

5. REMEDIAL INVESTIGATION TASKS

Several steps are needed to accomplish the RI. These steps are presented and discussed below.

5.1 PROJECT PLANNING

The project plans for the RI, as outlined in these management plans, will be implemented once finalized. Revisions will be updated as necessary and will go through appropriate review by oversight agencies.

5.2 SAMPLE COLLECTION AND ANALYSES

The field investigation at the former Rayonier Mill Site will be implemented as detailed in the SAPs (Volume II).

5.3 DATA VALIDATION AND EVALUATION

Several phases of data evaluation must be completed before the results of the RI can be used to determine the former Rayonier Mill Site characteristics and the associated human health and environmental risks. Initially, field analytical data must be validated and reviewed for compliance with quality control criteria. The data are then interpreted for chemical sources, fate and transport, and risks.

5.3.1 Data Validation

The analytical data generated during the sampling and laboratory analyses will be used for site characterization and risk assessment (RA). Data quality objectives are detailed in the QAPP, Volume III.

5.3.2 Data Interpretation

Data sets collected are statistically summarized and reported for further interpretation. Methods for specific analyses and inferences are detailed in the SAP (Volumes II).

5.4 ASSESSMENT OF RISKS

Risk assessment (RA) is a procedure used to estimate the probabilities of known adverse effects that may result from chemicals released into the environment. This section describes the approaches to human health and ecological risks at the Rayonier facility. Because the Rayonier facility is being evaluated pursuant to a deferral agreement, the RA guidance under MTCA is the preferred approach. However, there are certain categories of human

and ecological receptors that are not specifically defined by MTCA. Consequently, both the human health and ecological RAs for the Rayonier facility are a blend of the specific guidance provided under MTCA and additional guidance provided by EPA where no such guidance is otherwise available from the state. The approach to human health risk assessment is described in Section 5.4.1 and that for ecological risk is described in Section 5.4.2.

5.4.1 Human Health Risk Analyses

RA is a useful tool for evaluating potential hazards to humans and thus guiding the cleanup process at contaminated sites. RA can greatly increase the efficiency of a given site cleanup by optimizing the type and amount of data collected, and identifying the areas needed to be remediated. The RA process involves using available information to clean a site to a protective standard. Section 5.4.1.1 below is a general overview of the various components of a risk assessment and how they are integrated to make judgments concerning potential site-specific impacts to human health or to calculate site-specific cleanup levels. Subsequent sections provide descriptions of how the risk assessment process will be implemented to evaluate potential health risks associated with on-site soils (Section 5.4.1.2), off-site residential soils (Section 5.4.1.3), and marine sediments (Section 5.4.1.4).

5.4.1.1 Overview of Health Risk Assessment

Both EPA and Ecology provide guidance on the RA process. The two procedures are based on the same concepts and calculations, but differ fundamentally in how the risk assessment results are presented. EPA provides the more general guidance, and is based on providing a single estimate of risk that is summed across multiple chemicals and pathways of exposure. Ecology's approach focuses on how cleanup levels can be calculated for individual chemicals and exposure media (e.g., soils). EPA's general approach is described below to provide an overview of the fundamentals of risk assessment, which generally involves four components: (1) hazard identification and data evaluation, (2) exposure assessment, (3) toxicity assessment, and (4) risk characterization. Ecology's approach for calculating cleanup levels for various kinds of sites is described below.

Hazard Identification

The hazard identification process involves a review of historical activities at a site, as well as current and future uses in order to identify areas of concern and potentially exposed people. During the hazard identification process, COPCs are determined based on the analysis of sampling results. Sample results are contrasted against established regulatory standards in a process referred to as screening. Chemicals that exceed their respective

screening values are retained for further consideration. Chemicals that do not exceed their screening values may be eliminated from further consideration; however, it may be necessary to collect additional samples before a chemical can be ruled out as a COPC. Chemicals for which there are no established screening values may also be retained for further analysis, unless it can be demonstrated that these chemicals pose an insignificant human health risk.

Exposure Assessment

Exposure assessment is an estimate of a COPC's magnitude, frequency, duration, and route of exposure at a given location. The chemical exposure for a given population depends on the chemical concentration in the various media at a site, the amount of time spent at a site, and the behavior patterns of the exposed population. For instance, children living and playing near a hazardous site will have different exposure patterns than construction workers at the same location. For an exposure assessment, each population (e.g., children or industrial workers) should be evaluated individually. Physical characteristics of a site, such as the type of ground surface and the depth to groundwater, can impact the magnitude of the exposure.

People can be exposed to chemicals through a number of different pathways. According to EPA (1989), for a pathway to be considered complete, it must consist of four necessary components: (1) a source and mechanism of chemical release, (2) an environmental transport medium (e.g., air and water), (3) a point of potential contact with the impacted medium (referred to as an exposure point), and (4) an exposure route (e.g., inhalation or ingestion) at the contact point.

A CSM is a commonly used tool for summarizing potential exposure pathways at a given site. The CSM is a flow chart diagram that relates the exposure source, the transport pathway, and the receptor population. Among the more common exposure pathways are ingestion of water, inhalation of airborne dust, ingestion of soil, and dermal contact with soil. At many sites, however, less obvious pathways must also be considered. For instance, if a site includes offshore lake or marine sediment, people could be exposed to chemicals in this media indirectly by consuming fish with elevated chemical concentrations in their tissue. Once all of the relevant exposure pathways have been considered, a total exposure, referred to as a reasonable maximum exposure (RME), can be calculated for people at a given site by summing the chemical intake associated with each pathway.

Chemical intake can be quantified and expressed as a chronic daily intake by using the following general equation:

$$\text{Intake} = \frac{C \times CR \times ED \times EF \times FI \times F_{\text{abs}}}{BW \times AT}$$

where:

Intake = Expressed as a dose (mg chemical/kg body weight per day).

C = Exposure point concentration [expressed a concentration in a specific medium such as soils (mg/kg dw), water (mg/L), fish (mg/kg ww) (EPA 1992b)]

CR = Contact rate; the amount of contaminant medium contacted per unit time or event; this parameter may be a soil, sediment, or fish ingestion rate (for ingestion pathways (mg/day), an inhalation rate (m³/day), or a skin contact rate (mg/cm² skin) for dermal pathways.

EF = Exposure frequency (number of days per year)

ED = Exposure duration (years of exposure)

BW = Body weight over the exposure period (kg)

AT = Averaging time; period over which exposure is averaged (days); for carcinogens, exposure is averaged over a 70-year lifetime; for noncarcinogens, exposure is averaged over the exposure duration.

FI = Fraction ingested from contaminated source (unitless)

F_{abs} = Fraction of chemical absorbed.

Toxicity Assessment

The relationship between exposure and adverse health effects is established through the toxicity assessment. Information regarding toxic effects of chemicals to humans is developed from a number of sources. If adequate human data are available, this information is used as the basis for the toxicity criteria. When direct human data are inadequate, toxicity values are developed by interpreting animal studies. If the toxicological data are not available for a particular exposure route, available toxicity information from another exposure route is adjusted for application to the route of interest. In general, the objective of the toxicity assessment is to establish a dose-response relationship between the amount of chemical intake and the severity of toxic effects. The approach for calculating a chemical's relative toxicity varies, depending on whether a chemical is considered a cancer-causing agent (carcinogen) or a systemic toxicant (noncarcinogen).

Noncarcinogens

For noncarcinogens, the outcome of a toxicity assessment is the development of a reference dose (RfD) or a reference concentration (RfC), which is considered a safe exposure level for an upper percentage of a given population. The study on the most sensitive population or species (the population showing a toxic effect at the lowest dose) is the basis for developing RfDs. The effect characterized by the “lowest-observed-adverse-effect level” (LOAEL) is the lowest dose at which an adverse effect was observed. In addition, EPA identifies the experimental exposure level representing the highest level at which no adverse effects (including the critical toxic effect) were observed. The highest “no-observed-adverse-effect level” (NOAEL) or the LOAEL are used to develop RfDs.

RfDs are calculated as the NOAEL or LOAEL divided by the product of the appropriate uncertainty factors (UFs) and modifying factors (MFs). Uncertainty factors are included to account for the uncertainty that may be associated with various components of the RfD development process, including the following extrapolations: from animal data to humans, from high-dose to low-dose exposures, from one exposure route to another, from short-term to long-term effects, and/or from less-sensitive to more-sensitive individuals in the population. Depending upon the study on which the RfD was based, the UF may range from less than 10 to 10,000. The MF is an additional UF ranging from less than 0 to 10. The purpose of the MF is to account for uncertainties not addressed by the other categories mentioned above, or to account for limitations in the overall database (e.g., number of studies or number of species tested). The use of UFs and MFs generally results in toxicity values that are unlikely to underestimate risks.

Carcinogens

For carcinogens, EPA generally assumes that effects on a single cell can evoke changes that may lead to the onset of disease; therefore, no dose is considered risk-free. Carcinogens are categorized into weight-of-evidence categories that represent the amount of evidence available to suggest their carcinogenicity. The weight-of-evidence classification is based on EPA’s Proposed Guidelines for Carcinogen Risk Assessment (EPA, 1996) as described in Table 5-1.

The toxicity criteria that EPA has developed to quantify carcinogenic dose-response relationship are called cancer slope factors (CSFs). A CSF is a plausible, upper-bound estimate of the probability of developing cancer per unit intake of a chemical over a lifetime. At low levels of exposure, the probability of cancer cannot be measured, but must be extrapolated from higher dosages. In order to observe a quantifiable effect on the

exposed population, animals typically are exposed to concentrations that are orders of magnitude greater than what is likely to be encountered by human populations. EPA has calculated CSFs for many potential carcinogens in classes A, B1, and B2. For class C chemicals, quantitative estimates of CSFs must be performed on a case-by-case basis.

Table 5-1. Weight of Evidence Classification for Carcinogenic Substances.

Weight-of-Evidence Category	Description
A	Human carcinogen.
B1 or B2	Probable human carcinogen. B1 indicates that limited human data are available. B2 indicates sufficient evidence in animals and inadequate or no evidence in humans.
C	Possible human carcinogen.
D	Not classifiable as to human carcinogenicity.
E	Evidence of noncarcinogenicity for humans.

For chemicals sharing similar properties, such as CDDs and CDFs, EPA has developed toxicity values based on relative risk. For CDDs and CDFs, TEFs have been assigned to all CDDs and CDFs based on their cancer potency relative to 2,3,7,8-TCDD, the most toxic and widely studied chemical in this class of compounds. The magnitude of the TEFs is determined both by their structure (i.e., the number and position of the halogen atoms on the dioxin-like compound) and by in vivo and in vitro toxicity test results. A similar TEF approach is commonly used for carcinogenic PAHs with toxicity values expressed relative to benzo(a)pyrene.

Sources of Toxicity Data

EPA maintains and updates a list of toxicity values for several hundred chemicals in the Integrated Risk Information System (IRIS) catalogue. An additional source for toxicity values is the EPA's Health Effects Summary Tables (HEAST), which is updated quarterly. Both of these sources summarize interim and final RfDs and CSFs and other toxicity information for specified chemicals. Ecology provides its own summary tables of toxicity values in its Cleanup Levels and Risk Calculations (CLARC) publication, (Ecology, 1996). Many of the values presented in the CLARC tables are from IRIS and HEAST.

Risk Characterization

Risk characterization is the final step of the RA process, which involves calculating risks to exposed individuals by relating the chemical intake calculated in the exposure assessment to the dose-response values determined in the toxicity assessment. Separate risk characterizations are conducted by noncarcinogens and carcinogens.

Noncarcinogenic Risk

For noncarcinogens, potential health threats are estimated by comparing the estimated average daily exposure with the RfD value by calculating a hazard quotient (HQ):

$$\text{Hazard Quotient} = \frac{\text{Intake}}{\text{RfD}}$$

where:

Intake = Chronic daily intake, averaged over the exposure duration (mg/kg-day)
RfD = Reference dose (mg/kg-day).

If a person's average exposure is less than the RfD (i.e., if the hazard quotient is lower than one), the chemical is considered unlikely to pose a significant noncarcinogenic health hazard to individuals under the given exposure conditions. Unlike carcinogenic risk estimates, a hazard quotient is not expressed as a probability. Therefore, while both cancer and noncancer risk characterizations indicate a relative potential for adverse effects to occur from exposure to a chemical, a noncancer health threat estimate is not directly comparable with a cancer risk estimate.

If more than one noncarcinogen or pathway is evaluated, the hazard quotients for each chemical and each pathway are summed to determine whether exposure to a combination of pathways and chemicals poses a health concern. This sum of the hazard quotients is known as a hazard index (HI). Where HIs exceed a ratio of 1.0, noncarcinogenic COPCs are segregated according to target organ, effect, and mechanism of action.

Carcinogenic Risk

In the risk characterization, carcinogenic risk is estimated as the incremental probability of an individual developing cancer over a lifetime as a result of a chemical exposure.

Carcinogenic risks are evaluated by multiplying the estimated average exposure rate (i.e., the lifetime average daily intake calculated in the exposure assessment) by the chemical's CSF.

$$\text{Risk} = \text{CSF} \times \text{Intake}$$

where:

- Risk = Chemical-specific probability of cancer over a 70-year lifetime of exposure
Intake = Chronic daily intake, averaged over a 70-year lifetime (mg/kg-day)
CSF = Cancer slope factor (mg/kg-day).

The CSF converts estimated daily intakes averaged over a lifetime to incremental risk of an individual developing cancer. Because cancer risks are averaged over a person's lifetime, longer-term exposure to a carcinogen will result in higher risks than shorter-term exposure to the same carcinogen, if all other exposure assumptions are constant. Theoretical risks associated with low levels of exposure in humans are assumed to be directly related to an observed cancer incidence in animals associated with high levels of exposure. According to EPA (1989), this approach is appropriate for theoretical upper bound cancer risks of less than 1×10^{-2} .

The total risk to a given population at a site can be expressed as the sum of the individual risk for each chemical associated with each exposure pathway. For carcinogens, it is assumed that simultaneous exposures to multiple chemicals are additive, unless information is available that suggests interactions such as antagonism or synergism. Thus, the result of the assessment will be an upper-bound estimate of the total carcinogenic risk, which can be compared to EPA's risk range of 10^{-6} to 10^{-4} . In general, risks less than 10^{-6} are considered de-minimis and do not require a cleanup action. Site-management and clean up decisions are made on a case-by-case basis when risks in the range of 10^{-6} to 10^{-4} . Risks greater than 10^{-4} almost always require a site-management action.

Risk Assessment in the Model Toxics Control Act

Ecology's approach focuses on how cleanup levels can be calculated for individual chemicals and exposure media (e.g., soils) by assuming a fixed level of risk (e.g., 10^{-5} cancer risk), re-arranging the general algebraic equation for calculating risk, and solving the expression for the corresponding chemical concentration in an exposure medium (e.g., soils). Ecology's approach is summarized in MTCA (WAC 173-340-708) newly revised in February 2001.

Under MTCA, three approaches for determining cleanup levels are described, and are referred to as Method A, Method B, and Method C. Method A is the most basic approach for establishing cleanup levels at a site. Method A is applicable to sites meeting specific

criteria: the former Rayonier Mill Site must meet routine cleanup actions as defined in WAC 173-340-130; there are few hazardous substances present; and numerical cleanup values are available for the hazardous substances at the former Rayonier Mill Site, as provided in WAC 173-340-720, 740, or 745. Method B is a cleanup approach that can be applied to all sites and involves the calculation of cleanup levels using RA principles. Under Method B, Ecology allows for the use of either a standard approach, or one using modified assumptions. Under the standard approach, default assumptions are used to calculate cleanup levels, whereas under the modified approach, site-specific information can be incorporated to calculate cleanup levels that are more realistic for a given site.

Method C can be used to calculate cleanup levels that are protective of human health and the environment under certain site uses and conditions, including sites where usage is limited to industrial activities. Method C cleanup values can be applied to multiple environmental media (i.e., soil, groundwater, and air), however, each medium must be evaluated separately. In other words, even if the soil at a site is to be used for industrial activity, the groundwater cleanup level does not automatically meet Method C criteria.

5.4.1.2 Marine Sediments

Marine organisms that live in or near contaminated marine sediments may accumulate contaminants in their body tissue over time. Although humans are unlikely to directly contact marine sediments, they may be exposed to contaminants in marine sediments indirectly by ingesting organisms that have lived in the sediments. The conceptual site model for indirect exposure to sediments via the consumption of seafood is presented in Section 3.3 above. A summary of the relevant receptor population and exposure pathways is provided in Table 5-2. Certain subpopulations that consume larger quantities of seafood from contaminated sediments such as recreational anglers or subsistence tribes, may, therefore, have a greater exposure levels to contaminants than the general public.

The tendency of chemicals to bioaccumulate in the tissue of marine organisms is a function of a chemical's physical properties (e.g., K_{ow}), but also depends on how the chemical is metabolized in an organism's body (EPA, 1989). The surface water cleanup provisions of MTCA (WAC 173-340-730) provide guidance for evaluating chemical uptake by fish via water-only exposure and the application of an appropriate bioconcentration factor (BCF)³ [WAC 173-340-708(9)]. Because some of the COPCs in sediments are highly hydrophobic, they are unlikely to be detected in water. Hence, an alternative method is needed to evaluate potential human exposure to chemicals in fish and shellfish. The EPA provides

³ BCF is defined as the chemical concentration in fish tissue divided by the chemical concentration in water.

guidance on how to estimate human exposures to chemicals through seafood ingestion based on chemical concentrations in marine sediments, as well as on fish ingestion rates for the general public and certain subpopulations. A more direct measure of potential risk due to ingestion of potentially contaminated seafood is, however, to collect and analyze the tissue of selected organisms from a particular area. The distribution of site-specific marine biota tissue concentrations can then be combined with ingestion rates to predict chemical exposure through this pathway.

The following equation (EPA, 1989) is proposed to estimate risks from ingestion of seafood:

$$\text{Intake (mg/kg - day)} = \frac{\text{CF} \times \text{IR} \times \text{FI} \times \text{EF} \times \text{ED} \times \text{F}_{\text{abs}}}{\text{BW} \times \text{AT}}$$

where:

- CF = Chemical concentration in fish (mg/kg body weight)
- IR = Ingestion rate (kg fish/day)
- FI = Fraction ingested from contaminated source (unitless)
- EF = Exposure frequency (days/yr)
- ED = Exposure duration (years)
- F_{abs} = Fraction of chemical absorbed
- BW = Body weight (kg)
- AT = Averaging time (period over which exposure is averaged – days).

Appropriate values for the ingestion rate of fish and shellfish, the fraction of fish and shellfish potentially associated with the former Rayonier Mill Site, the duration of exposure, and the frequency of exposure will be developed pursuant to MTCA guidance (WAC 173-340-708 and WAC 173-340-730).

Risks associated with the ingestion of fish and shellfish will be estimated for carcinogenic and noncarcinogenic substances as indicated in Section 5.4.1.1.4 above and compared to appropriate risk-based benchmarks established under MTCA. No further evaluation of the data will be needed if cumulative carcinogenic risk or the HI for noncarcinogens are less than their respective benchmarks. However, if either benchmark is exceeded additional evaluation will be undertaken to assess the relationship between chemical concentrations in fish and shellfish and those in sediments. The purpose of this analyses will be to determine

Table 5-2. Human Health Exposure Pathways and Potentially Contaminated Media.

Location	Receptor	Route of Exposure	Surface		Sediment	Aquatic
			Water ^a	Sediments	Porewater	Biota
Off-Site	Recreational Anglers	Respiration				
		Ingestion	▲	●	▲	●
		Dermal Absorption	▲	▲	▲	
	Subsistence Anglers	Respiration				
		Ingestion	▲	●	▲	●
		Dermal Absorption	▲	▲	▲	

^a Includes indirect exposure via ground water that may be discharged to Ennis Creek or Port Angeles Harbor.

- = Primary evaluation pathway.
- ▲ = Secondary evaluation pathway.

scientifically valid correlations between chemical concentrations in fish or shellfish tissues and those in sediments. Where such correlations can be established, it may be appropriate to develop bio-sediment accumulation factors (BSAF values)⁴ to assist in establishing sediment cleanup goals.

5.4.2 Ecological Risk Analyses

In addition to safeguarding long-term human health, RIs are also required to evaluate environmental health. The approach for this evaluation is discussed below for marine sediments.

5.4.2.1 Marine Biota

Potential adverse effects to aquatic organisms in Port Angeles Harbor will be assessed using the Washington Sediment Management Standards (WAC 173-204). Contaminants may, however, also enter the marine food chain, causing semi-aquatic wildlife to become exposed. This pathway is particularly important when bioaccumulative chemicals such as dibenzodioxins and dibenzofurans are present. Consequently, this section describes the approach for assessing risk to wildlife that inhabit the marine environment in Port Angeles Harbor and may become exposed to sediment-borne contaminants that bioaccumulate and/or biomagnify in marine foodwebs. Therefore, the ecological risk assessment (ERA) for wildlife that use the nearshore marine habitat of Port Angeles Harbor will follow EPA's framework (EPA, 1997a, 1997b) that consists of three phases: problem formulation, analysis, and risk characterization. The EPA framework is closely emulated in Washington State, site-specific, terrestrial ecological evaluation procedures (WAC 173-340-7493), and elements of that regulation will be applied when appropriate.

Problem Formulation

Defining the problem is the first step of an ERA and may be reevaluated throughout the process as more information becomes available. Problem formulation is a formal process for generating and evaluating preliminary hypotheses about why ecological effects have occurred, or may occur, as a result of human activities. The scope and limitations of the assessment are established during problem formulation to maximize the collection of pertinent information within existing resource constraints. The primary components of problem formulation are as follows:

⁴ Bio-sediment accumulation factor (BSAF) is defined as the concentration of a substance in tissue divided by its concentration in sediment. For base-neutral, non-ionic organic compounds, BSAF values are often expressed as the lipid-normalized tissue concentration divided by the organic carbon-normalized sediment concentration.

- Identification of the ecosystems at risk
- Identification of stressor characteristics
- Identification of known effects
- Construction of a CSM
- Selection of assessment endpoints

During the initial phase of the risk assessment, the problem is formulated by presuming potential risk based on the characteristics of known stressors and direct observations of ecological effects in the system. Physical and chemical properties of the stressors (e.g., environmental persistence and water solubility) define potential exposure pathways, temporal and spatial boundaries of the assessment, and ecosystems at risk. Biological properties of the stressors (e.g., toxicity and effect on community structure) are directly relevant to the type of ecological responses that could be expected to occur. The identification of potential stressors, ecological effects, and ecosystems at risk are the key factors required initially to define the nature and extent of the problem. Once identified, these factors lead to the selection of appropriate endpoints for the assessment.

Identification of the Ecosystem at Risk

An ecosystem is composed of biological, physical, and chemical elements that function together in a complex and inter-dependent manner. Ecosystems are dynamic and change with alterations in one or more of their elements. The ecosystem at risk is Port Angeles Harbor, and it is characterized in detail in Section 2.2.6. The marine environment can broadly be classified based on its distance from shore and depth from the surface. Relative to distance from shore, the marine environment is often classified as follows:

- Intertidal (littoral) – The portion of the shoreline that is exposed during the lowest low tides and is covered with water during high tides. Distinctive communities have developed in these regions as a result of this alternation of exposure to air and inundation by seawater (McConnaughy and Zottoli, 1983)
- Subtidal (sublittoral) – The portion of the continental shelf that is permanently covered by water, but is shallow enough to be strongly influenced by wave action and turbulence, at least during storm events (McConnaughy and Zottoli, 1983)

The subtidal marine environment can be classified according to water depth:

- Coastal Pelagic – The water column environment within the neritic zone where the neritic zone is defined as the shallow surface water zone extending from the high-tide mark to the edge of the continental shelf (EPA, 1979)
- Epibenthic – The environment associated with the surface of the sediment sea floor and the water directly above the sea floor (EPA, 1979)
- Benthic – The environment immediately next to the sea floor where organisms occupy the thin sediment layer below the water column (Sumich, 1988)

Aerial photographs of the shoreline along the former Rayonier Mill Site (Ecology's shoreline aerial photos at <http://ecology.ecy.wa.gov/apps/shorephotos>) show intertidal habitat occurring at the mouth of Ennis Creek and along the log pond. Available information suggests that the intertidal zone next to the former Rayonier Mill Site is a high-energy environment comprised of a mixture of coarse sand and cobbles. The subtidal environment next to the former Rayonier Mill Site is relatively shallow (less than 30 feet) with sediments generally characterized as medium-grained (i.e., sandy) with moderate levels of organic matter. Sediments in the log pond have higher organic matter levels due to the presence of coarse woody debris. Coastal drift within the harbor is generally clockwise and easterly. The local water circulation pattern is dominated by a clockwise eddy located between the former Rayonier Mill Site and the tip of the Ediz Hook.

The biotic community of Port Angeles harbor consists of primary producers (marine algae and phytoplankton), primary consumers (e.g., clams, shrimp, and snails), secondary consumers (e.g., crabs and fish), and tertiary consumers (e.g., marine birds and mammals). A relatively diverse and abundant population of marine birds and mammals inhabits the harbor (see Section 2.2.6). The only listed species likely to use Port Angeles Harbor to any significant degree is the bald eagle.

Chemical exposure usually occurs via three routes: ingestion, dermal absorption, and inhalation. The contaminant exposure route of greatest concern for marine wildlife at the former Rayonier Mill Site is ingestion of contaminated surface water, sediment, and prey. Of particular concern is the bioaccumulation of contaminants in invertebrates and fish that are the prey base for most marine wildlife. The dermal absorption route of exposure will not be assessed because it is typically considered insignificant, and appropriate methods to evaluate this route for wildlife species do not currently exist. The inhalation route of exposure will not be assessed, because it is considered to be incomplete or insignificant (e.g., inhalation of volatile chemicals by marine wildlife is incomplete because the volatility

of chemicals associated with the marine environment is low, and any chemical that did volatilize would quickly dissipate in ambient air).

Ecological risk assessments must consider effects to multiple species. However, it is neither possible nor desirable to evaluate the risk posed to every potentially exposed species. Instead, a systematic approach will be used to identify representative marine wildlife species on which to base the ecological risk assessment for the former Rayonier Mill Site. The criteria used to select the representative species are as follows:

- Exposure frequency was evaluated based on the organism's home range and migratory behavior. Species with large home ranges or that are migratory will have less exposure to chemicals at a site than nonmigratory animals with small home ranges.
- Foraging habits were evaluated to determine the pathways by which wildlife would become exposed via the ingestion route of exposure. Species that forage on prey in the sediment will be exposed to contaminants through the incidental ingestion of sediments while foraging, while species that forage in the water column will not be exposed directly to sediment-borne contaminants.
- Feeding guilds were identified for individual species because they help define the pathways by which wildlife become exposed to sediment-borne contaminants. Wildlife that forage on invertebrates that live in the sediment may be exposed to higher concentrations of chemicals because their prey may bioaccumulate higher concentrations of contaminants due to their close contact with the sediments.
- Intake rates of water, sediment, and food were evaluated because they help determine the potential level of exposure. Within similar feeding guilds, smaller species within a feeding guild will tend to have greater exposure to contaminants because they have higher rates of food consumption relative to their body weight per day.

Table 5-3 identifies the receptors and routes of exposure that were selected to assess risks to marine wildlife inhabiting Port Angeles Harbor.

Table 5-3. Ecological Exposure Pathways and Contaminated Media

Location	Receptor	Route of Exposure	Surface Water ^a	Sediments	Sediment Porewater	Aquatic Biota	
Off-Site	Marine - Pelagic Biota	Respiration	●				
		Ingestion	●			●	
		Dermal Absorption	●				
	Marine - Benthic Biota	Respiration				●	
		Ingestion		●		●	●
		Dermal Absorption				●	
	Greater Scaup	Respiration					
		Ingestion		●	●		●
		Dermal Absorption					
	Double Crested Cormorant	Respiration					
		Ingestion		●			●
		Dermal Absorption					
	Black-bellied Plover	Respiration					
		Ingestion		●	●		●
		Dermal Absorption					
	Harbor Seal	Respiration					
		Ingestion					●
		Dermal Absorption					
Otter	Respiration						
	Ingestion		●	●		●	
	Dermal Absorption						

^a Includes indirect exposure via ground water that may be discharged to Ennis Creek or Port Angeles Harbor.

Identification of Stressor Characteristics.

A clear knowledge of the characteristics of a stressor helps to focus the definition of the problem and, thus, the way that it will be evaluated. Identification of potential stressors requires a review of past operations and sampling activities at the former Rayonier Mill Site. Past activities at the former Rayonier Mill Site and previous sampling results are detailed in the 1998 Expanded Site Inspection Report (E&E, 1998), the 1997 Current Situation/Site Conceptual Model Report (Foster Wheeler, 1997), a comparison of sediment chemistry data to the Washington State Sediment Management Standards numeric criteria, and a synopsis of contaminants found in marine biota in Port Angeles Harbor. Several classes of chemical stressors were associated with historical mill operations. They include metals, SVOCs, and dioxins and furans. Several metals (e.g., arsenic and mercury) and SVOCs (PAHs) were detected in sediment at concentrations exceeding Washington State criteria for the protection of aquatic organisms. Concentrations of arsenic, several SVOCs, dioxins and furans, several pesticides, and Aroclor 1260 (a commercial polychlorinated biphenyl [PCB] mixture) detected in red rock crabs and geoducks collected of Port Angeles Harbor in 1998 exceeded EPA human health screening values for consumption of seafood.

Chemicals that bioaccumulate in aquatic biota pose the greatest potential adverse effects on marine wildlife. Section 4.2.4 identified the COPCs in marine biota for Port Angeles Harbor based in large part on their historical occurrence in biota collected from the harbor at levels of potential human health concern. In addition, chemicals detected in sediments from the harbor with a potential to bioaccumulate were also identified as COPCs in marine biota. The COPCs that will be evaluated in the marine wildlife risk assessment include the following:

- Inorganic analytes (arsenic, cadmium, copper, and mercury)
- Dioxins/furans
- SVOCs (pentachlorophenol, pyridine)
- Pesticides/PCBs (BHC, DDD, DDE, DDT, and PCBs)

Chemical stressors can have effects on organisms ranging from biochemical alterations that may have no long-term impact on survival to mortality. If distributed over a wide enough area, they can affect an entire local wildlife population, potentially disrupting the food chain and, thereby, affecting higher trophic level populations. The ecological effects from.

chemical stressors depend on the intensity, frequency, and duration of the stress, as well as the specific properties of the chemicals that have been released. Characteristics of several representative contaminants (i.e., arsenic, mercury, PAHs, and dioxins and furans) detected in Port Angeles Harbor are presented. Metals, SVOCs, and dioxins and furans. Several metals (e.g., arsenic and mercury) and SVOCs (PAHs) were detected in sediment at concentrations exceeding Washington State criteria for the protection of aquatic organisms. Concentrations of arsenic, several SVOCs, dioxins and furans, several pesticides, and Aroclor 1260 (a commercial polychlorinated biphenyl [PCB] mixture) detected in red rock crabs and geoducks collected in Port Angeles Harbor in 1998 exceeded EPA human health screening values for consumption of seafood.

Chemicals that bioaccumulate in aquatic biota pose the greatest potential adverse effects on marine wildlife. Section 4.2.4 identified the COPCs in marine biota for Port Angeles Harbor based in large part on their historical occurrence in biota collected from the harbor at levels of potential human health concern. In addition, chemicals detected in sediments from the harbor with a potential to bioaccumulate were also identified as COPCs in marine biota. The COPCs that will be evaluated in the marine wildlife risk assessment include the following:

- Inorganic analytes (arsenic, cadmium, copper, and mercury)
- Dioxins/furans
- SVOCs (pentachlorophenol, pyridine)
- Pesticides/PCBs (BHC, DDD, DDE, DDT, and PCBs)

Chemical stressors can have effects on organisms ranging from biochemical alterations that may have no long-term impact on survival to mortality. If distributed over a wide enough area, they can affect an entire local wildlife population, potentially disrupting the food chain and, thereby, affecting higher trophic level populations. The ecological effects from chemical stressors depend on the intensity, frequency, and duration of the stress, as well as the specific properties of the chemicals that have been released. Characteristics of several representative contaminants (i.e., arsenic, mercury, PAHs, and dioxins and furans) detected in Port Angeles Harbor are presented.

Arsenic. Many arsenic compounds tend to adsorb to clays, iron oxides, aluminum hydroxides, manganese compounds, sulfides, and organic materials in sediments. Arsenate is one of the more tightly bound forms of arsenic in sediments. The degree of binding depends largely on the chemical concentration sediment characteristics, pH, and ionic

strength of other sediment compounds (Eisler, 2001). Consequently, migration of arsenic from sediments via leaching is not expected to be widespread.

Arsenic accumulates in the marine food chain, but is not usually biomagnified (ATSDR, 1997). In fact, most marine organisms commonly accumulate up to 10 mg organic arsenic/kg-wet weight. Plants are hypothesized to be able to control the amount of arsenic present in their tissues. Arsenic regulatory mechanisms for potential exposure of marine wildlife to arsenic at Port Angeles Harbor is likely to occur through ingestion of sediments and vegetation or prey. Significant exposure to higher marine food chain species is unlikely because of the lack of biomagnification. In animals, arsenic compounds are quickly absorbed through the intestine and then eliminated (Eisler, 2001).

Mercury. The fate of mercury in sediments depends largely on the total organic content and pH of the sediment, as well as on the form of the mercury. Elemental mercury is highly volatile and is, therefore, unlikely to be found in Port Angeles Harbor. Numerous other forms of mercury may be present, however, including organic and inorganic. The species of mercury found depends on the pH, Eh, alkalinity, and other factors (Eisler, 2001). The anionic forms of mercury ($\text{Hg}[\text{OH}]^-$, HgCl_3^- , HgCl_3^{2-}) are most often found in marine environments. In addition, mercury methylation by microbes is likely in aquatic environments. Methylmercury is both very persistent and toxic. Methylation can occur in sediments under aerobic or anerobic conditions, but anerobic conditions are favored (Eisler, 2001).

Mercury compounds are primarily associated with particulates in the sediments (ATSDR, 1997). Adsorption of mercury in sediments decreases with increasing pH and/or chloride ion concentrations. Leaching is a relatively insignificant transport process in sediments. Mercury, once methylated, can then enter and biomagnify within the food chain (Eisler, 2001). The primary transport pathway for mercury in the food chain is through ingestion of prey. Bioconcentration of mercury in fish species has been well documented. Bird species that contain the highest levels of mercury are those that eat fish or other birds. Tissue burdens are usually low in muscle and higher in kidney and liver of wildlife species. Marine mammals have also been shown to have elevated levels of mercury; however, the associated health risks of these accumulations have not been reported.

Polycyclic Aromatic Hydrocarbons. Transport and fate of PAHs in the environment are largely determined by their individual physical and chemical properties. Molecular weight in particular determines the distribution of PAHs in the environment. As the molecular weight of PAHs increases, solubility decreases and $\log K_{ow}$ increases (Eisler, 2001).

Primarily, the fate of PAHs in aquatic environments is to become sorbed to suspended particles or sediments. The degree of sorption depends on the organic carbon content and particle size (ATSDR, 1997). Sorption of PAHs to particulates increases with increasing organic carbon content of the particles and increasing molecular weight of the PAHs. Low and medium molecular weight PAHs are more likely to be transported through sediments by leaching or resuspended into the water column. At low humic acid concentrations (below 0.1 percent), hydrocarbons are adsorbed onto the hydrophobic portions of humic particles (Eisler, 2001). This sorption increases as the humic acid concentration increases. Above humic concentrations of 0.1 percent, solubilization of PAHs into humic acid aggregates sharply increases (Eisler, 2001). This solubility is also pH dependent. At a humic acid concentration of 0.05 percent, higher pH levels favor PAH solubilization (Eisler, 2001). Approximately 33 percent of PAHs do, however, remain dissolved in the water column (Eisler, 2001). These PAHs are expected to degrade rapidly through photo oxidation. Because of specific attributes of the region under examination (i.e., reduced solar radiation and low water temperatures), the rate of photo oxidation may be diminished.

Microbial metabolism is the major degradation process for PAHs in sediments. This biodegradation is affected by environmental inputs, characteristics of the microbial population, and the physical and chemical properties of the PAHs. Environmental inputs that may affect the rate of biodegradation include temperature, pH, dissolved oxygen, PAH concentrations, sediment type, moisture content, nutrients, and other substances that may act as substrate co-metabolites. The size and composition of microbial populations are also affected by these factors. The rate of biodegradation is also altered by the presence of other chemical stressors that may be toxic to biodegrading microorganisms. PAHs can be very persistent under low oxygen or anoxic conditions (Eisler, 2001).

Sorption of PAHs to organic matter and sediment particles influences their bioavailability and, hence, metabolism (ATSDR, 1997). PAHs may accumulate in benthic organisms, fish, and other organisms that consume sediments while feeding, but biomagnification is not expected. Although food-chain transfer of PAHs to higher trophic levels can occur, biomagnification is unlikely due to the high rate of PAH metabolism in fish, mammals, and birds. In general, bioaccumulation is greater for the higher molecular weight compounds than for the lower molecular weight compounds. Unsubstituted PAHs do not accumulate in mammalian adipose tissues, despite their high lipid solubility, because they are quickly metabolized (Eisler, 2001).

Dioxins and Furans. In 1989, ATSDR described dioxin as follows:

The chlorinated dibenzo-p-dioxins are a class of compounds that are loosely referred to as dioxins. There are 73 possible dioxins. The one with four chlorine atoms at positions 2, 3, 7, and 8 of the dibenzo-p-dioxin chemical structure is called 2,3,7,8-tetrachlorinated dibenzo-p-dioxin (2,3,7,8-TCDD). It is a colorless solid with no known odor. It can be inadvertently produced in very small amounts as an impurity during the manufacture of certain herbicides and germicides and has been detected in products of incineration of municipal and industrial wastes.

Dibenzodioxins and furans have also been associated with combustion of natural products, which may occur during forest fires.

Dioxins, especially TCDD, are characterized by extremely low vapor pressures, high log octanol-water coefficients ($\log K_{ow}$), high organic-carbon coefficients (K_{oc}), and extremely low water solubilities. These factors indicate a strong affinity for sediments, particularly sediments with high organic content. TCDD has been shown to biomagnify in marine organisms and associated avian and mammalian species.

Identification of Known Effects

Observed or measured effects on populations of animals at a site are important for problem formulation. Observed mortality or symptoms (e.g., egg shell thinning) can help to identify the source and/or nature of the problem. No written records have been found documenting marine wildlife morbidity or mortality close to the former Rayonier Mill Site.

Construction of a Conceptual Site Model

In order for an ecological risk to exist, there must be a chemical source, a mechanism for the chemical to migrate in the environment, an ecological receptor, and a plausible means for that receptor to be exposed to the chemical. Section 3.3 describes the CSMs for the former Rayonier Mill Site. A summary of the relevant receptor population and exposure pathways is provided in Table 5-4.

For marine wildlife, contaminants emitted from the former Rayonier Mill Site as stack, surface water, and/or groundwater emissions enter the nearshore marine environment where they are either dissolved in the water column, or they become associated with particulate matter that is deposited onto the sea floor as sediment. Contaminants present in the water column and sediments can accumulate in aquatic organisms. The marine wildlife may

become exposed to contaminants primarily through the ingestion of surface water, sediment, and prey.

Table 5-4. Wildlife Receptors for Port Angeles Harbor

Receptor	Route of Exposure	Comment
Greater Scaup	Ingestion of surface water, sediment, and benthic invertebrates	Common migrant that forages throughout the winter, spring, and fall; forages in the subtidal habitat primarily on mollusks that are highly exposed to sediment-borne contaminants; exposed through direct ingestion of sediment while foraging; relatively small body size.
Double-crested Cormorant	Ingestion of surface water and fish	Common throughout the area foraging in the subtidal habitat on fish that bioaccumulate contaminants
Black-bellied Plover	Ingestion of surface water, sediment, and benthic invertebrates	Common throughout the area, foraging on invertebrates in the intertidal environment; exposed through direct ingestion of sediment while foraging; relatively small body size
Harbor Seal	Ingestion of fish	Common throughout the subtidal area foraging on fish that accumulate contaminants
Otter	Ingestion of surface water, sediment, and benthic/epibenthic invertebrates	Uncommon resident in area foraging opportunistically in the subtidal zone on invertebrates that accumulate contaminants; exposure through direct ingestion of sediment while foraging; relatively small home range

Selection of Endpoints

Endpoints define the focus of the ecological risk assessment. Two types of endpoints are delineated for use in ecological risk assessments (Suter et al., 1993; EPA, 1992).

Assessment endpoints symbolize environmental conditions or processes that are valued, but that may not be quantifiable. Measurement endpoints represent quantifiable indicators of the state of the valued conditions or processes. The assessment and measurement endpoints for the Port Angeles Harbor ecological RA for wildlife are shown in Table 5-5.

Analysis

The analysis phase of the ecological risk assessment is composed of two principal activities: the characterization of exposure and the characterization of ecological effects. In exposure characterization, the available data are analyzed to describe the source, the distribution of the stressor in the environment, and the contact or co-occurrence of the stressor with the ecological receptors. In the ecological effect characterization, data are analyzed to describe the relationship between the stressor and response and to evaluate the evidence that exposure to the stressor causes the response (i.e., stressor-response analyses). In many cases, inference is necessary to link the measures of effect with the assessment endpoint.

Table 5-5. Assessment and Measurement Endpoints for the Marine Wildlife ERA

Assessment Endpoint	Measurement Endpoint	Linkage
Survival and reproductive success of marine birds within the foraging range of the nearshore habitat associated with the former Rayonier Mill Site.	Comparison of chemical doses as estimated by exposure modeling for the greater scaup, double-crested cormorant, and black-bellied plover to laboratory dose-response relationships to estimate HQs and HIs of risk.	Elevated HQs and HIs for target species using exposure models may indicate adverse impacts to the survival and/or reproductive success of marine birds in the former Rayonier Mill Site area.
Survival and reproductive success of marine mammals within the foraging range of the nearshore habitat associated with the former Rayonier Mill Site.	Comparison of chemical doses as estimated by exposure modeling for the harbor seal and otter to laboratory dose-response relationships to estimate HQs and HIs of risk.	Elevated HQs and HIs for target species using exposure models may indicate adverse impacts to the survival and/or reproductive success of marine mammals in the former Rayonier Mill Site area.

During the exposure analysis, spatial and temporal distributions of both the receptor and the chemical stressor are evaluated to estimate receptor exposure. Most commonly for wildlife, exposure is modeled by combining measured chemical concentrations in the environment and assumptions about receptor co-occurrence, contact rate, and uptake. When exposure occurs through the food chain, quantitative parameters can be added describing the frequency and magnitude of contact. The daily exposure of a wildlife receptor to a chemical can be expressed as the sum of the amount of chemical consumed during ingestion of food, water, and sediment. Mathematically, chemical ingestion can be expressed as shown in Equation 5.4.2.2.2-1. The variables used in this equation can be categorized as those that define exposure point concentrations and those that define intake rates.

Equation 5.4.2.2.2-1

$$\text{Dose}_{\text{ing}} = \left(\left[\sum (F_p \times I_f \times M_f \times P_x \times F_o) \right] + (S_x \times M_s \times F_s \times I_f \times F_o) + (I_w \times W_x \times F_o) \right) \times (A/HR) \times D_s$$

Where:

$$\begin{aligned} \text{Dose}_{\text{ing}} &= \text{Dose of chemical ingested (mg chemical/kg body weight-day)} \\ F_p &= \text{Fraction of food item p in diet (kg prey species dw/kg diet)} \\ I_f &= \text{Total amount of daily food intake (kg diet/kg BW-d)} \\ M_f &= \text{Wet to dry weight conversion factor for food (mg dry weight/mg wet weight)} \\ P_x &= \text{Concentration of chemical x in food item p (mg chemical/kg prey)} \\ F_o &= \text{Oral absorption efficiency of chemical from medium in gastrointestinal tracts (unitless)} \\ S_x &= \text{Concentration of chemical x in sediment (mg chemical/kg sediment)} \end{aligned}$$

M_s	=	Wet to dry weight conversion factor for sediment (mg dry weight/mg wet weight)
F_s	=	Fraction of sediment in diet (kg sediment/kg diet)
I_w	=	Total amount of daily water intake (liters water/kg BW-d)
W_x	=	Concentration of chemical x in water (mg chemical/liter sediment)
A	=	Area represented by sediment chemical concentration data used to calculate S_x (acres)
HR	=	Home range of receptor being evaluated (acres)
D_s	=	The proportion of the year that a species likely spends in its home range

Marine wildlife receptors foraging in the subtidal environment (i.e., white-winged scoter, double-crested cormorant, harbor seal, and otter) are assumed to become exposed to site-related contaminants when foraging uniformly throughout Port Angeles Harbor. The exposure point concentration of contaminants in sediment (S_x) and surface water (W_x) for these receptors will be estimated using the 95 percent upper confidence limit (95 UCL) of the average concentration. Only sediment data for the surface strata for both subtidal and intertidal locations will be used to calculate wildlife exposure. The marine wildlife that forages in the intertidal environment (i.e., the black-bellied plover) is assumed to be exposed to site-related contaminants when foraging on the intertidal habitat immediately adjacent to the former Rayonier Mill Site. The exposure point concentrations for sediment and surface water for the plover will also be estimated using the 95 percent UCL, and only sediment data for the surface strata for intertidal locations will be used to estimate contaminant exposure for the plover. Analytical data collected for surface water from locations next to the former Rayonier Mill Site will be used to estimate exposure for the plover if available; otherwise, data from the greater Port Angeles Harbor will be used.

The exposure point concentrations for contaminants in food items (P_x) will be estimated using site-specific empirical data and models. The double-crested cormorant and harbor seal consume fish, and the 95 percent UCL of contaminant concentrations, measured in English sole collected from Port Angeles Harbor as part of the remedial investigation, will be used to estimate their exposure. The greater scaup and otter have a diverse diet consisting of mollusks, insects, crustaceans, and plants, depending upon availability. For this assessment, the 95 percent UCL of contaminant concentrations measured in clams and crabs, collected from Port Angeles Harbor as part of the remedial investigation, will be used to estimate their exposure. It will be assumed that half the diet of the scaup and otter will be clams and half crabs (F_p is 0.5 for clams and crabs for the scaup and otter). The analytical data measured in English sole, clams, and crabs collected from Port Angeles Harbor are proposed as sufficient to estimate potential contaminant exposures for the double-crested

cormorant, harbor seal, greater scaup, and otter. Empirical data are, however, available to estimate exposure to the black-bellied plover foraging on benthic invertebrates in the intertidal environment. The concentrations of contaminants in benthic invertebrates in the intertidal environment will be predicted based on BSAFs. The most appropriate BSAFs will be selected from a review of literature and development of chemical-specific models to describe the potential for biotic uptake. Ecology has provided an overview of the type of models to be developed (Department of Ecology, 1995).

Intake parameters for the marine wildlife include food intake rate (I_f), fraction of sediment in diet (F_s), water intake rate (I_w), home range (HR), and proportion of year spent in its home range (D_s). Species-specific parameter values will be obtained from a literature review. If a suitable parameter value cannot be found in the scientific literature, allometric models will be used to estimate the values. Sample et al. (1997) provides allometric models for ingestion of water and food for various classes of animals and foraging guilds. For example, Equation 5.4.2.2.2-2 can be used to estimate the water intake rate for mammals.

Equation 5.4.2.2.2-2

$$I_w = (0.099 (BW)^{0.9})/BW$$

Where:

I_w = surface water ingestion rate (liters water/kilogram body weight/day)

BW = body weight (kilogram wet weight)

In the ecological response analyses, data used in the characterization of ecological effects are assessed to quantify the stressor-response relationship and evaluate the evidence for causality. A stressor response profile will be generated for each contaminant that summarizes their ecological effects. Next, species-specific toxicity reference values (TRVs) will be derived for each contaminant. TRVs are a daily dose (mg chemical/kg BW-d) of a chemical below which adverse effects are not expected to occur. TRVs will be derived using the methodology promulgated in MTCA (WAC 173-340-7490). Toxicity information is often presented as a concentration of chemical in the diet (mg chemical/kg diet). Toxicity data reported as a dietary concentration will be converted to a dose using published food ingestion rates accompanying the specific toxicity study or other published sources describing ingestion rates for test animals of similar size.

Risk Characterization

Risk characterization is the final phase of risk assessment, in which the likelihood of adverse effects occurring as a result of exposure to a contaminant is evaluated. Risk characterization consists of estimating and describing risk. Risk estimation is quantitative and includes a description of the uncertainties associated with the estimates. The risk description summarizes the risk estimates and discusses their ecological significance.

Quantitative risk estimates will be calculated for individual receptors and contaminants using Equation 5.4.2.2.2-3. HQs for individual chemicals that exceed 1.0 indicate that the chemical may pose a potential stress to the receptor.

Equation 5.4.2.2.2-3.

$$HQ = \text{Dose}_{\text{ing}}/\text{TRV}$$

Where:

$$HQ = \text{hazard quotient}$$

$$\text{Dose}_{\text{ing}} = \text{ingested dose of chemical x (mg chemical/kg BW-d)}$$

$$\text{TRV} = \text{toxicity reference value for chemical x (mg chemical/kg BW-d)}$$

Uncertainties are associated with all phases of the risk assessment, and they must be understood to properly interpret the quantitative risk estimates. The uncertainty analysis will describe uncertainties associated with the CSM, information and data used in the assessment, natural variability, and errors.

The risk description will summarize the results of the risk estimate and interpret the ecological significance of those results. The results of the risk estimate, and associated uncertainties will be summarized in a quantitative or qualitative manner. The confidence in the risk estimate will be discussed in terms of the sufficiency and quality of the data, corroborative information, and evidence of causality.

5.5 REMEDIAL INVESTIGATION REPORT

An RI report will be prepared that presents the results of all investigations conducted during the RI. All data will be reported in tabular form, and various map overlays and other plots will be used to present the information. The pertinent features of the RI report will be description of the investigations conducted, summary of the extent of contamination identified, characterization of potential migration pathways, and an RI. The RI report will follow the Ecology guidance.