanese Quail eggs. Health Physics 16: 657-663.

- Thomas, J.M., L.L. Cadwell, D.A. Cataldo, and T.R. Garland. 1986. Distribution of orally administered and chronically fed Tc-95m in Japanese Quail tissues and eggs. In: Desmet, G., and C. Myttenaere, Eds. *Technetium in the Environment*. Elsevier Applied Science Publishers, London, pp. 349–357.
- Thorne, M.C. 2003. Estimation of animal transfer factors for radioactive isotopes of iodine, technetium, selenium and uranium. *Journal of Environmental Radiology* 70: 3-20.
- Uchida, S., K. Tagami, and E. Wirth. 1999. Concentrations levels of technetium-99 in forest soils collected within the 30-km zone around the Chernobyl reactor. *Chemosphere* 39: 2757-2766.
- U.S. Environmental Protection Agency (EPA). Fact Sheet; Technetium-99 July 2002. Available at:
www.epa.gov/superfund/health/contaminants/radiation/pdfs/technetium.pdf
Accessed 4 November, 2010.
- U.S. Environmental Protection Agency (EPA). 2010. Last updated October 1, 2010. <http://www.epa.gov/rpdweb00/radionuclides/technetium.html> Accessed 4 November, 2010.
- Van Loon, L.R., G.M. Desmet, and A. Cremers. 1989. The uptake of TC04- by plants: A mathematical description. *Health Physics* 57: 309-314.
- Vázquez, M.D., A. Bennassar, C. Cabot, Ch. Poschenrieder, and J. Barcelo. 1990. Phytotoxic effects of technetium-99 in beans: influence of cotyledon excision. *Environmental and Experimental Botany* 30: 271-281.
- Watson, J.H.P. and D.C. Ellwood. 2003. The removal of the pertechnetate ion and actinides from radioactive waste streams at Hanford, Washington, USA and Sellafield, Cumbria, UK: the role of iron-sulfide-containing adsorbent materials. *Nuclear Engineering and Design* 226: 375-385.
- Wildung, R.E., T.R. Garland, and D.A. Cataldo. 1977. Accumulation of technetium by plants. *Health Physics* 32: 314-317.
- Willey, N.J., S. Tang, and A. McEwen. 2010. Effects of plant traits and physiology on soil to plant transfer of Tc-99. *Journal of Environmental Radiology* 101: 757-766.
- Wolterbeek, H. 2001. Evaluation of the transfer factor of technetium from water to aquatic plants. *Journal of Radioanalytical and Nuclear Chemistry* 249: 221-225.
- Wood, M.D., N.A. Beresford, D.V. Semenov, T.L. Yankovich, and D. Copplestone. 2010. Radionuclide transfer to reptiles. *Radiation and Environmental Biophysics* 49: 509-530.
- Woodard-Blankenship, B., J.W. Neel, and P.J. Papin. 1995. Localization and morphological effects of technetium-99 on higher plant cells. Department of

Physics and Department of Biology. San Diego State University, San Diego.
Pages 1-18.

Yoshihara, K. 1996. Technetium in the environment. In: Topics in Current Chemistry, Volume 176, Technetium and Rhenium Their Chemistry and Its Application. pp. 17-35.

Zeevaert, T., C.M. Vandercasteele, and R. Kirchmann. 1989. Assessment of dose to man from releases of ^{99}Tc in freshwater systems. Health Physics 51:337-343.

TRITIUM ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Tritium (radioactive hydrogen) is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Tritium (^3_1H or T_2 , atomic mass of three), has one proton and two neutrons in the nucleus. Oxidation causes tritium gas (HTO) to become the most common form in the environment. Tritium at room temperature in its gaseous form reacts with hydrogen to form HT. Tritium is a beta emitter, decaying to helium (^3He) by emitting a beta particle and an antineutrino from one of the neutrons in the nucleus. The energy of the beta particle varies from 0 to 18.6 kiloelectron-volts (keV) with an average energy of 5.69 keV. For scientific purposes, the generally accepted value for the half-life of tritium, as measured by Mound Laboratories, is 12.323 ± 0.004 years (4500.88 ± 1.46 days). For DOE accountability purposes, the half-life of tritium is 12.33 ± 0.06 years (NCRP 1979, DOE Handbook 2008). Tritium is generally accepted to have relatively low radiotoxicity and no chemical toxicity (ANL 2005).

II. SOURCES **NATURAL SOURCES**

Tritium is a naturally occurring isotope of hydrogen produced in the upper atmosphere by cosmic rays and in rocks by decay of naturally occurring radioactive elements. The natural steady-state global inventory of tritium is about 7.3 kg (ANL 2005). Tritium commonly occurs in nature as part of a water molecule (HTO), so when it is produced in the atmosphere, it falls to the earth as rain and enters the hydrological cycle.

ANTHROPOGENIC SOURCES

Tritium is a fission product in nuclear weapons tests, existing in worldwide fallout from historical testing. It is also produced in nuclear power reactors by ternary fission and by activation of light elements such as boron, which is used for reactivity control in pressurized water reactors, and lithium, used to control corrosion (NCRP 1979). Tritium has a fission yield of one atom of tritium per approximately 10,000 fissions, or 0.01% (ANL 2005). In 1979 it was estimated that by 1986 emissions from the nuclear fuel cycle would become more important than residue from weapons testing (NCRP 1979).

Tritium also has a variety of uses. It is a key element in nuclear fusion, in which energy is produced by the controlled fusion of tritium with deuterium. Tritium is also used as an agent in luminous paints such as those used to make building exit signs, airport runway lights and watch dials, and even in novelty items such as glow sticks; it is thought that the presence of tritium leachate in municipal and other landfills is a result of these commercial uses (Mutch and Mahony 2008).

The United States is estimated to have a current inventory (as of 2005) of approximately 75 kg of tritium (ANL 2005). Discharges from medical and research facilities contribute various forms of organically bound tritium (OBT) as well as tritiated water (e.g., Williams *et al.* 2001).

At Hanford, the processing of zirconium alloy clad fuel released about five to nine percent of the dissolved tritium to the atmosphere, less than one percent to recovered fuel, five to 16 percent to waste storage, and 73-90 percent to ponds (NCRP 1979).

Tritium is one of the most widespread contaminant at the Hanford site, originating from the central part of the site, which includes the 200 Area and the high level radioactive waste tanks. The 200 Area, located on Hanford's Central Plateau, is where chemical processing, plutonium finishing, and defense waste management activities took place. The tritium plume from Hanford's 200 Area is the largest known contaminant plume associated with the site, and it extends east to the Columbia River. The sediments in this area are highly permeable, and the plume has traveled more than 20 km, whereas plumes originating in the less permeable aquifer of west-central Hanford have moved only about three km (Hartman 2003). The 200 Area plume extends under the 400 Area and has historically affected tritium concentrations in all 400 Area drinking wells, though the concentrations have decreased since 1990. In 2009 all samples from these wells were below the state and Federal drinking water standard of 20,000 pCi/L (Poston *et al.* 2010). However a total tritium-contaminated groundwater plume of 126.5 km² still exists at Hanford that exceeds the drinking water standard. Elevated tritium in the 300 Area of Shoreline Springs indicates that the groundwater plume is still evident from the 200 Area (Poston *et al.* 2010).

One source of tritium to the 200 Area is the onsite disposal of tritium-containing wastewater at the State-Approved Land Disposal Site (SALDS) located north of the 200 West Area. Wastewater containing tritium has been discharged to the ground in this area since 1995 (Caron 2008), and in 2009, approximately 96.2 million liters (25.4 million gallons) was disposed of in this fashion (Poston *et al.* 2010). Tritium is discharged to SALDS because no known economically reasonable method of removal has been identified (Barnett *et al.* 2004). Tritium (usually in stacks and vents) has also been released at the permitted discharge points in the 100, 200, 300, 400 and 600 Areas of Hanford Site (Poston *et al.* 2010).

Tritium was detected at low levels in some samples from food products at locations near the Hanford Site, including alfalfa, apples, leaf vegetables, milk, potatoes, tomatoes and wine collected in the vicinity of the site in 2009 (Poston *et al.* 2010).

III. ENVIRONMENTAL CHEMISTRY

Tritium is the only radioactive isotope of hydrogen. It has one proton and two neutrons in the nucleus, in contrast to the nucleus of an ordinary hydrogen atom (which consists solely of a proton) and a deuterium atom (which consists of one proton and one neutron). Ordinary hydrogen comprises over 99.9% of all naturally occurring hydrogen. Deuterium comprises about 0.02%, and tritium comprises about a billionth of a billionth (10^{-16} percent) of natural hydrogen (ANL 2005). Tritiated water

is generally indistinguishable from normal water and can move rapidly through the environment in the same way as water.

The electronic configuration and the chemical properties of tritium are essentially the same as protium and deuterium. Due to the fact that the different isotopes have very different atomic masses, the rates of reaction for the different isotopes vary. The energy provided by the radioactive decay of tritium provides the activation energy required so that some reactions will occur with tritium that will not occur with deuterium or hydrogen (NCRP 1979).

Tritium in the form of HTO may be difficult to store for long periods due to its corrosive properties. This corrosiveness is likely due to tritium oxide generating free radicals (OH^\cdot) from radiolytic decomposition of water in addition to extra energy from beta decay impinging on surrounding molecules (DOE 2008).

When tritium replaces the hydrogen atoms of compounds other than water, they are defined as special tritium compounds (STCs). Examples of STCs are metal tritides and organically bound tritium (OBT). The physical properties of special tritium compounds may make their detection, characterization, and assessments of hazards and exposure effects (i.e., individual dose assessments) difficult (DOE 2008).

FATE AND TRANSPORT

Tritiated water will replace water when released in natural systems, and it behaves much like water in the environment. It is thus one of the most widely distributed contaminants at nuclear facilities. Tritium can transfer to and from the atmosphere from surface runoff, direct evaporation from the surfaces of vegetation, standing pools of water, and the soil surface. Tritiated water that infiltrates the soil is cycled more slowly, and movement in soil may occur both laterally and vertically with losses due to transpiration, evaporation, recharge of surface streams, and direct groundwater flow into oceans. Some of the water that recharges the groundwater moves so slowly that a portion of this tritium is effectively lost from circulation (NCRP 1979).

Soils/Sediments

Because hydrogen is a major constituent of biotic material (along with carbon, oxygen and nitrogen), tritium can become a component of organic molecules within cells and can become involved in biologic processes in the soil and sediments. Differences in bonding forces within organic matter mean that there are two types of organically bound tritium (OBT): exchangeable OBT (exchangeable with hydrogen in cell water) and non-exchangeable OBT (specifically, tritium bound to carbon). The exchange processes can be slowed or even stopped after the death of an organism, causing the organic molecules to remain for a certain time in the soil or sediments (ASN 2010). NCRP (1979) summarizes the soil parameters that affect tritiated water transport: state of the soil (cultivated or fallow), type of soil (clay, loam, or sand), water content, and organic matter. In general, the more water that is already present in the soil, the more slowly the tritiated water will move. Water associated with minerals in the ground will have different degrees of mobility and will be affected by pore size of the sediments.

It is generally assumed that tritium's reactivity with ligands and solids in aquatic systems is limited. Recent studies have shown that the sorption of tritiated water in river water and seawater appears to be influenced by its affinity for organic matter, and that understanding the behavior of OBT is critical. Although tritium equilibrates quickly with dissolved organic ligands and with suspended sediment particles, a fraction of sorbed tritium associates with proteinaceous material that is potentially available to sediment-feeding organisms (Turner *et al.* 2009). High concentrations of organic tritium have been measured in the sediment of water courses affected by discharges from the watchmaking industry, with the organically bound tritium (OBT) content varying with respect to HTO content in the river water by factors of between 1,000 and 10,000 (Jean-Baptiste *et al.* 2007, ASN 2010).

Groundwater

Tritiated groundwater behaves like water, except for a slight change in vapor pressure (NCRP 1979). As a contaminant, it is of most concern in the vadose zone, as it travels through the environment as rapidly as water. At Hanford approximately two-thirds of the tritium produced by fission was released into the groundwater (Haney *et al.* 1962 in NCRP 1979). Studies of the hydrologic and geologic conditions at Hanford have aided in predictions of contaminant migration pathways based on integration of various parameters into three-dimensional hydrogeologic models or "hydrofacies" using tritium as a model because of its ubiquitous, conservative nature. Aspects of the hydrogeological profile such as grain size and sorting, degree of cementation, packing arrangement, sedimentary structures and grain shape affected movement of tritium through the aquifer, as water would be also affected by these parameters (Poeter and Gaylord 1990).

Barnett *et al.* (2004) modeled and ground-truthed the behavior of the tritium plume at Hanford based on lithostratigraphic and hydrostratigraphic conditions. Predictions based on this model and updated increases in future tritium inventories suggest that concentration levels would not drop below the 500 pCi/L level until about the year 2140.

Belot *et al.* (2005) conducted an exercise revealing how much variation can exist in groundwater modeling of tritium depending on different factors. Tritium concentration varies dramatically over short distances and is very sensitive to many interactive factors including rainfall amount, evapotranspiration rate, rooting depth and water table position. Therefore modeling of tritium movement close to the ground surface generally requires rather complex models and detailed input. Others have identified air emissions as a pathway to groundwater contamination, which may be worth further exploration in environmental assessments (Lyness 2000).

Water

Tritium is naturally present in surface waters at about 10 to 30 pCi/L (ANL 2005). Tritium released from nuclear facilities ends up in the environment mainly as tritiated water, which, if dispersed in rivers, is rapidly taken up by biota. In many cases only a few minutes are required for equilibrium conditions to be reached. Most tritium ends

up in the aqueous environment, and cycles through the hydrosphere (Blaylock *et al.* 1986). The relative net flux of HTO from pond water into air with greater than eight percent humidity is greater than that of H₂O (NCRP 1979).

Air

A Tritium Unit (TU) is a metric of atmospheric tritium, representing one atom of tritium per 1×10^{18} atoms of hydrogen. The level of tritium in atmospheric hydrogen increased from 3,800 tritium units in 1948 and 1949 to 490,000 TU in 1959 (Katz 2005). Extensive monitoring of atmospheric tritium concentrations have taken place in Japan from 1984-1999 to establish a general database on the behavior of tritium in the atmosphere (Okai *et al.* 1999). There are three main forms in the atmosphere: tritiated water vapor (HTO), tritiated hydrogen (HT) and tritiated hydrocarbons (primarily tritiated methane, CH₃T). HTO concentrations have a strong correlation with the atmospheric humidity, typically high in the summer and low in winter. In the case of HT and CH₃T, no seasonal variations were observed, and specific activities of the three forms vary, and are much lower for HTO than for the other two (Okai *et al.* 1999).

The transformation of tritium released to the atmosphere into tritiated water vapor is a complex process, with the rate of oxidation depending upon the presence of catalysts as well as on HT and HTO concentrations (NCRP 1979). In order to model tritium's behavior in the global hydrologic cycle, assumptions are typically made about tritium's distribution in the mixed layer of the oceans in the northern hemisphere, as well as food and water consumption by the world's population. Others have put tritium into three (or up to seven) compartments (e.g., atmosphere, oceans, and surface water) in order to model its dynamic behavior (references within NCRP 1979). HTO essentially mimics the hydrological cycle if certain assumptions are made about surface runoff, evaporation, precipitation, absence of HTO releases directly to groundwater, and latitudinal fluxes (NCRP 1979).

BIOACCUMULATION POTENTIAL

Aquatic Systems

Tritium has high food chain mobility because it essentially replaces hydrogen in water and organic compounds. It is not typically thought to bioaccumulate, but recent results from marine studies (e.g., Hunt *et al.* 2010, ASN 2010) indicate that there is potential for bioaccumulation/biomagnification of organically bound tritium, particularly in the marine environment.

Earlier studies of aquatic organisms, however, showed no evidence of bioaccumulation at higher trophic levels (e.g., Blaylock *et al.* 1986). Tritium is rapidly taken up by organisms, but it is also rapidly excreted, with a small fraction that can be bound in tissues and incorporated into proteins and DNA. It is this organically bound tritium (OBT) that is of biomagnification concern, and incorporation of tritium from HTO into the organic matter of algae, for example, occurs mainly by photosynthesis, which causes the splitting of the tritiated molecule (references within Blaylock *et al.*

1986). The specific activity of the total organic matter of algae depends on the external tritium concentration and medium. The marine algae *Acetabularia* attained a specific activity in its organic matter of about six percent of the external medium; in comparison the freshwater algae *Chlamydomonas* reached a specific activity about five times higher for the same concentration of tritiated water. The fate of OBT depends largely on the chemical form of the molecule, with certain biological molecules such as specific amino acids and nucleic acids showing very different concentration ratios (Blaylock *et al.* 1986).

Aquatic plants have not been researched as extensively as aquatic algae, but Harrison and Koranda (1973, in Blaylock *et al.* 1986) found that cattails (*Typha angustifolia*) grown in a pool of tritiated water for 230 days never equilibrated with levels of tritium in the water. In contrast, tissue free water of the submergent pond weed (*Potamogeton foliosus*) was at equilibrium with the lake water; lack of equilibrium for cattails was attributed to exchange with atmospheric water by the exposed leaves, similar to terrestrial plants.

In a more recent study of cattails and carp (*Cyprinus carpio*) in a reservoir contaminated by the cooling operations of a nuclear power plant, tritium levels fluctuated throughout the study period (Baeza *et al.* 2009). Incorporation of tritium to bulrushes and carp was fairly similar, the respective mean concentration factors being 0.74 and 0.8. The temporal changes in tritium levels fairly closely followed that observed for the surface water tritium, although evapotranspiration and the seasonal growth of cattails produced an annual periodicity for the levels of tritium in the plant.

Most studies reviewed by Blaylock *et al.* (1986) to determine trophic transfer and bioaccumulation were experimental manipulations. Organically bound tritium is accumulated slowly and does not reach concentrations as high as those in tissue free water, but organisms that consume tritiated food accumulate OBT at a faster rate and attain a higher concentration than those only exposed to tritiated water. There was no indication of biomagnification of tritium through food chains from these studies.

The recent white paper from the Tritium Working Group of the French Autorité de Sûreté Nucléaire (ASN 2010) attempts to begin to address the potential for biomagnification in the marine environment, and the authors make the distinction between bioconcentration, bioaccumulation, and biomagnification¹²⁶

¹²⁶ Bioconcentration is generally the presence of a substance in an organism (e.g., aquatic organism) at a higher concentration than is measured in the environment (e.g., water). Bioconcentration factors (ratio between the contaminant concentration in the organism and the surrounding environment) are usually defined in the laboratory and do not take into account transmission up trophic levels. Bioaccumulation is often used with the same meaning as bioconcentration, and includes food as a source of contaminant increase, but also takes into account accumulation of the contaminant from other sources in the environment (atmosphere, water, etc.). If the phenomenon of increased contamination occurs at each trophic level, with cumulative increase in concentration of the substance as it moves up the food chain, the term used is biomagnification. Another important consideration with regard to tritium is the idea of remnance, where an organism was exposed to higher environmental concentrations in the past than in the present, leading to a higher concentration of the contaminant as compared to the environment because it fixes in the organism at a particular concentration and remains there for a period of time. Remnance can also apply to abiotic aspects of the

Concentrations of tritium in seawater and marine biota have been elevated as a result of discharges from nuclear facilities in the UK (Cardiff, Sellafield, Hartlepool). Near Cardiff, concentration factors above a priori values have been attributed to discharges of OBT from a health care facility (e.g., Williams *et al.* 2001). Concentration factors increased to values near 7,000 in flounders and about 4,000 in mussels, but since 2001, have been decreasing.¹²⁷ The tritium concentration factors are still high near Cardiff, but not the other two sites; the variation between years, sites, and organisms could be due to changes in discharges. Differences in the organic content of effluent and uptake by different organisms near the different sites may be contributing (Hunt *et al.* 2010).

For the Sellafield site (fuel reprocessing facility which discharges tritiated water), tritium content found as free tritium (HTO) or organically bound tritium (OBT) in marine fauna (fish, crustaceans and mollusks) varies by a factor of 10 with respect to the concentrations found as HTO in the sea water with a one to two year time lag between the maximum discharge values and the maximum tritium values in mollusks and flat fish (ASN 2010). The ASN (2010) Working Group discussed various hypotheses to account for these findings: “Some consider that the abnormally high concentrations measured in fish near Sellafield may result either from remnance in sediment labelling following previously large discharges or from the existence of tritiated organic molecules in the same water outflow. Others consider that this is a case of bioaccumulation which is related to the discharge of tritiated water. According to proponents of this view, the hypothesis of marine currents carrying tritium-labelled organic molecules discharged by the Cardiff radiochemical plant is refuted by the fact that analysis carried out near the Wylfa nuclear power plant (NPP), on the west coast of the UK between Cardiff and Sellafield did not find any detectable presence of tritium in the marine fauna.”

Concentration ratios (CRs), or the whole organism-to-water activity concentrations, are typically used to estimate radionuclide concentrations in biota at particular bodies of water over time based on surface water measurements. An international model validation study has begun to address the complexities of developing and standardizing methods of assessing impacts of radionuclide contamination on diverse biota and whole ecosystems rather than just humans (e.g., Yankovich *et al.* 2009). Numerous models were tested with field measurements of tritium at Perch Lake, Atomic Energy of Canada Limited (AECL)’s Chalk River Laboratories site. In general, modeled values were 1-3-fold less than measured values based on a concentration ratio (CR) of one, though two models (RESRAD-BIOTA, NRPA and

environment such as sediments, when biophysical processes can cause a substance to be fixed in the environment over the long-term (ASN 2010).

¹²⁷ Since tritiated water is effectively chemically identical to normal water, the concentration of tritiated water in an organism is expected to be very close to the concentration of tritiated water in the water from which the organisms obtains its water. When a large breakdown in this relationship is observed, it is likely due to a temporal change in the concentration of tritium in the water resource and the organisms has not had a chance to re-establish equilibrium between the organism and its source of water.

USDOE 2002, in Yankovich *et al.* 2009) over- and under-estimated tritium activity concentrations in fish by 10- and five-fold, respectively (Yankovich *et al.* 2009).

Additional discussion of bioconcentration factors and accumulation within tissues of aquatic organisms continues below (in the section ‘Accumulation Within Tissues’).

Terrestrial Systems

Tritium is readily incorporated into plants during photosynthesis and is distributed throughout the food chain by consumption of plants (e.g., Choi and Aranoff 1966, Kanazawa *et al.* 1982 in Driver 1994). Retention in terrestrial systems depends on rates of catabolism at various trophic levels and the forms and extent of OBT incorporated into organisms (Driver 1994). Neither plants nor animals are traditionally thought to concentrate tritium in their tissues, and hence its biomagnification potential is generally discounted (Driver 1994). Boyer *et al.* (2009) review the literature on tritium in plants, including the processes of uptake, incorporation and conversion of tritium into plants.

Tritium concentration in plants is strongly correlated to the HTO concentration in water vapor and precipitation, and plants obtain tritium from both water vapor and soil-water. The relative humidity of the atmosphere strongly influences where plants get most of their tritium: if humidity is high, most tritium originates from the air, and if humidity is low, most tritium enters plants via the root system (Kumar *et al.* 2010). These authors also reiterate the need for additional study of OBT in plants in order to understand the behavior of tritium in terrestrial systems.

Vichot *et al.* (2008) studied plants and lichens continuously exposed to tritium in the atmosphere in order to test exposure models. The concentration of organically bound tritium in tree rings was strongly correlated with timing of tritium releases, and distance from releases could explain some variation. Lichens are recognized as bioindicators, and for very contaminated areas, OBT activity in lichens has been measured at levels 1,000 times higher than background, and still 10-100 times higher at a distance of 20 km from the tritium release source (Daillant *et al.* 2004). Their slow metabolism makes them suitable for tracing of tritium incorporated by photosynthesis. Other authors argue that lichens cannot be used to determine tritium integration time and integration appears to be variable depending on lichen species, however, so their use as bioindicators may be limited (Vichot *et al.* 2008).

Momoshima *et al.* (2000) found that tritium concentrations in organic fractions of dead and degraded pine needles accumulated on the forest floor was higher than the water fractions of tritium. The higher tritium concentration in the organic fraction was thought to be caused by decomposition, particularly microbial oxidation of atmospheric hydrogen and methane. Rain was the primary source of water fractions in the samples (with larger water fractions in fresh pine needles, but lower tritium concentrations in the fresh needles).

ACCUMULATION WITHIN TISSUES

Photosynthesis readily incorporates tritium into plant tissues, but neither plants nor animals are thought to concentrate tritium in their tissues (Driver 1994). The biological half-life for plants can be thought of in three phases: the first component is rapidly excreted (about 90%) within 0.3 to 2.0 hours, the organically bound tritium (OBT) is excreted within 17 to 30 hours, and tritium in the soil water has a half-life of 80 to 270 hours (references within Driver 1994). Plant concentration ratios are less than 1.0 for plants exposed to tritiated water (Diabete *et al.* 1990 in Driver 1994).

Ingested HTO in animals is almost completely absorbed from the gastrointestinal tract and is quickly incorporated into the blood (Pinson and Langham 1957 in NCRP 1979). Within minutes it can be found in varying concentrations in the various organs, fluids, and tissues of the body (references within NCRP 1979). Most mammalian studies on tritium ingestion and accumulation have been done on mice, rats or agricultural animals (references within Driver 1994). The biological half-life of tritium is known to vary with animal species, the age of the animal, and the tissue or organ under consideration (NCRP 1979). Most relevant to the effects of tritium and its half-life within an organism is how much of it is incorporated into DNA. In one study with mice and rats given tritiated water for 41-147 days, the nonexchangeable OBT was 25-40% of the tissue free water tritium. The mouse liver DNA non-exchangeable OBT was 12% of that of the tissue water. In another study (Mewissen and Rust 1975 in NCRP 1979) incorporation of tritium into RNA was five-fold greater than in DNA.

Calves and pigs show a 10-60-fold increase of tritium in their tissues if they ingest tritiated plant material vs. drinking tritiated water (Kirchmann *et al.* 1977 within Driver 1994). Rats show an increase in tritium in their tissues related to the protein content of the plant materials they ingest, and food with higher fat content meant higher tritium content in the rats' fat tissue (Takeda and Iwakura 1992 in Driver 1994). Rats, mice, and rabbits fed either tritiated food or tritiated water had specific activity ratios ranging from 0.2-1.0 after continuous ingestion of tritium (references in Driver 1994). Only kangaroo rats had higher concentration of tritium because of their unique water metabolism (1.2-1.6). Biological half-lives of the first compartment of tritium in body water of mice, pigs, and cows range from 1.1 hours (mouse) to over four hours for pigs and cows. The second OBT compartment was 33 hours for lactating cows and 40 hours for non-lactating cows (references within Driver 1994). There are differences in the residual specific activity of tritium in various organs and tissues, with higher specific activity typically seen in brain and body fat, but it is always well below (10-40%) the specific activity of the tritiated water from which it was derived (Thompson 1971 in NCRP 1979).

Studies on birds are limited, with most on domestic fowl, with human ingestion as the focus of most investigations. Peak activity of the free water fraction of chicken eggs occurred one day after hens received a single dose of tritiated water, and decreased with an average half-life of 3.65 days (Mullen *et al.* 1975). Peak activity of tritium incorporated into the organic constituents was recorded after six days for albumen and

eight days for yolk with half-lives very similar to the free water fraction (Mullen *et al.* 1975).

As described above, the exchangeable form of tritium contains hydrogen molecules bound to elements other than carbon. About 70% of the body's hydrogen is in this form, as body water, whereas non-exchangeable hydrogen makes up the remainder in compounds such as proteins, lipids, carbohydrates, and nucleic acids. These strong carbon-hydrogen bonds are stable and only broken down with enzymatic reactions, so once tritium is incorporated into these components of cells as organically bound tritium (OBT), it has a longer biological half-life (e.g., Galeriu *et al.* 2005). Other studies in birds have focused on tritiated thymidine because of its use in molecular tracing studies. Party *et al.* (1997) found that birds injected with tritiated thymidine eliminated 98% of the radioactivity within 28 hours, via excreta.

The majority of marine/aquatic studies focus on the dose effects on humans (e.g., Hodgson *et al.* 2005, Harrison 2009), but some assays that have shown genotoxic effects on mussels (*Mytilus edulis*), have also shown that inorganic tritium accumulated differently in mussel tissues, with the gut accumulating the highest amount of radioactivity, followed by the gill, mantle, muscle, food and byssus thread (Jha *et al.* 2005). Differential accumulation within tissues of aquatic organisms has implications for biomonitoring assays.

Numerous studies have focused on dose coefficients for humans, typically using the rat model, but with renewed interest in risk from seafood ingestion because of high tritium releases in areas like Cardiff Bay, U.K. (e.g., Hodgson *et al.* 2005, Harrison 2009). For marine animals biological half-lives vary from 110-290 days (NCRP 1979). Particular concern exists about ingestion of OBT directly, and estimates of incorporation into body tissues (particularly DNA) because of uncertainties in biokinetics and reliance on animal models. For intakes of OBT by adults it is generally assumed that 50% of tritium is non-exchangeably incorporated into organic molecules in body tissues. Substantial uncertainty is associated with this estimate because it is based on the behavior of selected forms of OBT in animals (Hodgson *et al.* 2005). Dose coefficients for ingestion of HTO or OBT also assume that absorption of tritium from the alimentary tract to blood is complete, and that tritium is then uniformly distributed throughout all body tissues. For intakes of HTO, the two components (HTO and OBT) are taken to account for 97% and 3% of tritium reaching blood, with half-times of retention in adults of 10 days and 40 days, respectively (ICRP 1993 and Harrison *et al.* 2002 in Hodgson *et al.* 2005).

Devol and Powell (2004) investigated theoretical vs. experimentally-based dose ratios of OBT to free water tritium from ingestion of various foods, with theoretical values from grains in particular being 261% above the experimentally-derived value. The OBT vs. free water tritium content of different foods and the T:H ratio is a function of the kinetics associated with the assimilation of tritium into the tissues (Devol and Powell 2004).

IV. TYPICAL MAJOR EXPOSURE ROUTES As discussed in other sections of this profile, tritiated water, atmospheric tritium including tritiated water vapor and food products containing tritium (particularly organically bound tritium) are all routes of exposure for organisms including humans and other animals directly or indirectly. Though its beta energy is weak, tritium exposure through food is of concern (especially food containing OBT); if tritium is incorporated into DNA, genotoxicity can result (e.g., Balonov *et al.* 1993). Tritium can be absorbed either as a gas or water vapor via skin or lungs and when humans inhale gaseous tritium a very small fraction is converted to HTO (about 0.004%) and retained as free water (Pinson 1951 in NCRP 1979). A small fraction of the tritium atoms from HTO can be incorporated into OBT, but most is turned over from the free water pool quickly. The uptake of tritium via inhalation of tritiated water vapor is efficient, with 99% of that inhaled taken into the body water within seconds (Hutchins and Vaughn 1965 in NCRP 1979). Skin uptake of tritiated water is correlated with skin temperature and is typically about equal to intake by inhalation (Osborne 1966 in NCRP 1979), and the amount of HTO absorbed through the skin is dependent on humidity as well. Hot weather and high humidity means more absorption of HTO, as would occur with regular water moving through the skin (ANL 2005).

V. CHEMICAL ECOTOXICITY Tritium, especially in the form of tritiated water, which is by far its most common form in the environment, is generally not considered to be chemically toxic because it is, for all intents and purposes, water. However, according to NCRP (1979): “when radioactive decay of tritium occurs, the emission of the beta particle gives the resulting species a recoil momentum that is very large in chemical terms.” The so-called “hot atoms” can be used for labeling organic compounds (e.g., thymidine) and the recoil momentum of the helium ion formed by beta decay is sufficient to break C-H bonds and allow substitution of tritium at any position occupied by a hydrogen atom. In this respect, tritiated water can be considered to be chemically toxic, but, in fact, it is its radiological decay that is responsible for this particular type of biochemical insult.

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

There are no known beneficial or protective properties of tritium, although it does have beneficial uses. Tritium is used as a tracer in biological and environmental studies, and as an agent in luminous paints such as those used to make building exit signs, airport runway lights and watch dials (ANL 2005, Mutch and Mahony 2008). Tritium is also widely used as a tracer in molecular biology experiments in which tritium-labeled thymidine is typically used (Katz 2005).

VI. RADIOLOGICAL ECOTOXICITY **MECHANISM(S) AND LOCI OF TOXICITY** Radioecological damages to aquatic and terrestrial organisms due to tritium in the environment result from ionization caused by the interaction of its beta particles with living tissue and to a lesser degree the recoil of the tritium atom incorporated into DNA and the associated changes in the base pair sequences.

Upon each disintegration, tritium emits a beta particle with a maximum energy of 0.0186 MeV and an average energy of 0.0057 MeV (Shleien *et al.* 1998). In all organisms, tritium is metabolized as water, and distributed throughout the body as water, where all of its beta energy is deposited.

Internal Beta Exposures

Figure 5.8.1 of Shleien *et al.* 1998 indicates that beta particles of this energy have a range in water (which is equivalent to tissue in terms of stopping power) of about 0.0001 cm²/g, which is about 0.0001 cm in tissue. Given that the typical energy required to ionize a molecule (i.e., eject an electron from its orbit) is about 34 to 35 eV (see Casarett 1968, page 17), the total number of ion pairs produced by the energy deposited in tissue from the typical energy beta particle emitted by tritium is about 2163 ion pairs (i.e., ~0.0057 MeV/35 eV).

The pattern of energy deposition for beta particles is described in Morgan and Turner (1973) as follows:

$$\text{Mean linear ion density} = T/R_t \times W$$

Where:

T = average energy of electron liberated

R_t = range or electrons of energy T

W = average energy to form an ion pair

For tritium, the equation is 0.0057 MeV × 1,000,000 eV/MeV ÷ 0.0001 cm × 35 eV/ion pair = 1.6 × 10⁶ ion pairs per cm or about 160 ion pairs per micron. Given that a typical cell is on the order of tens of microns (see page 102 of Curtis and Barnes 1989), a single cell might experience about 1600 ion pairs produced by the passage of a typical tritium beta particle. It is primarily this deposited energy in living tissue that results in biological damage. Also of concern is the replacement of hydrogen atoms with tritium in nucleic acids, and resulting genotoxic effects.

Jones *et al.* (2010) summarizes the impact of ionizing radiation on cells:

“Ionizing radiation is now recognized to be a significant risk for carcinogenic events. By its nature, radiation exposure to living system of energy sufficient to produce atomic ionization, can damage key cellular molecules and organelles, especially nuclear and mitochondrial nucleic acids. Significant exposure or high energy radiation produces single and double strand breaks in the nucleic acids. Damaged cellular molecules can result in perturbed cellular function, altered transcription, translation, and reproduction. These perturbations are the root for cellular genotypic and phenotypic changes that lead to neoplastic transformation. Recent studies are beginning to unfold possible mechanisms of ionizing radiation-induced carcinogenesis... Epidemiological data has been accumulating from numerous sources of animal and human ionizing radiation exposure, which clearly links these exposures with subsequent carcinogenesis. At sub-lethal doses, ionizing radiation is a powerful carcinogen, even though at high doses it is lethal

to both normal and neoplastic cells and tissues. Since at least one of the molecular events seems to occur via radiation-induced reactive oxygen species (ROS) formation, then a possible means to reduce the risk of radiation-induced cellular damage may be via free radical scavengers, antioxidants, stimulators of ROS clearance and other radioprotectors and immune modulators.”

FACTORS AFFECTING TOXICITY

Because tritium in the environment and incorporated into biota is chemically identical to water, there are no factors that can affect its uptake or toxicity. However, in higher organisms, drinking lots of water increases the rate at which tritiated water is excreted from the body, thereby reducing the dose to organisms that have absorbed tritium. As far as combined effects, very little is known about the combined action of other stressors on the adverse effects of radiation, including internal exposure to tritium, on biota.

PLANTS

Boyer *et al.* (2009) review the literature on tritium in plants, including the processes of uptake, incorporation and conversion of tritium into plants. Little work has been done on the effects of beta-emitters such as tritium alone on plants, as most ionizing radiation exposure includes gamma- and alpha-emitters. However, numerous studies have investigated the effects of chronic exposure to multiple radionuclides at sites such as Chernobyl (e.g., Grihikh and Shevchenkovv 1992, Bourbriak *et al.* 2008). Chromosomal aberrations are typically evident in root meristematic cells during the first mitosis metaphases of seed germination, with especially high frequency soon after initial radiation release (Grihikh and Shevchenkovv 1992). Both diploid cells (e.g., seed embryos of the evening primrose) and haploid cells (e.g., birch pollen) from plants in fall-out sites show DNA damage as well as improved capacity to repair DNA damage over time; however the ability to repair DNA damage does not seem to improve at sites exposed to combined alpha- and gamma/beta- emissions (Bourbriak *et al.* 2008).

AQUATIC INVERTEBRATES AND FISH

According to NCRP (1979) the vertebrates with the shortest half-life for free water tritium probably are freshwater fish, with a two component half-life of 0.2 hours (96%) and 0.9 hours (4%) and for OBT a half-life of 8.7 days, except for small remaining residue.

One of the largest spawning populations of fall Chinook salmon (*Oncorhynchus tshawytscha*) occurs on the Hanford Reach (Dauble and Watson 1997) and other nearby ‘Ecologically Significant Units’ of this species are listed as threatened or endangered under the federal Endangered Species Act. Steelhead trout (*Oncorhynchus mykiss*) within the Hanford Reach are considered part of the upper Columbia River Evolutionarily Significant Unit and are listed as endangered under the federal Endangered Species Act (Mueller 2009). The early life stages of these species are potentially exposed to radiological contaminants that enter the river via shoreline seeps and upwelling through the river substrate (Poston *et al.* 2003). Poston *et al.*

(2003) performed dose assessments for developing salmonid embryos for hypothetical exposure to tritium at specific sites on the Hanford Reach, and found that at the Tier I screening level, no site approached the dose guideline of 10 mGy/d established with the RAD-BCG calculator, but cumulative impacts should be monitored.

Jha *et al.* (2005, 2006) investigated the genotoxic effects of tritium on adult mussel (*Mytilus edulis*) haemocytes. A dose dependent increase was seen in induction of micronuclei and single strand DNA breaks/alkali labile sites (Comet assay), and less than 500 $\mu\text{Gy/h}$ (0.05 rad/h) of tritium is capable of inducing genetic damage.

MAMMALS

Tritium ingested in organic form in food is approximately three times more radiotoxic than tritiated water (dose coefficient per unit of activity ingested is approximately three times higher) due to the biological (elimination) half-life (ASN 2010).

Consistent with this, the frequency of dominant lethal mutations induced by OBT in the form of labeled lysine, thymidine, and deoxycytidine is three to 12 times higher than those induced by equal HTO activity (Balonov *et al.* 1993).

Even tritiated water has the potential to have genotoxic effects, however. Ribas *et al.* (1994) found that low concentrations of beta-radiation administered in the form of tritiated water to human blood lymphocyte cultures induced a significant increase in the frequency of chromosome aberrations, though it did not induce sister-chromatid exchanges. Other studies have investigated the effects of tritiated water on DNA damage and repair in blood cells, mainly using mice and rats as models (e.g., Balonov *et al.* 1993). Because tritium is readily absorbed into the bloodstream from all routes of exposure, radiological effects are comparable to those of whole body exposure (Osborn 1972 in Driver 1994). Studies in mice and rats indicate that the radiobiological effects of tritium beta radiation in the form of HTO is two to six times higher than gamma radiation of Cesium-137 (Balonov *et al.* 1993). This is believed to occur because the linear energy transfer (LET) of the betas from tritium are higher than the LET from other beta and gamma emitters. Hence, the same dose in rad from tritium is more damaging than the dose from other beta/gamma emitters.

Genetic consequences, especially in ovaries and testes are of primary concern (Driver 1994). Again, the mouse model is most common in studies of tritium effects on oocytes. For example, a particular dose response relationship is defined for frequencies of chromosome aberrations in mouse eggs at the pronuclear stage exposed to beta-particles via tritiated water (Matsuda *et al.* 1986).

Tritium has also been studied in mammals in the context of relative biological effectiveness (RBE) experiments (Little and Lambert 2008). The RBE is a way to estimate the absorbed dose of particular radiation under consideration by using reference doses of other types of radiation (gamma, x-ray). Little and Lambert (2008) compiled tritium RBE studies that evaluated endpoints including carcinogenesis, chromosomal aberration, cell death, and others; however, reference radiations vary widely in the published literature, and doses and dose rates studied in organisms other than man are frequently much higher than those normally received by humans. The

authors caution that the RBE values summarized in their review should not be used as RBE_{max} (the maximum relative biological effects observed experimentally) because of flaws and varied interpretations in many of the studies. Challenges with understanding risk to humans from tritium radiation are many, and ecologically relevant wildlife studies are few (Little and Lambert 2008). It is important to keep in mind that experimentally observed RBEs are unique to the radionuclide, organism, biological end point, and circumstances that apply to a given experiment. Hence, caution must be used when extending an RBE determined from one experiment to another.

BIRDS

Driver (1994) summarizes general effects of ionizing radiation on birds: “As a group, birds appear to be at greater risk of beta-gamma radiation exposure than other wild animals. About 33% of birds collected from a contaminated area had radiation counts above the background level, whereas only 7% of the mammals collected, and 5% of the reptiles collected had higher-than-background counts. The higher rate of contamination was attributed to the grit-use behavior of birds (Bellamy *et al.* 1949).”

Information on tritium’s specific effects on birds is limited. Driver (1994) summarizes Hanson and Watson’s (1960) concentration factors for beta-emitters for birds that might inhabit the Columbia River area, including shorebirds, diving ducks, river ducks, grebes, gulls, and mergansers. They concluded that omnivorous and fish-eating birds have lower concentration factors than invertebrate- or larvae-feeders or herbivores. Driver (1994) also reviewed LD50/30s for a variety of avian species (passerines and waterfowl) and found that LD50/30s ranged from 400-1060 rad. Types of radiation were not always defined in this review, but impacts of ionizing radiation on a wide range of species, behavioral impacts, genotoxic effects, and abnormalities are summarized. For some studies of sublethal effects, no reproductive effects were observed, even at relatively high cumulative doses (e.g., 500 to 5316 R did not affect egg production, plumage coloration, or ovarian tissue structure (Greb 1955, Greb and Morgan 1961 in Driver 1994). For those studies that focused on genotoxic and other cellular effects, these ranged from increased mitotic abnormalities and inhibition of cell division of the cornea to testicular damage- and arrested germ cell maturation. Driver (1994) concludes that gross congenital abnormalities induced by radiation are relatively uncommon in birds.

VII. EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

There have been few studies on tritium’s synergistic effects with other contaminants. One would expect additive and synergistic effects of exposure to tritium with other environmental toxicants, such as heavy metals. Also because tritium’s effects are only radiological, exposure to tritium usually comes with exposure to other radionuclides that might be more toxic, especially cumulatively (e.g., Balonov *et al.* 1993). Concerns over the possible synergistic effects of exposure to radiation and chemical toxins have been extensively raised and reported in the scientific literature (Burkart *et al.* 1997, Prasad *et al.* 2004) but little consensus has been achieved in quantifying

these effects in humans, except possibly for radon and smoking (BEIR IV 1988) and certainly in the enhancement of the therapeutic effects of radiotherapy used to treat cancer (e.g., Lew *et al.* 2002). UNSCEAR (2000) Annex H explores the combined effects of radiation and chemical agents, including heavy metals. Only a few data are available from combined exposures of radiation and metals in human populations and no firm evidence of interactions has been observed.

There is some literature on observed synergistic adverse effects of radiation and toxic chemicals on organisms other than humans (e.g., salmon, Mothersill *et al.* 2007). Examples of ionizing radiation and metals producing combined effects in other biological systems include synergistic effects on soil microbial activity from cadmium and zinc in combination with gamma radiation (summarized in UNSCEAR 2000).

Overall, there is a clear need for additional research on synergistic effects of multiple stressors in radioecotoxicology (e.g., Salbu and Skipperud 2007, Mothersill and Seymour 2007). In particular, these authors raise the issue of pesticides, organics, and endocrine disruptors and synergistic effects with radioactive materials, particularly with long-term exposure to various biological systems. Manti and D'Arco (2010) summarize the *in vitro* and animal-model studies and epidemiological surveys with two or more stressors, including radionuclides (DNA-damaging agents). They also emphasize that most research focuses only on the short-term effects of combined single exposures to animal models, and more work is needed to understand chronic exposure to trace contaminants and radioactive elements in the environment, including impacts to long-term genome stability. Specific research is lacking on tritium effects with multiple stressors on biological systems, particularly non-human systems.

VIII. DATA GAPS The recent Autorité de Sûreté Nucléaire (ASN) Working Group on Tritium in France (ASN 2010) put out several recommendations for further study surrounding tritium in the environment including: 1) standardizing measurement protocols for the various forms of tritium (particularly exchangeable and non-exchangeable), 2) improvement of monitoring of discharges (particularly of OBT) and reference species for environmental sampling, 3) improvement of RBE estimates, 4) standardizing dose assessment methods according to the forms of tritium, contamination pathways and length of exposure, 5) focusing studies on the effects of tritium exposure on embryos and fetuses, and investigate potential induction of hereditary effects, and 6) assessing the feasibility of epidemiological studies. This group also focused on the need for more work on the possible biomagnifications of OBT, as some suspect may occur in marine systems.

Studies of ecologically relevant doses and effects of tritium to wildlife species are lacking, particularly for non-laboratory, non-agricultural mammals, birds, amphibians, and reptiles.

IX. REFERENCES

- Argonne National Laboratory (ANL). 2005. Human Health Fact Sheet. <http://www.ead.anl.gov/pub/doc/tritium.pdf>. Accessed 4 January, 2001.
- Autorité de Sûreté Nucléaire (ASN). 2010. Summary of work and recommendations by the “Tritium impact” working group. Smeesters, P., Chairman. 8 July 2010, English Version, White Paper.
- Baeza, A., E. Garcia, J.M. Paniagua, and A. Rodriguez. 2009. Study of the comparative dynamics of the incorporation of tissue free-water tritium (TFWT) in bulrushes (*Typhalatifolia*) and carp (*Cyprinus carpio*) in the Almaraz nuclear power plant cooling reservoir. *Journal of Environmental Radioactivity* 100: 209-214.
- Balonov, M.I., K.N. Muksinova, and G.S. Mushkacheva. 1993. Tritium radiobiological effects in mammals – review of experiments of the last decade in Russia. *Health Physics* 65:713-726.
- Barnett, D.B., M.P. Bergeron, and E.J. Freeman. 2004. Results of groundwater modeling for tritium tracking at the Hanford Site 200 Area State-Approved Land Disposal Site – 2004. Pacific Northwest National Laboratory, PNNL-14898.
- Belot, Y., B.M. Watkins, O. Edlund, D. Galeriu, G. Guinois, A.V. Golubev, C. Meurville, W. Raskob, M. Taschner, and H. Yamazawa. 2005. Upward movement of tritium from contaminated groundwaters: a numerical analysis. *Journal of Environmental Radioactivity* 84:259-270. [abstract only]
- Blaylock, B.G., F.O. Hoffman, and M.L. Frank. 1986. Tritium in the aquatic environment. *Radiation Protection Dosimetry* 16:65-71.
- Boubriak, I.I., D.M. Grodzinsky, V.P. Polischuk, V.D. Naumenko, N.P. Gushcha, A.N. Micheev, S.J. McCready, and D.J. Osborne. 2008. Adaptation and impairment of DNA repair function in pollen of *Betula verrucosa* and seeds of *Oenothera biennis* from differently radionuclide-contaminated sites of Chernobyl. *Annals of Botany* 101:267-276.
- Boyer, C., L. Vichot, M. Fromm, Y. Losset, F. Tatin-Froux, P. Guetat, and P.M. Badot. 2009. Tritium in plants: a review of current knowledge. *Environmental and Experimental Botany* 67:34-51. [abstract only]
- Burkart, W., G.L. Finch, and T. Jung. 1997. Quantitative health effects from the combined action of low-level radiation and other environmental agents can new approaches solve the enigma. *Science of the Total Environment* 205:51-70.
- Caron, M.E. 2008. Results of tritium tracking and groundwater monitoring at the Hanford Site 200 Area State-Approved Land Disposal Site. Fiscal Year 2008. Revision 0. CH2M Hill Plateau Remediation Company. SGW-38802.
- Committee on the Biological Effects of Ionizing Radiation (BEIR). 1988. Health Risks of Radon and Other Internally Deposited Alpha-Emitters, BEIR IV, Biological Effects of Ionizing Radiation, National Research Council, National Academy of Sciences, National Academy Press, Washington, D.C. 1988.

- Casarett, A.P. 1968. Radiation Biology. Englewood Cliffs, New Jersey: Prentice-Hall, Inc.
- Curtis, H. and N.S. Barnes. 1989. Biology. Worth Publishers, Inc.
- Daillant, O., G. Kirchner, G. Pigree, and J. Porstendorfer. 2004. Lichens as indicators of tritium and radiocarbon contamination. *Science of the Total Environment* 323:253-262.
- Dauble, D.D. and D.G. Watson. 1997. Status of fall chinook salmon populations in the mid-Columbia River: 1948-1992. *North American Journal of Fisheries Management* 17:283 - 300. [abstract only]
- Department of Energy (DOE). 2008. DOE Handbook: Tritium Handling and Safe Storage. DOE-HDBK-1129-2008. December, 2008. Washington, DC.
- Devol, T.A. and B.A. Powell. 2004. Theoretical organically bound tritium dose estimates. *Health Physics* 86: 183-186.
- Driver C.J. 1994. Ecotoxicity Literature Review of Selected Hanford Site Contaminants. PNL-9394, Pacific Northwest Laboratory, Richland, Washington. <http://www.osti.gov/energycitations/servlets/purl/10136486-6sLptZ/native/10136486.pdf>
- Galeriu, D., R. Heling, and A. Melintescu. 2005. The dynamics of tritium – including OBT – in the aquatic food chain. *Fusion Science and Technology* 48: 779-782.
- Galeriu, D., A. Melintescu, N.A. Beresford, H. Takeda, and N.M.J. Crout. 2009. The dynamic transfer of ³H and ¹⁴C in mammals: A proposed generic model. *Radiation and Environmental Biophysics* 48:29-45.
- Grinikh, L.I. and V.V. Shevchenkova. 1992. Cytogenetic effects of ionizing-radiation in *Crepis tectorum* growing within 30 km of the Chernobyl atomic power-station. *Science of the Total Environment* 112:9-18.
- Harrison, J. 2009. Doses and risks from tritiated water and environmental organically bound tritium. *Journal of Radiological Protection* 29:335-349.
- Hartman, M. 2003. Groundwater contamination at U.S. Department of Energy's Hanford site. 2003 Seattle Annual Meeting (November 2-5, 2003), Geological Society of America Abstracts with Programs, Vol. 35, No. 6, September 2003, p. 531
- Hodgson, A., J.E. Scott, T.P. Fell, and J.D. Harrison. 2005. Doses from the consumption of Cardiff Bay flounder containing organically bound tritium. *Journal of Radiological Protection* 25:149-159.
- Hunt, G.J., T.A. Bailey, S.B. Jenkinson, and K.S. Leonard. 2010. Enhancement of tritium concentrations on uptake by marine biota: experience from UK coastal waters. *Journal of Radiological Protection* 30:73-83.
- Jean-Baptiste, P., D. Baumier, E. Fourre, A. Dapoigny, and B. Clavel. 2007. The distribution of tritium in the terrestrial and aquatic environments of the Creys-

- Malville nuclear power plant (2002-2005). *Journal of Environmental Radioactivity* 94:107-118.
- Jha, A.N., Y. Dogra, A. Turner, and G.E. Millward. 2005. Impact of low doses of tritium on the marine mussel, *Mytilus edulis*: Genotoxic effects and tissue-specific bioconcentration. *Mutation Research, Genetic Toxicology and Environmental Mutagenesis* 586: 47-57.
- Jha, A.N., Y. Dogra, A. Turner, and G.E. Millward. 2006. Are low doses of tritium genotoxic to *Mytilus edulis*? *Marine Environmental Research* 62:S297-S300.
- Jones, J.A., R.C. Casey, and F. Karouia. 2010. Ionizing radiation as a carcinogen. *Comprehensive Toxicology* 14:181-228.
- Katz, J.K. 2005. Deuterium and Tritium. In: Kirk-Othmer Encyclopedia of Chemical Technology. New York, NY: John Wiley & Sons. [abstract accessed only]
- Kumar, A., Y.P. Gautam, V. Kumar, K.S. Rao, S. Sharma, A.K. Sharma, and A.G. Hegde. 2010. Uptake study of tritiated water (HTO) by plants after long-term continuous normal release around Narora Atomic Power Station. *Journal of Radioanalytical Nuclear Chemistry*, available online 6 Jan 2011.
- Lew, Y.S., A. Kolozsvary, S.L. Brown, and J.H. Kim. 2002. Synergistic interaction with arsenic trioxide and fractionated radiation in locally advanced murine tumor. *Cancer Research* 62: 4202-4205.
- Little, M.P. and B.E. Lambert. 2008. Systematic review of experimental studies on the relative biological effectiveness of tritium. *Radiation and Environmental Biophysics* 47: 71-93.
- Lyness, L.S. 2000. Assessing air emission impacts on groundwater quality. In: Singhal, R.K. and A.K. Mehrotra. Environmental issues and management of waste in energy and mineral production. 6th International Conference on Environmental Issues and Management of Waste in Energy and Mineral Production, Calgary, Canada, May 30-June 2, 2000.
- Manti, L. and A. D'Arco. 2010. Cooperative biological effects between ionizing radiation and other physical and chemical agents. *Mutation Research* 704:115-122.
- Matsuda, Y., I. Tobar, and T. Yamada. 1986. Chromosome aberrations induced by tritiated water or ^{60}Co γ -rays at early pronuclear stage in mouse eggs. *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 160:87-93.
- McNaughton, M. 2005. Biota dose assessment at Los Alamos National Laboratory. LA-UR-05-4699. July 7, 2005.
- Momoshima, N., H. Kakiuchi, T. Okai, S. Hisamatsu, and Y. Maedal. 2000. Tritium in a pine forest ecosystem: Relation between fresh pine needles, organic materials on a forest floor and atmosphere. *Journal of Radioanalytical and Nuclear Chemistry* 243:479-482.

- Morgan, K.Z. and J.E. Turner. 1973. Principles of Radiation Protection. Huntington, New York: Robert E. Krieger Publishing Company.
- Mothersill, C., B., Salbu, L.S. Heier, H.C. Teien, J. Denbeigh, D. Ougton, B.O. Rosseland, and C.B. Seymour. 2007. Multiple stressor effects of radiation and metals in salmon (*Salmo salar*). Journal of Environmental Radioactivity 96:20-31.
- Mothersill, C. and C. Seymour. 2007. Radiation risks in the context of multiple stressors in the environment – issues for consideration. In: Mothersill, C., I. Mosse, and C. Seymour, eds. Multiple stressors: a challenge for the future. Book Series: NATO Science for Peace and Security Series C – Environmental Security. Pp. 235-246.
- Mueller, R.P. 2009. Steelhead – 2009. Environmental Report Section 8.14.1.3. Pacific Northwest National Laboratory.
http://www.pnl.gov/ecomon/2009_report/pnnl_19455_fw_8.14.1.3.asp Accessed 12 January 2011.
- Mullen, A.A., R.E. Stanley, S.R. Lloyd, and A.A. Moghissi. 1975. Biological half-life of tritium in chickens and eggs. Health Physics 29:917-918. [abstract only]
- Mutch, R.D. and J.D. Mahony. 2008. Study of tritium in municipal solid waste leachate and gas. Fusion Science and Technology 54:305-310.
- National Council on Radiation Protection and Measures (NCRP). 1979. Tritium in the Environment. NCRP Report No. 62. Washington, DC.
- Nyhan, J.W., P.R. Fresquez, W.R. Velasquez, and E.A. Lopez. 2000. Radionuclide concentrations in soils and vegetation at low-level radioactive waste disposal area G. LA-13771-PR Progress report, Los Alamos National Laboratory, New Mexico.
- Okai, T., N. Momoshima, and Y. Takashima. 1999. Variation of atmospheric tritium concentrations in three different chemical forms in Fukuoka, Japan. Journal of Radioanalytical and Nuclear Chemistry 239: 527-531.
- Party, E., A. Barnea, I. Linins, and E.L. Gershey. 1997. Elimination of tritiated thymidine from birds. Health Physics 72:633-635.
- Poeter, E. and D.R. Gaylord. 1990. Influence of aquifer heterogeneity on contaminant transport at the Hanford site. Ground Water 28:900-910.
- Poston, T.M. and D.C. Klopfer. 1985. A literature review of the concentration factors of selected radionuclides in freshwater and marine fish. PNL-5484, Pacific Northwest Laboratory, Richland, Washington.
- Poston, T.M., E.J. Antonia, and R.E. Peterson. 2003. Application of biota dose assessment committee methodology to assess radiological risk to salmonids in the Hanford reach of the Columbia River. Conference Information: 3rd International Symposium on the Protection of the Environment from Ionising

- Radiation (SPEIR 3), July 22-26, 2002 Darwin, Australia. In: Book Series 17: 397-405. [abstract only]
- Poston, T.M., J.P. Duncan, and R.L. Dirkes, eds. 2010. Hanford Site Environmental Report for Calendar Year 2009. PNNL-19455. Pacific Northwest National Laboratory, Richland, WA. 393 pp.
- Prasad, K.N., W.C. Cole, and G.M. Hasse. 2004. Health Risks of Low Dose Ionizing radiation in Humans: A Review. *Experimental Biology and Medicine* 229:378-382.
- Ribas, G., E. Carbonell, N. Xamena, A. Creus, and R. Marcos. 1994. Genotoxicity of tritiated water in human lymphocytes. *Toxicology Letters* 70:63–69.
- Salbu, B. and L. Skipperud. 2007. Challenges in radioecotoxicology. In: Mothersill, C., I. Mosse, and C. Seymour, eds. *Multiple stressors: a challenge for the future*. Book Series: NATO Science for Peace and Security Series C – Environmental Security. Pp. 3-12.
- Shleien, B., L.A. Slaback, Jr., and B.K. Birky. 1998. *Handbook of Health Physics and Radiological Health, Third Edition*. Williams & Williams, A Waverly Company, 1998.
- Turner, A., G.E. Millward, and M. Stemp. 2009. Distribution of tritium in estuarine waters: the role of organic matter. *Journal of Environmental Radioactivity* 100: 890-895.
- United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2000. *Sources and Effects of Ionizing Radiation*.
- Vichot, L., C. Boyer, T. Boissieux, Y. Losset, and D. Pierrat. 2008. Organically bound tritium (OBT) for various plants in the vicinity of a continuous atmospheric tritium release. *Journal of Environmental Radioactivity* 99:1636-1643.
- Williams, J.L., R.M. Russ, D. McCubbin, and J.F. Knowles. 2001. An overview of tritium behaviour in the Severn Estuary(UK). *Journal of Radiological Protection* 21:337–44
- Yankovich, T.L., J. Vives I Batlle, S. Vives-Lynch, N.A. Beresford, C.L. Barnett, K. Beaugelin-Seiller, J.E. Brown, J.-J. Cheng, D. Coplestone, R. Heling, A. Hosseini, B.J. Howard, S. Kamboj, A.I. Kryshev, T. Nedveckaite, J.T. Smith, and M.D. Wood. 2009. An international model validation exercise on radionuclide transfer and doses to freshwater biota. *Journal of Radiological Protection* 30: 299-340.

URANIUM (U) ECOTOXICOLOGICAL PROFILE

I. INTRODUCTION Uranium (U) is one of the hazardous substances (as defined by Sections 101(14) and 101(33) of CERCLA and listed in 40 CFR §302.4) to which natural resources have been exposed as a result of operations and cleanup efforts over the past 60 years at the U.S. Department of Energy (DOE) Hanford Site in the State of Washington. Uranium is a naturally occurring element in rocks and minerals in the earth's crust and has the highest atomic weight of any naturally occurring element. Uranium is unusual in that it is both radiologically and chemically toxic.

Natural uranium is made up of three isotopes¹²⁸: U-238, U-234 and U-235. U-238 is the most abundant of the three isotopes, making up 99.27% of the total mass of natural uranium. U-234 and U-235 make up only a fraction of a percent of the total mass: 0.0005% and 0.72%, respectively. Although the U-238 isotope makes up the majority by mass of natural uranium, it is not particularly radioactive, with a very long half-life of 4.5×10^9 years (ATSDR 1999). The radioactive properties of natural uranium are due partially to the presence of the other two less stable isotopes. U-234 has a half life of 2.4×10^5 years, which is several orders of magnitude lower than that of U-238, and U-235 has a half-life of 7.0×10^8 years (ATSDR 1999). Since U-235 is the only naturally occurring fissile isotope, the concentration of U-235 is often increased through the enrichment process in order for uranium to be used as nuclear fuel, as was the case at the Hanford Site. This is typically accomplished by chemically separating the uranium from the ore matrix, converting the uranium to uranium oxide (U_3O_8), and then processing the oxide in various manners, including fluorination to uranium gas (UF_6), which is passed through a diffusion process in order to produce the enriched uranium.

II. SOURCES **NATURAL SOURCES**

Uranium is found in rocks and soil and is released into the environment by natural weathering of the rocks, erosion by wind, and by volcanic activity (ATSDR 1999). Typical concentrations of uranium in common rock types are 0.5 to 4.7 ppm (Eisenbud and Gesell 1997). Exhibit 1 summarizes typical concentrations of natural uranium in air, drinking water, and a variety of food products.

¹²⁸ A nuclide of an element having the same number of protons but a different number of neutrons. Nuclide is a general term applicable to all atomic forms of an element. Nuclides are characterized by the number of protons and neutrons in the nucleus, as well as by the amount of energy contained within the atom (<http://www.epa.gov/radiation/glossary/index.html>).

EXHIBIT 1 URANIUM CONCENTRATIONS IN AIR AND FOOD PRODUCTS (UNSCEAR 2000)

| | URANIUM CONCENTRATION (PCI/M3) |
|----------------------------|-----------------------------------|
| Air | 2.43e-5 - 1.35e-4 |
| | URANIUM CONCENTRATION (PCI/KG) |
| Milk | 0.019 |
| Meat | 0.02 - 0.06 |
| Grain | 0.08 - 0.6 |
| Leafy vegetables | 0.65 |
| Root vegetables and Fruits | 0.024 - 0.21 |
| Fish products | 0.35 - 51.3 |
| Drinking water | 0.008 - 2.1 |

EXHIBIT 2 RADIOACTIVE DECAY SERIES FOR U-238

| NUCLIDE AND RADIATION TYPE | APPROXIMATE HALF-LIFE |
|---|--------------------------------|
| Uranium-238 $\alpha\downarrow$ | 4.5 x 10 ⁹ years |
| Thorium-234 $\beta\downarrow$ | 24 days |
| Protactinium-234 $\beta\downarrow$ | 1 minute |
| Uranium-234 $\alpha\downarrow$ | 2.4 x 10 ⁵ years |
| Thorium-230 $\alpha\downarrow$ | 7.7 x 10 ⁴ years |
| Radium-226 $\alpha\downarrow$ | 1600 years |
| Radon-222 $\alpha\downarrow$ | 3.8 days |
| Polonium-218 $\alpha\downarrow$ | 3 minutes |
| Lead-214 $\beta\downarrow$ | 27 minutes |
| Bismuth-214 $\beta\downarrow$ | 20 minutes |
| Polonium-214 $\alpha\downarrow$ | 1.6 x 10 ⁻⁴ seconds |
| Lead-210 $\beta\downarrow$ | 22 years |
| Bismuth-210 $\beta\downarrow$ | 5 days |
| Polonium-210 $\alpha\downarrow$ | 140 days |
| Lead-206 | Stable |
| Source: http://pubs.usgs.gov/of/2004/1050/uranium.htm | |

When present in the environment, uranium is often accompanied by the other members of its naturally occurring decay series¹²⁹. Exhibit 2 presents the U-238 decay series. When present naturally in the environment (such as uranium ore), all these radionuclides are also often present in radioactive equilibrium. This means that each radionuclide in the series is present in the same concentration (e.g., if 1 pCi of U-238 is present in a gram of soil, it is likely that all the other radionuclides in the decay chain are also present at comparable concentrations). When uranium is chemically separated from its ore, the short-lived progeny of U-238 (Th-234 and Pa-234) grow in quickly and will also be present.

ANTHROPOGENIC SOURCES

The anthropogenic sources of uranium in the environment surrounding the Hanford Site originated from releases from the nuclear fuel cycle processes at that site. The Hanford operations included plutonium (Pu) production and research reactors, chemical separation facilities, and fuel fabrication facilities, all of which involved processing and storing of various uranium compounds.

The 100 Area at Hanford contained the nine reactors where the uranium targets were bombarded with neutrons produced from uranium fuel to produce plutonium. “Because only a fraction of the uranium in fuel and targets was converted to plutonium during each cycle through a reactor, workers at Hanford and Savannah River Site processed hundreds of thousands of tons of uranium” (DOE 1996).

Plutonium was subsequently separated from the uranium at the Hanford 200W and 200E Areas. The separation facilities consisted of the B Plant, T Plant, U Plant, Reduction-Oxidation (REDOX), the Plutonium-Uranium Extraction (PUREX), UO₃, and C Plant (ORAU 2010).

Uranium was also processed and fabricated in the 300 Area. The facilities in that area included the Uranium Metal Fuels Fabrication Facility, which processed and encased rods in aluminum, the Uranium Metal Extrusion Facility, which processed raw uranium billets into rods, and the 303 Facilities Fresh Metal Storage Facility, which consisted of 10 buildings reserved for the storage of fresh uranium, chemicals, uranium scrap and plutonium. Building 303-L was an additional building built in 1961 for the purpose of burning uranium metal scrap to uranium oxide so the leftover usable uranium could be recovered. The burning resulted in elevated airborne uranium concentration levels that exceeded the maximum allowable levels at that time. The 303-L building was subsequently shut down in 1971 (ORAU 2010). To varying degrees, these processes resulted in the release of uranium into the atmosphere and later deposition on the soil and surface water. Some of the wastes from these processes leached into the groundwater and entered into the Columbia River (Delistraty and Yokel 1998).

¹²⁹ Most naturally occurring radioactive materials and many fission products; undergo radioactive decay through a series of transformations rather than in a single step. Until the last step, these radionuclides emit energy or particle with each transformation and become another radionuclide. This decay chain, or decay series, ends in a stable nuclide (<http://www.epa.gov/radiation/understand/chain.html>).

III. ENVIRONMENTAL CHEMISTRY

As a preface to this section, it is appropriate to point out that the subject of the environmental chemistry of uranium is vast, and can include both the physical and chemical behavior of uranium in the environment as well as its transport through aquatic and terrestrial ecosystems. Many compendia have been published that address this broad subject. Till and Meyer (1983) is one of the earliest compilations and describes the environmental behavior of radionuclides, including uranium. A recent addition to the literature on this subject is the International Atomic Energy Agency's (IAEA) Handbook of Parameter Values for the Prediction of Radionuclide transfer in Terrestrial and freshwater Environments (2010). In addition, the U.S. Department of Energy Richland Operations Office (DOE-RL) periodically updates the System Assessment Capability (SAC), which is an environmental assessment tool that can be used to assist in the analysis of the movement of contaminants from waste sites at the Hanford Site into and through the vadose zone, groundwater, atmosphere, and Columbia River. These reports can serve as a convenient starting point for compiling generic and site-specific models and modeling parameters for assessing the behavior of uranium in the environment at Hanford.

The ATSDR report on uranium describes, in general, the interaction of uranium compounds with the environment:

“Uranium deposited by wet or dry precipitation will be deposited on land or in surface waters. If land deposition occurs, the uranium can be reincorporated into the soil, resuspended in the atmosphere (typically factors are around $10^{-6}/m$), washed from the land into surface water, incorporated into groundwater, or deposited on or adsorbed onto plant roots (little or none enters the plant through leaves or roots). Conditions that increase the rate of formation of soluble complexes and decrease the rate of sorption of labile uranium in soil and sediment enhance the mobility of uranium. Significant reactions of uranium in soil are formation of complexes with anions and ligands (e.g. CO_3^{-2} , OH^{-1}) or humic acid, and reduction of U^{+6} to U^{+4} . Other factors that control the mobility of uranium in soil are the oxidation-reduction potential, the pH, and the sorbing characteristics of the sediments and soils” (p. 259 of ATSDR 1999).”

The uranyl ion (UO_2^{2+}) is the most stable form of uranium and therefore the most common form found in the environment (Sheppard 2005). Sheppard (2005) summarizes the oxidation states of uranium and their interaction with the environment:

“The oxidized U (+VI) (uranyl) ion complexes readily with carbonate, phosphate or sulfate ions. In these forms, U is soluble and readily transported. In contrast, under reducing conditions, such as those found in anoxic waters and sediment, U occurs in the tetravalent (U(+IV)) state which has a strong tendency to bind to organic material and to precipitate, and is therefore immobile. Metallic U and particles of insoluble U compounds are not very bioavailable.”

FATE AND TRANSPORT

A comprehensive review of binding coefficients for a broad range of elements, including uranium, is provided by Sheppard and Thibault (1990), as shown in Exhibit 3.

EXHIBIT 3 URANIUM K_d VALUES (SHEPPARD AND THIBAUT 1990)

| SOIL TYPE | GEOMETRIC MEAN OR BEST ESTIMATE OF K_d VALUE | RANGE |
|---|--|--------------|
| Sand | 35 | 0.03 - 2,200 |
| Loam | 15 | 0.2 - 4,500 |
| Clay | 1600 | 46 - 395,100 |
| Organic | 410 | 35 - 7,350 |
| Agricultural Soils & Clays, pH of 4.5 - 9 (Baes & Sharp 1983) | 45 | 10.5 - 4,400 |

Given the wide range in values, it is clear that K_d values are of limited use unless a great deal of information is available regarding the chemical properties and types of soil at the site.

Serne *et al.* (2002) performed experiments to assess the leaching and sediment sorption of uranium in soil samples taken from the Hanford 300 Area. Their laboratory results show that “uranium sorption onto the uncontaminated sediment was highly variable and the sorption was dependent on the solution concentrations of inorganic carbon, pH, and to a lesser extent total dissolved solids.” They also found that the “uranium adsorption K_d values ranged from 0 ml/g to >100 ml/g depending on which solution parameter was being adjusted.”

The uranium K_d values may also range widely in the actual environment surrounding Hanford since Serne *et al.* (2002) describe that the natural vadose porewater and the groundwater composition vary according to rain and snowfall and the “fluctuations of the [Columbia] River which cause groundwater and river water to mix at different proportions at different times of the year and different times of the day.”

BIOACCUMULATION POTENTIAL

Bioconcentration factors for uranium vary widely among different types of organisms, with the highest bioaccumulation observed in low trophic organisms, such as bacteria and algae. Bioconcentration factors for water bacteria range from 2,794 to 354,000 (Driver 1994). A bioconcentration factor of 1,576 has been measured in fresh water algae, and a factor of 439 has been measured in plankton (Driver 1994; ATSDR 1999). Uranium bioaccumulation studies involving a variety of fish species showed that the bioconcentration factors for fish are no greater than 38. It appears that, in the case of bacteria, the observed bioconcentration is a result of the adsorption of uranium on the cell wall and not the absorption of uranium within the cell. A similar observation regarding the adsorption of uranium has been made for plants. Studies show that uranium adheres to the outer layer of the roots but does not penetrate the inner tissue of the plant. Therefore, root vegetables grown in soil containing elevated

levels of uranium are potential sources of uranium exposure to humans and animals (ATSDR 1999).

A recent IAEA (2010) publication reports uranium soil-to-plant transfer factors. Exhibit 4 is excerpted and summarized directly from Table 17 of IAEA 2010.

EXHIBIT 4 URANIUM SOIL-TO-PLANT TRANSFER FACTORS (DRY WEIGHT) FOR THE TEMPERATE ZONE (ALL SOIL GROUPS) (IAEA 2010)

| PLANT GROUP | COMPARTMENT | N | MEAN | MIN | MAX |
|-------------------|------------------------------|-----|--------|--------|--------|
| Root crops | Roots | 48 | 8.4e-3 | 4.9e-4 | 2.6e-1 |
| | Stems & Shoots | 37 | 2.8e-2 | 2.0e-3 | 7.0e-1 |
| Tubers | Tubers | 28 | 5.0e-3 | 1.8e-4 | 8.0e-2 |
| | Stems & shoots | 1 | 1.9e-1 | - | - |
| Herbs | | 9 | 3.6e-2 | 8.6e-1 | 4.1e-1 |
| Other crops | Sunflower leaves | 39 | 7.1e-2 | 8.9e-3 | 7.8 |
| | Sunflower grain | | 1.5e-2 | 8.2e-3 | 2.9e-2 |
| Grasses | Stems & shoots | 2 | 1.7e-2 | 2.0e-4 | 5.5 |
| Pasture | Stems & shoots | 147 | 4.6e-2 | 1.3e-3 | 14 |
| Leguminous fodder | Stems & shoots | 53 | 1.5e-2 | 2.0e-3 | 1.6 |
| Unspecified | | 1 | 0.1 | - | - |
| Cereals | Grain | 5 | 5.0e-4 | - | - |
| Leafy veg | Leaves | 1 | 2.0e-3 | - | - |
| Non-leafy vegs | Fruits, beads, berries, buds | 1 | 2.0e-3 | - | - |
| Root crops | Roots | 1 | 2.0e-3 | - | - |
| Tubers | Tubers | 1 | 1.0e-3 | - | - |
| Pasture | Stems & shoots | 1 | 5.0e-3 | - | - |

The unabbreviated version of IAEA (2010)'s Table 17 (2010) provides more detailed information, where the soil-to-plant uptake factors for different types and parts of plants are further divided into different types of soil. Other compendia of this type are provided in earlier publications, such as Table 5.17 of Till and Meyer (1983). These values may be useful in predicting the concentration of uranium in plants given the concentration of uranium in soil. However, it is not clear from a review of IAEA (2010) whether a distinction has been made between uranium adsorbed to plant surfaces as opposed to uranium that has been absorbed within the cells. In addition, given the variability of soil-to-plant uptake factors, it is important to use site-specific values if at all possible.

Regarding the trophic transfer of uranium, Driver (1994) states "Uranium enters the food chain via adsorption on surfaces of plants and small animals. Because of membrane discrimination against uranium, little uranium is accumulated internally in biota. Consequently, concentration factors for uranium decline substantially with trophic level."

IAEA (2010) also presents information on the gastrointestinal (GI) absorption of uranium in cows, uranium transfer factors for beef, expressed in units of pCi/kg beef per pCi/day ingested by cows. These values could be useful in predicting the uptake of uranium by some animals following ingestion of uranium. However, as previously stated, the use of site-specific values is preferable.

ACCUMULATION WITHIN TISSUES

Barillet *et al.* (2007) observed the accumulation of uranium in the gills and liver of the zebrafish *Danio rerio*. This study also observed that since the gills serve as the uptake route for pollutants, “they could also serve as an effective barrier to uranium uptake, particularly if uranium is trapped within the protective mucus layer, as previously shown for other divalent metals in fish.”

IAEA (2010) presents fresh weight concentration ratios for freshwater invertebrates (a minimum of 360 and a maximum of 400) and for freshwater fish (1.5 and 3.3). IAEA (2010) provides a number of transfer factors (for a variety of aquatic and terrestrial organisms) that might be useful in predicting the uptake of uranium by organisms other than man, given information on the concentration of uranium in soil, sediment, water and food items at Hanford. However, though useful, site-specific values for these parameters are always preferred.

Driver (1994) summarizes the accumulation of uranium in mammalian tissue following acute and chronic exposure. Uranium has been found to accumulate in mammalian kidneys, liver, bone, cardiovascular and central nervous system. In particular: “Kidney and bone tissues are the main targets of both the radiation and chemical toxicity of uranium in vertebrate organisms. Of these two tissues, kidney tissue is the most sensitive and is considered to be the key target organ for hazard assessment.” The lung and the skeletal systems are critical target organs for radiological damage from uranium carcinogenesis.

Uranium contamination of both surface and groundwater has occurred in the vicinity of the Hanford Site due to discharges of wastes into the Columbia River and into the ground, which leached down into the groundwater (Delistraty and Yokel 1998). Sediment samples taken near the 300 Area were found to have elevated levels of uranium (Serne *et al.* 2002), and sediment samples taken from the river bank and river bank areas in the vicinity of the 100 B, N, D, and F areas, as well as the 300 Area, were found to have contaminants (Delistraty and Yokel 1998). Schnug *et al.* (2006) states that uranium that has entered the environment through anthropogenic activities “is easily mobilized and transported into the food chain.”

IV. TYPICAL MAJOR EXPOSURE ROUTES Aquatic invertebrates and fish could ingest uranium that was adsorbed onto sediment particles in the water. Plants adsorb uranium onto their roots from contaminated soil (ATSDR 1999). Terrestrial animals, including birds and mammals, can be exposed to uranium in the environment by inhalation, skin contact, and most commonly, through ingestion of contaminated water, plants, and animals (Schnug *et al.* 2006). In fact,

Schnug *et al.* (2006) states that ingestion of contaminated water “accounts for more than 80% of the total uranium ingestion” by animals.

V. CHEMICAL ECOTOXICITY

Exposure to uranium can cause lethal and sub-lethal effects on both aquatic and terrestrial organisms, although most research has focused on lethality. Driver (1994) summarizes these toxic effects and LC50 values for various aquatic and terrestrial biota, and notes that the toxicity of uranium is due primarily to the chemical properties of the uranyl ion rather than its radiological properties (Driver 1994). In all cases, the discussion of toxicity in this section refers to chemical and not radiological toxicity. For a discussion of radiological toxicity, see Section VI.

KNOWN BENEFICIAL OR PROTECTIVE PROPERTIES

There are no known beneficial or protective properties of uranium.

MECHANISM(S) AND LOCI OF TOXICITY

Labrot *et al.* (1999) describes the two types of effects that exposure to heavy metals can have on biota: (1) an indirect effect, which is “due to the additional metabolic cost of accumulating, transporting, storing, and excreting the contaminant, and (2) a direct [effect], on cellular membranes and/or specific biochemical pathways.” As was previously mentioned, uranium is not usually found in the inner tissues of plants and does not usually cross the cell membrane of most organisms. The soluble uranyl ion can form complexes with proteins and anions (Schnug *et al.* 2006). The detrimental effects of exposure to uranium appear to be the result of the adsorption of uranium on the cell membranes, which results in a disruption of normal cellular processes.

Monleau *et al.* (2005), Darolles *et al.* (2009), and Lerebours *et al.* (2009) explore the genotoxic effects of depleted and enriched uranium. These investigators provide insight into the interaction of uranium as both a radionuclide and a heavy metal at the molecular level. These studies are concerned with the effects (both direct and indirect) of uranium on gene expression, a relatively more recent area of inquiry.

FACTORS AFFECTING TOXICITY

As discussed in more detail in the following sections, a variety of factors can affect the ecotoxicological properties of uranium. In aquatic systems, harder water reduces the toxicity of uranium to aquatic organisms (Driver 1994). Dissolved organic matter may also reduce uranium’s toxicity. The toxic effects of uranium on mammals depend on its chemical and physical form, route of intake, and level of enrichment (ATSDR 1999).

PLANTS

Sheppard *et al.* (1992) comments on the conflicting information in the literature on the toxicity of uranium to plants. Toxicity to plants has been reported beginning at background levels of uranium, while others report no toxicity at 100 to 1000 times higher than background. Sheppard *et al.* (1992) attempted to resolve some of these discrepancies by conducting a series of experiments. Plant seedlings, including lettuce, tomato, corn, pine, and *Brassica rapa* (commonly known as field mustard or turnip mustard), were grown in soil contaminated with varying levels of uranium. Their results show that all plants species observed showed reduced rate of germination

when grown in uranium contaminated soil. Notably, toxicological studies of terrestrial plants have been largely limited to human food crops (Driver 1994).

AQUATIC INVERTEBRATES AND FISH

There are numerous publications addressing the toxicity of uranium to algae, daphnia, and other aquatic organisms (Poston *et al.* (1978); Parkhurst, *et al.* 1983; Hogan *et al.* 2005). These studies were initiated due to concern over uranium mine effluents entering the freshwater environment, and measuring the growth rate of a variety of aquatic organisms as indicators of potential ecological damage.

In aquatic microorganisms, uranium exposure has been found to inhibit growth, cell division, and food intake. For the algae *Scenedesmus*, cell division was inhibited by uranium exposure. For the protozoan *Microregma*, food intake was inhibited following exposure to uranium. The freshwater hydrae (*Hydra viridissima*) has been found to be particularly sensitive to uranium exposure. Elevated uranium concentrations were found to be lethal to hydrae after 48 hours of exposure, and growth was inhibited at relatively low concentrations. Further analysis demonstrated that the observed growth inhibition was most likely due to the accumulation of uranium on the hydra nematocysts, which was disrupting the organism's ability to capture prey (Driver 1994). For various species of freshwater water fleas, LC50 toxicity values were reported over a range of values, depending on the species and the water hardness level, which is indicated by the amount of CaCO₃/L.

In aquatic systems, exposure to uranium is more toxic in soft water than in hard water. In fact, the hardness of the water is a key factor in the toxicity of uranium, where the tolerance of uranium by aquatic organisms increases dramatically with the increased level of CaCO₃ and the increased hardness of the water (Driver 1994). Studies performed by Charles *et al.* (2002) involving the tropical freshwater alga *Chlorella* sp. showed that a "50 fold increase in water hardness resulted in a 5 fold decrease in the toxicity of uranium to *Chlorella* sp." The authors determined that this observation was mostly likely due to competition between uranium and calcium and/or magnesium for the binding sites on the cell surface.

As with aquatic invertebrates, the uranium LC50 values for freshwater fish vary with the hardness of the water. Driver (1994) presents an extensive list of LC50 values for freshwater fish found in the northern hemisphere.

The toxicity of uranium to aquatic organisms was also found to be dependent on the presence of dissolved organic matter. Hogan *et al.* (2005) grew the alga *Chlorella* sp. in both natural water from the Magela Creek in northern Australia and synthetic water, which contained no organic matter. "The toxicity of uranium to *Chlorella* sp. in NMCW [natural creek water] was approximately two to four times lower than in SMCW [synthetic creek water]. Based on geochemical speciation modeling, this difference corresponded to a four-fold decrease in the proportion of free uranyl ion in NMCW [natural water] compared to SMCW [synthetic water], most likely due to the presence of dissolved organic carbon."

A variety of lethal and sublethal effects have been observed in fish exposed to varying levels of uranium, including damage to the liver, kidneys, brain, and olfactory centers.

In studies performed by Cooley *et al.* (2000), lake whitefish (*Coregonus clupeaformis*) were fed a uranium contaminated diet with three concentrations of uranium, 100, 1000, and 10,000 µg U/g. Results showed elevated levels of serum lipid peroxides indicating damage to cellular membranes. Following prolonged exposure to uranium in their diet, the lake whitefish exhibited numerous types of renal and liver lesions and pathologies. Lerebours *et al.* (2009) observed genotoxic effects, with a change of gene expression in brain, liver and muscle tissue of zebrafish following waterborne exposure to uranium. In the liver they observed the induction of genes involved in detoxification, apoptotic mechanism and immune response. In the skeletal muscles, genes involved in mitochondrial metabolism and production of reactive oxygen species were induced. Finally, the results of this study showed an increase of the expression of genes involved in neural transmission. Barillet *et al.* (2007) observed similar effects on brain activity with an increase in acetylcholinesterase activity following uranium exposure in zebrafish. In their 2010 study, Lerebours *et al.* also observed damage to the olfactory bulb of the zebrafish following exposure to uranium and mentions previous studies suggesting that uranium may cross the blood-brain barrier. The sensory nerves of the olfactory bulb are nearly in direct contact with the surrounding water, and damage to the olfactory center in fish has the potential to disrupt various life functions and behavior. For fish, olfaction plays a key role in detection of mates and relatives through pheromones, detection of prey or predators, homing, and detection of changes in the surrounding environment (Tierney *et al.* 2009).

TERRESTRIAL INVERTEBRATES

Sheppard *et al.* (1992) found that earthworm growth and survival was compromised following exposure to uranium at concentrations of 1000 mg U/kg. There appears to be limited investigation of the toxicity of uranium to terrestrial invertebrates.

MAMMALS

The toxic effects of uranium on mammals vary with the solubility of the uranium compound and the route of the exposure. Effects include teratogenic effects, reproductive effects, genotoxic effects, and damage to various organs and tissues, particularly to the kidneys, liver, bone and blood. Several laboratory studies have been performed that examine the effects of uranium exposure on reproduction and fetal development in mice and rats. Domingo *et al.* (2001) and Driver (1994) summarize these results, which include skeletal malformations and other developmental anomalies, decreased birth weight, and body length and smaller litter size. Reproductive effects of uranium exposure include damage to testes. Damage to other various organs and tissues, including kidneys, liver, bone and blood, was observed. Acute uranium exposures can result in damage to the nephron, which is the functioning unit of the kidney. Specifically, Driver (1994) describes the renal damage in mammals as “injury and necrosis of the terminal segments of the renal proximal tubule [of the nephron].” Driver also reports injury of the glomerulus, which is the group of filtering capillaries associated with the nephron.

The extent of toxicity to mammals depends on a variety of factors, including:

“[its] chemical and physical form, route of intake, and level of enrichment. The chemical form of uranium determines its solubility and, thus, transportability in body fluids as well as retention in the body and various organs.” (ATSDR 1999).

Driver (1994) provides LC50 values for some small mammals exposed acutely to airborne uranium for 10 minute periods and chronic airborne exposures over the course of a few months to one year. The exposures resulted in kidney damage and death to guinea pigs, rabbits, and dogs.

Monleau *et al.* (2005) examine the genotoxic effects of depleted uranium on rat lung tissue and found that rats exposed to depleted uranium by inhalation experienced DNA damage in the lung cells, which may be a result of inflammation and oxidative stress.

Little information is available about the toxicity of uranium to wild mammalian species.

BIRDS

Little is known about the ecotoxicity of uranium to birds. However, a 2010 study of marine birds in the Baltic Sea (Borylo *et al.* 2010) found that uranium accumulates in the liver, rest of viscera, and on the feathers of these animals. The highest amount of uranium was found in plant-eating birds, while carnivorous birds accumulated a lower amount. A uranium toxicity study involving chicks provides LC50 values and also the lowest lethal dose expressed in units of mg U/kg body weight (Sheppard *et al.* 2005).

VI. RADIOLOGICAL ECOTOXICITY Uranium is somewhat unique among the radionuclides with respect to its ecotoxicity in that it is generally accepted that its ecotoxicity is limited by its chemical toxicity, as opposed to its radiotoxicity. This occurs because uranium has a very low specific activity (i.e., relatively large quantities of uranium, in terms of mass, have a relatively low decay rate). Nevertheless, uranium can represent a radioecological stressor, contributing to the overall ecotoxicity of uranium due to its ability to cause damage to DNA, primarily from alpha radiation that is deposited within living tissue (discussed below).

MECHANISM OF ACTION

Radioecological damages to aquatic and terrestrial organisms due to uranium in the environment are due to ionization caused to the interaction of highly energetic alpha particles with living tissue. In particular, typical alpha particles, which have an energy of about 5 MeV, have a range in air of only about 4 cm (see Figure 6.7 of Shleien *et al.* 1998). In addition, a layer of tissue of 0.07 mm will stop a 7.5 MeV alpha particle (see Table 3-1 of Shleien *et al.* 1999). Hence, unless the uranium alpha particle is in intimate contact with living tissue or is intracellular, it has a limited potential for biological damage. However, if it is in close proximity to living tissue and/or inside the cytoplasm or the nucleus of a cell, it deposits its 5 MeV alpha energy over a relatively short distance. For example, the typical binding energy of a hydrogen electron is about 13.6 eV and the typical energy required to ionize a molecule (i.e.,

eject an electron from its orbit) is about 34 to 35 eV (see Casarett 1968). Hence, in a relatively short distance, a large number of ion pairs are produced by the passage of a 5 MeV alpha particle through tissue. For example, the total number of ion pairs produced by the energy deposited in tissue from a 5 MeV alpha particle is about 140,000 ion pairs (i.e., 5 MeV/35 eV). The pattern of energy deposition for a 5 MeV alpha particle is about 110 keV/micron, also referred to as the linear energy transfer (Casarett 1968). Hence, about 3000 ion pairs are produced per micron. Given that a typical cell is on the order of tens of microns (Curtis and Barnes 1989), a single cell might experience 30,000 ion pairs produced by the passage of a single 5 MeV alpha particle. It is this deposited energy in living tissue that results in biological damage.

More specifically, radiation causes damage through the production of free radicals:

“Furthermore, uranium can enhance the production of free radicals via the ionization phenomenon induced by alpha particle emissions. The damage, in this case, would not be direct result of radiation, but rather an indirect consequence as a result of reactive oxygen species stemming from radiation. When quantity of free oxygen species generated exceeds the level that the cell’s protective system can control, cell proteins, nucleic acids, and lipids can be damaged” (Jones *et al.* 2003 and Jelka *et al.* 2005, as cited in Barillet *et al.* 2007).

RADIOECOTOXICOLOGICAL EFFECTS

The literature on the effects of radiation on organisms other than man is extensive (see NCRP 1991, IAEA1992, ICRP 2008, Driver 1994), but the literature explicitly addressing the radiotoxicity of uranium on organisms other than man is limited. However, a number of review documents, primarily Till *et al.* (1976), Driver (1994), and Sheppard *et al.* (2005) summarize the literature. In particular, Sheppard *et al.* (2005) summarizes the literature in order to set “PNECs (predicted no-effect concentrations) for chemical toxicity to uranium for non-human biota.”

Aquatic Biota

Considerable research has been dedicated to studying the effect of radiation on fish eggs and developing fish embryos (e.g., see the literature reviewed in Till *et al.* 1976); however, data on uranium in particular are few. Till *et al.* (1976) cite studies that found no adverse effects on egg hatching of carp at uranium concentrations of 60 ppm. However, other investigations involving plutonium reported by Till *et al.* (1976) did observe adverse effects. This is important to note because, like plutonium, uranium is an alpha emitter, and in principle could have similar radioecological effects. There are, of course, differences in the environmental chemistry and behavior of uranium and plutonium that make it difficult to draw firm conclusions regarding the radiotoxicity of uranium based on the results of plutonium investigations.

Because uranium is an alpha emitter, and therefore has a potential to cause radioactive damage primarily when it absorbed within cells, Till *et al.* (1976) provide a qualitative discussion on the ability of uranium and plutonium to penetrate the chorion of fish eggs. However, quantitative information was not presented, and the question remains

as to whether the approximate 5 MeV alpha particles emitted by uranium and some of its progeny could penetrate the chorion of fish eggs. The range of a 7.5 MeV alpha particle in tissue is about 70 microns. Eggs of some fish species appear to have chorions (or zona radiata, ZR) that are less than 70 microns thick. Even within salmonids, there seems to be substantial variation in the structure of the surface of the eggs (see Schemehl and Graham 1987). Some species seem to have very variable ZR structures, with protrusions and pores, making it difficult to judge chorion thickness. Baldacci *et al.* (2001), which addresses ice fish, states that the unfertilized egg has a chorion that is 50 microns thick.

As noted previously, Driver (1994) reviews the levels of uranium in aquatic ecosystems observed to adversely affect aquatic plants, invertebrates, and fish. In general, the observed effects have been attributed to the element's chemical toxicity. However, it is difficult to judge the degree to which the radiation emitted by the uranium and its short-lived progeny may have contributed to the observed effects.

Terrestrial Biota

Little information is available that explicitly addresses the radioecotoxicological effects of uranium on terrestrial biota. As is the case for aquatic biota, it is difficult to separate the chemical from the radiological effects of exposure. Hence, the literature described above addressing the chemical effects of uranium in the terrestrial environment has a degree of applicability to the radiotoxic effects of uranium in the terrestrial environment. However, it appears that, at least for mammals, the toxicity of uranium is primarily related to kidney damage caused by uranium as a heavy metal and not the radiological exposures (HPS 2002).

VII. EFFECTS IN THE PRESENCE OF OTHER CONTAMINANTS

In theory, one would expect additive and synergistic effects of exposure to uranium with other environmental toxicants, similar to those observed between other heavy metals. For example, Schubert *et al.* (1978); Tabata *et al.* (2003); Traore *et al.* (1999); and Sanchez *et al.* (2001) address the synergistic cytotoxic and nephrotoxic effects of a number of heavy metals. In addition, there may also be synergistic effects associated with the combined action of exposure to uranium as a chemical and radiological toxin. Concerns over the possible synergistic effects of exposure to radiation and chemical toxins have been extensively raised and reported in the scientific literature (Prasad *et al.* 2004; Burkart *et al.* 1997) but little consensus has been achieved in quantifying these effects in humans, except possibly for radon and smoking (BEIR IV, 1988) and certainly in the enhancement of the therapeutic effects of radiotherapy used to treat cancer (e.g., Lew *et al.* 2002). There is considerable literature on observed synergistic adverse effects of radiation and toxic chemicals on organisms other than man (e.g., Mothersill *et al.* 2007).

There is some literature on observed synergistic adverse effects of radiation and toxic chemicals on organisms other than humans (e.g., salmon, Mothersill *et al.* 2007). Examples of ionizing radiation and metals producing combined effects in other biological systems include synergistic effects on soil microbial activity from cadmium and zinc in combination with gamma radiation (summarized in UNSCEAR 2010). Also, combined effects of cesium-134/137 and lead found in highly contaminated

habitats in the Russian Federation increased the mutation rate in the plant *Arabidopsis thaliana* (summarized in UNSCEAR 2010). However, the authors clearly indicate that the relative importance of different damage-inducing mechanisms of metals for combined exposures in human and non-human populations remains to be elucidated.

Overall, there is a clear need for additional research on synergistic effects of multiple stressors in radioecotoxicology (e.g., Salbu and Skipperud 2007, Mothersill and Seymour 2005). In particular, these authors raise the issue of pesticides, organics, and endocrine disruptors and synergistic effects with radioactive materials, particularly with long-term exposure to various biological systems. Manti and D'Arco (2010) summarize the *in vitro* and animal-model studies and epidemiological surveys with two or more stressors, including radionuclides (DNA-damaging agents). They also emphasize that most research focuses only on the short-term effects of combined single exposures to animal models, and more work is needed to understand chronic exposure to trace contaminants and radioactive elements in the environment, including impacts to long-term genome stability. Specific research is lacking on uranium effects with multiple stressors on biological systems, particularly non-human systems.

VIII. DATA GAPS In general, a review of the literature on uranium ecotoxicity reveals that there are significant data gaps including the following: absence of data on wild species and absence of field-based studies of effects. In addition it appears that the research focus in many cases (e.g., aquatic organisms) has been on acutely lethal endpoints rather than sublethal endpoints.

One of the largest data gaps in understanding the chemical and radiological toxicity of uranium is its synergistic effects with other toxins, as briefly described above and in ATSDR (1999), which states “No information was located regarding the modulation of the toxicity of uranium by other chemicals or vice versa. It is possible that co-exposure to other heavy metal nephrotoxicants (e.g., lead, cadmium) could have an additive effect on uranium toxicity.” ATSDR (1999) indicates that animal studies designed to examine the combined effects on the kidney of uranium and other heavy metal nephrotoxicants (lead, cadmium) would be useful to determine whether effects are less than expected on the basis of individual toxicity, additive, or synergistic.

Durakoviae (1999) explores the complex intracellular chemical and radiological toxicity of uranium when it interacts with complexing agents in body fluids, and refers to the need for a better understanding of the radiological and chemical toxicity of depleted uranium (primarily U-238).

Driver (1994) indicates that toxicological studies of terrestrial plants were limited to human food crops (i.e., soybeans and Swiss chard). Driver (1994) also indicates that no studies were found on the chemical or radiation toxicity of uranium in amphibians or reptiles, which are ecologically important species in the Columbia River watershed.

There appears to be limited information on the toxic effects of uranium on birds, but Sheppard *et al.* (2005) indicates that birds may not be “critical vertebrates for effects of uranium.”

No animal tests have been conducted to study cancer incidence following oral exposure to uranium, which is the most likely route of exposure to animals from contamination originating on the Hanford site. Although non-neoplastic kidney damage has been observed in numerous feeding studies, no tumors in any organs have been observed during these tests. It is noteworthy that, based on the linear no-threshold model that forms the basis of current guidelines and regulations for protection from radiation induced cancer, any exposure to a radioactive substance involves some increase in the risk of cancer. Thus, enriched uranium would be expected to present a higher risk for cancer than natural uranium (Driver 1994). ATSDR (1999) suggests that research investigating the radiotoxicity of uranium would be more beneficial for the less available, high specific activity isotopes such as U-233 and U-234 that are formed during energy production or associated with weapons-grade uranium.

IX. REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for Uranium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.
- Baes, C.F. and R.D. Sharp. 1983. A proposal for estimation of soil leaching and leaching constants for use in assessment models. *J. Environ. Qual.* 12(1):17-28.
- Balducci, A., A.R. Taddei, M. Mazzini, A.M. Fausto, F. Buonocore, and G. Scapigliati. 2001. Ultrastructure and proteins of the egg chorion of the arctic fish *Chionodraco hamatus* (Teleostei, Notothenioidei). *Polar Biology* 23: 417-421.
- Barillet, S., C. Adam, O. Palluel, and A. Devaux. 2007. Bioaccumulation, oxidative stress, and neurotoxicity in *Danio rerio* exposed to different isotopic compositions of uranium. *Environmental Toxicology and Chemistry* 26 (3):497-505.
- Borylo, A., B. Skwarzec, and J. Fabisiak. 2010. Bioaccumulation of uranium U-234 and U-238 in marine birds. *Journal of Radioanalytical and Nuclear Chemistry.* 284:165-172.
- Burkart, W., G.L. Finch, and T. Jung. 1997. Quantitative health effects from the combined action of low-level radiation and other environmental agents can new approaches solve the enigma? *Science of the Total Environment*, 205(1):51-70.
- Brandom W.F., G. Saccomanno, V.E. Archer, P.G. Archer and A.D. Bloom. 1978. Chromosome aberrations as a biological dose-response indicator of radiation exposure in uranium miners. *Radiat. Res.* 79: 159-171.
- Committee on the Biological Effects of Ionizing Radiation (BEIR). 1988. Health Risks of Radon and Other Internally Deposited Alpha-Emitters, BEIR IV, Biological Effects of Ionizing Radiation, National Research Council, National Academy of Sciences, National Academy Press, Washington, D.C. 1988.

- Cooley, H.M., R.E. Evans, and J.F. Klaverkamp. 2000. Toxicology of dietary uranium in lake whitefish (*Coregonus clupeaformis*). *Aquatic Toxicology* 48:495–515.
- Darolles, C., D. Broggio, A. Feugier, S. Frelon, I. Dublineau, M. De Meo, and F. Petitot. 2010. Different genotoxic profiles between depleted and enriched uranium. *Toxicology Letters*. 192(3):337-338.
- Delistraty, D.A. and J. Yokel. 1998. Ecotoxicity of River and Spring Sediment Along the Hanford Reach. *Bulletin of Environmental Contamination and Toxicology*. 61:754-761.
- Domingo, J. 2001. Reproductive and developmental toxicity of natural and depleted uranium: a review. *Reproductive Toxicology* 15:603-609.
- Driver C.J. 1994. Ecotoxicity Literature Review of Selected Hanford Site Contaminants. PNL-9394, Pacific Northwest Laboratory, Richland, Washington. <http://www.osti.gov/energycitations/servlets/purl/10136486-6sLptZ/native/10136486.pdf>
- Durakovias, A. 1999. Medical Effects of internal Contamination with Uranium, *Croatian Medical Journal* Volume 40, No. 1, March.
- Eisenbud, M. and T. Gesell. 1997. Environmental Radioactivity from Natural, Industrial, and Military Sources, Fourth Edition, Academic Press, San Diego, CA.
- Health Physics Society (HPS). 2002. Ask the Experts: Alpha Emitters – Uranium. <http://www.hps.org/publicinformation/ate/q1906.html>, posted April 30, 2002.
- Hogan, A.C., R.A. van Dam, S.J. Markich, and C. Camillieri 2005. Chronic toxicity of uranium to tropical green algae (*Chlorella* sp) in natural waters and the influence of dissolved organic carbon. *Aquatic Toxicology* 75(4):345-353.
- International Atomic Energy Agency (IAEA). 1992. Effects of Ionizing Radiation on Plants and Animals at Levels Implied by Current Radiation Protection Standards. Technical Report Series No. 332. Vienna.
- International Atomic Energy Agency (IAEA). 2010. Handbook of Parameter Values for the Prediction of Radionuclide transfer in Terrestrial and freshwater Environments, Technical Report Series No. 472, International Atomic Energy Agency, Vienna.
- International Commission on Radiological Protection (ICRP). 2008. Environmental Protection: The Concept and use of Reference Animals and Plants, *Annals of the Internal Commission on Radiation Protection*. ICRP Publication 108. Volume 38, Nos. 4-6, ,
- Labrot, F. J.F. Narbonne, P. Ville, M. Saint Denis, and D. Ribera. 1999. Acute toxicity, toxicokinetics, and tissue target of lead and uranium in the clam *Corbicula fluminea* and the worm *Eisenia fetida* : Comparison with the fish

Brachydanio rerio. *Archives of Environmental Contamination and Toxicology* 36:167-178.

- Lerebours, A., J.-P. Bourdineaud, K. Van der Ven, T. Vandenbrouck, P. Gonzolez, V. Camilleri, M. Floriani, J. Garnier-Laplace, and C. Adam-Guillermine. 2010. Sublethal effects of waterborne uranium exposures on the zebrafish brain: Transcriptional responses and alterations of olfactory bulb ultrastructure. *Environmental Science Technology* 44:1438-1443.
- Lerebours, A, P. Gonzolez, C. Adam, V. Camilleri, J., Bourdineaud, J. Garnier-Laplace. 2009. Comparative analysis of gene expression in brain, liver, skeletal muscles, and gills of zebrafish (*Danio rerio*) exposed to environmentally relevant waterborne uranium concentrations. *Environmental Toxicology and Chemistry* 28(6):1271-1278.
- Lew, Y.S., A. Kolozsvary, S.L. Brown, and J.H. Kim. 2002. Synergistic interaction with arsenic trioxide and fractionated radiation in locally advanced murine tumor. *Cancer Res* 62: 4202-4205.
- Manti, L. and A. D'Arco. 2010. Cooperative biological effects between ionizing radiation and other physical and chemical agents. *Mutation Research* 704: 115-122.
- Monleau, M, M. DeMeo, F. Paquet, V. Chazel, G. Dumenil, M. Donnadiou-Claraz, 2006. Genotoxic and inflammatory effects of depleted uranium particles inhaled by rats. *Toxicological Sciences* 89(1):287-295.
- Mothersill, C., B., Salbu, L.S. Heier, H.C. Teien, J. Denbeigh, D. Oughton, B.O. Rosseland, and C.B. Seymour. 2007. Multiple stressor effects of radiation and metals in salmon (*Salmo salar*). *Journal of Environmental Radioactivity*. 96(1-3):20-31.
- Mothersill, C. and C. Seymour. 2007. Radiation risks in the context of multiple stressors in the environment – issues for consideration. In: Mothersill, C., I. Mosse, and C. Seymour, eds. Multiple stressors: a challenge for the future. Book Series: Nato Science for Peace and Security Series C – Environmental Security. Pp. 235-246.
- National Council on Radiation Protection & Measurements (NCRP). 1991. Effects of Ionising Radiation on Aquatic Organisms. Report No. 109. Bethesda, MD.
- Oak Ridge Associated Universities (ORAU). 2010. Technical Basis Document for the Hanford Site – Site Description. ORAUT-TKBS-006-2, Rev. 01.
- Parkhurst, B.R., R.G. Elder, J.S. Meyer, D.A. Sanchez, R.W. Pennak, and W.T. Waller. 1984. An environmental hazard evaluation of uranium in a Rocky Mountain Stream. *Environmental Toxicology and Chemistry* 3(1):113-124.
- Poston, T.M., J. P. Duncan and R. L. Dirkes. 2010. Hanford Site Environmental Report for Calendar Year 2009, Prepared for the U.S. Department of Energy by Pacific Northwest Laboratory under contract DE-AC05-76RL01830, PNL, Richland, Washington.

- Poston, T.M., R.W. Hanf, and M.A. Simmons. 1983. Toxicity of uranium to *Daphnia magna*/ *Water, Air and Soil Pollution* 22(3):289-298.
- Prasad, K.N., W.C. Cole, and G. M. Hasse. 2004. Health Risks of Low Dose Ionizing radiation in Humans: A Review. *Exp Biol Med* 229: 378-382.
- Salbu, B. and L. Skipperud. 2007. Challenges in radioecotoxicology. In: Mothersill, C., I. Mosse, and C. Seymour, eds. Multiple stressors: a challenge for the future. Book Series: Nato Science for Peace and Security Series C – Environmental Security. Pp. 3-12.
- Sanchez D.J., M. Belles, M.L. Albina, J.J. Sirvent, and J.L. Domingo. 2001. Nephrotoxicity of simultaneous exposure to mercury and uranium in comparison to individual effects of these metals in rats. *Biol Trace Elem Res* 84(1-3):139-54.
- Schnug, E., R.B.M. Sparovek, M.C. Lamas, S.L. Kratz. J. Fleckenstein and S. Schroetter. 2006. Uranium Contamination. *Encyclopedia of Soil Science* 1 (1):1811-1813.
- Serne, R.J., C.F. Brown, H.T., Schaef, E.M. Pierce, J. Lindberg, Z. Wang, P. Gassman, and J. Catalano. 2002. 300 Area Uranium Leach and Adsorption Project. PNNL-14022. Richland, WA: Pacific Northwest National Laboratory, U.S. Department of Energy. November 25.
- Schmehl, M.K. and E.F. Graham. 1987. Comparative ultrastructure of the zona radiata from eggs of six species of salmonids. *Cell and Tissue Research* 250:513-519.
- Schubert, J., E.J. Riley and S.A. Tyler. 1978. Combined effects in toxicology. A rapid systematic testing procedure: cadmium, mercury, and lead. *Toxicol Environ Health* 4(5/6):763-776.
- Sheppard, S.C. and D.H. Thibault. 1990. Default soil solid/liquid partition coefficients, K_{ds} , for four major oil types: a compendium. *Health Physics* 59(4):471-482.
- Sheppard, S.C., W.G. Evenden, and A.J. Anderson. 1992. Multiple Assays of Uranium Toxicity in Soil. *Environmental Toxicology and Water Quality: An International Journal* 7:275-294.
- Sheppard, S.C, M.I. Sheppard, M.O. Galler and B. Sanipelli. 2005. Derivation of ecotoxicity thresholds for uranium. *Journal of Environmental Radioactivity* 79:55-83.
- Tabata M., S.A. Kumar, and E. Nyarko. 2003. Enhanced conformational changes in DNA in the presence of mercury(II), cadmium(II) and lead(II) porphyrins. *J Inorg Biochem* 94(1-2):50-8.
- Tierney, K.B. D.H. Baldwin, T.J. Hara, P.S. Ross, N.L. Scholz and C.J. Kennedy. 2010. Review: Olfactory toxicity in fishes. *Aquatic Toxicology* 96: 2-26.
- Till, J.E. and H. R Meyer. 1983. Radiological Assessment – A Textbook on Environmental Dose Analysis, NUREG/CT-3332, ORNL-5968, Prepared for the

Division of Systems Integration, Office of Nuclear Regulatory Regulation, U.S. Nuclear Regulatory Commission, Washington, D.C. NRC FIN B0766.

Traore A, M. Bonini, S.D. Dano, and E.E. Creppy. 1999. Synergistic effects of some metals contaminating mussels on the cytotoxicity of the marine toxin okadaic acid. *Arch Toxicol* 73(6):289-95.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2000. Sources and Effects of Ionizing Radiation.

U.S. Department of Energy (DOE). 1996. Closing the Circle on the Splitting of the Atom. Washington, D.C.: U.S. Department of Energy Office of Environmental Management. <http://ndep.nv.gov/lts/close/circle.htm>.

APPENDIX C | SPECIES DOCUMENTED ON THE HANFORD SITE

The following tables present information on wildlife species present on the Hanford Site, highlighting species of special conservation status. Note these tables are not exhaustive; additional information on species found on the Hanford Site is documented in numerous publications such as Gray and Dauble 1977, Fitzner and Gray 1991, Downs *et al.* 2004, CRICIA 1998, TNC 1999, TNC 2003, Burk *et al.* 2007, USFWS 2008, and information from the Hanford Site Environmental Monitoring and Compliance Project (presented in Downs *et al.* 1993 and the annual Hanford Site Environmental Monitoring Reports).¹³⁰

EXHIBIT C-1 COMMON VASCULAR SHRUB-STEPPE PLANTS ON THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME |
|--------------------------|--|
| Big sagebrush | <i>Artemisia tridentata</i> |
| Bitterbrush | <i>Purshia tridentata</i> |
| Gray rabbitbrush | <i>Ericameria nauseosa</i> |
| Green rabbitbrush | <i>Chrysothamnus viscidiflorus</i> |
| Snow buckwheat | <i>Eriogonum niveum</i> |
| Spiny hopsage | <i>Grayia (=Atriplex) spinosa</i> |
| Threetip sagebrush | <i>Artemisia tripartita</i> |
| Bluebunch wheatgrass | <i>Pseudoroegneria spicata</i> |
| Bottlebrush squirreltail | <i>Elymus elymoides</i> |
| Indian ricegrass | <i>Oryzopsis (=Achnatherum) hymenoides</i> |
| Needle-and-thread grass | <i>Hesperostipa comata</i> |
| Prairie junegrass | <i>Koeleria macrantha</i> |
| Sand dropseed | <i>Sporobolus cryptandrus</i> |
| Sandberg's bluegrass | <i>Poa secunda (sandbergii)</i> |
| Thickspike wheatgrass | <i>Elymus lanceolatus</i> |
| Buckwheat milkvetch | <i>Astragalus caricinus</i> |
| Carey's balsamroot | <i>Balsamorhiza careyana</i> |
| Cusick's sunflower | <i>Helianthus cusickii</i> |
| Gray's desert parsley | <i>Lomatium grayi</i> |
| Hoary aster | <i>Machaeranthera canescens</i> |

¹³⁰ These lists show many, but not all, species present at the Hanford Site. Note that inclusion of a species in this assessment plan does not imply an obligation on the part of the Trustees to evaluate it, nor does omission of a species preclude the Trustees from evaluating potential injury to that species.

| COMMON NAME | SCIENTIFIC NAME |
|----------------------------------|------------------------------|
| Annual Jacob's ladder | <i>Polemonium micranthum</i> |
| Pink microsteris | <i>Microsteris gracilis</i> |
| Tarweed fiddleneck | <i>Amsinckia lycopsoides</i> |
| Threadleaf scorpion weed | <i>Phacelia lycopsoides</i> |
| Source: Burk <i>et al.</i> 2007. | |

EXHIBIT C-2 THREATENED AND ENDANGERED PLANT SPECIES AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS | STATE STATUS |
|---------------------------------|--|--------------------|--------------|
| Awned halfchaff sedge* | <i>Lipocarpa (= Hemicarpha) aristulata</i> | | Threatened |
| Beaked spike-rush | <i>Eleocharis rostellata</i> | | Sensitive |
| Canadian St. John's wort | <i>Hypericum majus</i> | | Sensitive |
| Chaffweed | <i>Anagallis (= Centunculus) minimus</i> | | Threatened |
| Columbia milkvetch | <i>Astragalus columbianus</i> | Species of concern | Sensitive |
| Columbia yellowcress* | <i>Rorippa columbiae</i> | Species of concern | Endangered |
| Coyote tobacco | <i>Nicotiana attenuata</i> | | Sensitive |
| Desert cryptantha | <i>Cryptantha scoparia</i> | | Sensitive |
| Desert dodder | <i>Cuscuta denticulata</i> | | Threatened |
| Desert evening-primrose | <i>Oenothera caespitosa</i> | | Sensitive |
| Dwarf evening primrose | <i>Camissonia (= Oenothera) pygmaea</i> | | Sensitive |
| Fuzzytongue penstemon | <i>Penstemon eriantherus whitedii</i> | | Sensitive |
| Geyer's milkvetch | <i>Astragalus geyeri</i> | | Threatened |
| Grand redstem* | <i>Ammannia robusta</i> | | Threatened |
| Gray cryptantha | <i>Cryptantha leucophaea</i> | Species of concern | Sensitive |
| Great Basin gilia | <i>Aliciella (= Gilia) leptomeria</i> | | Threatened |
| Hoover's desert parsley | <i>Lomatium tuberosum</i> | Species of concern | Sensitive |
| Loeflingia | <i>Loeflingia squarrosa var. squarrosa</i> | | Threatened |
| Lowland toothcup* | <i>Rotala ramosior</i> | | Threatened |
| Piper's daisy | <i>Erigeron piperianus</i> | | Sensitive |
| Rosy pussypaws | <i>Cistanthe (= Calyptridium) roseum</i> | | Threatened |
| Small-flowered evening-primrose | <i>Camissonia (= Oenothera) minor</i> | | Sensitive |

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS | STATE STATUS |
|--|---|----------------|--------------|
| Snake River cryptantha | <i>Cryptantha spiculifera</i> (= <i>C. interrupta</i>) | | Sensitive |
| Suksdorf's monkey flower | <i>Mimulus suksdorfii</i> | | Sensitive |
| Umtanum desert buckwheat | <i>Eriogonum codium</i> | Candidate | Endangered |
| White Bluffs bladderpod | <i>Physaria</i> (= <i>Lesquerella</i>) <i>tuplashensis</i> | Candidate | Threatened |
| White eatonella | <i>Eatonella nivea</i> | | Threatened |
| <p>Notes:</p> <ol style="list-style-type: none"> * indicates that species is aquatically-linked. Endangered - Species in danger of extinction within all or a significant portion of its range. Threatened - Species likely to become endangered in the foreseeable future. Candidate - Species that are believed to qualify for threatened or endangered species status, but for which listing proposals have not been prepared. Sensitive - Taxa that are vulnerable or declining and could become endangered or threatened without active management or removal of threats. Species of concern - Species that are not currently listed or candidates under the Endangered Species Act of 1973, but are of conservation concern within specific U.S. Fish and Wildlife Service regions. <p>Source: Poston, Duncan and Dirkes, eds. 2010.</p> | | | |

EXHIBIT C-3 REPTILE AND AMPHIBIAN SPECIES DOCUMENTED AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | STATUS | FEDERAL/STATE STATUS | HABITAT |
|-----------------------|--------------------------------|--------|----------------------|---------|
| Reptiles | | | | |
| Painted turtle | <i>Chrysemys picta</i> | C | | P |
| Short-horned Lizard | <i>Phrynosoma douglassi</i> | UC | | E |
| Sagebrush Lizard | <i>Sceloporous graciosus</i> | R | State Candidate | S, LE |
| Side-blotched Lizard | <i>Uta stansburiana</i> | A | | LE |
| Western Rattlesnake | <i>Crotalus viridis</i> | C | | E, BS |
| Gopher Snake | <i>Pituophis melanoleucus</i> | A | | E |
| Night Snake | <i>Hypsiglena torquata</i> | UC | | BS, E |
| Striped Whipsnake | <i>Masticophis taeniatus</i> | R | State Candidate | LE |
| Racer | <i>Coluber constrictor</i> | A | | E |
| Amphibians* | | | | |
| Great Basin spadefoot | <i>Scaphiopus intermontana</i> | C | | R |
| Woodhouse's toad | <i>Bufo woodhousei</i> | C | | R |
| Western toad | <i>Bufo boreas</i> | R | Species of | |

| COMMON NAME | SCIENTIFIC NAME | STATUS | FEDERAL/STATE STATUS | HABITAT |
|---|-----------------|--------|--------------------------|---------|
| | | | concern /State Candidate | |
| <p>Notes:</p> <p>R = Rare C = Common UC = Uncommon A = Abundant R = Riparian</p> <p>S = Sandy Areas P = Pond LE = Low Elevation E = Entire Site BS = Basalt Outcroppings</p> <ol style="list-style-type: none"> * indicates that species is aquatically-linked. Candidate - Species that are believed to qualify for threatened or endangered species status, but for which listing proposals have not been prepared. Species of concern - Species that are not currently listed or candidates under the Endangered Species Act of 1973, but are of conservation concern within specific U.S. Fish and Wildlife Service regions. <p>Source: Fitzner and Gray 1991 and Poston, Duncan, and Dirkes, eds. 2010.</p> | | | | |

EXHIBIT C-4 INSECTS IDENTIFIED AT THE HANFORD SITE

| ORDER | NUMBER OF SPECIES IDENTIFIED | APPROXIMATE NUMBER OF SPECIES REMAINING TO BE IDENTIFIED AT TIME OF PUBLICATION |
|---|------------------------------|---|
| Aranae (spiders) | 0 | 50-100 |
| Chilopoda (centipedes) | 2 | 0 |
| Coleoptera (beetles) | 242 | 50-70 |
| Dermaptera (earwigs) | 1 | 0 |
| Diptera (flies) | 322 | 100-150 |
| Hemiptera (bugs) | 86 | 20-40 |
| Homoptera (leafhoppers and relatives) | 112 | 25-40 |
| Hymenoptera (bees, wasps, and ants)* | 364 | 75-150 |
| Lepidoptera (butterflies) | 50 | 0 |
| Lepidoptera (moths) | 320 | 50-100 |
| Mantodea (mantids) | 1 | 2 |
| Neuroptera (lacewings) | 26 | 0 |
| Odonata (dragonflies and damselflies) | 8 | 0 |
| Orthoptera (grasshoppers and relatives) | 1 | 10-20 |
| Scorpiones (scorpions) | 1 | 0 |
| Siphonaptera (fleas) | 0 | 1-2 |
| Total | 1,536 | 383-672 |
| * Combines figures from Ensor 1997 and Zack undated (1998 field season) | | |
| Source: TNC 1999 | | |

EXHIBIT C-5 THREATENED AND ENDANGERED INSECT SPECIES AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS(A) | STATE STATUS(A) |
|--|-----------------------------------|-------------------|-----------------|
| Columbia River tiger beetle* | <i>Cicindela columbica</i> | | Candidate |
| Silver-bordered fritillary butterfly | <i>Boloria selene atrocotalis</i> | | Candidate |
| <p>Notes:</p> <ol style="list-style-type: none"> *Species is likely present, but has not been observed on site. Candidate - Species that are believed to qualify for threatened or endangered species status, but for which listing proposals have not been prepared. <p>Source: Poston, Duncan and Dirkes, eds. 2010.</p> | | | |

EXHIBIT C-6 THREATENED AND ENDANGERED MOLLUSK SPECIES AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS | STATE STATUS |
|--|--|--------------------|--------------|
| California floater | <i>Anodonta californiensis</i> | Species of Concern | Candidate |
| Giant Columbia River spire snail | <i>Fluminicola (= Lithoglyphus) columbiana</i> | Species of Concern | Candidate |
| Shortfaced lanx | <i>Fisherola nuttalli</i> | | Candidate |
| <p>Notes:</p> <ol style="list-style-type: none"> Candidate - Species that are believed to qualify for threatened or endangered species status, but for which listing proposals have not been prepared. Species of concern - Species that are not currently listed or candidates under the Endangered Species Act of 1973, but are of conservation concern within specific U.S. Fish and Wildlife Service regions. <p>Source: Poston, Duncan and Dirkes, eds. 2010.</p> | | | |

EXHIBIT C-7 FISH SPECIES IN THE HANFORD REACH

| COMMON NAME | SCIENTIFIC NAME |
|---|----------------------------------|
| Paddlefishes, spoonfishes, sturgeons (family <i>Acipenseridae</i>) | |
| White sturgeon | <i>Acipenser transmontanus</i> |
| Anchovies, herrings (family <i>Clupeidae</i>) | |
| American shad | <i>Alosa sapidissima</i> |
| Suckers (family <i>Catostomidae</i>) | |
| Bridgelip sucker | <i>Catostomus columbianus</i> |
| Largescale sucker | <i>Catostomus macrocheilus</i> |
| Mountain sucker | <i>Catostomus platyrhynchus</i> |
| Carp, minnows (family <i>Cyprinidae</i>) | |
| Chiselmouth | <i>Acrocheilus alutaceus</i> |
| Common carp | <i>Cyprinus carpio</i> |
| Peamouth | <i>Mylocheilus caurinus</i> |
| Northern pikeminnow | <i>Ptychocheilus oregonensis</i> |
| Longnose dace | <i>Rhinichthys cataractae</i> |
| Leopard dace | <i>Rhinichthys falcatus</i> |
| Redside shiner | <i>Richardsonius balteatus</i> |
| Speckled dace | <i>Rhinichthys osculus</i> |
| Tench | <i>Tinca tinca</i> |
| Livebearers (family <i>Poeciliidae</i>) | |
| Western mosquitofish | <i>Gambusia affinis</i> |
| Cods (family <i>Gadidae</i>) | |
| Burbot | <i>Lota lota</i> |
| Pipefishes, sticklebacks (family <i>Gasterosteidae</i>) | |
| Threespine stickleback | <i>Gasterosteus aculeatus</i> |
| Perch-like fishes (family <i>Centrarchidae</i>) | |
| Pumpkinseed | <i>Lepomis gibbosus</i> |
| Bluegill | <i>Lepomis macrochirus</i> |
| Smallmouth bass | <i>Micropterus dolomieu</i> |
| Largemouth bass | <i>Micropterus salmoides</i> |
| Yellow perch | <i>Perca flavescens</i> |
| White crappie | <i>Pomoxis annularis</i> |
| Black crappie | <i>Pomoxis nigromaculatus</i> |
| Walleye | <i>Sander vitreus</i> |
| Trout perches (family <i>Percopsidae</i>) | |
| Sand roller | <i>Percopsis transmontana</i> |
| Lampreys (family <i>Petromyzontidae</i>) | |

| COMMON NAME | SCIENTIFIC NAME |
|--|---------------------------------|
| River lamprey | <i>Lampetra ayresii</i> |
| Pacific lamprey | <i>Lampetra tridentata</i> |
| Salmonids, salmon, trouts (family <i>Salmonidae</i>) | |
| Lake whitefish | <i>Coregonus clupeaformis</i> |
| Bull trout | <i>Salvelinus confluentus</i> |
| Cutthroat trout | <i>Oncorhynchus clarkii</i> |
| Coho salmon | <i>Oncorhynchus kisutch</i> |
| Rainbow trout (steelhead) | <i>Oncorhynchus mykiss</i> |
| Sockeye salmon | <i>Oncorhynchus nerka</i> |
| Chinook salmon | <i>Oncorhynchus tshawytscha</i> |
| Mountain whitefish | <i>Prosopium williamsoni</i> |
| Chabots, sculpins (family <i>Cottidae</i>) | |
| Prickly sculpin | <i>Cottus asper</i> |
| Mottled sculpin | <i>Cottus bairdii</i> |
| Piute sculpin | <i>Cottus beldingii</i> |
| Reticulate sculpin | <i>Cottus perplexus</i> |
| Torrent sculpin | <i>Cottus rhotheus</i> |
| Bullhead catfishes, North American freshwater catfishes (family <i>Ictaluridae</i>) | |
| Yellow bullhead | <i>Ameiurus natalis</i> |
| Brown bullhead | <i>Ameiurus nebulosus</i> |
| Black bullhead | <i>Ameiurus melas</i> |
| Channel catfish | <i>Ictalurus punctatus</i> |
| Source: Gray and Dauble (1977) as cited in Duncan (2007). | |

EXHIBIT C-8 THREATENED AND ENDANGERED FISH SPECIES AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS | STATE STATUS |
|---|---------------------------------|--------------------|--------------|
| Bull trout* | <i>Salvelinus confluentus</i> | Threatened | Candidate |
| Leopard dace* | <i>Rhinichthys flacatus</i> | | Candidate |
| Mountain sucker* | <i>Catostomus platyrhynchus</i> | | Candidate |
| River lamprey* | <i>Lampetra ayresi</i> | Species of Concern | Candidate |
| Spring-run Chinook | <i>Oncorhynchus tshawytscha</i> | Endangered | Candidate |
| Steelhead | <i>Oncorhynchus mykiss</i> | Threatened | Candidate |
| <p>Notes:</p> <ol style="list-style-type: none"> * indicates the species has been reported, but is seldom observed on the Hanford Site. Endangered - Species in danger of extinction within all or a significant portion of its range. Threatened - Species likely to become endangered in the foreseeable future. Candidate - Species that are believed to qualify for threatened or endangered species status, but for which listing proposals have not been prepared. Species of concern - Species that are not currently listed or candidates under the Endangered Species Act of 1973, but are of conservation concern within specific U.S. Fish and Wildlife Service regions. <p>Source: Poston, Duncan and Dirkes, eds. 2010.</p> | | | |

EXHIBIT C-9 COMMON AVIAN SPECIES AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | SEASON OF HIGHEST ABUNDANCE |
|----------------------------|------------------------------|-----------------------------|
| Eared grebe | <i>Podiceps nigricollis</i> | Winter |
| Horned grebe | <i>Podiceps auritus</i> | Winter |
| Pied-billed grebe | <i>Podilymbus podiceps</i> | Winter |
| Double-crested cormorant | <i>Phalacrocorax auritus</i> | All year |
| American green-winged teal | <i>Anas crecca</i> | All year |
| American wigeon | <i>Anas americana</i> | Winter |
| Barrow's goldeneye | <i>Bucephala islandica</i> | Winter |
| Blue-winged teal | <i>Anas discors</i> | Breeding |
| Common goldeneye | <i>Bucephala clangula</i> | Winter |
| Common merganser | <i>Mergus merganser</i> | All year |
| Northern pintail | <i>Anas acuta</i> | All year |
| American coot | <i>Fulica Americana</i> | All year |
| California gull | <i>Larus californicus</i> | All year |
| Black-crowned night heron | <i>Nycticorax nycticorax</i> | Breeding |
| Greater yellowlegs | <i>Tringa flavipes</i> | Migration |
| Spotted sandpiper | <i>Actitis macularia</i> | Breeding |

| COMMON NAME | SCIENTIFIC NAME | SEASON OF HIGHEST ABUNDANCE |
|----------------------------------|--------------------------------|-----------------------------|
| Ring-necked pheasant | <i>Phasianus colchicus</i> | All year |
| American kestrel | <i>Falco sparverius</i> | All year |
| Merlin | <i>Falco columbarius</i> | Migration |
| Prairie falcon | <i>Falco mexicanus</i> | All year |
| Red-tailed hawk | <i>Buteo jamaicensis</i> | All year |
| Common barn owl | <i>Tyto alba</i> | All year |
| Great horned owl | <i>Bubo virginianus</i> | All year |
| Bank swallow | <i>Riparia riparia</i> | Breeding |
| Blue-headed vireo | <i>Vireo solitarius</i> | Migration |
| Bullock's oriole | <i>Icterus galbula</i> | Breeding |
| Common raven | <i>Corvus corax</i> | All year |
| Dark-eyed junco | <i>Junco hyemalis</i> | All year |
| Eastern kingbird | <i>Tyrannus tyrannus</i> | Breeding |
| Golden-crowned kinglet | <i>Regulus satrapa</i> | Migration |
| House finch | <i>Haemorhous mexicanus</i> | All year |
| Rufous-sided towhee | <i>Pipilo erythrophthalmus</i> | Breeding |
| White-crowned sparrow | <i>Zonotrichia leucophrys</i> | Winter |
| Source: Burk <i>et al.</i> 2007. | | |

EXHIBIT C-10 THREATENED AND ENDANGERED AVIAN SPECIES AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS(A) | STATE STATUS(A) |
|------------------------|----------------------------------|--------------------|-----------------|
| American white pelican | <i>Pelecanus erythrorhynchos</i> | | Endangered |
| Bald eagle | <i>Haliaeetus leucocephalus</i> | Species of concern | Sensitive |
| Burrowing owl | <i>Athene cunicularia</i> | Species of concern | Candidate |
| Clark's grebe | <i>Aechmophorus clarkii</i> | | Candidate |
| Common loon | <i>Gavia immer</i> | | Sensitive |
| Ferruginous hawk | <i>Buteo regalis</i> | Species of concern | Threatened |
| Flamulated owl* | <i>Otus flammeolus</i> | | Candidate |
| Golden eagle | <i>Aquila chrysaetos</i> | | Candidate |
| Greater sage grouse | <i>Centrocercus urophasianus</i> | Candidate | Threatened |
| Lewis's woodpecker* | <i>Melanerpes lewis</i> | | Candidate |
| Loggerhead shrike | <i>Lanius ludovicianus</i> | Species of concern | Candidate |
| Northern goshawk* | <i>Accipiter gentilis</i> | Species of | Candidate |

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS(A) | STATE STATUS(A) |
|--|----------------------------------|--------------------|-----------------|
| | | concern | |
| Olive-sided flycatcher | <i>Contopus cooperi</i> | Species of concern | |
| Peregrine falcon | <i>Falco peregrinus</i> | Species of concern | Sensitive |
| Sage sparrow | <i>Amphispiza belli</i> | | Candidate |
| Sage thrasher | <i>Oreoscoptes montanus</i> | | Candidate |
| Sandhill crane | <i>Grus canadensis</i> | | Endangered |
| Western grebe | <i>Aechmophorus occidentalis</i> | | Candidate |
| <p>Notes:</p> <ol style="list-style-type: none"> * Indicates that species has been reported, but is seldom observed on the Hanford site. Endangered - Species in danger of extinction within all or a significant portion of its range. Threatened - Species likely to become endangered in the foreseeable future. Candidate - Species that are believed to qualify for threatened or endangered species status, but for which listing proposals have not been prepared. Sensitive - Taxa that are vulnerable or declining and could become endangered or threatened without active management or removal of threats. Species of concern - Species that are not currently listed or candidates under the Endangered Species Act of 1973, but are of conservation concern within specific U.S. Fish and Wildlife Service regions. <p>Source: Poston, Duncan and Dirkes, eds. 2010.</p> | | | |

EXHIBIT C-11 MAMMALIAN SPECIES DOCUMENTED AT THE HANFORD SITE

| FAMILY | SCIENTIFIC NAME | COMMON NAME | STATUS | DISTRIBUTION |
|------------------|----------------------------------|--------------------------|--------|--------------|
| Soricidae | <i>Sorex vagrans</i> | Vagrant shrew | UC | Ri |
| | <i>S. merriami</i> | Merriam's shrew | UC | UE, ALE |
| Vespertilionidae | <i>Lasionycteris noctivagans</i> | Silver-haired bat | C | Ri, ALE |
| | <i>Lasiurus cinereus</i> | Hoary bat | C | Ri, ALE |
| | <i>Antrozous pallidus</i> | Pallid bat | C | BC |
| | <i>Myotis lucifugus</i> | Little brown myotis | C | BC |
| | <i>M. yumanensis</i> | Yuma myotis | C | BC |
| | <i>M. californicus</i> | California myotis | C | BS, ALE |
| Leporidae | <i>Lepus townsendii</i> | White-tailed jack rabbit | UC | UE, ALE |
| | <i>L. californicus</i> | Black-tailed jack rabbit | C | E |
| | <i>Sylvilagus nuttallii</i> | Nuttall's cottontail | C | E |
| | <i>S. idahoensis</i> | Pygmy rabbit | EX | |

Final Hanford Natural Resource Damage Assessment Injury Assessment Plan

| FAMILY | SCIENTIFIC NAME | COMMON NAME | STATUS | DISTRIBUTION | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|----------------------------------|----------------------------|--------|--------------|-------------------------------|---|------|----|---|-------------------------------|----|---|----------|---|---|-------------|---|---|--------|----|---|----------------|---|---|----------|-----|---|-------------|----|---|------------|----|---|------------------|----|---|----------|---|---|-----------|----|---|---------------------|--|--|--|
| Sciuridae | <i>Spermophilustownsendii</i> | Townsend ground squirrel | C | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Marmotaflaviventris</i> | Yellow-bellied marmot | R | UE, ALE | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Eutamias minimus</i> | Least chipmunk | R | UE, ALE | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Geomyidae | <i>Thomomys talpoides</i> | Northern pocket gopher | A | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Heteromyidae | <i>Perognathus parvus</i> | Great Basin pocket mouse | A | E, LE | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Castoridae | <i>Castor canadensis</i> | Beaver | C | CR | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Cricetidae | <i>Reithrodontomys megalotis</i> | Western harvest mouse | R | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Peromyscus maniculatus</i> | Deer mouse | A | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Onychomys leucogaster</i> | Northern grasshopper mouse | C | ALE | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Neotoma cinerea</i> | Bushy-tailed woodrat | C | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Lagurus curtatus</i> | Sagebrush vole | UC | UE, Ri | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Microtus montanus</i> | Montane meadow mouse | R | Ri | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Ondatra zibethica</i> | Muskrat | R | CR | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Muridae | <i>Rattus norvegicus</i> | Norway rat | C | B | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Mus musculus</i> | House mouse | C | B | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Erethizontidae | <i>Erithizon dorsatum</i> | Porcupine | C | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Canidae | <i>Canis latrans</i> | Coyote | UC | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Procyonidae | <i>Procyon lotor</i> | Raccoon | UC | Ri, CR | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Mustelidae | <i>Mustela vison</i> | Mink | UC | Ri, CR | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>M. frenata</i> | Long-tailed weasel | UC | Ri, CR | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>M. erminea</i> | Short-tail weasel | R | Ri | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Lutra canadensis</i> | Otter | R | CR | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Taxidea taxus</i> | Badger | C | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Mephitis mephitis</i> | Striped skunk | C | R | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Felidae | <i>Lynx rufus</i> | Bobcat | UC | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Cervidae | <i>Odocoileus hemionus</i> | Mule deer | C | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>O. virginianus</i> | White-tailed deer | R | CR | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | <i>Cervus elaphus</i> | Elk | C | E | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Antilocapridae | <i>Antilocapra americana</i> | Pronghorn | EX | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| <p>Notes:</p> <table> <tr> <td>R</td> <td>=</td> <td>Rare</td> <td>BS</td> <td>=</td> <td>Basalt Outcropping and Cliffs</td> </tr> <tr> <td>UC</td> <td>=</td> <td>Uncommon</td> <td>E</td> <td>=</td> <td>Entire site</td> </tr> <tr> <td>C</td> <td>=</td> <td>Common</td> <td>CR</td> <td>=</td> <td>Columbia River</td> </tr> <tr> <td>A</td> <td>=</td> <td>Abundant</td> <td>ALE</td> <td>=</td> <td>ALE Reserve</td> </tr> <tr> <td>EX</td> <td>=</td> <td>Extirpated</td> <td>UE</td> <td>=</td> <td>Upper elevations</td> </tr> <tr> <td>Ri</td> <td>=</td> <td>Riparian</td> <td>B</td> <td>=</td> <td>Buildings</td> </tr> <tr> <td>BC</td> <td>=</td> <td>Buildings and Caves</td> <td></td> <td></td> <td></td> </tr> </table> | | | | | R | = | Rare | BS | = | Basalt Outcropping and Cliffs | UC | = | Uncommon | E | = | Entire site | C | = | Common | CR | = | Columbia River | A | = | Abundant | ALE | = | ALE Reserve | EX | = | Extirpated | UE | = | Upper elevations | Ri | = | Riparian | B | = | Buildings | BC | = | Buildings and Caves | | | |
| R | = | Rare | BS | = | Basalt Outcropping and Cliffs | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| UC | = | Uncommon | E | = | Entire site | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| C | = | Common | CR | = | Columbia River | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| A | = | Abundant | ALE | = | ALE Reserve | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| EX | = | Extirpated | UE | = | Upper elevations | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Ri | = | Riparian | B | = | Buildings | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| BC | = | Buildings and Caves | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Source: Fitzner and Gray (1991). | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |

EXHIBIT C-12 THREATENED AND ENDANGERED MAMMALIAN SPECIES AT THE HANFORD SITE

| COMMON NAME | SCIENTIFIC NAME | FEDERAL STATUS(A) | STATE STATUS(A) |
|---|---------------------------------|--------------------|-----------------|
| Black-tailed jackrabbit | <i>Lepus californicus</i> | | Candidate |
| Merriam's shrew | <i>Sorex merriami</i> | | Candidate |
| Townsend's ground squirrel | <i>Spermophilus townsendii</i> | Species of concern | Candidate |
| Washington ground squirrel* | <i>Spermophilus washingtoni</i> | Candidate | Candidate |
| White-tailed jackrabbit | <i>Lepus townsendii</i> | | Candidate |
| <p>Notes:</p> <ol style="list-style-type: none"> * indicates that species has been reported, but is seldom observed on the Hanford site. Candidate - Species that are believed to qualify for threatened or endangered species status, but for which listing proposals have not been prepared. Species of concern - Species that are not currently listed or candidates under the Endangered Species Act of 1973, but are of conservation concern within specific U.S. Fish and Wildlife Service regions. <p>Source: Poston, Duncan and Dirkes, eds. 2010.</p> | | | |