

CHAPTER 11
EFFECTS OF THE ACTION – INTRODUCTION
TABLE OF CONTENTS

11	Effects of the Action	11-2
11.1	Stressors Associated with the Proposed Action	11-2
11.1.1	Formulated products	11-3
11.1.2	Tank mixtures and environmental mixtures.....	11-4
11.2	Important Habitat Use and Life History Considerations for Anadromous Fish	11-5
11.3	Analyzing Exposure	11-7
11.3.1	Estimating Aquatic Exposure Concentrations Associated with Pesticide Uses.....	11-7
11.3.2	Estimating Terrestrial Exposure Concentrations Associated with Pesticide Uses.....	11-11
11.3.3	Estimating Co-Occurrence Associated with Pesticide Uses	11-12
11.3.4	Mitigation to Minimize or Avoid Exposure.....	11-12
11.3.5	Analyzing Exposure to 1,3-D and chloropicrin	11-12
11.3.6	Analyzing Exposure to Metolachlor	11-20
11.4	Analyzing Responses	11-29
11.4.1	Data Quality Requirements	11-30
11.4.2	Direct Effects	11-31
11.4.3	Indirect Effects.....	11-35
11.4.4	Environmental Factors That Modify Pesticide Toxicity	11-37
11.4.5	Analyzing Response to 1,3-D and its degradates.....	11-39
11.4.6	Analyzing Response to Metolachlor	11-50
11.5	Assessing Risk	11-62
11.6	Weighing the uncertainties in the best commercial and scientific information	11-67

11 EFFECTS OF THE ACTION

Our analysis of the effects of the action to threatened and endangered species includes three primary components which are integrated into the risk analysis: exposure analysis, response analysis, and species life-history considerations.

Section 7 regulations define “effects of the action” as all consequences to listed species or critical habitat that are caused by the proposed action, including the consequences of other activities that are caused by the proposed action.

A consequence is caused by the proposed action if it would not occur but for the proposed action and it is reasonably certain to occur. Effects of the action may occur later in time and may include consequences occurring outside the immediate area involved in the action (Sec § 402.02). This effects analyses section is organized following the stressor, exposure, response, risk assessment framework.

11.1 Stressors Associated with the Proposed Action

For this consultation, the Environmental Protection Agency’s (EPA’s) proposed action encompasses all currently approved product labels containing the active ingredients 1,3-D and metolachlor. This opinion evaluates these separately to avoid the misinterpretation that the analysis is comparing the two herbicides. The potential stressors we expect to result from the proposed action include 1,3-D and metolachlor; other ingredients of these product formulations (including “inert” ingredients and other active ingredients); label recommended tank mixtures (including other pesticide formulations and adjuvants); and toxic metabolites and degradates of product formulation ingredients. We also consider abiotic stressors (e.g. temperature) and aquatic parameters (e.g., water hardness) that influence the response of the species to stressors associated with the proposed action.

Here, we describe our approach to assessing the toxicity of pesticide mixtures containing 1,3-D or metolachlor. Consideration of the toxicity resulting from exposure to pesticide mixtures is an important part of the Effects Analysis of this Opinion. This is due in part to the identified need to consider all effects of the action when making jeopardy determinations and establishing RPAs and RPMs. Pesticide mixtures are explicitly permitted on EPA-authorized product labels, and are therefore part of the action under consultation here. Additionally, monitoring data showing that pesticide mixtures are common in aquatic habitats throughout the United States (Gilliom et al 2007; Bradley et al 2017; Lisa et al. 2018) supports the expectation that ESA-listed species will be exposed to complex pesticide mixtures. Methods of predicting mixture toxicity are widely available and utilize readily available exposure and toxicity data. Finally, failing to consider mixtures may underestimate pesticide risk to such an extent as to lead to erroneous conclusions and ineffective protections for listed species.

11.1.1 Formulated products

Pesticide mixtures can be divided into three categories; formulated products, tank mixes, and environmental mixtures. Formulated products are produced and sold as one product containing multiple active ingredients. Since the exact types and amounts of the active ingredients are shown on the product labels, it is possible to predict the resulting aquatic concentrations following their use. Several formulated products containing 1,3-D and metolachlor have been identified as part of this action and are shown in Tables 3 and 5 of Chapter 5. Tank mixes refer to a situation where the pesticide user applies multiple pesticides simultaneously at the use site. Tank mixes are explicitly allowed on product labels and their use is often encouraged to increase pesticide efficacy. Environmental mixtures result from unrelated pesticide use over the landscape and are typically detected in ambient water quality monitoring efforts. Quantitative and qualitative estimates of risk from mixtures were generated here using current product labels, routine toxicity data, and expected exposure concentrations. These estimates of risk contribute to the overall qualitative mixtures analysis.

Current methodologies for calculating mixture toxicity indicate that additivity is the appropriate initial assumption (Cedergreen and Streibig 2005) unless available data suggest antagonism (less than additive toxicity) or synergism (greater than additive toxicity) is more appropriate. We found no published data showing antagonism or synergism in mixtures containing 1,3-D or metolachlor. Therefore, additive toxicity is the default assumption in this Opinion. Additive toxicity can be calculated by using either dose-additive or response-additive equations, depending on the nature of the pesticides under consideration. For chemicals with similar modes of action (i.e., organophosphate pesticide that inhibit AChE), dose-addition is appropriate. Conversely, response-addition is appropriate for chemicals with dissimilar modes of action. The preponderance of evidence supports this approach and is consistent with the best available scientific information and peer-reviewed publications.

Estimates of additive toxicity utilize two main pieces of information - exposure concentrations and taxa-specific toxicity values. For metolachlor, exposure concentrations were generated using EPA's Pesticide Water Calculator (PWC), which incorporates chemical-specific parameters (e.g., breakdown rates in water and soil) and application-specific parameters (e.g., application method and rate) to calculate anticipated water concentrations over several different averaging durations (e.g. 1-day and 4-day average peak concentrations). For 1,3-D, exposure concentrations were based on extrapolations from a field study assessing run-off (Heim et al., 2002) as recommended by the EPA (2019). Likewise, standard measures of toxicity (typically the LC50, or the concentration that is lethal to 50% of the test organisms) were gathered from various EPA sources for the relevant taxa groups to which NMFS listed species belong. Details regarding exposure and toxicity data can be found below. Calculating toxicity at the taxa level is important, since taxa groups can have vastly different sensitivities to a given pesticide. For example, aquatic invertebrates are more sensitive to organophosphates than are mammals (i.e.,

much lower LC50 values), and therefore will have different estimates of expected risk following exposure to the same mixture concentrations. Calculations of taxa-level toxicity are also useful for representing species for which no species-specific toxicity data are available.

Formulated products containing metolachlor were assessed qualitatively given the variety of additional active ingredients (Chapter 5). A semi-quantitative assessment was determined to be appropriate for 1,3-D given the frequency that it is co-formulated with the active ingredient chloropicrin and chloropicrin's toxicity. Estimates of toxicity were calculated for the formulated products containing 1,3-D that are part of EPA's action under consideration here. All of the formulated products assessed here contain the pesticides chloropicrin and 1,3-D. Since these two chemicals are toxic by different biological mechanisms, response-addition is the appropriate method for calculating mixture toxicity.

Calculations of response-addition of chemicals A and B (i.e., TOXmix), or the sum of the toxic response, were done using the following equation:

$$\text{TOXmix} = 100 * ((\text{mortality A} + \text{mortality B}) - (\text{mortality A} * \text{mortality B}))$$

Where mortality is a function of taxa-specific 48-hr or 96-hr LC50 values, chemical-specific EECs, and the standard probit slope of 4.5 for mortality. A summary of the expected mixture toxicity of a few of the currently-registered formulated products is shown below in Table 1.

Table 1. Predicted mixture toxicity of select formulated products to fish.

Formulated Product	EEC (ug/l)		Single Chemical Toxicity (%mortality)		Mixture Toxicity (%mortality)
	1,3-D	Chloropicrin	1,3-D	Chloropicrin	
Telone® C-35	5.84	3.20	0%	0.8%	0.8%
Pic-Clor 30	11.43	5.03	0%	6.3%	6.3%
Tri-form 40	11.83	7.94	0%	26.3%	26.3%

Predicted mixture toxicity, as measured by the percent of exposed organisms experiencing mortality, ranged from zero to nearly 30%. Nearly all of the expected mortality to fish is caused by exposure to chloropicrin, the other pesticide constituent of all current 1,3-D formulated products. Predicted mixture mortality to aquatic invertebrates is negligible due to that taxa group being less sensitive to both 1,3-D and chloropicrin. Mixture toxicity calculations for all 1,3-D formulated products at all use sites for both taxa groups are shown in Appendix B.

11.1.2 Tank mixtures and environmental mixtures

While pesticide labels explicitly allow, and sometimes even recommend, mixing the product with additional ingredients, including other pesticides, they typically do not define which ingredients to add at the time of application. So, while tank mixtures need to be considered as a

part of the action, unlike formulated products it is not feasible to develop a list of all tank mixtures. Suggested tank mixtures from available product labels for 1,3-D and metolachlor were not summarized in this Opinion. Rather, all tank mixtures are assumed to produce additive toxicity and are described qualitatively. Sources of historical use data are available to provide some information about likely tank mixtures, with the CalDPR database (<http://calpip.cdpr.ca.gov/main.cfm>) being the most extensive. Environmental mixtures are also assumed to produce additive toxicity and are described qualitatively in this Opinion. Consequently, the effects that these other ingredients may have on listed salmonids and designated critical habitat remain an uncertainty and are a recognized data gap in EPA’s action under this consultation. Remaining areas of uncertainty, and recognized data gaps in EPA’s action under this consultation, include the toxic effects of degradates and metabolites, as well as the effects of abiotic stressors such as elevated temperature.

11.2 Important Habitat Use and Life History Considerations for Anadromous Fish

Anadromous fish are born in freshwater and spend a portion of their life cycle in marine habitats. Generalized life history characteristics for listed anadromous fish are described in Table 2.

Table 2. General life histories of anadromous fish

Species (number of listed ESUs or DPSs ¹)	General Life History Descriptions		
	Spawning Migration	Spawning Habitat	Juvenile Rearing and Migration
Chum (2)	Mature adults (usually three to four years old) enter rivers as early as July, with arrival on the spawning grounds occurring from September to January. Chum salmon are semelparous ³	Generally spawn from just above tidewater in the lower reaches of mainstem rivers, tributary stream, or side channels to 100 km upstream.	The alevin life stage primarily resides just below the gravel surface until they approach or reach the fry stage. Immediately after leaving the gravel, swim-up fry migrate downstream to estuarine areas. They reside in estuaries near the shoreline for one or more weeks before migrating for extended distances, usually in a narrow band along the Pacific Ocean’s coast. Preferred prey: fish, invertebrates

Species (number of listed ESUs or DPSs ¹)	General Life History Descriptions		
	Spawning Migration	Spawning Habitat	Juvenile Rearing and Migration
Chinook (9)	<p>Mature adults (usually three to five years old) enter rivers (spring through fall, depending on run). Adults migrate and spawn in river reaches extending from above the tidewater inland hundreds of miles from the Pacific.</p> <p>Migrating adults typically follow the thalweg. Chinook salmon migrate and spawn in four distinct runs (spring, fall, summer, and winter).</p> <p>Chinook salmon are semelparous.</p>	<p>Generally spawn in the middle and upper reaches of main stem rivers and larger tributary streams.</p>	<p>The alevin life stage primarily resides just below the gravel surface until they approach or reach the fry stage.</p> <p>Immediately after leaving the gravel, fry distribute to floodplain habitats that provide refuge from fast currents and predators.</p> <p>Juveniles exhibit two general life history types: Ocean-type fish migrate to sea in their first year, usually within six months of hatching. Ocean-type juveniles may rear in the estuary for extended periods. Stream-type fish migrate to the sea in the spring of their second year. Preferred prey: fish, invertebrates</p>
Coho (4)	<p>Mature adults (usually two to four years old) enter the rivers in the fall. The timing varies depending on location and other variables. Coho salmon are semelparous.</p>	<p>Spawn throughout smaller coastal tributaries, usually penetrating to the upper reaches to spawn. Spawning takes place from October to March.</p>	<p>Following emergence, fry move to shallow areas near stream banks. As fry grow they distribute up and downstream and establish territories in small streams, lakes, and off-channel ponds and other floodplain habitats. Here they rear for 12-18 months. In the spring of their second year juveniles rapidly migrate to sea. Initially, they remain in nearshore waters of the estuary close to the natal stream following downstream migration. Preferred prey: fish, invertebrates</p>
Sockeye (2)	<p>Mature adults (usually four to five years old) begin entering rivers from May to October. Sockeye are semelparous.</p>	<p>Spawn along lakeshores where springs occur and in outlet or inlet streams to lakes.</p>	<p>The alevin life stage primarily resides just below the gravel surface until they approach or reach the fry stage.</p> <p>Immediately after leaving the gravel, swim-up fry migrate to nursery lakes or intermediate feeding areas such as floodplain habitats along the banks of rivers. Populations that migrate directly to nursery lakes typically occupy shallow beach areas of the lake's littoral zone; a few cm in depth. As they grow larger they disperse into deeper habitats. Juveniles usually reside in the lakes for one to three years before migrating to off shore habitats in the ocean. Some are residual, and complete their entire lifecycle in freshwater.</p> <p>Preferred prey: fish, invertebrates</p>

Species (number of listed ESUs or DPSs ¹)	General Life History Descriptions		
	Spawning Migration	Spawning Habitat	Juvenile Rearing and Migration
Steelhead (11)	Mature adults (typically three to five years old) may enter rivers any month of the year, and spawn in late winter or spring. Migrating adults typically follow the thalweg. Steelhead are iteroparous.	Usually spawn in fine gravel in a riffle above a pool.	The alevin life stage primarily resides just below the gravel surface until they approach or reach the fry stage. Immediately after leaving the gravel, swim-up fry usually inhabit shallow water along banks of stream or floodplain habitats on streams margins. Steelhead rear in a wide variety of freshwater habitats, generally for two to three years, but up to six or seven years is possible. They smolt and migrate to sea in the spring. Preferred prey: fish, invertebrates

1 Evolutionarily Significant Unit (ESU), Distinct Population Segment (DPS)

2 spawn only once

3 may spawn more than once

11.3 Analyzing Exposure

In this section we describe the methods used to characterize pesticide exposure to listed species. The procedures rely on models that identify potential interactions of pesticides with listed species and quantify the magnitude of exposure based on how the pesticides and the listed species behave in the environment. We begin with a description of the development of aquatic habitat bins, linking physical characteristics that define aquatic habitats used by listed species with modeling parameters used to predict exposure. Finally, we describe incident reporting for pesticide uses that resulted in effects on non-target species.

11.3.1 Estimating Aquatic Exposure Concentrations Associated with Pesticide Uses

The National Research Council Committee of the National Academy of Sciences recommended that fate and transport models be used to estimate time-varying and space-varying pesticide concentrations in generic habitats relevant to listed species (NRC 2013). Physical characteristics of aquatic habitats, including depth, width, and flow rate affect the environmental concentrations and dissipation patterns of pesticides. A generic habitat defines these physical parameters and uses them to derive Estimated Environmental Concentrations (EECs). The 2-meter deep, static “Farm Pond” that is routinely used by EPA in screening level assessments is an example of a generic habitat. Defining generic habitats to represent all listed species is a challenge given the diversity in the habitats they occupy. Ultimately, the Services identified 10 habitat “bins,” a

number EPA felt could feasibly be evaluated given the scope of the analysis (Table 3)¹. The generic habitats included one aquatic-associated terrestrial habitat, three static freshwater habitats of varying volume, three flowing water habitats of variable volume and flow rates, and three marine/estuarine habitats representative of nearshore tidal, nearshore subtidal, and offshore habitats.

Table 3. Generic aquatic habitats parameters for exposure modeling

Generic Habitat Bins	Depth (meters)	Width (meters)	Length (meters)	Flow (m ³ /second)
1 – Aquatic-associated terrestrial habitats	NA	NA	NA	NA
2- Low-flow	0.1	2	length of field ¹	0.001
3- Moderate-flow	1	8	length of field	1
4- High-flow	2	40	length of field	100
5 – Low-volume	0.1	1	1	0
6- Moderate-volume	1	10	10	0
7- High-volume	2	100	100	0
8- Intertidal nearshore	0.5	50	Length of field	NA
9- Subtidal nearshore	5	200	Length of field	NA

¹length of field – The habitat being evaluated is the reach or segment that abuts or is immediately adjacent to the treated field. The habitat is assumed to run the entire length of the treated area.

The Services identified the bin(s) representative of habitats utilized by each listed species. A single species may occur in a range of habitats represented by multiple bins. The EPA Preliminary Ecological Risk Assessments identify each of the species bin assignments (EPA 2017a; EPA 2017b; EPA 2017c). Bin 1 represents habitats in the terrestrial-aquatic transition zone, such as riparian habitats and rocky shorelines. These habitats are important to water quality and habitat structure and function. In particular, riparian vegetation acts as a buffer trapping pollutants in stormwater runoff and provides shade and allochthonous materials² to aquatic food webs.

Flowing water habitats represented by bins 2, 3, and 4 vary considerably in depth, width, and velocity, which influence both initial concentration and rates of dissipation. These bins are defined by differing flow rates that are products of velocity (influenced by the gradient and other factors) and habitat volume (width and depth). Flow rates vary temporally and spatially in these habitats and are influenced by several factors. For example, bends in the shoreline, shoreline

¹ Interim Approaches for National-Level Pesticide Endangered Species Act Assessments Based on the Recommendations of the National Academy of Sciences April 2013 Report. Available at <https://www.epa.gov/sites/production/files/2015-07/documents/interagency.pdf>

² In ecology, allochthonous material is something from outside an ecosystem that contributes organic matter and nutrients to that ecosystem. For example, leaves and branches from riparian vegetation fuel the invertebrate community which, in turn, feed larger invertebrates and fish.

roughness, and organic debris can create back currents or eddies that can concentrate allochthonous inputs. Dams and other water control structures would also significantly influence flow. Some small streams and channels are intermittent and can become static and temporally cut off from connections with surface water flows during dry seasons. Low flow habitats may also occur on the margins of higher flow systems (e.g. floodplain habitats associated with higher flowing rivers).

Bin 2 is intended to represent habitats with flow rates occurring of 0.001-1 m³/second including springs, seeps, brooks, small streams, and a variety of floodplain habitats (oxbows, side channels, alcoves, etc.) used by salmonids. Pacific salmonids inhabit lower flow habitats in some phase of their lifecycle for activities such as spawning, rearing, or migration. Bin 3 flow rates are representative of small to large streams (1-100 m³/second) and bin 4 definitions (larger volumes and flow rates exceeding 100 m³/second) correspond with larger riverine habitats. These habitats are used by listed salmonids during spawning migrations.

Bins 5, 6, and 7 represent freshwater habitats that are relatively static, where flow is less likely to substantially influence the rate of pesticide dissipation. Examples of bin 5 habitats (volumes <100 m³) include vernal pools, small ponds, floodplain habitats that are cut off from main channel flows, and seasonal wetlands. Salmonid juveniles use a variety of small volume floodplain habitats to forage, over-winter, and shelter from larger predators such as backwater areas and off-channel ponds that are relatively static and may temporarily lose connection to the main stream channel. Bin 6 volumes (100 – 20,000 m³) correspond with many ponds, vernal pools, wetlands, and small shallow lakes and Bin 7 represents larger volume habitats (>20,000 m³) such as lakes, impoundments, and reservoirs. Impoundments are frequently encountered by anadromous fish during spawning migrations of adults and out-migrations of juveniles. Ponds and lakes are also utilized by salmonids for rearing, particularly juvenile sockeye salmon which rear in lakes for one to three years.

Bins 8, 9, and 10 were designed to characterize marine habitats. Marine habitats are generally defined by water depth and distance from shoreline. The nearshore, or neritic zone is the relatively shallow area that extends from the coastlines to the edge of the continental shelf at depths of approximately 200 meters. Nearshore habitats are subdivided into the intertidal zone (Bin 8, the area between shoreline and mean low tide mark), and the subtidal zone (Bin 9, nearshore habitats that extend from the mean low tide mark to the continental shelf and are generally submerged). Bin 10 is intended to represent the deep offshore habitats (>200 meters in depth) that extend beyond the continental shelf. Depths within the intertidal zone are variable between locations but generally range from 0 to <10 meters. Depth within the intertidal habitat depends on the tidal cycle and tidal range. Surface waters can persist during low tides and are used by listed salmonids. Offshore habitats are also used by listed salmonids.

In addition to the above aquatic habitat Bins 2-10, NMFS also estimated pesticide concentrations present in direct runoff from a site following a pesticide application (Bin "0"). This aquatic bin

does not represent a ‘habitat’ where salmon may reside, but does provide useful information regarding the concentration of pesticide entering aquatic habitats. Note that the runoff concentration (Bin 0) does not capture dilution upon entering an aquatic habitat Bin (which would decrease the exposure concentration) or the contribution of drift to an aquatic habitat Bin (which would increase the exposure concentration).

EPA’s PWC (PWC version 1.52, available from <https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/models-pesticide-risk-assessment>) was used to generate aquatic exposure estimates for the different habitat bins for each of the labeled uses. Detailed information on the PWC is available at the above URL. The PWC is an edge-of-field exposure model that estimates the concentration of pesticide in a water body adjacent to a single use site (e.g. a field of crops) resulting from drift and runoff following applications. The PWC incorporates factors that influence exposure concentrations including the pesticide’s physical properties, application rates and methods, precipitation, and soil type. NMFS uses PWC EECs to calculate exposure concentrations that individuals could experience when located immediately adjacent to a use site following an authorized use of a pesticide. PWC EECs do not reflect the contribution to exposure risk due to any additional use to other sites within the range of the species.

The PWC scenarios were chosen from ESA Scenarios developed by EPA for previous assessments (EPA, 2017a) and that were developed for specific regions (Hydrologic Units at the HUC-2 scale). Generic habitat bins (rather than the standard Farm Pond or Reservoir) were used based on the dimensions of the aquatic habitats used by salmon and discussed above. The field length varied with the HUC2 region associated with the PWC Scenario.

Application efficiencies of 0.95 and 0.99 were used for aerial and ground applications (respectively). Application drift values for aerial and ground applications were calculated for each habitat bin using AgDRIFT (2.1.1). Like the PWC, AgDRIFT is a field-scale model in that it estimates the amount of pesticide transported off-site following application to a single use site. NMFS uses AgDRIFT as an additional exposure model to estimate the contribution of spray drift only to water bodies that are not immediately adjacent to a single use site. The model inputs and the estimated deposition rates of 1,3-D and metolachlor are presented in Table 4.

Table 4. Average estimated deposition as a fraction of the application rate (AgDRIFT 2.1.1)

AgDRIFT Simulation (bin range*)	Bin 2 (0-2 m)	Bin 5 (0-1 m)	Bin 6 (0-10 m)	Bin 7 (0-100 m)
Ground Tier 1 ¹	0.2448	0.3833	0.0704	0.0101
Aerial Tier 1 ²	0.4372	0.4686	0.2968	0.0925

*Bin range = distance to near-side and far-side of habitat from treatment area

¹ High Boom, ASAE fine-medium course, 50th percentile distribution

² Fine-Medium Droplet Distribution (EPA default)

Note that these values differ from the standard Farm Pond used by EPA in their Ecological Risk Assessments (EPA 2004). For some PWC inputs, NMFS choose to rely on values described in this Chapter as more representative of the habitats specific to the listed-species considered in this Opinion. These included the drift fractions and applications rates (summary of pesticide labels in Tables 1&2 in Chapter 5). For other PWC inputs, NMFS relied on information provided in the EPA assessments (e.g. application timing and pesticide properties). The PWC inputs specific to 1,3-D and metolachlor are described below.

Estimates for runoff (Bin 0) are not directly available from the output of the PWC. Calculating the runoff concentrations (Bin 0) used the *.zts files generated as part of the PWC runs (i.e. by the PRZM component). The runoff concentration leaving the field can be calculated based on the runoff estimate (RUNF0 column) and the pesticide mass estimate (RFLX1 column).

NMFS did not calculate EECs for the larger flowing water bodies (Bins 3 & 4) or the marine water bodies (Bins 8-10). Adequate exposure models for these water bodies are not currently available. For example, NMFS considers the PWC to be a field-scale model and not appropriate for estimating pesticide concentrations at a watershed scale where multiple application sites will combine to produce an aggregate exposure. NMFS relied on estimates for Bins 0 & 2 as qualitatively representing upper estimates for EECs in Bins 3 & 4. Contributions from other sites within the watershed that did not see applications will serve to reduce these EECs via dilution.

In relying on field scale modeling NMFS did not assume that use will occur to every authorized use site, nor did NMFS assume that all uses are applied at the same day and time. The EECs NMFS derived with exposure modeling do not assume application to more than one site at a time and do not factor in potential increased risk from applications to multiple use sites. Rather than relying on watershed models which require making highly uncertain assumptions regarding the presence/absence and timing of multiple pesticide applications, we relied on field scale models which are intended to generate realistic exposure estimates for treatment to a single use site. The EECs generated represent concentrations that are expected to occur in an aquatic habitat at the edge of the treated field when the pesticide is applied according to product labeling (e.g. application rate specified on label). While they are quantitative in nature, we apply them qualitatively recognizing that they represent only the modeled situation. As discussed in the uncertainty section, use sites receiving lower application rates, or aquatic habitats that are not immediately adjacent to the treated sites are expected to have lower EECs. Ultimately, we look at several lines of evidence (such as the density of use sites within a species range, the proximity of use sites to species habitat, chemical persistence, etc.) to weigh the information for our qualitative determinations.

11.3.2 Estimating Terrestrial Exposure Concentrations Associated with Pesticide Uses

Products containing 1,3-D. Given the application methods (e.g. soil injection) and physical characteristics of the active ingredients in 1,3-D product formulations, the primary exposure

pathways for non-target riparian vegetation are anticipated to include runoff, and aerial transport of vapor-phase. Information from field studies, monitoring data, and modeling efforts from previous risk assessments were used to estimate exposure from these two transport pathways as described later in this document.

Products containing metolachlor. AgDRIFT (Version 2.1.1) was used to generate estimates for pesticide drift deposition in riparian habitats for characterizing potential impacts to riparian plants. Application rates and methods were based on information from the pesticide labels summarized in the Master Use Summary Tables in Chapter 5 (e.g. a label will specify the maximum application rate and approved methods for authorized use). These estimates predict exposure from drift that would be expected in the 10 meters downwind of the target site. Labels do not currently require any buffer to aquatic habitats or riparian zones. The estimates were based on a single application.

Terrplant (Version 1.2.210-29-9009) was used to generate additional estimates for terrestrial exposures in riparian habitats. Inputs included the pesticide solubility in water as well as runoff and drift fractions specified below.

11.3.3 Estimating Co-Occurrence Associated with Pesticide Uses

NMFS evaluated co-occurrence of listed salmonids with the stressors of the actions by comparing the spatial distribution of salmonids with the labeled uses of the two a.i.s. We relied on previous analyses performed by EPA and provided as part of three recent Biological Evaluations (EPA 2017a; EPA 2017b; EPA 2017c). Details of the procedure and rationale are available in sections of the EPA BEs. In brief, use sites described on the pesticide labels (e.g. carrots) were assigned to land use categories. Some use sites were grouped into an aggregate category (e.g. carrots as part of Vegetables and Ground Fruit), while some crops (e.g. corn) were kept as an individual land use category. Geo-spatial information associated with the use sites and the land use categories were primarily based on 2010-2015 data from the National Land Cover Database and the NASS Cropland Data Layer. The use of aggregate land use categories for some use sites accounted for uncertainties associated with the spatial location of pesticide use. Over the 15-year period of the action, cropping patterns for many crops may change due to market demand or crop rotations. Additionally, there is the potential for mis-classification of crops. Relying on broader aggregate land use categories for specific use sites was considered conservative and less likely to undergo significant changes during the 15-year interim.

11.3.4 Mitigation to Minimize or Avoid Exposure

Mitigation has not been proposed beyond the restrictions described in product labeling that would minimize or avoid exposure of ESA-listed species to the potential stressors of the action.

11.3.5 Analyzing Exposure to 1,3-D and chloropicrin

Table 5 shows the extent of overlap for different authorized uses with each species' range. The GIS layers are based on information provided by EPA and used in previous assessments (EPA

2017a; EPA 2017b; EPA 2017c). Since the GIS location information is not specific to a.i., but to land use, it is applicable to 1,3-D applications. Each authorized use was assigned to a GIS layer (Table 6). The overlap data represent upper estimates of the area within a species range where authorized use of 1,3-D could occur. NMFS does not know the actual extent of use that will occur over the 15-years of the action. The uncertainty in the actual extent of use is discussed below and handled qualitatively in the assessment. Also, NMFS recognizes that authorized use sites may only represent a subset of a GIS layer. For example, 1,3-D is authorized for use on “Fruit and Nut Crops” that will be only a subset of the “Orchard and vineyards” and “Vegetables and ground fruit” GIS layers. NMFS does not have a method to refine the location of these authorized uses within these GIS layers. These uncertainties in estimating the overlap between use and species ranges will be addressed in the Risk Characterization section.

Table 5. Percent of an ESU range that overlaps with a GIS Layer associated with 1,3-D uses (mean over 2010-2015).

Species	Corn	Cotton	Soybeans	Wheat	Other Grains	Vegetables	Orchards	Pasture	Nursery	Cultivated
Chum salmon, Columbia River ESU	0.10	0.00	0.00	0.41	0.03	0.16	0.55	9.82	0.06	2.47
Chum salmon, Hood Canal summer-run ESU	0.01	0.00	0.00	0.00	0.01	0.00	0.00	4.17	0.01	0.28
Chinook salmon, California coastal ESU	0.00	0.00	0.00	0.00	0.01	0.00	0.94	9.52	0.00	1.28
Chinook salmon, Central Valley spring-run ESU	2.90	1.08	0.00	2.41	1.22	2.65	14.37	33.52	0.05	41.22
Chinook salmon, Lower Columbia River ESU	0.06	0.00	0.00	0.05	0.02	0.11	0.30	6.04	0.04	1.09
Chinook salmon, Puget Sound ESU	0.44	0.00	0.00	0.05	0.05	0.60	0.01	5.76	0.05	1.80
Chinook salmon, Sacramento River winter-run ESU	2.72	0.03	0.00	1.82	1.43	2.06	8.21	24.65	0.05	39.69
Chinook salmon, Snake River fall-run ESU	0.76	0.00	0.00	6.38	0.44	2.66	1.14	19.31	0.02	17.50
Chinook salmon, Snake River spring/summer run ESU	0.20	0.00	0.00	3.51	0.39	0.99	0.30	14.26	0.01	8.51
Chinook salmon, Upper Columbia River spring-run ESU	0.78	0.00	0.00	2.46	0.14	1.69	2.47	8.99	0.02	12.37
Chinook salmon, Upper Willamette River ESU	0.29	0.00	0.00	1.02	0.11	1.06	0.64	14.16	0.07	6.68
Coho salmon, Central California coast ESU	0.00	0.00	0.00	0.02	0.27	0.02	1.87	12.75	0.04	2.96
Coho salmon, Lower Columbia River ESU	0.06	0.00	0.00	0.05	0.02	0.11	0.30	6.13	0.04	1.10
Coho salmon, Oregon coast ESU	0.02	0.00	0.00	0.01	0.00	0.00	0.01	8.51	0.01	0.08
Coho salmon, S. Oregon and N. Calif coasts ESU	0.00	0.00	0.00	0.03	0.02	0.00	0.00	7.04	0.00	0.85
Sockeye, Ozette Lake ESU	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2.71	0.00	0.00
Sockeye, Snake River ESU	0.66	0.00	0.00	3.70	0.19	1.74	1.00	14.58	0.02	12.26
Steelhead, California Central Valley ESU	2.45	1.20	0.00	2.29	1.22	2.42	12.09	33.56	0.04	36.29
Steelhead, Central California coast ESU	0.00	0.00	0.00	0.12	0.39	0.03	2.45	17.25	0.05	4.30

Species	Corn	Cotton	Soybeans	Wheat	Other Grains	Vegetables	Orchards	Pasture	Nursery	Cultivated
Steelhead, Lower Columbia River ESU	0.06	0.00	0.00	0.05	0.02	0.11	0.31	6.03	0.04	1.14
Steelhead, Middle Columbia River ESU	0.48	0.00	0.00	5.44	0.19	1.10	1.19	6.49	0.01	15.31
Steelhead, Northern California ESU	0.00	0.00	0.00	0.00	0.00	0.00	0.00	8.14	0.00	0.03
Steelhead, Puget Sound ESU	0.45	0.00	0.00	0.05	0.05	0.64	0.01	5.94	0.05	1.87
Steelhead, Snake River Basin ESU	0.20	0.00	0.00	3.51	0.39	0.99	0.30	14.26	0.01	8.51
Steelhead, South-Central California coast ESU	0.06	0.02	0.00	0.17	0.66	0.73	2.76	34.32	0.03	8.11
Steelhead, Southern California ESU	0.00	0.00	0.00	0.12	0.05	0.37	0.47	12.16	0.10	1.54
Steelhead, Upper Columbia River ESU	0.88	0.00	0.00	2.55	0.14	1.78	2.66	9.08	0.02	13.07
Steelhead, Upper Willamette River ESU	0.40	0.00	0.00	1.60	0.24	1.34	1.07	17.45	0.10	10.18

Estimates of Aquatic EECs following Uses of 1,3-D and chloropicrin

NMFS generated aquatic EECs for each authorized use of 1,3-D. Due to its presence in most formulated products, NMFS also generated EECs for chloropicrin that would be expected from use of products containing both pesticides. However, NMFS did not perform, or rely, on exposure models such as the PWC to derive EECs for both 1,3-D and chloropicrin. NMFS agrees with EPA's draft risk assessment for 1,3-D (2019) that those models should not be considered reliable for estimating EECs for highly volatile fumigants such as these pesticides. Instead, NMFS relied on extrapolations from a field study assessing 1,3-D run-off (Heim et al., 2002) as recommended by EPA (2019). Heim et al. (2002) reported maximum 1,3-D concentrations in run-off of 17.2 ppb following an application rate of 327.4 lbs/acre. NMFS considered this to be equivalent to a 1-d bin 0 EEC resulting from that application rate. 1-d bin 0 EECs associated with 1,3-D uses at other application rates were extrapolated from these values (i.e. 17.2 ppb per 327.4 lbs/acre). The maximum application rate for each use and extrapolated 1-d bin 0 EEC are shown in Table 6.

Table 6. Data used in estimating exposures to uses of 1,3-D.

Use Site	GIS Overlap Layers	Maximum Application Rate (lbs a.i./A)	1-d bin 0 EEC (µg/L)
Vegetable Crops	Vegetables and Ground Fruit	580.29	30.48
Field Crops	Corn, Cotton, Other grains, Pasture hay, Soybeans, Wheat	580.29	30.48
Fruit and Nut Crops	Orchards and vineyards, Vegetables and ground fruit	580.29	30.48
Nursery Crops	Nursery	580.29	30.48
Mint	Vegetables and Ground Fruit	295.5	15.52
Idaho potato – USDA Potato Cyst Nematode Eradication Program	Vegetables and Ground Fruit	354.6	18.63
Unspecified cropland in Idaho – certain weed control	Cultivated	246.25	12.94

Use Site	GIS Overlap Layers	Maximum Application Rate (lbs a.i./A)	1-d bin 0 EEC (µg/L)
Unspecified cropland in Oregon – certain weed control	Cultivated	394	20.70
Unspecified cropland in Washington – certain weed control	Cultivated	246.25	12.94

Similar to 1,3-D, NMFS did not rely on modeled estimates for chloropicrin EECs. NMFS is unaware of any equivalent field study for chloropicrin. A comparison of the physical/chemical and environmental fate properties of chloropicrin to those of 1,3-D found them to be sufficiently similar for the results of the 1,3-D study (Heim, 2002) to be a reasonable surrogate for chloropicrin run-off estimates (Attachment A). Therefore, NMFS applied the same extrapolation used for 1,3-D (17.2/327.4) to chloropicrin application rates to generate chloropicrin EECs. This approach makes the assumption that the relationship between application rate and runoff concentration is the same for both compounds. NMFS recognizes the uncertainties associated with this assumption and was careful to consider these uncertainties when making risk characterizations. In general, we anticipate that chloropicrin concentrations will be no greater than those estimated for 1,3-D with similar application rates. The maximum application rate for chloropicrin across all uses was 350.2 lbs/acre (Chapter 5) leading to a maximum 1-d bin 0 EEC for all uses of 18.4 ppb.

NMFS used the EECs extrapolated from the field study (Heim, 2002) to represent direct run-off at the edge of a field (i.e. 1-d bin 0 EECs). As mentioned above, NMFS does not consider bin 0 to be representative of an aquatic habitat but of the run-off contribution to aquatic habitats (e.g. bin 2). Given the application methods employed for 1,3-D (e.g. injection rather spray), NMFS does not consider that drift will contribute to an increase in exposures to aquatic habitats. However, NMFS does expect that pesticide concentrations will decrease with factors such as time, dilution, and degradation (i.e. for the same application the 4-d bin 2 EEC will be less than the 1-d bin 0 EEC). To estimate reduction factors that could be applied to the initial 1-d bin 0 EEC to estimate other EECs NMFS did use the PWC. A small set of PWC runs were done specifically to compare the 1-d bin 0 EECs to other EECs from the same application. NMFS recognizes the uncertainty introduced by this approach. The focus, however, is on the impact of aquatic dilution and degradation processes on EECs for which the model may be more reliable for 1,3-D and chloropicrin. Details of the PWC runs and calculations are in Appendix C The resulting reduction factors are shown in Table 7 and can be seen in the Risk Characterization (e.g. in the Risk Plots).

Table 7. Reduction Factors for converting 1-day bin 0 EECs

	Time-weighted-average		
	1-day	4-day	21-day
Bin 0	1.000	0.296	0.070
Bin 2	0.435	0.142	0.035
Bin 7	0.044	0.037	0.017

Analyzing terrestrial exposure to 1,3-D and chloropicrin

For reasons mentioned earlier, and described in more detail by EPA's Problem Formulation (2013) and Draft Risk Assessment (2019), NMFS did not generate modeled exposures for terrestrial riparian vegetation using AgDRIFT or Terrplant. Nonetheless, exposure to riparian terrestrial vegetation is considered from both run-off and vapor-phase transport routes.

Numerous sources of information are available to characterize the range of expected 1,3-D and chloropicrin concentrations in the vapor phase. For 1,3-D, the highest air concentrations (841 mg/m³, derived from a field study) are substantially less than concentrations at which no adverse effects were observed in the available vegetative vigor study (MRID 50883601). The vegetative vigor study investigated the potential adverse effects to ten different terrestrial plant species from a four hour vapor exposure of 1,3-D. Although some adverse effects were observed, the EC₂₅, EC₅₀, and NOEC values were all greater than the highest concentrations tested which ranged from 250ppm to 528ppm. For chloropicrin, the highest air concentrations available are those described by exposure models, in particular the ISCST3 model as described in EPA's 2008 Reregistration Eligibility Decision document. These concentrations are comparable to seedling emergence and vegetative vigor EC₂₅ values for terrestrial plants which range from 0.0021 mg/L to >0.068 mg/L. Other available exposure estimates, including monitoring data and refined exposure models suggest environmental exposures substantially lower than those generated with the ISCST3 model. Below are high level summaries of available data from field studies, ambient monitoring as well as modeled concentrations.

EPA considered a number of different approaches to modeling vapor exposure in the problem formulation (EPA, 2013). The 2019 DRA, however, concluded that available field studies are considered the best available information to estimate this exposure. According to the DRA, the highest potential exposure reported in these studies is 4.556mg/m³ based on an application rate of 51 lbs. a.i./acre (MRID 45222501). Note, however, the application method associated with this vapor concentration (shallow application to turf) is not necessarily representative of agricultural applications. Another field volatility study (MRID 42545101) found 1,3-D concentrations at 15cm above the soil to be 0.533mg/m³. This concentration was associated with an 18-inch injection (more typical of agricultural applications) of 121 lb ai/acre. Another study examined air

concentrations following an application of 5.12 gallons/acre at a depth of 5 inches on a golf course. Air concentrations were measured on the site of application as well as 100 and 300 feet off site. The average concentration detected on site, 100 feet, and 300 feet off site were 30.9, 1.9, and 3.3 ug/m³ respectively (Barnekow et al. 1999).

The ambient air monitoring effort by California Department of Pesticide Regulation (CDPR, 2018) on 1,3- D shows highest 1-day concentrations of 5.0 ppb (6 µg/m³) in Santa Maria, 2.8 ppb (3.4 µg/m³) in Watsonville, 8.7 ppb (10 µg/m³) in Oxnard, and 3.1 ppb (3.7 µg/m³) in Camarillo. For chloropicrin, the highest 1-day concentration was 1.1 ppb (0.16 ug/m³) in Santa Maria and 1.0 ppb (0.15 ug/m³) in Watsonville. In 2011, CDPR implemented an Air Monitoring Network (AMN) to weekly measure 32 pesticides, including 1,3-D and chloropicrin, in three agricultural communities: Ripon, Salinas, and Shafter. The highest 24-hour and 4-week exposure measurements were 45 µg/m³ and 18 µg/m³ for 1,3-D and 6.38 µg/m³ and 3.02 µg/m³ for chloropicrin (EPA, 2019).

EPA's 2019 draft human health risk assessment for the registration review of 1,3-D includes modeled ambient air concentrations which were generated using the Soil Fumigant Exposure Assessment (SOFEA) modeling system. The maximum 24-hour concentrations estimated for the Pacific Northwest region over the time periods modeled were 0.105 and 0.089 ppm (473 and 401 ug/m³ based on the conversion factor provided in the human health assessment). For chloropicrin, the 2008 RED included modeled concentrations based on the Industrial Source Complex Short Term version 3 (ISCST3) model as well as PERFUM. The highest concentrations estimated with these models were 19 mg/m³ and 0.004219 mg/m³ respectively.

Exposure of 1,3-D and chloropicrin to terrestrial non-target plants is also possible via surface runoff and subsurface flow. The 2019 DRA for 1,3-D identifies a run-off field study (Heim et al. 2002) as the best currently available information on run-off concentrations given the limitations in existing models. The maximum concentration detected in the field study was 17.2 ppb. Although the application rates in the field study do not represent the highest allowed, extrapolations based on maximum application rates authorized by product labeling suggest that run-off concentrations would be unlikely to exceed around 50 ppb. EECs generated using the Pesticide in Water Calculator ranged in the tens to hundreds of ppb, depending on the application scenario. For chloropicrin, maximum aquatic EECs calculated using PRZM/EXAMS in previous assessments (USEPA 2007c and 2009a) were 79, 19, and 6.8 µg/L for peak, 21-day average, and 60-day average, respectively. Aquatic EECs for chloropicrin are anticipated to be similar to those of 1,3-D, given similar application rates and methods.

11.3.6 Analyzing Exposure to Metolachlor

Table 8 shows the extent of overlap for different authorized uses of metolachlor with each species' range. The GIS layers are based on information provided by EPA and used in previous assessments (EPA 2017a; EPA 2017b; EPA 2017c). Since the GIS location information is not specific to a.i., but to land use, it is applicable to metolachlor applications. Each authorized use was assigned to a GIS layer (Table 10). The overlap data represent upper estimates of the area within a species range where authorized use of metolachlor could occur. NMFS does not know the actual extent of use that will occur over the 15-years of the action. The uncertainty in the actual extent of use is discussed below and handled qualitatively in the assessment. Also, NMFS recognizes that authorized use sites may only represent a subset of a GIS layer. For example, while metolachlor is authorized for use on a number of vegetables, they still represent a subset of all possible "Vegetables and ground fruit" within the GIS layer. Also, use on alfalfa in Oregon will occur on only a portion of "Pasture" land. For this use site, additional information from the NASS was used to inform the overlap. This uncertainty in estimating the overlap between use and species ranges will be considered in the Risk Characterization section of this Opinion.

Table 8. Percent of an ESU range that overlaps with GIS Layers associated with metolachlor uses (mean percent over 2010-2016).

Species	Corn	Cotton	Soybeans	Vegetables	Other Grains	Other Row Crops	Other Crops	Pasture	Nursery
Chum salmon, Columbia River ESU	0.10	0.00	0.00	0.16	0.03	0.00	0.52	9.82	0.06
Chum salmon, Hood Canal summer-run ESU	0.01	0.00	0.00	0.00	0.01	0.00	0.00	4.17	0.01
Chinook salmon, California coastal ESU	0.00	0.00	0.00	0.00	0.01	0.00	0.00	9.52	0.00
Chinook salmon, Central Valley spring-run ESU	2.90	1.08	0.00	2.65	1.22	0.31	5.42	33.52	0.05
Chinook salmon, Lower Columbia River ESU	0.06	0.00	0.00	0.11	0.02	0.00	0.12	6.04	0.04
Chinook salmon, Puget Sound ESU	0.44	0.00	0.00	0.60	0.05	0.01	0.10	5.76	0.05
Chinook salmon, Sacramento River winter-run ESU	2.72	0.03	0.00	2.06	1.43	0.95	7.65	24.65	0.05
Chinook salmon, Snake River fall-run ESU	0.76	0.00	0.00	2.66	0.44	0.01	3.55	19.31	0.02
Chinook salmon, Snake River spring/summer run ESU	0.20	0.00	0.00	0.99	0.39	0.02	1.52	14.26	0.01
Chinook salmon, Upper Columbia River spring-run ESU	0.78	0.00	0.00	1.69	0.14	0.01	2.21	8.99	0.02
Chinook salmon, Upper Willamette River ESU	0.29	0.00	0.00	1.06	0.11	0.08	6.43	14.16	0.07
Coho salmon, Central California coast ESU	0.00	0.00	0.00	0.02	0.27	0.00	0.08	12.75	0.04
Coho salmon, Lower Columbia River ESU	0.06	0.00	0.00	0.11	0.02	0.00	0.12	6.13	0.04
Coho salmon, Oregon coast ESU	0.02	0.00	0.00	0.00	0.00	0.00	0.03	8.51	0.01
Coho salmon, S. Oregon and N. California coasts ESU	0.00	0.00	0.00	0.00	0.02	0.00	0.11	7.04	0.00
Sockeye, Ozette Lake ESU	0.00	0.00	0.00	0.00	0.00	0.00	0.00	2.71	0.00
Sockeye, Snake River ESU	0.66	0.00	0.00	1.74	0.19	0.00	2.77	14.58	0.02
Steelhead, California Central Valley ESU	2.45	1.20	0.00	2.42	1.22	0.27	5.13	33.56	0.04

Steelhead, Central California coast ESU	0.00	0.00	0.00	0.03	0.39	0.00	0.22	17.25	0.05
Steelhead, Lower Columbia River ESU	0.06	0.00	0.00	0.11	0.02	0.00	0.12	6.03	0.04
Steelhead, Middle Columbia River ESU	0.48	0.00	0.00	1.10	0.19	0.12	4.35	6.49	0.01
Steelhead, Northern California ESU	0.00	0.00	0.00	0.00	0.00	0.00	0.00	8.14	0.00
Steelhead, Puget Sound ESU	0.45	0.00	0.00	0.64	0.05	0.01	0.10	5.94	0.05
Steelhead, Snake River Basin ESU	0.20	0.00	0.00	0.99	0.39	0.02	1.52	14.26	0.01
Steelhead, South-Central California coast ESU	0.06	0.02	0.00	0.73	0.66	0.00	1.30	34.32	0.03
Steelhead, Southern California ESU	0.00	0.00	0.00	0.37	0.05	0.00	0.10	12.16	0.10
Steelhead, Upper Columbia River ESU	0.88	0.00	0.00	1.78	0.14	0.01	2.23	9.08	0.02
Steelhead, Upper Willamette River ESU	0.40	0.00	0.00	1.34	0.24	0.10	8.35	17.45	0.10

Estimates of Aquatic EECs following Uses of Metolachlor

NMFS generated aquatic EECs for each authorized use of metolachlor using the PWC. Exposure modeling focused on racemic metolachlor as the applied chemical. While several formulated products consist of a mixture of racemic metolachlor and S-metolachlor, EECs were not generated for S-metolachlor since the chemical properties are similar to racemic metolachlor (EPA 2014). Any differences in EECs were considered likely to be minor. The chemical inputs for the PWC runs for metolachlor are shown in Table 9. Application information for the PWC runs are summarized in Table 10. Application rates are based on maximum rates allowed by the labels. Application timing information is based on information from EPA (2014). Efficiency and drift inputs were summarized earlier (Table 4). The PWC runs for metolachlor were performed using external batch files (Appendix E). The EECs generated by NMFS for metolachlor are displayed in the Risk Characterization and are also in Appendix E.

Table 9. Chemical Inputs Parameters for PWC runs.

Physical / Chemical Property	Metolachlor
Sorption Coefficient(mL/g)	132.4
Koc flag	TRUE
Water Column Metabolism Halflife (days)	39.7
Water Reference Temperature (°C)	25
Benthic Metabolism Halflife (days)	234
Benthic Reference Temperature (°C)	25
Aqueous Photolysis Halflife (days)	70
Photolysis Reference Latitude (°)	40
Hydrolysis Halflife (days)	0
Soil Halflife (days)	98.4
Soil Reference Temperature (°C)	25
Foliar Halflife (days)	0
Molecular Weight (g/mol)	283.8
Vapor Pressure (torr)	2.78E-05
Solubility (mg/L)	530

Table 10. Inputs used in estimating exposures to uses of Metolachlor.

Use Site	PWC Scenarios	GIS Overlap Layer	Application Rate(s) (kgs a.i./A)	Application Date(s) (Relative)	Application Efficiency/Drift
Beans and other pod crops	VegetableESA17a.scn VegetableESA17b.scn VegetableESA18a.scn VegetableESA18b.scn	Vegetables and Ground Fruit	2.19	-7	Ground (0.99) Air (0.95)
			1.1	3	
Horseradish; Rhubarb			1.43	-7	Ground (0.99) Air (0.95)
Potato			3.01 1.05	-7 3	Ground (0.99) Air (0.95)
Pumpkin			1.43	-7	Ground (0.99)
Tomato			2.23 2.23 2.23	-24 96 216	Ground (0.99)
Corn	CornESA17a.scn CornESA17b.scn CornESA18a.scn CornESA18b.scn	Corn	2.99 1.34	-7 3	Ground (0.99) Air (0.95)
Safflower	OtherGrainESA17a.scn OtherGrainESA17b.scn OtherGrainESA18a.scn OtherGrainESA18b.scn	Other Grains	2.15	-7	Ground (0.99) Air (0.95)
Sorghum			1.87	-7	
Soybean	SoybeanESA17a.scn SoybeanESA17b.scn SoybeanESA18a.scn SoybeanESA18b.scn	Soybean	3.08	-7	Ground (0.99) Air (0.95)
Sugarbeet	OtherRowESA17a.scn OtherRowESA17b.scn OtherRowESA18a.scn OtherRowESA18b.scn	Other Row Crops	1.78 1	-14 47	Ground (0.99) Air (0.95)
Sunflower			2.14	-7	
Turf – commercial, residential, sod farms	OtherCropESA17a.scn OtherCropESA17b.scn OtherCropESA18a.scn OtherCropESA18b.scn	Other Crops	2.78 1.7	7 49	Ground (0.99) Air (0.95)
Nursery and landscape plantings	NSLandcoverESA17a.scn NSLandcoverESA17b.scn NSLandcoverESA18a.scn NSLandcoverESA18b.scn	Nursery	2.78 1.71	-7 3	Ground (0.99) Air (0.95)
California Only: Swiss chard; Subgroup 1-B (beet, carrot, turnip, etc.) and 1-C (artichoke, ginger, yam, etc.)	VegetableESA18a.scn VegetableESA18b.scn	Vegetables and Ground Fruit	1.43	-7	Ground (0.99)
California Only: Pepper; Seeded and transplanted tomato			1.79	-7	

Use Site	PWC Scenarios	GIS Overlap Layer	Application Rate(s) (kgs a.i./A)	Application Date(s) (Relative)	Application Efficiency/Drift
California Only: Spinach			1.07	-7	
California Only: Dry bulb onion			1.43 1.43	-14 8	
California Only: Celery			1.43 0.71	-7 3	
California Only: Cotton	CottonESA18a.scn CottonESA18b.scn	Cotton	1.49 1.49 1.49	-24 -7 7	Air (0.95)
Idaho Only: Carrot, collard, radish, beet, kale, mustard, parsnip, rutabaga, turnip	VegetableESA17a.scn VegetableESA17b.scn	Vegetables and Ground Fruit	0.72	-7	Ground (0.99)
Idaho Only: Pepper			1.79	-7	
Idaho Only: Dry bulb onion			1.43 1.43	-14 8	
Oregon Only: Seed crops including radish, spinach, beets, and Swiss chard; blueberry, blackberry, and raspberry; Sweet potato	VegetableESA17a.scn VegetableESA17b.scn	Vegetables and Ground Fruit	1.43	-7	Ground (0.99)
Oregon Only: Transplanted bell pepper			1.79	-7	
Oregon Only: Strawberry			1.06	-7	
Oregon Only: Alfalfa for seed	GrasslandESA17a.scn GrasslandESA17b.scn	Pasture	3.56	-7	Ground (0.99)

Estimates of Terrestrial EECs following Uses of Metolachlor

AgDRIFT (version 2.1.1) was used to generate estimates for pesticide drift deposition in riparian habitats for characterization of potential impacts to riparian plants and invertebrates. Application rates and methods were based on information summarized in the Master Use Summary Table in Chapter 5. These estimates predict exposure from drift that is expected to occur in the 10 meters downwind of the target site. Labels do not currently require any buffer to aquatic habitats or riparian zones. The estimates were based on a single application. Drift estimates for ground applications assumed a high boom, ASAE fine-medium course droplet size. The Estimated

Environmental Concentrations (EECs) provided in Table 1 below represent a 50th percentile distribution. Aerial estimates assumed the EPA default, fine-medium droplet size distribution. These assumptions predict an average drift deposition fraction of 0.0704 and 0.2968 for ground and aerial applications when the wind is blowing 10 miles per hour. Additional terrestrial EECs were generated using EPA's Terrplant model (version 1.2.210-29-9009). Inputs included the solubility of metolachlor (530 mg/L) as well as runoff and drift fractions (0.05 and 0.01, respectively). Table 11 presents the resulting terrestrial EECs.

Table 11. Estimated drift deposition onto riparian habitat adjacent to field following application of metolachlor.

Use Site	Maximum Single Application Rate (lbs a.i./A)	AgDRIFT EECs (lbs a.i./A)		Terrplant EECs (lbs a.i./A)			
		Ground	Aerial	Ground		Aerial	
				Dry	Semi-aquatic	Dry	Semi-aquatic
Beans and other pod crops	1.95	0.137	0.579	0.117	0.9945	0.195	1.0725
Corn	2.67	0.188	0.792	0.1602	1.3617	0.267	1.4685
California Cotton	1.33	0.094	0.395	0.0798	0.6783	0.133	0.7315
Horseradish	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
Potato	2.68	0.189	0.795	0.1608	1.3668	0.268	1.474
Pumpkin	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
Rhubarb	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
Safflower	1.91	0.134	0.567	0.1146	0.9741	0.191	1.0505
Sorghum	1.67	0.118	0.496	0.1002	0.8517	0.167	0.9185
Soybean ^c	2.74	0.193	0.813	0.1644	1.3974	0.274	1.507
Sugarbeets	1.59	0.112	0.472	0.0954	0.8109	0.159	0.8745
Sunflower	1.91	0.134	0.567	0.1146	0.9741	0.191	1.0505
Tomato	1.99	0.140	0.591	0.1194	1.0149	0.199	1.0945
Turf – commercial, residential, sod farms	2.48	0.175	0.736	0.1488	1.2648	0.248	1.364
Nursery and landscape plantings	2.47	0.174	0.733	0.1482	1.2597	0.247	1.3585
California - Pepper	1.59	0.112	0.472	0.0954	0.8109	0.159	0.8745
California - Seeded and transplanted tomato	1.59	0.112	0.472	0.0954	0.8109	0.159	0.8745

Use Site	Maximum Single Application Rate (lbs a.i./A)	AgDRIFT EECs (lbs a.i./A)		Terrplant EECs (lbs a.i./A)			
		Ground	Aerial	Ground		Aerial	
				Dry	Semi-aquatic	Dry	Semi-aquatic
California - Swiss chard	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
California - Spinach	0.95	0.067	0.282	0.057	0.4845	0.095	0.5225
California - Dry bulb onion	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
California - Celery	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
California - Subgroup 1-B (beet, carrot, turnip, etc.) and 1-C (artichoke, ginger, yam, etc.)	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
Idaho - Carrot, collard, radish, beet, kale, mustard, parsnip, rutabaga, turnip	0.64	0.045	0.190	0.0384	0.3264	0.064	0.352
Idaho - Pepper	1.59	0.112	0.472	0.0954	0.8109	0.159	0.8745
Idaho - Dry bulb onion	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
Oregon - Alfalfa for seed	3.17	0.223	0.941	0.1902	1.6167	0.317	1.7435
Oregon - Seed crops including radish, spinach,	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985

Use Site	Maximum Single Application Rate (lbs a.i./A)	AgDRIFT EECs (lbs a.i./A)		Terrplant EECs (lbs a.i./A)			
		Ground	Aerial	Ground		Aerial	
				Dry	Semi-aquatic	Dry	Semi-aquatic
beets, and Swiss chard							
Oregon – Transplanted bell pepper	1.59	0.112	0.472	0.0954	0.8109	0.159	0.8745
Oregon – blueberry, blackberry, and raspberry	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
Oregon – Sweet potato	1.27	0.089	0.377	0.0762	0.6477	0.127	0.6985
Oregon - Strawberry	0.95	0.067	0.282	0.057	0.4845	0.095	0.5225

11.4 Analyzing Responses

The response analysis of this opinion evaluates toxicity information from the stressors of the action and organizes them into assessment endpoints which target potential effects to individual salmonids and their supporting habitats. The assessment endpoints represent biological and habitat attributes that, when adversely affected, lead to reduced fitness of individual salmonids or degrade the Physical and Biological Features (PBFs) essential to the conservation of the species. For the reasons described in the following sections, we determine that in total the toxicity information included in this summary provides the best available scientific information for quantitative concentrations that would trigger a response. We place higher weight on those studies that are well-designed, more relevant to our species and habitat, and conducted with stressors of the action. Uncertainties in the available toxicity information are discussed as they are encountered and identified at the end of this section. Following the response analysis, the risk analysis compares anticipated environmental concentrations described in the exposure analysis with assessment endpoints to evaluate whether individual fitness or habitat endpoints might be compromised. Salmonid and designated critical habitat risk hypotheses are evaluated separately in the *Effects of the Proposed Action on Designated Critical Habitat Section*.

The EPA provided three documents to support NMFS' evaluation of 1,3-D: *1,3-Dichloropropene Analysis of Risks to Endangered and Threatened Salmon and Steelhead (1,3-D Biological*

Evaluation), *Draft Risk Assessment (DRA) in Support of Registration Review* (1,3-D Draft Risk Assessment), and *Problem Formulation for the Environmental Fate, Ecological Risk, Endangered Species, and Drinking Water Exposure Assessments in Support of the Registration Review of 1,3-Dichloropropene (Telone)* (1,3-D Problem Formulation). Collectively, this section calls these three documents “the 1,3-D risk analyses.” Three documents were also provided for metolachlor: *Risks of Metolachlor Use to 26 Evolutionarily Significant Units of Endangered and Threatened Pacific Salmon and Steelhead* (Metolachlor Biological Evaluation), *Draft Ecological Risk Assessment for the Registration Review of Metolachlor/(S)-Metolachlor* (Metolachlor Draft Risk Assessment), and *Registration Review Problem Formulation for Metolachlor and S-Metolachlor* (Metolachlor Problem Formulation). These are collectively referred to as the “Metolachlor Risk Analyses” in this section. We relied on the information in these assessments and supplemented with data from the ECOTOX and EPA OPP’s Pesticide Ecotoxicity Database, the open literature, and information provided by the applicant.³ The OPP database includes the MRID submissions reviewed by EPA in conjunction with pesticide registrations or reregistrations that have been evaluated by EPA biologists and judged acceptable for use as core or supplemental data to support an ecological assessment. Here we describe the types of data that reflect effects that can influence the persistence of populations exposed to environmental toxicants and factors that affect the toxicity and vulnerability of salmonids to pesticides.

11.4.1 Data Quality Requirements

The ESA mandates the use of the best available scientific and commercial data when determining the effects of pesticides on threatened and endangered species. The following paragraphs describe NMFS’ data quality acquisition and review process for the information used in in this assessment. Sources of information include ecological effects data for pesticides provided by the registrants as part of the 40 CFR Part 158 guideline requirements, compiled in EPA databases, and found through searches of the open literature. For most pesticides, a substantial amount of ecological effects data are identified through using the ECOTOX as its search engine to access relevant data compiled from scientific journals, books, government reports, and theses and dissertations.

Data acceptable for inclusion into the ECOTOX must be from an English-language primary data source reporting measurable adverse responses occurring concurrently with exposures of ecologically relevant and taxonomically verifiable species to ambient concentrations, doses, or application rates over a discrete exposure duration. The ECOTOX reports these exposures in standardized environmentally relevant units of exposure intensity (i.e., mg active ingredient per liter for aquatic organisms) and exposure duration in days. NMFS also applies the additional data

³ NMFS accessed the most recent version of Pesticide Ecotoxicity Database. The database is a preliminary copy presently under development. The data continues to receive additional quality assurance checks. NMFS reports these data with this consideration in mind. Overall EPA asserts that the majority of data accurately reflects the Agency data evaluation reports for these studies. EPA OPP is expected to review and make any additional corrections to the data reported in this opinion from this database prior to finalization of the opinion.

acceptability requirements required by OPP: the entire article must be a publically available document published in English, the information must be presented as a full article, treatments must be compared to an acceptable control, and the paper must clearly indicate whether the exposure occurred in the laboratory or field. Failure of data acceptability criteria means the data cannot be used in a quantitative assessment, it does not mean the data cannot inform the assessment in some other way. For example, exposures that are not expressed in environmentally relevant exposure units can still be used to inform the Effects Characterization.

A second tier of review may be applied to ECOTOX data, depending on how a study will be used in the assessment:

- Studies establishing an effects threshold concentration above which mortality or sublethal effects occur.
- Studies providing data used to assemble a species sensitivity distribution (SSD), with particular emphasis on studies providing influential data for the distribution (i.e., values near the 5th and 95th percentiles and the median).
- Studies that represent the most sensitive response thresholds for assessment endpoints (e.g., reproduction, behavior, or sensory effects).
- Other studies in the arrays that contain data influential in describing how a species may be affected by the registration of the pesticide.

Searches of the open literature are necessary to supplement data acquired through the ECOTOX for a number of reasons. The ECOTOX attempts to be comprehensive, but searches for content to populate the database do not locate all relevant literature and, once content is identified, it can take up to six months or more for it to be acquired and encoded into ECOTOX. Data included in ECOTOX are limited to single chemical exposures of substances with verifiable chemical abstract numbers. This means information on mixtures like pesticide products and tank mixes need to be identified through the open literature. The ECOTOX content identifies primarily adverse biological effects in live, whole organisms, so information describing mechanisms of effect at sub-organism levels or from in-vitro tests also need to be identified through open literature searches.

11.4.2 Direct Effects

Direct effects on survival resulting from exposure to pesticides that are deposited in surface waters through runoff and drift transport pathways are described by dose-response data from laboratory toxicity studies with results reported as median lethal concentrations (LC50s), median lethal doses (LD50s), slopes of dose response curves, and species sensitivity distributions (SSDs) showing variability in lethal responses among tested species. Effects on other responses affecting population persistence are described as statistically significant thresholds obtained from dose-response data with results reported as the Lowest Observed Effect Concentration (LOEC) and

No Observed Effect Concentration (NOEC) tested in the study along with and the magnitude of effects observed at these thresholds. These responses include, but are not limited to:

- reproduction (e.g., percent hatch, egg viability),
- impaired growth that could increase individual mortality (e.g., predation risk and gape limitation on prey selection) or decrease reproduction (e.g., delayed sexual maturation, gonad size),
- behaviors and impaired motor function (i.e., swimming, ability to migrate) that could increase individual mortality (e.g., predator avoidance), or decrease growth or reproduction (e.g. feeding, reproductive behavior),
- impaired sensory function that could increase individual mortality, or decrease growth or reproduction (e.g. predator or prey detection, homing ability)

Survival

Individual survival is typically measured by incidences of death at the end of 96-hour (h) exposures (acute test⁴) and incidences of death at the end of 21 d, 30 d, 32 d, and “full life cycle” exposures (chronic tests⁵) to a subset of freshwater and marine fish species reared and exposed in laboratories under controlled conditions (temperature, pH, light, salinity, etc.; EPA 2004). The LC50 is the statistically derived concentration sufficient to kill 50% of the test population. It is derived from the number of surviving individuals at each concentration tested at the end of a 96 h exposure and is usually estimated by probit or logit analysis and more recently by non-linear curve fitting techniques. Ideally, to maximize the utility of a given LC50 study, a slope, variability around the LC50, and a description of the experimental design, such as experimental concentrations tested, number of treatments and replicates used, solvent controls, etc., are needed. The slope of the observed dose response relationship is particularly useful in interpolating incidences of death at concentrations below or above an estimated LC50. The variability of an LC50 is usually depicted by a confidence interval (95% CI) or error (standard deviation or standard error) and is illustrative of the degree of confidence associated with a given LC50 estimate (i.e., the smaller the range of uncertainty, the higher the confidence in the estimate). Without an estimate of the variability, it is difficult to infer the precision of the estimate. Furthermore, survival experiments are of most utility when conducted with the most sensitive life stage of a listed species or a representative surrogate. In the case of ESA-listed Pacific salmonids, there are several surrogates including hatchery reared coho salmon, Chinook salmon, steelhead, and chum salmon, as well as rainbow trout.⁶ We consider the range in

⁴ Organisms are exposed for 96 hours in static or flowing water (flow-through) to varying concentrations of the chemical. At 96 hours, dead organisms are counted in each treatment. Concentrations may be renewed at various intervals (24, or 48 hr) or maintained through continuous introduction of the chemical.

⁵ Organisms are exposed for longer than 96 hours, typically more than 14 days.

⁶ Rainbow trout and steelhead are the same genus species (*Oncorhynchus mykiss*), with the key differentiation that steelhead migrate to the ocean while rainbow trout remain in freshwaters. Rainbow

response of these surrogates to specified exposures to characterize the likely response of listed salmonids.

In addition to laboratory tests of survival, a summary of reported lethality incidents are provided from in EPA's incident database (Sections 11.4.5.7). Section 6(a)(2) of the Federal Insecticide, Fungicide and Rodenticide Act requires pesticide product registrants to report adverse effects information, such as incident data involving fish and wildlife. Criteria require reporting of large-scale incidents. For example, pesticide registrants are required to report the following (40 CFR part 159):

- Fish – Affecting 1,000 or more individuals of a schooling species or 50 or more individuals of a non-schooling species.
- Birds – Affecting 200 or more individuals of a flocking species, or 50 or more individuals of a songbird species, or 5 or more individuals of a predatory species.
- Mammals, reptiles, amphibians – Affecting 50 or more individuals of a relatively common or herding species or 5 or more individuals of a rare or solitary species.

The number of documented incidents is believed to be a very small fraction of total incidents caused by pesticides for a variety of reasons. Incident reports for non-target organisms typically provide information only on mortality events and plant damage. Sub-lethal effects in organisms such as abnormal behavior, reduced growth and/or impaired reproduction are rarely reported, except for phytotoxic effects in terrestrial plants. An absence of reports does not necessarily equate to an absence of incidents given the nature of the incident reporting.

Information on unintended pesticide effects on non-target plants and animals is compiled in the Ecological Incident Information System (EIIS). The EIIS is a database containing adverse effect reports, typically mortality of non-target organisms where such effects have been associated with the use of pesticides. Other Ecological Incident databases used are the Incident Data System (IDS), Aggregated Incident Database, and Avian Information Monitoring System (AIMS).

Each incident record indicates whether the incident occurred due to a misuse, registered use, or whether it is undetermined. Each incident is additionally classified with a certainty of the association with the identified a.i. and are classified as: “highly probable,” “probable,” “possible,” and “unlikely.”

Growth and Reproduction

The FIFRA guideline tests that EPA requires pesticide registrants to conduct evaluate select growth and reproduction endpoints (chronic tests). In these tests, fish are exposed to the a.i. for variable durations depending on the species tested and may have static renewal or flow through

trout are therefore good toxicological surrogates for freshwater life stages of steelhead, but are less useful as surrogates for the life stages that use estuarine and ocean environments.

exposures, both techniques to maintain an exposure concentration. Fish are fed twice daily, ad libitum (i.e., an overabundance of food is available at time of feeding). The lowest concentration eliciting a statistically significant difference from controls (no treatment) to growth or reproductive endpoints is recorded (i.e., the LOEC), as well as the lowest exposure concentration tested that is not different than the control (i.e., the NOEC). Many researchers have commented on the poor application of environmental statistics and laboratory testing regarding NOECs and LOECs (Baas et al. 2009; Chapman et al. 1996; Landis and Chapman 2011; Laskowski 1995; Suter 1996). Prominent limitations include: (1) NOECs and LOECs are statistically derived, a function of the concentrations selected by the experimenters, and often are highly variable among studies; (2) ignore the fundamental model of toxicology i.e., does not use the dose-response relationship; (3) ignore critical data at other treatment concentrations i.e., effects at higher treatment concentrations are not reported; (4) use a lack of evidence as a no-effect; and (5) are limited to the concentrations tested. NOECs typically correspond to an EC10 to EC30 on an exposure response curve (Moore and Caux 1997). A 30% effect rate within a population can be striking, particularly if the effect is on a critical biological endpoint such as reproduction, growth, migration, or olfactory-mediated behaviors. Previous salmonid population modeling suggests that when 14% mortality occurs to juveniles population growth rate is substantially affected (NMFS 2009). We therefore exercise caution in interpreting a NOEC as a true “no response” to an exposure.

Growth of individual organisms is an assessment endpoint derived from the chronic fish and invertebrate toxicity tests described above. Reproduction, at the scale of an individual, can be measured by the number of eggs produced per female (fecundity), and at the population scale by measuring the number of offspring per female in a population over multiple generations. The EPA Preliminary Ecological Risk Assessments summarized reproductive endpoints at the individual scale from chronic, freshwater fish experiments described above. Other assessment measures of reproduction include egg size, spawning success, sperm and egg viability, gonadal development, and hormone levels-most of which are rarely measured in standardized toxicity tests conducted pursuant to pesticide registration.

Other Effects

Responses that are not typically evaluated in laboratory toxicity studies have significant implications for survival in the wild. Swimming is a critical function for anadromous salmonids to complete their life cycle. Impairment of swimming may affect feeding, migrating, predator avoidance, and spawning. It has been used to assess behavioral responses of fish to various toxicants, including pesticides (Little and Finger 1990). Swimming capacity is a measure of orientation to flow as well as the physical capacity to swim against it (Dodson and Mayfield 1979; Howard 1975). Swimming activity includes measurements of frequency and duration of movements, speed and travel distance, frequency and angle of turns, position in the water column, and form and pattern of swimming. Little and Finger (1990) concluded that swimming-

mediated behaviors are frequently adversely affected at 0.3 – 5.0 % of reported fish LC50s, and that 75% of reported adverse effects to swimming occurred at concentrations lower than reported LC50s.

Olfaction conveys critical environmental information that fishes use to mate, locate food, discriminate kin, avoid predators, and home (i.e., navigate). Any or all of these essential olfactory-mediated behaviors may be affected by exposure to contaminants such as pesticides (reviewed by Tierney et al. 2010)(Tierney et al. 2009). For example, copper impairs and destroys salmonid olfactory sensory neurons in a matter of minutes at low $\mu\text{g/L}$ levels and effects persist for hours to weeks depending on exposure concentration and duration. Measured behavioral effects in salmonids from impaired olfaction include compromised alarm response, loss of ability to avoid copper, interrupted spawning migrations, loss of homing ability, and delayed and reduced downstream migration of juveniles (Baldwin et al. 2003; Baldwin et al. 2011; Hansen et al. 1999; McIntyre et al. 2008; Mebane and Arthaud 2010; Sandahl et al. 2004). Disruption of these essential behaviors reduces the likelihood of an individual salmonid completing its life cycle.

Certain critical biochemical responses can indicate organism-level responses affecting survival and fitness in the wild. For example estrogen mimics like nonylphenol, used as a surfactant in tank mixes and fracking, has been linked to endocrine disrupting effects in aquatic systems (Arsenault et al. 2004; Brown et al. 2003; Brown et al. 1999; Brown et al. 2005; Madsen et al. 2004; Schoenfuss et al. 2008). Another example is impaired neurotransmitter function through changes in acetylcholinesterase levels. Acetylcholinesterase is a crucial enzyme in the proper functioning of cholinergic synapses in the central and peripheral nervous systems of vertebrates and invertebrates. Of consequence to salmon, anticholinesterase insecticides have been shown to interfere with salmon swimming behavior (Beauvais et al. 2000; Brewer et al. 2001; Sandahl et al. 2005), feeding behavior (Sandahl et al. 2005), foraging behavior (Morgan and Kiceniuk 1990), homing and antipredator behaviors (Scholz et al. 2000), and reproductive physiology (Moore and Waring 1996; Scholz et al. 2000; Waring et al. 1996).

We located no study results that evaluated swimming effects or olfactory responses in fish following exposure to the pesticides evaluated in this opinion. However, the absence of such information does not mean these effects do not occur. For example, one study reported metolachlor potentiation of organophosphate acetylcholinesterase inhibition in earthworms (Stepić et al. 2013).

11.4.3 Indirect Effects

Indirect effects to fish and habitats exposed to the pesticides evaluated in this opinion are evaluated using toxicity tests of species representing the prey and habitat salmonids depend on.

Invertebrate Prey

Fish can consume a very high proportion of the invertebrate community in aquatic habitats (Huryn 1998; Huryn, 1996 #82). Juvenile salmonids consume a wide range of invertebrates, including those from all functional feeding groups. Changes in abundance of any of these groups could change prey availability for these fish. Pesticides may kill or injure aquatic insects and other macroinvertebrates that serve as food for rearing juvenile salmonids of all five species and adult steelhead. Lack of food may affect a salmonid's growth and development, ultimately affecting their ability to complete their life cycle. Juvenile salmonids are generally opportunistic drift-feeders, and are therefore sensitive to factors that influence the general quantity and quality of invertebrate prey items. If, for instance, there were reductions in the production of invertebrate grazers or the inputs of invertebrate prey from riparian vegetation, salmonids may be forced to alter their foraging behavior (e.g., take more risks, select less energy-rich prey). Alternatively, changes in abundance and composition may have minimal impacts to salmonids if they do not alter the overall quality or quantity of prey, or impact foraging behaviors. Whether or not production of prey decreases or shifts (or increases) after exposure to pesticides will depend in part on the composition of the community (structure and function) and the relative sensitivities of those taxa. Multiple experiments conducted in mesocosms have demonstrated that the particular composition of the community at the time of pesticide exposure influences the magnitude of the impact as well as the trajectory of the recovery (Colville et al. 2008; Downing et al. 2008; Heckmann and Friberg 2005; Hessian et al. 1994; Lytle and Lytle 2002; Maund et al. 2009; Rohr and Crumrine 2005; Schulz et al. 2003a; Schulz et al. 2003b; Van den Brink et al. 2007; Van den Brink et al. 2006) and this would likely be the case in salmonid habitats.

Mixtures of pesticides present a particular challenge in assessing impacts on salmon habitat. Most of the experiments described above were conducted in mesocosms with a single exposure of a single pesticide, something that rarely occurs in salmonid habitat. In streams and rivers of the United States pesticides frequently co-occur with other pesticides (Gilliom 2007). A final consideration in assessing how pesticides may impact salmonids and their habitats is the question of resiliency of these aquatic ecosystems. The recovery of secondary production, to rates observed prior to exposure, depends on the communities themselves and the exposure. For example, univoltine species of macroinvertebrates (i.e. that produce one generation per year) will require a long time to recover. Additionally, if pesticides persist in the landscape, exposures may occur repeatedly (or continuously) depending on application rate, precipitation, and conditions in the watershed. In habitats that receive pesticidal inputs repeatedly throughout the year, salmonid prey may be chronically suppressed.

Riparian Vegetation and Aquatic Primary Producers

We evaluate the available information to assess whether riparian vegetation and aquatic primary producers may be affected by the a.i.s. Riparian vegetation is important for providing shade to

the stream, stabilizing the stream banks, reducing sedimentation, and providing organic material inputs, both in terms of plant material and terrestrial insects. Riparian vegetation is a major focus of restoration efforts of salmonid habitat throughout their range to help reduce pesticide loading into aquatic resources. Riparian vegetation is an important assessment endpoint for herbicidal impacts on salmon habitats. Generally there are sparse data regarding the effects of herbicides (and much less with insecticides, aracnicides, or miticides) on wild plants within riparian systems, other than weed species. The EPA requires submission of crop effects data as part of the registration process for herbicides (EPA 1996). This information currently provides the only basis for evaluating effects on herbaceous plants unless data are available from other sources. The overall assumption is that the sensitivity of plant species tested (typically plants used in agriculture) in the registrant-provided guideline studies will be representative of riparian species. There is no way to know this is the case, therefore a high degree of uncertainty regarding the toxicity of the a.i.s to riparian vegetation exists. We also evaluate if and to what extent aquatic primary producers are affected by the stressors of the action. Primary producers including periphyton, diatoms, macrophytes, and plankton are integral components of aquatic food chains, serving as food for salmonid prey. Reductions in primary productivity may lead to impacts to salmonid prey. Although typically not tested for effects to freshwater and marine primary producers, we search for and evaluate any information on pesticide effects to primary producers.

11.4.4 Environmental Factors That Modify Pesticide Toxicity

The physical and chemical properties of water, its temperature, hardness, pH, oxidation/reduction potential, and content of naturally occurring substances like carbon, organic acids, can influence pesticide toxicity. The information submitted by the EPA only discussed these factors in context of pesticide transformation, fate, and transport because these factors influence pesticide degradation half-life and biological availability. For example pesticide half-lives are longest at the optimum pH, with increasing hydrolysis at lower and higher pH values. Substances like minerals, silt, and organic acids can bind to pesticides, reducing their bioavailability to target and non-target organisms.

Searches of the open literature for the influence of environmental factors that modify the toxicity of 1,3-D and metolachlor only identified information on effects of salinity and temperature on metolachlor toxicity. Exposure to s-metolachlor at concentrations as low as 0.01 µg/L and temperatures that were four degrees above or four degrees below the optimal developmental temperature of 24° C (75.2° F) significantly increased frequency of larval abnormalities in Pacific oyster (Gamain et al. 2017). Salinities below 33 p.s.i. also synergistically impaired larval development at 0.01 µg/L S-metolachlor (Gamain et al. 2016).

Increased toxicity for fish at elevated temperatures is a generally accepted principle. As ectotherms, the metabolism of aquatic organisms increases at higher temperatures. This includes metabolism for life functions (e.g. oxygen consumption, excretion, homeostasis) and biotransformation of toxicants. For example, gold fish exposed to environmentally realistic

mixtures of herbicides and fungicides, including S-metolachlor, exhibited concentration and temperature-dependent increases in molecular indicators of stressor injury, defense, repair, and cellular replacement (Gandar et al. 2017; Jacquin et al. 2019). A toxicant that effects energy metabolism or respiratory gas exchange may make it difficult for organisms to meet increased metabolic needs under higher temperatures. Increased metabolism requires higher rates of active uptake and diffusion of water and solute moving over the gills, increasing uptake and excretion of aquatic toxicants (Cairns et al. 1975).

We expect elevated temperatures across the freshwater habitats of listed cold-water fish to co-occur with both a.i.s. As shown in the Environmental Baseline, many listed cold-water fish reside in watersheds listed on State 303(d) lists as impaired due to temperature exceedances. We expect that cold-water fish and their prey exposed to both elevated temperature and the two herbicides and their degradates in the environment will be adversely affected at relatively lower concentrations compared to exposures to the two herbicides and their degradates at non-elevated temperatures in laboratory and field assays. While we cannot quantify the degree to which elevated temperature may increase toxicity of 1,3-D, we will treat temperature qualitatively as a factor expected to increase the risk of reregistration of both 1,3-D and metolachlor, to cold-water fish.

It is also important to note that the hardness of waters in much of the range of listed anadromous species is below 60 mg CaCO₃/L; this suggests that responses within the freshwater habitats of listed salmonids will be comparable or potentially more sensitive than responses observed under laboratory conditions (Figure 1).

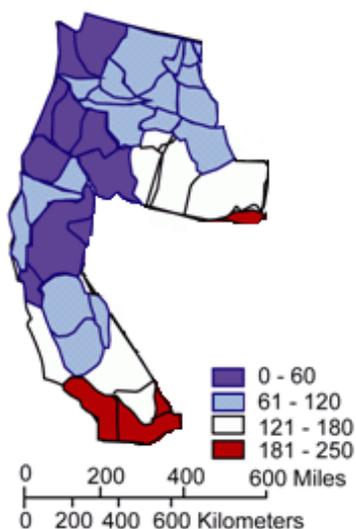


Figure 1. Water hardness among watershed accounting units (6 digit HUCs) within the range of ESA-listed salmonids (mg/L CaCO₃).

11.4.5 Analyzing Response to 1,3-D and its degradates

The soil fumigant 1,3-D restricts the function of vital enzymes of nematodes through substituting a sulfhydryl, ammonia or hydroxyl group of functioning enzyme systems with a 1,3-D chlorine. Restriction of these enzyme systems results in the paralysis and death of exposed nematodes (Cox 1992). Information on the mechanism by which 1,3-D exerts toxic effects on aquatic animals or other species groups was not found in EPA assessments or a search of the open literature.

The most significant aquatic degradation route for 1,3-D is aerobic aquatic metabolism formation of 3-chloroallyl alcohol and, to a lesser extent, 3-chloroacrylic acid (Figure 2). The 1,3-D aerobic aquatic metabolism half-life of 5 days contrasts with the hydrolysis half-life of 196 hours at pH 7 and 20°C. The degradate 3-chloroallyl alcohol is formed at a maximum 6.4% of applied 1,3-D one day after treatment. In the absence of metabolic activity, 3-chloroallyl alcohol formed at up to 77% of applied 1,3-D via hydrolysis upon termination of a 22-day study (MRID 44975503 as cited in USEPA, 2013). The degradate 3-chloroacrylic acid forms at a maximum of 9.5% of applied 1,3-D seven days after treatment. Long term exposure to both degradates is not expected because they dissipate rapidly in metabolically active waters, with half lives of 1.2 and 3.96 hours for 3-chloroallyl alcohol and 3-chloroacrylic acid, respectively.

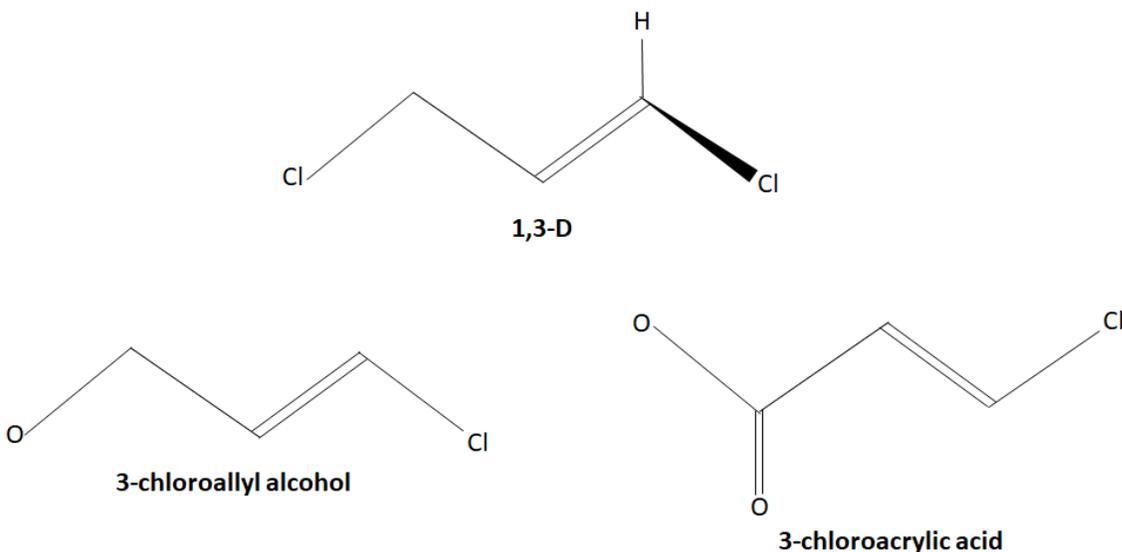


Figure 2. Structures of 1,3-D and degradates

Not all endpoint estimates were provided with confidence limits and exposure-response slopes. The ECOTOX does not include data for 3-chloroallyl alcohol or 3-chloroacrylic acid and does not report exposure response slopes. With the exception of the 1,3-D data from Mayer and Ellersieck (1986) and Buccafusco et al. (1981), the studies entered into ECOTOX have not undergone review by EPA, so they have not been classified as *acceptable*, *core*, or *supplemental*.

11.4.5.1 *Salmonid Lethality*

The 1,3-D lethality data reported in both the ECOTOX and EPA's Pesticide Ecotoxicity Database are presented in Table 12. The fish LC50s in EPA's risk analyses for 1,3-D were not adjusted for purity or recalculated from the original data. The 2013 1,3-D Problem Formulation used the Walleye LC50 of 1,080 ppb while the 2019 Draft Risk Assessment reported updated data which included an LC50 of 2,780 ppb for rainbow trout. The ECOTOX also included LC50s for fathead minnow that were lower than those LC50s for rainbow trout, the lowest of which, and LC50 of 239 ppb (Geiger et al. 1990). Nonetheless, NMFS considers rainbow trout to be the most suitable surrogate species for ESA-listed salmonids. Further, rainbow trout 96-hour LC50s are available for 3-chloroallyl alcohol and 3-chloroacrylic acid. This allows within-species comparison of the parent compound toxicity to these degradates. The LC50 of 986 ppb for 3-chloroallyl alcohol is about one third the 1,3-D LC50, while the LC50 for 3-chloroacrylic acid, at 69,500 ppb, is 25 times the LC50 for the parent compound.

The 1,3-D Problem Formulation stated that the degradates are sufficiently mobile and persistent to reach estuarine and marine environments. While there are no LC50 data for estuarine or marine fish exposures to the 1,3-D degradates, the sheepshead minnow LC50 for 1,3-D of 870 ppb is about one third the LC50 for rainbow trout. Taking in freshwater degradate toxicity into consideration, it is reasonable to expect LC50s for estuarine and marine fish exposed to the more toxic degradate, 3-chloroallyl alcohol, would be lower still.

Table 12 Fish LC50 data for 96 hour exposures to 1,3-dichloropropene and degradates.

Species	Purity	Exposure	Toxicity Value (ppb)	MRID or Author, year (ECOTOX number)	EPA data quality designation
1,3-Dichloropropene					
Rainbow trout	100	static	LC50=2,780 ^a (2,130-3,620); NOEC / LOEC =1,460 / 2130 ^a	49382003	core
	92	static	LC50=3,940 (3,100-5,000)	39692	core
	not reported	static	LC50=5,360	Birge et al., 1982 (45758)	
Walleye	100	static	LC50=1,080 (990-1,200)	40098001; Mayer, Jr. and Ellersiek, 1986 (6797)	supplemental
Bluegill	96	flow through	LC50=3,700 (2,800-4,800); NOEC=1,000	44849101	core
	92	static	LC50=6,700 (5,800-7,760); NOEC=4,200	TN 1118	core
	92	static	LC50=7,090 (5,160-9,700)	39692	core
	80+	static	LC50=6,100 (5,100-6,800)	117043; Buccafusco et al., 1981 (5590)	supplemental
Carp	not reported	static	LC50=9000 (8000-11000)	Shell Oil Co, 1987 (93891)	not coded ^b

Fathead minnow	100	static	LC50=4,100 (3,400-4,970)	40098001; Mayer, Jr. and Ellersiek, 1986 (6797)	supplemental
	95	flow through	LC50=239 (211-271)	Geiger et al., 1990 (3217)	not coded
	not reported	flow through	LC50=1400 (1200-1500)	Turner, 1982 (9994)	not coded
	not reported	static	LC50=1600 (1400-1900); LOEC=710; NOEC=670	Turner, 1982 (9994)	not coded
	not reported	static	LC50=2320 (1520-2680)	Birge et al., 1982 (45758)	not coded
Goldfish	100	static	LC50<7500	Mayer, Jr. and Ellersiek, 1986 (6797)	not coded
Largemouth bass	100	static	LC50=3,650 (3,500-3,780)	40098001, Mayer, Jr. and Ellersiek, 1986 (6797)	supplemental
Sheepshead minnow	96	flow through	LC50=870 (570-1100); NOEC=570	44843901	core
	80	static	LC50=1800 (700-4500); NOEC=1200	Heitmuller et al., 1981 (10366)	not coded
3-chloroallyl alcohol					
Rainbow trout	not reported	static renewal	LC50=986 ^a (747-1320), slope=6.5 (ppm); NOEC=303	44940306	supplemental
3-chloroacrylic acid					
Rainbow trout	not reported	static	LC50=69,500 ^a (49,200-98,100); NOEC=49,200	44940307	core

^a Value appears in Risk-plots within Chapters 12 & 15

Not coded = EPA has not classified this study (e.g. "core", "supplemental", etc.)

11.4.5.2 *Salmonid Growth And Fitness*

Thresholds for growth and fitness effects were only available for 1,3-D and not the degradates (Table 13). The 2019 1,3-D Draft Risk Assessment included an early life stage fathead minnow growth LOEC of 15 ppb. The difference in mean dry weight at the 15 ppm treatment group from the pooled controls was considered slight, at 8.3% (MRID 49682401). NMFS also identified NOEC of 1,460 ppb and LOEC of 2,130 ppb for effects of 1,3-D on rainbow trout swimming behavior from the same study reporting the LC50 at 2,780 ppb in MRID 49382003. Data for the effects of chronic exposures to 1,3-D on estuarine and marine fish species were not available. The 2019 1,3-D Draft Risk Assessment estimated chronic values for sheepshead minnow based by applying fathead minnow and sheepshead minnow data in acute to chronic ratios.

Table 13 Fish LOEC and NOEC data for growth and fitness responses to 1,3-D exposures.

Response	Species	Purity	Exposure design	Toxicity Value (ppb)	MRID	Fulfills guideline?
Growth	Fathead Minnow	96.8	flow through, chronic early life stage at 28 days	NOEC = 15 ^a LOEC = 34	49682401	core
Behavior	Rainbow Trout	100	flow through, 96 hours	NOEC = 1,460 ^a LOEC = 2,130 (erratic swimming)	49382003	core
ACR estimate	Sheepshead minnow	N.A.	N.A.	NOEC = 1.8 LOEC = 3.2	N.A.	N.A.

^a Values in this table appear in Risk-plots within Chapters 12 & 15

N.A. = not applicable (threshold is an ACR estimate, not empirical data).

11.4.5.3 *Invertebrate Prey*

The 1,3-D problem formulation classified the 1,3-D as very highly toxic to freshwater invertebrates and highly toxic to estuarine and marine invertebrates. There were abundant data for the effects of acute exposures to 1,3-D on invertebrates (Table 14). The 2013 1,3-D Problem Formulation applied an acute LC50 of 90 ppb for the water flea in its analysis. This LC50 is one or more orders of magnitude lower than the 1,3-D LC50s for other invertebrates and the water flea LC50s for both 3-chloroallyl alcohol and 3-chloroacrylic acid. LC50s for marine species ranged from 640 ppb for 96 hour flow through exposure of eastern oyster to 3,900 ppb for a 48 hour static exposure of opossum shrimp.

Table 14 Toxicity data for acute exposures of invertebrates to 1,3-D and degradates.

Response	Species	Purity	Exposure design	Toxicity Value (ppb)	MRID
Dichloropropene					
Midge	92	48 hours, static	LC50=1,350 (1,080-1,670)	Horne and Oblad, 1983 (14396)	not coded
Scud	92	96 hours, static	LC50=2,000		
Marsh rams-horn snail	92	96 hours, static	LC50=8,100 (7,520-8,720)		
Stonefly	92	96 hours, static	LC50=5,420 (4,800-6,120)		
Water Flea	100	48 hours, static	EC50=90 ^a (63-129)	40098001, Mayer and Ellersieck 1986 (6797)	supplemental
	80+	48 hours, static	EC50=6,200 ^a (4,300-9,000); NOEC=410	00117044	supplemental
	80	24 hours, static	LC50=7,200 (5,100-11,000)	LeBlanc, 1980 (5184)	not coded
		48 hours, static	NOEC=410; LC50=6,200 (4,300-9,000)		not coded
	not reported	24 hours, static	LC50>6,800	Turner, 1982 (9994)	not coded
		48 hours, static	NOEC=1,600; LOEC=2,600; LC50=4,500 (4,200-5,000)		not coded
		48 hours, flow through	NOEC<990; LOEC=990; LC50=2,800 (2,400-3,400)		not coded
	24 hours, flow through	LC50=6,000 (5,600-6,500)		not coded	
Eastern oyster	96	96 hours, flow through	EC50=640 (570-710); NOEC=350	44843903	Core
Opossum Shrimp	96	96 hours, flow through	LC50=700 (600-850), slope=6.9 (ppm); NOEC=170	44843904	Core
	not reported	96 hours, static	NOEC=410; LOEC=800; LC50=1,200 (650-2,300)	Turner, 1982 (9994)	not coded
		24 hours, static	LC50=3,900 (2,200-3,900)		not coded
		72 hours, static	LC50=1,400 (690-2,400)		not coded
		48 hours, static	LC50=1,700 (770-2,500)		not coded
		48 hours, flow through	LC50=1,300 (1,200-1,400)		not coded
		24 hours, flow through	LC50>1,700		not coded
		96 hours, flow through	NOEC=230; LOEC=400; LC50=640 (560-730)		not coded
		72 hours, flow through	LC50=940 (690-1,200)		not coded
3-Chloroacrylic acid					
Water flea	100	48 hours, static renewal	EC50=56,900 ^b (49,500-65,400); NOEC=24,900	44940308	core
3-Chloroallyl alcohol					
Water flea		48 hours, static	EC50=2,300 (1,200-4,200); NOEC=1,200	44843902	supplemental

^aValue appears in Risk-plots within Chapters 12 & 15

^bThe data in this table are as reported in the OPP database. The 1,3-D Problem formulation adjusted this value to 55,000 ppb and this is the value reported in the Risk-plot.

Not coded = EPA has not classified this study (e.g. "core", "supplemental", etc.)

There were two core studies available to assess chronic toxicity to invertebrate prey. These were a single study reporting chronic effects for invertebrates exposed to 1,3-D and one study for 3-chloroacrylic acid. The 18-day LOEC of 105 ppb (MRID 450075801) for water flea exposures to 1,3-D was similar to the 48 hour LC50 of 90 ppb (MRID 40098001). The degradate 3-chloroacrylic acid was substantially less toxic, with an 18-day LOEC of 5,080 ppb (MRID 49382005).

Table 15 Toxicity data for chronic exposures of aquatic invertebrates to 1,3-D and 3-chloroacrylic acid.

Species	Purity (%)	Exposure Duration	Toxicity Values (ppb)	MRID	EPA data quality designation
1,3 Dichloropropene					
Water flea	96	18 days, flow through	LOEC=105; NOEC=70	45007501	core
3-Chloroacrylic acid					
Water flea	100	18 days, static renewal	LOEC=5,080; NOEC=2,530	49382005	core

11.4.5.4 *Phytoplankton And Aquatic Vascular Plants*

The data in Table 16 are from the OPP database, but some of these data, denoted with “b” in superscript, do not match the values attributed to the same MRID in the 1,3-D Problem Formulation. Both the OPP database and the 1,3-D Draft Risk Assessment report the freshwater diatom (*Navicula pelliculosa*) 5-day EC₅₀ from MRID 44843909 as 1,390 ppb, but the 1,3-D Problem Formulation reports a much higher EC₅₀ for this study, at 7,900 ppb. This difference could not be attributed to a correction for percent purity and it was unclear whether the difference was due to a recalculation from the original exposure-response data. Both EC₅₀ estimates indicate the freshwater diatom as is more sensitive than other aquatic plant species to 1,3-D. This opinion uses the EC₂₅ of 30 ppb for *Navicula pelliculosa* in the Risk-plots as reported in MRID 44843909.

The relative toxicity of 1,3-D metabolites to aquatic plant life differs from that of fish and invertebrates. Data for 3-chloroacrylic acid indicate that it is actually more toxic. The EC₅₀ for 3-chloroacrylic acid is an order of magnitude lower than the 1,3-D EC₅₀ for duckweed, with EC₅₀s of 220 and 20,000 ppb, respectively. This metabolite is also more toxic than the parent compound to green algae, with EC₅₀s of 432 ppb for exposure to 3-chloroacrylic acid and 15,000 ppb for exposure to 1,3-D. While the 3-chloroallyl alcohol EC₅₀ for duckweed was an order of magnitude lower than the 1,3-D EC₅₀ for this species. Freshwater diatom and green algae were more sensitive to 1,3-D than to 3-chloroallyl alcohol.

Table 16 Toxicity data for phytoplankton and aquatic plants exposed to 1,3-D and degradates.

Species	Purity (%)	Exposure Duration	Toxicity Values (ppb)	MRID	EPA data quality designation
1,3 Dichloropropene					
Blue-green algae	96	5 days, static	EC50=108,000 (50,000-232,000); NOEC=11,300	44843911	core
Duckweed	96	7 days, static	EC25 = 1310 ^a ; EC50=20,000 (14,000-29,000); NOEC=1,200	44843914	core
Freshwater diatom	96	5 days, static	EC25 = 30 ^a ; EC50=1,390 (1,060-1,810); NOEC<74	44843909	supplemental
Freshwater green algae	96	96 hours, static	EC25 = 7850 ^a ; EC50=15,000 (10,200-22,000); NOEC=9,500	44940314	core
Marine diatom	96	5 days, static	EC50=15,500 (10,800-22,300); NOEC=8,800	44843910	core
3-Chloroacrylic acid					
Blue-green algae	not reported	5 days, static	EC50=4,200 (3,000-3,600), slope=4,400; NOEC=3,200	44940318	supplemental
Duckweed	not reported	196 hours, static	EC50=220 (120-400)	45007504	core
Freshwater diatom	not reported	5 days, static	EC50=5,400 (5,100-5,700), slope=8,800; NOEC=2,500	44940317	supplemental
Freshwater green algae	not reported	96 hours, static	EC50=432 (271-688); NOEC=181	44940319	supplemental
Marine diatom	not reported	5 days, static	EC50=50,200 (47,700-52,900); NOEC=23,700	45007503	core
3-Chloroallyl alcohol					
Blue-green algae	not reported	5 days, static	EC50>101,000; NOEC=52,000	44843912	supplemental
Duckweed	not reported	196 hours, static	EC50=1,694 (926-3,100); NOEC=42	44940320	supplemental
Freshwater diatom	not reported	5 days, static	EC50=32,900 (12,850-84,400); NOEC=48,000	44843913	supplemental
Freshwater green algae	not reported	96 hours, static	EC50=49,000 (38,000-63,000); NOEC=14,000	44940315	supplemental
Marine diatom	not reported	5 days, static	EC50=140 (43-490), slope=821; NOEC=22	44940316	supplemental

^aValue appears in Risk-plots within Chapters 12 & 15

11.4.5.5 Terrestrial (Riparian) Vegetation

Riparian vegetation is important for providing shade to the stream, stabilizing the stream banks, reducing sedimentation, and providing organic material inputs, both in terms of plant material and terrestrial insects. Riparian vegetation is a major focus of restoration efforts within California, and when present can reduce pesticide loading into aquatic resources. Riparian

vegetation is an important assessment endpoint for herbicidal impacts on salmon habitats. Generally, there are sparse data regarding the effects of herbicides (and much less with insecticides, arachnicides, or miticides) on wild plants within riparian systems, other than weed species. The EPA requires submission of crop effects data as part of the registration process for herbicides. This information currently provides the only basis for evaluating effects on herbaceous plants unless data are available from other sources. The overall assumption is that the sensitivity of plant species tested (typically plants used in agriculture) in the registrant-provided guideline studies will be representative of riparian species. There is no way to know this is the case, therefore a high degree of uncertainty regarding the toxicity of the a.i.s to riparian vegetation exists.

Currently there are gaps in information on the effects of 3-chloroallyl alcohol and 3-chloroacrylic acid on terrestrial plants. The EC25 estimates from the OPP database for MRID 45007502 were converted from the ppm to pounds per acre for the 1,3-D Problem Formulation (Table 17).

Table 17. Toxicity data for terrestrial plants exposed to 1,3-D and degradates.

Study Type	% AI	Species	Lowest reported EC ₂₅ (dataset size) in lb ai/A	Most Sensitive Endpoint/ Measured Endpoint	MRID or ECOTOX reference	EPA data quality designation
Seedling emergence	not reported	dicot (tomato)	4.81	Shoot weight	45007502	core
		monocot (onion)	>11.69	--		
Vegetative vigor	not reported	dicot (tomato)	6.86	Shoot weight	45007502	core
		monocot (onion)	3.5	Shoot length		
Development	not reported	monocot (garden ginger)	>446.09 (n=1)	Emergence	Smith et al., 2011 (174802)	not coded
Population	not reported	dicot (Canada thistle)	>249 (n=5)	Abundance	Ogg, Jr., 1975 (89203); Schneider et al., 2009 (153245); Hanson et al., 2010 (153138)	not coded
	not reported	monocot (garden ginger)	>446.09 (n=1)	Biomass	Smith et al., 2011 (174802)	not coded
		Dicot (beet)	>15 (n=5)	Biomass	Schwartz and Gale, 1979 (155570)	not coded
Reproduction	not reported	monocot (yellow nutsedge)	>332 (n=1)	Viability	Hanson et al., 2010 (153138)	not coded
		dicot (multiple)	>332 (n=8)	Viability	Hanson et al., 2010 (153138); Shrestha et al., 2008	not coded

11.4.5.6 Field Studies

Field studies on the effects of 1,3-D on aquatic life were not identified in the ECOTOX or a search of the open literature.

11.4.5.7 Field Incidents

The 1,3-D Problem Formulation reported incidents from the Ecological Incident Information System (EIIS) database involving terrestrial plants (13), aquatic plants (1), and wildlife (1). Most plant incidents were attributed to applications of 1,3-D plus chloropicrin, with a few attributed to 1,3-D alone. Certainties for these incidents ranged from “possible” to “highly probable.” Certainty of a causal relationship between 1,3-D and the reported incident was not included for the wildlife incident or 5 of the 13 plant incidents. According to the 1,3-D Problem Formulation, the wildlife incident (#I016738-016) occurred when 1,3-D and chloropicrin applied to strawberry fields via irrigation accidentally spilled into a nearby creek, resulting in 1000 fish killed. Residues taken from the fish confirmed the exposure.

The 2019 1,3-D Draft Risk Assessment Since publication of the 1,3-D Problem Formulation, registrants reported three new minor plant incidents between 2017 and 2018 in the aggregate incident reports. No additional details are available for these incidents. The new terrestrial plant incident (#I029870-0007) reported in EIIS database occurred in 2017. A tomato crop was treated on several farms with Telone EC in Lazio, Italy. Transplanted seedlings were affected after the subsequent planting cycle. The certainty that this incident is attributed to Telone EC is classified as “possible.”

While incidents represent evidence of environmental exposures to 1,3-D, NMFS does not consider them contributing appreciably to the effects of the action.

11.4.5.8 Bioconcentration And Bioaccumulation

The ECOTOX database does not report data for bioconcentration or bioaccumulation of 1,3-D and this information is not typically reported in the OPP database. The 1,3-D Draft Risk Assessment concluded that 1,3-D is not likely to bioconcentrate in tissues of aquatic organisms due to the low octanol/water partition coefficient (log K_{ow}) of 1.82.

11.4.5.9 Degradate Toxicity

The 1,3-D degradates 3-chloroallyl alcohol and 3-chloroacrylic acid are important considerations in this analysis because, as shown by the data summarized in Table 12, the alcohol degradate may be more toxic to salmonid species than 1,3-D (Table 18). To further evaluate the potential for increased risk of direct lethality to salmon we considered the available environmental fate data. 1,3-D and its metabolites are expected to dissipate rapidly in surface waters. Aerobic aquatic metabolism studies reveal comparable half-lives at 25° C (EPA 2008; 1,3-D 4.9 days, 3-chloroallyl alcohol 1.2 days, and 3-chloroacrylic acid 3.4 days). EPA reports that 1,3-D is

hydrolyzed to the alcohol at a rate of 72 percent of the applied parent. However, based on aquatic metabolism studies, no degradate has been found to exceed 6.5% of the applied the 1,3-D (cite EPA 2008 RLF BE). A study evaluating environmental concentrations of 1,3-D and its degradates on a Florida golf course found that the peak concentrations of the alcohol in water collected in drains immediately below golf course fairways were <10% of the peak concentrations observed for 1,3-D (cite study labeled attachment 16 - provided by Dow April 14, 2020). In ponds, the alcohol was only detected only once, at a trace concentration of 0.025 ppb or <2% of the corresponding concentration observed for the parent 1,3-D. The available information to characterize exposure suggests the peak concentrations of the 1,3-D in surface waters are likely to be at least times 10 times greater than that of the alcohol degrade. Whereas, salmonid acute toxicity data suggest the sensitivity of 1,3-D and the alcohol metabolite vary by a factor of < 3. Taken together, this suggests that 1,3-D likely poses a greater risk of direct lethality to salmonids than the alcohol degrade.

Table 18 Relative toxicity of 1,3-D and its degradates to salmonids and aquatic invertebrates.

Endpoint	Duration	Test Species	Toxicity Value (ppb)		
			1,3-D	3-chloroallyl alcohol	3-chloroacrylic acid
Direct Mortality	96-hr	Rainbow Trout	LC50 = 2780	LC50 = 986	LC50 = 69,500
Prey	48-hr	Water flea	EC50 = 747*	EC50 = 2,300	EC50 = 55,000
	48-hr	Water flea	EC50 = 90-6200		

*geometric species mean

The available toxicity data suggests that 3-chloroacrylic acid is more toxic to aquatic plants than 1,3-D (Table 19). Based on EC50 values, the sensitivities between the parent and acid degrade vary by a factor of 1.5-91 (1.5, 35, and 91, for non-vascular plants, algae, and vascular plants, respectively).

Table 19. Relative toxicity of 1,3-D and its degradates to aquatic plants

Aquatic Plants	7-day, 14-day	Vascular (Duckweed)	EC50 = 20,000 7-day	EC50 = 1,694 14-day	EC50 = 220 14-day
	5-day	Non-Vascular (Freshwater diatom)	EC50 = 7850		EC50 = 5,400 Slope = 8.8
	96-hr	Green Algae	EC50 = 15,000	EC50 = 49,000	EC50 = 432

However, the magnitude of exposure to the acid degradate is expected to be less than that of the parent. EPA reports that the acid degradate is formed at a rate of 1-6% of the applied parent, which equates to a reduction in potential peak exposure by a factor of 17-100 (EPA 2008 RLF BE). Therefore, the ratio of peak exposure to toxicity in aquatic plants is expected to be

comparable between 1,3-D and the acid degradate. Given these considerations, we determined it is not necessary to derive quantitative estimates of exposure to the alcohol and acid degradates. Rather, risk of these degradates can be characterized by comparing expected exposure and responses of the parent compound.

11.4.5.10 Companion pesticide: Chloropicrin

NMFS' review of pesticide labels and products found that about 80 percent of products containing 1,3-D also contain chloropicrin as an active ingredient. Reregistration of 1,3-D is reasonably certain to result in continued co-application of chloropicrin within the action area. Table 20 summarizes the available toxicity data for chloropicrin from ECOTOX and the OPP database. Searches of the open literature did not identify additional papers. The data suggest that chloropicrin is at least an order of magnitude more toxic than 1,3-D to these freshwater fish and invertebrates.

Table 20 Toxicity of chloropicrin to fish, invertebrates, and plant species

Species	Purity (%)	Response	Exposure duration	Endpoint (ppb)	MRID or ECOTOX reference	EPA data quality designation
Fishes						
Bluegill	99.00	Mortality	96 hours	NOEL<75; LC50<105	MRID 2035127/ECOTOX 344	S
	99.80	Mortality	96 hours	LC50=50; NOEL=19	MRID 48442406	C
	99.90	Mortality	96 hours	LC50=44.1; NOEL=28.5	MRID 2079912/ECOTOX 344	S
Rainbow trout	99.00	Mortality	48 hours	LC50=16.5	U.S. EPA 1992 ECOTOX 344	not coded
			96 hours	NOEL<11.5; LC50<16.8	MRID 2035129/ECOTOX 344	S
	99.80	Mortality	96 hours	LC50=11 ^a ; NOEL=7.7	MRID 48442405	C
	99.90	Mortality	96 hours	NOEL=3.15; LC50=5.14	MRID 2079911/ECOTOX 344	S
Sheepshead minnow	99.80	Mortality	96 hours	LC50=100; NOEL=67	MRID 48442402	C
Invertebrate prey						
Daphnia magna	99.80	Immobilization	48 hours	EC50=120 ^a ; NOEL=46	MRID 48442401	C
	99.90	Intoxication	48 hours	EC50=170; NOEL=109	MRID 2079913/ECOTOX 344	S
Daphnia pulex	96.50	Immobilization	48 hours	NOEL<9	MRID 2035128	S
		Intoxication	48 hours	EC50<71; NOEL<5; NOEL<8; EC50=63	MRID 2032423/MRID 2035128/ECOTOX 344	C/S
Eastern oyster	99.80	Shell deposition	96 hours	LC50=10; NOEL=1.4	MRID 48442404	S
Mysid	93.00	Mortality	96 hours	LC50=30; LC50=257.8	Carr,R.S. 1987 ECOTOX 17308	not coded
	94.00	Mortality	96 hours	LC50=30; LC50=258	Carr,S. 1987 ECOTOX 155283	not coded
	99.80	Mortality	96 hours	LC50=27; NOEL=14	MRID 48442403	C
Aquatic Plant						

Duckweed	99.70	Growth and reproduction	7 days	EC25 = 4.6 ^a ; EC50=6.5; NOEL=0.309	MRID 48442801	A
Green Algae	not reported		NS	IC50 = 120 ^a	MRID 49559701	S
Terrestrial Plant						
Rapeseed	99.30	seedling emergence vegetative vigor (dry wt)	48 hours 21 days	EC25>10,082; NOEL=10,082 EC25=312; NOEL<204	MRID 48442802	S
Cucumber	99.30	seedling emergence vegetative vigor (chlorosis)	48 hours 21 days	EC25>10,082; NOEL=10,082 EC25>1,046; NOEL=1,046		
Soybean	99.30	seedling emergence vegetative vigor (dry wt)	48 hours 21 days	EC25>10,082; NOEL=10,082 EC25=866; NOEL=204		
Sunflower	99.30	seedling emergence vegetative vigor (dry wt)	48 hours 21 days	EC25>10,082; NOEL=10,082 EC25=2,094; NOEL=1,046		
Ryegrass	99.30	seedling emergence vegetative vigor (dry wt)	48 hours 21 days	EC25>10,082; NOEL=10,082 EC25=9,049; NOEL=1,046		
Corn	99.30	seedling emergence vegetative vigor	48 hours 21 days	EC25>10,082; NOEL=10,082 EC25>9,880; NOEL=9,880		

^aValue appears in Risk-plots within Chapters 12 & 15

11.4.6 Analyzing Response to Metolachlor

The molecular structures of metolachlor and S-metolachlor are illustrated in Figure 3. Metolachlor is a broad spectrum chloroacetamide herbicide that impairs seedling shoot and meristematic growth by inhibiting chlorophyll and biomolecule synthesis. Biosynthesis of fatty acids and lipids, protein, isoprenoids, and flavonoids is thought to be inhibited by conjugation with acetyl coenzyme A and other sulfhydryl-containing biomolecules (EPA 1997). EPAs 2014 problem formulation and 2019 Draft Risk Assessment both cite EPA's *Review of Documents Related to the Equivalency of Racemic Metolachlor (Metolachlor) and S-Metolachlor for Environmental Fate and Ecotoxicity* (EPA 2002), which concluded that it is appropriate to bridge the fate and toxicity data for metolachlor and S-metolachlor, but not the degradates metolachlor entansulfonic acid, metolachlor oxanilic acid. However, in evaluating the toxicity data for these structurally similar metabolites, EPA's 2019 Draft Risk Assessment concluded that they are far less toxic than the parent metolachlor and were thus not residues of concern for ecological exposure. Accordingly, NMFS did not include these metabolites in its analyses.

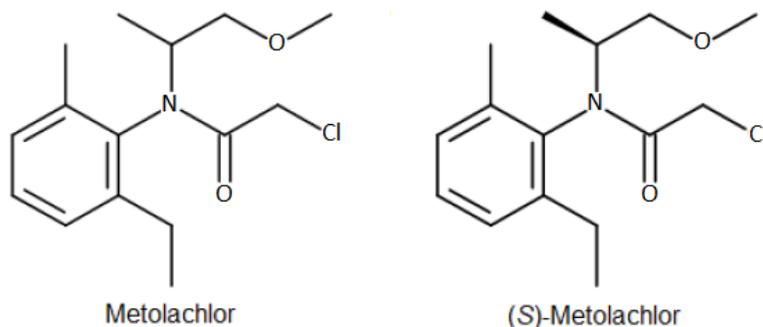


Figure 3. Molecular structure of metolachlor and (S)-metolachlor

Metolachlor acute toxicity is classified as “up to moderately toxic” for fish and aquatic invertebrates. With a Koc of 21.6-369 (L/kgOC), metolachlor is mobile to moderately mobile and is non-volatile from water and intermediate-to-nonvolatile on dry non-adsorbing surfaces (USEPA, 2010a). Metolachlor is unlikely to be significantly degraded via aqueous photolysis in clear water or on moist leaf surfaces (aqueous photolysis half-life = 70 d). The octanol-water partition coefficient (Kow) of 3.05 is high enough to have the potential to bioconcentrate in aquatic organisms, yet the measured bioconcentration factor BCF of 69X in fish and depuration value of 93% in 196 hours once fish were transferred to untreated water suggests that the potential for bioconcentration is low (EPA 2019).

In the absence of usable anaerobic aquatic metabolism data, EPA applied a 3x factor to the available anaerobic aquatic metabolism rate data in its assessment. Half-lives for aerobic metabolism in soils ranged from 13.9 to 2324 hours at 20 °C, placing it between non-persistent and persistent on the Goring persistence scale (Goring et al. 1975). Aerobic aquatic metabolism degradation half-life values ranged from 23.3 to 49.5 days over four soils and 2 temperatures (9 and 20 °C). Anaerobic aquatic metabolism data was only provided for a single soil, with a half-life of 78 days.

11.4.6.1 Salmonid Lethality

The metolachlor lethality data reported in both the ECOTOX and EPA’s Pesticide Ecotoxicity Database are presented in Table 21. The fish LC50s in EPA’s risk analyses for metolachlor were not adjusted for purity or recalculated from the original data. The 2013 Metolachlor Problem Formulation applied a rainbow trout LC50 of 3,800 ppb (MRID 00018722) for metolachlor and a bluegill LC50 of 3,200 for S-metolachlor (MRID 43928910). However, the Metolachlor Draft Risk Assessment applied the most sensitive endpoints from registrant-submitted guideline studies or open literature studies regardless of whether the endpoint was derived from a study conducted with metolachlor or S-metolachlor because EPA had determined that both the

environmental fate and ecotoxicity data submitted for racemic metolachlor and S-metolachlor are comparable.⁷

Table 21. Fish LC50 data for 96 hour exposures to metolachlor and s-metolachlor.

Species	Purity (%)	Exposure	Endpoint	MRID or ECOTOX reference	EPA data quality designation
Metolachlor					
Chinook Salmon Rainbow Trout Silver Salmon	97.2		LC50=13,000	Wan et al., 2006 (89626)	not coded
Rainbow trout	Tech	static	LC50=3,900 ^a (3,300-4,600); NOEC<2,800	00018722	Core
Fathead minnow	87EC	static	LC50=8,400 (6,400-11,000)	40098001; Mayer, Jr. and Ellersiek, 1986 (6797)	Supplemental
	95.4	static	LC50=8,000 (5,400-12,000)		
Bluegill	Tech	static	LC50=10,000 (8,600-12,000); NOEC=6,000	00018723	Core
Channel catfish	Tech	static	LC50=4,900 (3,600-6,800); NOEC<2,100	00015534	core
Crucian carp	Tech	static	LC50=4,900 (3,600-6,800); NOEC<2,100	00015534	supplemental
Guppy	Tech	static	LC50=8,600 (7,400-10,500), slope=11.0 (ppm); NOEC<6,500	00015534	supplemental
Sheepshead minnow	97.3	flow through	LC50=9,800 (8,500-11,400); NOEC=3,600	43487101	core
	97	static	LC50=7,900 (4,400-inf); NOEC=4,400	43044602	supplemental
	Tech		NOEC = 1,300 LOEC = > 1.300	Sousa, 2000	NA
S-Metolachlor					
Rainbow trout	97.6	static	LC50=11,900 ^a (8,300-15,000); NOEC=2,500	43928911	core
Bluegill	N.R.	static	LC50=3,200 (2,800-4,600), slope=14.8 (ppm); NOEC=1,500	43928910	Core
Zebra Danio	98.4	static	LC50=46,210 (40,800-52,730)	Quintaneiro et al., 2017 (178065)	not coded
Sheepshead minnow	98.9	static renewal	LC50=17,000 (12,100-23,300); NOEC=6,000	46829506	Supplemental

^aValue appears in Risk-plots within Chapters 12 & 15

11.4.6.2 *Salmonid Growth And Fitness*

Only two thresholds for statistically significant impacts to growth (i.e., LOECs) were reported in the OPP database: one for a sheepshead minnow exposure to metolachlor and one for a fathead minnow exposure to S-metolachlor. A LOEC of >1,300 ppb and a NOEC of 1,300 ppb was reported for 34-day exposures of sheepshead minnow to metolachlor, technical (Sousa, 2000). A

⁷ Federal Register. Volume 68, Number 63, Rules and Regulations, pp 15945-15958. April 2, 2003

LOEC of 56 ppb and NOEC of 30 ppb was reported for a 30-day flow through study exposing fathead minnow to 98.6 percent S-metolachlor (MRID 44995903 – core). Searches of ECOTOX and the open literature did not identify additional data on the effects of chronic exposures to metolachlor. Additionally, behavioral impacts were observed in bluegill sunfish, rainbow trout, and sheepshead minnow (see Table 22).

Table 22 Fish LOEC and NOEC data for growth and fitness responses to metolachlor.

Response	Species	Toxicity Value (ppb)	MRID	Fulfills guideline?
Growth	Fathead Minnow	NOEC = 30 ^a LOEC = 56	44995903	Acceptable
Behavior	Bluegill sunfish	NOEC = 2590 ^a LOEC = 3290	43928910	Acceptable
	Rainbow Trout	NOEC = 2500 ^a LOEC = 5300	43928911	Acceptable
	Sheepshead Minnow	NOEC = 6040 ^a LOEC = 12100	46829506	Acceptable

^aValue appears in Risk-plots within Chapters 12 & 15

Invertebrate Prey

Metolachlor is considered slightly to moderately toxic to aquatic invertebrates upon acute exposure, with marine invertebrates more sensitive than freshwater invertebrates. Data for the effects of acute and chronic exposures to metolachlor on invertebrate prey are presented in Table 23 and Table 24, respectively. An LC50 of 1,100 ppb for water flea (Foster et al. 1998), was applied quantitatively in the Metolachlor Draft Risk Assessment and Problem Formulation, but was not used in the Metolachlor BE. The S-metolachlor LC50 of 26,000 ppb was applied in all three of the EPA Metolachlor Risk Analyses.

Table 23 Acute toxicity data for aquatic invertebrates exposed to metolachlor.

Species	Purity (%)	Exposure Duration	Endpoint	MRID or ECOTOX reference	EPA data quality designation
Metolachlor					
Water Flea	87	48 hours, static	EC50=26,000 (19,400-34,900)	40098001; Mayer and Ellersiek, 1986 (6797)	not coded
	95.4	48 hours, static	EC50=23,500 ^a (18,700-29,500)		supplemental
	97.2	24 hours,	LC50=80,000	Wan et al., 2006 (89626)	not coded
	97.2	48 hours,	LC50=13,000		
	not reported	24 hours, static	EC50=5,100 (1,600-16,000)	EO67777; Foster et al., 1998 (67777)	Supplemental; qualitative
		48 hours, static	EC50=1,100 (900-1,400)		
		48 hours, static	EC50=2,000 (1,600-2,400)		
		87EC	48 hours, static	EC50=23,500 ^a (19,400-34,900)	40098001
	Tech	48 hours, static	EC50=25,100 (21,600-29,200); NOEC=5,600	00015546	core

Midge	87	48 hours, static	EC50=4,400 (3,200-6,100)	40098001; Mayer, Jr. and Ellersiek, 1986 (6797)	not coded
	95.4	48 hours, static	LC50=3,800 (2,100-10,300)		supplemental
	95.4	48 hours, static	EC50=3,800 (2,100-10,300)		not coded
	97	72 hours, static	LOEC=1,000; NOEC=100	Jin-Clark et al., 2008 (105238)	not coded
	97.1	48 hours, static	NOEC=200	Perez et al., 2013 (165182)	not coded
	not reported	96 hours, static	LC50=13,282 (12,612-13,983)	Osano et al., 2002 (65836)	not coded
	87E	48 hours, static	LC50=4,400 (3,200-6,100)	40098001	supplemental
Rusty Crayfish	96.1	96 hours, renewal	LOEC=80; NOEC=70	Cook and Moore, 2008 (109340)	not coded
Scud	97.2	96 hours,	LC50=6,000	Wan et al., 2006 (89626)	not coded
Snail	84.4	24 hours, static	NOEC=100	Elias and Bernot, 2017 (175884)	not coded
European Physa	84.4	24 hours, static	LOEC=100	Elias and Bernot, 2017 (175884)	not coded
Eastern oyster	97.3	96 hours, flow through	EC50=1,600 (1,400-1,900), slope=4,970; NOEC=710	43487102	core
Mysid	97.3	96 hours, flow through	LC50=4,900 (4,200-5,900), slope=6,060; NOEC=2,300	43487103	core
S-Metolachlor					
Water flea	97.6	48 hours, static	EC50=26,000 ^b (23,000-30,000), slope=9,100; NOEC=4,800	43928912	core
Amphipod	98.4	96 hours, static	EC50=42,900 (40,040-46,530)	Maazouzi et al., 2016 (174634)	not coded
Aquatic Sowbug	98.4	96 hours, static	EC50=11,780 (9,110-14,650)	Maazouzi et al., 2016 (174634)	not coded
Scud	98.4	96 hours, static	EC50=8,470 (6,870-10,430)	Maazouzi et al., 2016 (174634)	not coded
	98.4	96 hours, static	EC50=10,590 (9,390-12,770)		not coded
	98.4	96 hours, static	EC50=11,210 (9,600-13,490)		not coded
Eastern oyster	98.9	96 hours, flow through	EC50=4,000 (3,500-4,100); NOEC=645	46829505	Core

^aValue appears in Risk-plots within Chapters 12 & 15

Among chronic data, a growth and reproduction LOEC of 6,900 ppb and NOEC of 3,200 ppb were applied from a supplemental study (MRID 43802601). Due to variability in the measured concentrations for this study, the LOEC and NOEC endpoints applied are the lowest measured replicate concentration at each respective treatment level (nominal concentrations of 10,000 ppb and 5,000 ppb, respectively).

Table 24 Chronic toxicity data for aquatic invertebrates exposed to metolachlor.

Species	Purity (%)	Exposure	response	Endpoint (ppb)	MRID or ECOTOX reference	EPA data quality designation
Metolachlor						
Water flea	97	21 days, flow through	Growth and reproduction	LOEC=6,900;	43802601	supplemental
	97.2	21 days, flow through		NOEC=3,200 ^b		
	97.2	21 days, flow through		EC50=12,400 (10,300-15,300); NOEC=9,400		
S-Metolachlor						
Midge	98.5	28 days, spiked water, static	Growth	LOEC=7,200;	49579501	supplemental
	98.5	30 days, overwater, static	Growth	NOEC=3,200 LOEC>5,300; NOEC=5,300		
Mysid	98.6	28 days, flow through	Growth	LOEC=250;	44995902	core
Water flea	98.9	21 days, flow through	Growth	LOEC=10,000;	46829507	Core
	96	21 days,	Population & Reproduction	NOEC=5,170 LOEC=500;		
			Growth	NOEC=100 LOEC=1,000; NOEC=500		
			Survival	LOEC=10,000; NOEC=5,000		Supplemental; Test substance was not quantified during test, qualitative use in risk characterization

^b The Metolachlor Problem Formulation applied the lowest measured concentration at each treatment level due to variability in the measured concentrations.

11.4.6.3 *Phytoplankton And Aquatic Vascular Plants*

The ECOTOX contained abundant data for aquatic plant life (Table 25). The quality of this data varied, with some studies exposing test organisms to a single metolachlor concentration and more detailed studies, such as Vallotton et al. (2008), which reported responses at several concentrations over multiple points on the pre-exposure-exposure-recovery time scale. The ECOTOX data have not been coded as either core, supplemental, or invalid. Included here, these data place the coded data from the OPP database in context of the breadth of available information, particularly information about nonstandard lab species and the variability in sensitivity even within species groups (e.g., freshwater diatoms within the Larras et al. 2012 study). The lowest EC50 reported in ECOTOX was 50 ppb (St-Laurent et al. 1992) and about half of the EC50s reported in ECOTOX were below 380 ppb.

Table 25. Toxicity data for aquatic plants exposed to metolachlor.

Species	Purity (%)	Exposure	Response	Endpoint (ppb)	MRID or ECOTOX reference	EPA data quality designation
Metolachlor						
Algae	not reported	91.32 days, lotic	Ecosystem respiration	LOEC=274	Day, 1993 (13325)	not coded
Aquatic Macrophyte	not reported	196 hours, static	Growth	LOEC>3,000, NOEC>3,000	Fairchild et al., 1994 (152770)	not coded
Blue-green algae						
Unspecified species	97.3	5 days, static	Growth and reproduction	EC50=1,200 (900-1,600), slope=1,220, NOEC=63	43487104	core
<i>Anabaena flosaquae</i> (also <i>Microcystis</i> sp.)	95	96 hours, static	Population Chl-a	EC50>3,000	Fairchild et al., 1998 (19461)	not coded
<i>Anabaena</i> sp.	not reported	96 hours, static	Abundance	LOEC>3,000, NOEC>3,000	Fairchild et al., 1994 (152770)	not coded
<i>Microcystis</i> sp.			Abundance	LOEC=1,500, NOEC=750		
Chrysophyte	not reported	renewal	Population-growth rate	NOEC=2	Wei et al., 2013 (164067)	not coded
Coon-Tail	95	196 hours, static	Biomass	EC50=70 (62-78)	Fairchild et al., 1998 (19461)	not coded
	not reported	196 hours, static	Growth	LOEC=94, NOEC=47	Fairchild et al., 1994 (152770)	not coded
Diatoms						
<i>Skeletonema marinoi</i>	not reported	9 days, static	Photosynthesis and population growth rate	LOEC=15, NOEC=5	Fiori and Pistocchi, 2014 (166984)	not coded
<i>Ulnaria ulna</i>	98	96 hours, static	Population Chl-a	EC05=60 (52-68) EC50=3,314 (2609-3570)	Larras et al., 2012 (161002)	not coded
<i>Achnanthydium minutissimum</i> , <i>Cyclotella meneghiniana</i> , <i>Encyonema silesiacum</i> , <i>Gomphonema parvulum</i> , and <i>Mayamaea fossalis</i>	98	96 hours, static	Population Chl-a	EC05= 54 to 5,957 EC50= 3,476 to 10,313		
<i>Eolimna minima</i> , <i>Fragilaria capucina</i> ssp. <i>Rumpens</i> , <i>Nitzschia palea</i> , and <i>Fragilaria capucina</i> var. <i>vaucheriae</i>	98	96 hours, static	Population Chl-a	EC50>50,000		
Duckweed	95	96 hours, static	Abundance	EC50=360 (323-398)	Fairchild et al., 1998 (19461)	not coded
	97.3	196 hours, static	Growth and reproduction	EC50=48 (43-56), NOEC=8	43487105	core
	not reported	96 hours, static	Population changes	EC50=343 (187-872), LOEC=375, NOEC=187	Fairchild et al., 1997 (18093)	not coded
			Abundance	LOEC=375, NOEC=187	Fairchild et al., 1994 (152770)	not coded
		Static	Biomass	LOEC=75, NOEC=187	Fairchild et al., 1997 (18093)	not coded

Species	Purity (%)	Exposure	Response	Endpoint (ppb)	MRID or ECOTOX reference	EPA data quality designation
Floating Moss	not reported	28 days, static	Population Biomass	EC50=150	Goncz and Sencic, 1994 (13738)	not coded
Freshwater diatom	97.3	5 days, static	Growth and reproduction	EC25=42*; EC50=380 (270-560) slope=890, NOEC=4	43541302	core
Green algae	97.3	5 days, static	Growth and reproduction	EC50=10 (6-20), slope=1,700, NOEC=1	43541301	core
<i>Chlamydomonas moewusii</i>	95	12 days, static	Biomass and growth rate	LOEC=6,300, NOEC=63	Kotrikla et al., 1997 (178703)	not coded
<i>Chlamydomonas reinhardtii</i>	95	96 hours, static	Population Chl-a	EC50=1,138 (987-1290)	Fairchild et al., 1998 (19461)	not coded
<i>Chlamydomonas</i> sp.	not reported	96 hours, static	Abundance	LOEC=375, NOEC=188	Fairchild et al., 1994 (152770)	not coded
<i>Chlorella fusca</i>	95	12 days, static	Biomass, growth rate and abundance	EC50=101 to 108	Kotrikla et al., 1997 (20116)	not coded
<i>Chlorella fusca</i> ssp. <i>fusca</i>	95	96 hours, static	Population growth rate	EC50=157 to 178	Kotrikla et al., 1999 (174736)	not coded
<i>Chlorella fusca</i> var. <i>vacuolata</i>	97	24 hours, static		EC50=232 (217-247), NOEC=120	Junghans et al., 2006 (163051)	not coded
<i>Chlorella pyrenoidosa</i>	50	96 hours, static		EC50=12,704	Ma et al., 2002 (158793)	not coded
	96	96 hours, static	Abundance	EC50=152, Chl-a	Liu and Xiong, 2009 (118860)	not coded
	not reported	0.67 hours static	Photosynthesis	LOEC=28,380, NOEC=2,838	Pillai and Davis, 1975 (41594)	not coded
	not reported	1 hour static		LOEC=2,838, NOEC=284		
	not reported	1.3 to 2.3 hours		LOEC=28,380, NOEC=2,838		
<i>Chlorella</i> sp.	not reported	96 hours, static	Abundance	LOEC=150, NOEC=75	Fairchild et al., 1994 (152770)	not coded
<i>Chlorella vulgaris</i>	50	96 hours, static	Population growth rate	EC50=18,926	Ma et al., 2002 (65938)	not coded
	95	96 hours, static	Population Chl-a	EC50=203 (160-246)	Fairchild et al., 1998 (19461)	not coded
<i>Pseudokirchneriella subcapitata</i>	50	96 hours, static	Abundance	EC50=5,508	Ma et al., 2006 (83543)	not coded
	95	48 hours, static	Population growth rate	EC10=14 (5-36), EC50=210 (140-310)	Kusk et al., 2018 (180320)	not coded
	95	96 hours, static	Population Chl-a	EC50=84 (72-95)	Fairchild et al., 1998 (19461)	not coded
	97.1	48 hours	Population growth rate	EC50=159	Perez et al., 2011 (165277)	not coded
	97.1	72 hours		EC50=98, LOEC=77, NOEC=25		
	not reported	72 hours, static	Abundance	EC50=72 (44-119), NOEC=30	Sbrilli et al., 2005 (98204)	not coded

Species	Purity (%)	Exposure	Response	Endpoint (ppb)	MRID or ECOTOX reference	EPA data quality designation
<i>Scenedesmus acutus</i> var. <i>acutus</i>	50	96 hours, static	Population changes	EC50=77 (70-84), LOEC=75, NOEC=38	Fairchild et al., 1994 (152770)	not coded
		96 hours, static	Abundance	EC50=50.9-55.5	St. Laurent et al., 1992 (45196)	not coded
		static	Biomass	LOEC=75, NOEC=38	Fairchild et al., 1997 (18093)	not coded
		96 hours, static	Population growth rate	EC50=19,381	Ma and Liang, 2001 (61984)	not coded
<i>Scenedesmus quadricauda</i>	50	96 hours, static		EC50=600	Ma et al., 2003 (71458)	not coded
<i>Scenedesmus</i> sp.	97		Population Chl-a	EC50>3,000	Fairchild et al., 1998 (19461)	not coded
		24 hours, static	Abundance	EC50=232, NOEC=120	Junghans et al., 2003 (73426)	not coded
	not reported	96 hours, static		LOEC>3,000, NOEC>3,000	Fairchild et al., 1994 (152770)	not coded
Marine diatom	97.3	5 days, static	Growth and reproduction	EC50=61 (49-76), slope=1,000, NOEC=2	43487106	core
Pennate Diatom	98	96 hours, static	Population Chl-a	EC05=2,575 (1729-2999), EC50=30,147 (17,134- 44,657)	Larras et al., 2012 (161002)	not coded
Plant Kingdom	97.1	16 - 36 days, lentic	Population Chl-a and Biomass	NOEC=7.4	Relyea, 2009 (114296)	not coded
Sago Pondweed	not reported	3 hours static	Photosynthesis	IC50>10, LOEC=5	Fleming et al., 1995 (70739)	not coded
Two-Leaf Water-Milfoil	95	196 hours, static	Population Biomass	EC50>3,000	Fairchild et al., 1998 (19461)	not coded
Water Milfoil	98	196 hours, static	Growth (various conditions)	IC25=150-675, IC50=580- 1,896, NOEC=36.9-2,990,	Roshon, 1997 (74985)	not coded
	not reported	196 hours, static	Growth	LOEC>3,000, NOEC>3,000	Fairchild et al., 1994 (152770)	not coded
Water Nymph	95	196 hours, static	Population Biomass	EC50=242 (164-321)	Fairchild et al., 1998 (19461)	not coded
	not reported	196 hours, static	Growth	LOEC>750, NOEC>750	Fairchild et al., 1994 (152770)	not coded
Waterweed	95	196 hours, static	Population Biomass	EC50=2,355 (2,118-2,593)	Fairchild et al., 1998 (19461)	not coded
S-Metolachlor						
Wavyleaf Sealavender	not reported	29 days, foliar spray and 39 days, direct application	Growth	NOEC=2	Gilreath, 1985 (121097)	not coded
Blue-green algae	98.9	96 hours, static	Growth and reproduction	EC50=21,000 (19,000- 23,000), slope=5,680, NOEC=9,600	46829510	core
Diatom Class	not reported	6 days, static	Cell density	NOEC< and LOEC= from t0=5.1 to 1.6 ppb at day 6	Debenest et al., 2009 (118861)	not coded

Species	Purity (%)	Exposure	Response	Endpoint (ppb)	MRID or ECOTOX reference	EPA data quality designation
	not reported	72 hours exposed, 72 hours recovery, static	Cell density	NOEC from t0=24.2 to 1.8 ppb at day 6		
Diatom Family	not reported	96 hours, static	Population Chl-a	EC50=10,271 (6,642-15,279), EC50=5,888 (4,337-7,607)	Roubeix et al., 2012 (178311)	not coded
Diatom: <i>Nitzschia obtusa</i> var. <i>nana</i>	not reported	96 hours, static	Population Chl-a	EC50=18,000, EC50=18,179 (15,823-20,522), EC50=20,580 (18,966-22,072), LOEC=11,850	Roubeix et al., 2012 (178311)	not coded
Duckweed	97.6	14 days, static	Growth and reproduction	EC25=13 ^a ; EC50=23 (frond density); EC50=31 (frond biomass)	43928931	core
	87.4	7-day, semi-static	Growth	EC50 growth rate/yield = 133/37 (frond numbers); EC50 growth rate/yield = >916/75 (dry weight) ^b	Eckenstein, 2014	not coded
Freshwater diatom	98.9	96 hours, static	Growth and reproduction	EC50=18,000 (17,000-20,000), slope=3,730, NOEC=4,080	46829509	core
Green algae	97.6	5 days, static	Growth and reproduction	EC25=4.8 ^a ; EC50=8 (2.6-25) slope=3, NOEC=2	43928929	core
<i>Chlamydomonas reinhardtii</i>	not reported	48 hours, static	Reproduction	EC50=1,958 (1,760-2,157)	Korkaric et al., 2015 (172697)	not coded
<i>C. reinhardtii</i> strains	not reported	48 hours, static	Population growth rate	EC50=1,419 to 7,265	Fischer et al., 2012 (172723)	not coded
<i>Chlorella fusca</i> var. <i>vacuolata</i>	98.4	24 hours, static (t0 to t24)	Population growth rate	EC50=341 (300-389) EC50s for segments within exposure period	Vallotton et al., 2008 (112203)	not coded
<i>Chlorella pyrenoidosa</i>	96	96 hours, static	Abundance	EC50=68	Liu and Xiong, 2009 (118860)	not coded
<i>Scenedesmus acutus</i> var. <i>acutus</i>	96	96 hours, static	Population growth rate	EC50=156 (107-227)	Bian et al., 2009 (118780)	not coded
<i>Pseudokirchneriella subcapitata</i>	88.7	96 hours, static	Growth	EC50 = 32 (biomass), 77 (growth rate) ^c	Memmert, 2006	not coded
Marine diatom	97.6	5 days, static	Growth and reproduction	EC50=110 (91-128), NOEC=21	43928930	core
Red foxtail watermilfoil	98.9	21 days, static renewal	Growth and reproduction	EC50>1,000, NOEC<100	46861401	core

^a Value appears in Risk-plots within Chapters 12 & 15

^b Recovery after continuous exposure to test concentrations observed after 2-6 weeks.

^c Recovery observed after 3-12 days following exposure to highest test concentration.

11.4.6.4 Terrestrial (Riparian) Vegetation

Riparian vegetation is important for providing shade to the stream, stabilizing the stream banks, reducing sedimentation, and providing organic material inputs, both in terms of plant material and terrestrial insects. Riparian vegetation is a major focus of restoration efforts within California, and when present can reduce pesticide loading into aquatic resources. Riparian vegetation is an important assessment endpoint for herbicidal impacts on salmon habitats. Generally, there are sparse data regarding the effects of herbicides (and much less with insecticides, arachnidicides, or miticides) on wild plants within riparian systems, other than weed species. The EPA requires submission of crop effects data as part of the registration process for herbicides. This information currently provides the only basis for evaluating effects on herbaceous plants unless data are available from other sources. The overall assumption is that the sensitivity of plant species tested (typically plants used in agriculture) in the registrant-provided guideline studies will be representative of riparian species. There is no way to know this is the case, therefore a high degree of uncertainty regarding the toxicity of the a.i.s to riparian vegetation exists.

The standardized and coded studies from the OPP database (Table 26) show that metolachlor is generally more toxic to monocot seedling emergence and vegetative vigor than dicots with the most sensitive endpoint being dry weight. S-metolachlor seedling emergence EC25 concentrations for both dicots and monocots were an order of magnitude lower than seedling emergence EC25s for metolachlor. Shoot weight was the most sensitive endpoint for dicots and visible evidence of toxicity was the most sensitive endpoint for monocots. Visible evidence of toxicity was the sensitive endpoint for both dicot and monocots in vegetative vigor tests. At >0.02 pounds per acre for both dicots and monocots, the seedling emergence EC25s for S-metolachlor emulsified concentrate did not differ greatly from the metolachlor seedling emergence EC25s. The EC25s for vegetative vigor were an order of magnitude higher at >0.533 and >0.357 for dicots and monocots, respectively. The LOECs for seedling emergence ranged from 1.3 to 2.7 pounds per acre for Stoke's aster.

Table 26 Toxicity of metolachlor to terrestrial plants.

Study Type	% AI	Species group	Lowest EC25 (lb ai/A)	Most Sensitive Endpoint	MRID #	EPA data quality designation
Metolachlor						
seedling emergence	97.3	dicots monocots	>0.09 (n=6) >0.02 (n=4)	dry weight	43487107	core
vegetative vigor	97.3	dicots monocots	>0.03 (n=6) >0.016 (n=4)	dry weight	43487108	core
S-Metolachlor						
seedling emergence	97.6	dicots monocots	>0.0057 (n=2) >0.0048 (n=4)	shoot weight toxicity/chlorosis	43928932	Supplemental

vegetative vigor	97.6	dicots monocots	>0.27 (n=2) >0.021 (n=4)	toxicity/chlorosis	43928933	Supplemental
S-Metolachlor EC						
seedling emergence	86.3	dicots monocots	>0.021 (n=6) >0.0223 (n=6)	shoot weight	49930012 ^a	core
vegetative vigor	86.3	dicots monocots	>0.533 (n=6) >0.357 (n=6)	shoot height shoot weight	49930013 ^a	core

^aValues in this study appear in Risk-plots within Chapters 12 & 15

Data for terrestrial plants reported in ECOTOX as growth or population response EC50s ranging from 0.0022 pounds per acre for foxglove to 3.6 pounds per acre for bachelors button, a LOEC from the same study at 0.022 pounds per acre for catmint (artificial soil, Boutin et al. 2004) up to 3.6 pounds per acre for soybean (field exposure, Bowman 1985) and NOECs from 0.022 pounds per acre for black bindweed (artificial soil, Boutin et al. 2004) up to 8.8 pounds per acre for holly (natural soil, field exposure, Catanzaro et al. 1993). While these endpoints are not relatable to the endpoint data for the coded studies in the OPP database, they illustrate the breadth in response thresholds among non-standard test species and study designs and illustrate that the controlled studies reported in the OPP database are representative of the most sensitive responses.

11.4.6.5 Field Studies

Field studies on the effects of metolachlor or S-metolachlor on aquatic life were not identified in the ECOTOX or a search of the open literature.

11.4.6.6 Field Incidents

The Metolachlor Draft Ecological Risk Assessment summarized the results of an Incident Data System (IDS) query conducted on 6/5/2019. The IDS is an integrated summary of the EIIS and aggregate incident reports submitted by registrants to EPA since registration. The search returned a total of 623 reported ecological incidents associated with the use of S-metolachlor and metolachlor, most of which were reviewed in the Metolachlor Problem Formulation. Reports include 14 fish incidents; however, there is little other information on these and most are classified as unlikely or possible and involved products that included other active ingredients (e.g., atrazine). A few of the fish incidents, classified as highly-probable or probable, indicated metolachlor as the cause of fish kills following mis-use, no other details were provided. A total of 597 incidents were related to crop (e.g., corn, cotton, and soybean) damage following direct treatment of an agricultural field. While these incidents represent evidence of environmental exposures to metolachlor, NMFS does not consider them contributing appreciably to the effects of the action.

11.4.6.7 *Bioconcentration And Bioaccumulation*

Bioconcentration and bioaccumulation information is not typically reported in the OPP database. The ECOTOX database includes three records for accumulation of metolachlor, but the controls for these studies were considered to be insufficient and magnification factors were not calculated. Compounds with a log KOW of three and above are generally considered to have the potential to bioconcentrate in aquatic organisms. The potential for bioconcentration of metolachlor in organisms is considered low given the measured bioconcentration factor (BCF) of 69X in fish and depuration value of 93% in 14 days once fish were transferred to untreated water. (MRID 41154201). The Metolachlor Draft Risk Assessment concluded that, based on the octanol-water partition coefficient (KOW) of 3.05, there is potential for exposure to sediment dwelling organisms.

11.4.6.8 *Degradate Toxicity*

In evaluating the toxicity data for these structurally similar metabolites, EPA's 2019 Draft Risk Assessment concluded that they are far less toxic than the parent metolachlor and were thus not residues of concern for ecological exposure. Accordingly, NMFS did not include these metabolites in its analyses.

11.5 Assessing Risk

Population Models

Sufficient data were available to construct population models for four Pacific salmon life history strategies. We ran life-history matrix models for ocean-type and stream-type Chinook salmon (*O. tshawytscha*), coho salmon (*O. kisutch*), and sockeye salmon (*O. nerka*). The basic salmonid life history we modeled consisted of hatching and rearing in freshwater, smoltification in estuaries, migration to the ocean, maturation at sea, and returning to the natal freshwater stream for spawning followed shortly by death. An acute toxicity model was constructed that estimated the population-level impacts of sub-yearling juvenile mortality resulting from exposure. For specific information on the construction and parameterization of the models see Appendix A. Potential population-level impacts resulting from mortality following freshwater exposure to pesticides were integrated into the models as alterations in the first year survival rate. We also evaluated population level responses resulting from varying the proportion of the population exposed. Population level impacts were assessed as changes in the intrinsic population growth rate and quantified as the percent change in population growth rate. The results of the models are shown in Table 27, Table 28, Table 29, and Table 30. Changes that exceeded the variability in the baseline (*i.e.*, a standard deviation) were considered to be different. Importantly, the acute toxicity models excluded sublethal and indirect effects of the pesticide exposures. For example, the potential population-level impacts of reduced prey abundance are not captured by these models.

Table 27. Acute mortality model output for ocean-type Chinook. Shown are the percent changes in population growth rate (λ) with the standard deviations in parentheses. The toxicity values were applied as direct mortality on first year survival (left column). The percent of the population exposed was also varied (top row). Bold indicates a percent change in population growth rate of greater than one standard deviation from control values. The baseline values for ocean-type Chinook are: $\lambda=1.09$, standard deviation of 0.1, standard deviation as a percent of λ is 9, and first year survival $S1=5.64E-03$. Bold indicates values greater than or equal to one standard deviation away from baseline.

% population experiencing mortality					
% mortality	10	25	50	80	100
5	0 (12.9)	0 (12.9)	-1 (12.8)	-1 (12.8)	-1 (12.7)
10	0 (13.0)	-1 (12.9)	-1 (12.8)	-3 (12.6)	-3 (12.4)
15	0 (12.9)	-1 (12.9)	-2 (12.8)	-4 (12.5)	-5 (12.2)
20	-1 (13.0)	-2 (13.0)	-3 (12.9)	-5 (12.5)	-6 (12.1)
25	-1 (13.1)	-2 (13.0)	-4 (13.3)	-6 (12.7)	-8 (11.8)
30	-1 (13.0)	-2 (13.3)	-5 (13.4)	-8 (12.7)	-10 (11.5)
35	-1 (13.3)	-3 (13.8)	-6 (13.9)	-9 (13.0)	-12 (11.4)
40	-1 (13.4)	-3 (14.0)	-7 (14.3)	-11 (13.5)	-14 (11.1)
45	-1 (13.6)	-4 (14.3)	-8 (15.4)	-13 (14.1)	-16 (10.7)
50	-2 (13.6)	-5 (14.9)	-9 (16.0)	-15 (15.3)	-18 (10.5)
55	-2 (14.0)	-5 (15.5)	-11 (17.5)	-17 (16.5)	-21 (10.2)
60	-2 (14.2)	-6 (16.9)	-12 (18.6)	-20 (17.9)	-23 (9.7)
65	-2 (14.3)	-7 (16.9)	-14 (19.8)	-22 (19.1)	-26 (9.5)
70	-3 (14.6)	-7 (17.8)	-16 (21)	-24 (20.3)	-29 (8.9)
75	-3 (15.2)	-8 (18.4)	-17 (22.1)	-27 (21.6)	-33 (8.5)
80	-3 (15.3)	-9 (19.7)	-18 (23.2)	-30 (22.3)	-37 (8.1)
85	-4 (15.8)	-10 (20.4)	-20 (24)	-32 (23.1)	-42 (7.3)
90	-4 (16.1)	-10 (21.5)	-21 (24.9)	-34 (23.4)	-48 (6.6)
95	-4 (16.5)	-11 (22.7)	-22 (25.3)	-36 (23.2)	-56 (5.5)
100	-4 (17.1)	-12 (23.0)	-23 (25.9)	-38 (23.6)	-100 (NA)

Table 28. Acute mortality model output for stream-type Chinook. Shown are the percent changes in population growth rate (λ) with the standard deviations in parentheses. The toxicity values were applied as direct mortality on first year survival (left column). The percent of the population exposed was also varied (top row). Bold indicates a percent change in population growth rate of greater than one standard deviation from control values. The baseline values for stream-type Chinook are: $\lambda=1.00$, standard deviation of 0.03, standard deviation as a percent of λ is 3, and first year survival $S_1=6.43E-03$. Bold indicates values greater than or equal to one standard deviation away from baseline.

		% population experiencing mortality				
% mortality		10	25	50	80	100
5		0 (4.4)	0 (4.4)	-1 (4.4)	-1 (4.4)	-1 (4.3)
10		0 (4.5)	-1 (4.5)	-1 (4.5)	-2 (4.4)	-3 (4.3)
15		0 (4.6)	-1 (4.7)	-2 (4.7)	-3 (4.6)	-4 (4.2)
20		-1 (4.7)	-1 (4.9)	-3 (5.1)	-4 (4.8)	-5 (4.1)
25		-1 (4.8)	-2 (5.1)	-3 (5.5)	-6 (5.1)	-7 (4.1)
30		-1 (4.9)	-2 (5.6)	-4 (6.0)	-7 (5.6)	-8 (4.0)
35		-1 (5.1)	-2 (6.0)	-5 (6.8)	-8 (6.1)	-10 (4.0)
40		-1 (5.4)	-3 (6.5)	-6 (7.5)	-10 (6.9)	-12 (3.9)
45		-1 (5.6)	-3 (7.0)	-7 (8.5)	-11 (7.8)	-14 (3.7)
50		-2 (5.8)	-4 (7.5)	-8 (9.8)	-13 (9.3)	-16 (3.7)
55		-2 (6.2)	-4 (8.3)	-9 (11.1)	-15 (10.9)	-18 (3.6)
60		-2 (6.5)	-5 (9.3)	-11 (13.0)	-17 (13.1)	-20 (3.5)
65		-2 (6.9)	-6 (10.1)	-12 (14.7)	-19 (14.7)	-23 (3.4)
70		-2 (7.2)	-6 (11.1)	-13 (15.7)	-22 (16.7)	-26 (3.2)
75		-3 (7.7)	-7 (12.4)	-15 (17.5)	-24 (17.9)	-29 (3.1)
80		-3 (8.1)	-8 (13.5)	-15 (18.3)	-27 (18.8)	-33 (2.9)
85		-3 (8.6)	-8 (14.6)	-17 (19.3)	-29 (19.7)	-37 (2.7)
90		-3 (9.1)	-9 (15.4)	-18 (20.2)	-30 (20.0)	-43 (2.4)
95		-4 (9.5)	-10 (16.4)	-20 (21.1)	-32 (20.2)	-52 (2.0)
100		-4 (10.3)	-11 (17.6)	-21 (21.4)	-33 (20.0)	-100 (NA)

Table 29. Acute mortality model output for sockeye. Shown are the percent changes in population growth rate (λ) with the standard deviations in parentheses. The toxicity values were applied as direct mortality on first year survival (left column). The percent of the population exposed was also varied (top row). Bold indicates a percent change in population growth rate of greater than one standard deviation from control values. The baseline values for sockeye are: $\lambda=1.01$, standard deviation of 0.06, standard deviation as a percent of λ is 6, and first year survival $S_1=2.57E-02$. Bold indicates values greater than or equal to one standard deviation away from baseline.

% population experiencing mortality					
% mortality	10	25	50	80	100
5	0 (8.0)	0 (7.9)	-1 (7.9)	-1 (7.8)	-1 (7.8)
10	0 (8.0)	-1 (8.0)	-1 (8.0)	-2 (7.9)	-3 (7.7)
15	0 (8.0)	-1 (8.0)	-2 (8.1)	-3 (7.9)	-4 (7.7)
20	-1 (8.0)	-1 (8.2)	-3 (8.2)	-4 (8.1)	-5 (7.5)
25	-1 (8.1)	-2 (8.4)	-3 (8.5)	-5 (8.2)	-7 (7.4)
30	-1 (8.2)	-2 (8.8)	-4 (9.0)	-7 (8.4)	-8 (7.3)
35	-1 (8.4)	-2 (8.9)	-5 (9.6)	-8 (8.8)	-10 (7.1)
40	-1 (8.6)	-3 (9.2)	-6 (10.1)	-9 (9.6)	-11 (7.0)
45	-1 (8.7)	-3 (9.7)	-7 (10.9)	-11 (10.4)	-13 (6.9)
50	-1 (9.0)	-4 (10.4)	-8 (12.0)	-13 (11.2)	-15 (6.7)
55	-2 (9.2)	-4 (10.9)	-9 (13.4)	-15 (12.9)	-17 (6.5)
60	-2 (9.4)	-5 (11.9)	-10 (14.4)	-17 (14.4)	-19 (6.4)
65	-2 (9.7)	-5 (12.3)	-12 (16.1)	-19 (15.7)	-22 (6.2)
70	-2 (10.0)	-6 (13.4)	-13 (16.9)	-21 (17.3)	-25 (5.9)
75	-3 (10.4)	-7 (14.3)	-14 (18.2)	-23 (18.1)	-28 (5.6)
80	-3 (10.9)	-8 (15.6)	-16 (19.0)	-26 (19.1)	-32 (5.4)
85	-3 (11.3)	-8 (16.3)	-17 (19.9)	-28 (19.7)	-39 (5.0)
90	-3 (11.6)	-9 (17.0)	-18 (20.8)	-29 (19.8)	-42 (4.5)
95	-3 (12.3)	-10 (17.7)	-19 (20.9)	-30 (19.9)	-51 (3.8)
100	-4 (12.7)	-10 (18.3)	-20 (21.5)	-32 (19.8)	-100 (NA)

Table 30. Acute mortality model output for coho. Shown are the percent changes in population growth rate (λ) with the standard deviations in parentheses. The toxicity values were applied as direct mortality on first year survival (left column). The percent of the population exposed was also varied (top row). Bold indicates a percent change in population growth rate of greater than one standard deviation from control values. The baseline values for coho are: $\lambda=1.03$, standard deviation of 0.05, standard deviation as a percent of λ is 5, and first year survival $S_1=2.97E-02$. Bold indicates values greater than or equal to one standard deviation away from baseline.

% mortality	% population experiencing mortality				
	10	25	50	80	100
5	0 (7.4)	0 (7.5)	-1 (7.5)	-1 (7.4)	-2 (7.4)
10	0 (7.5)	-1 (7.6)	-2 (7.6)	-3 (7.4)	-3 (7.2)
15	0 (7.6)	-1 (7.7)	-3 (7.8)	-4 (7.5)	-5 (7.1)
20	-1 (7.7)	-2 (8.0)	-4 (8.1)	-6 (7.7)	-7 (7.0)
25	-1 (7.9)	-2 (8.4)	-5 (8.5)	-7 (8.0)	-9 (6.9)
30	-1 (7.9)	-3 (8.5)	-6 (9.1)	-9 (8.4)	-11 (6.6)
35	-1 (8.2)	-3 (9.2)	-7 (9.9)	-11 (8.9)	-13 (6.5)
40	-1 (8.5)	-4 (9.7)	-8 (10.7)	-13 (9.8)	-16 (6.4)
45	-2 (8.8)	-4 (10.3)	-9 (11.8)	-14 (11.0)	-18 (6.1)
50	-2 (9.1)	-5 (11.1)	-10 (13.4)	-17 (12.2)	-21 (5.9)
55	-2 (9.5)	-6 (11.7)	-12 (14.9)	-20 (14.2)	-23 (5.8)
60	-3 (9.9)	-6 (12.6)	-14 (17.0)	-23 (16.5)	-26 (5.5)
65	-3 (10.3)	-7 (14.1)	-15 (18.5)	-25 (18.7)	-30 (5.3)
70	-3 (10.7)	-8 (15.1)	-17 (20.6)	-28 (20.6)	-33 (5.0)
75	-3 (11.2)	-9 (16.4)	-19 (22.3)	-31 (22.4)	-37 (4.7)
80	-4 (11.6)	-9 (17.7)	-20 (23.6)	-34 (23.7)	-42 (4.4)
85	-4 (12.3)	-11 (19.3)	-22 (25.0)	-37 (24.5)	-47 (4.0)
90	-4 (12.9)	-12 (20.4)	-24 (26.0)	-39 (25.2)	-54 (3.4)
95	-4 (13.4)	-13 (21.6)	-25 (27.3)	-42 (25.2)	-63 (2.8)
100	-5 (14.1)	-14 (22.9)	-27 (27.6)	-43 (25.7)	-100 (NA)

In analyzing risk, we integrate the exposure and response information to evaluate the likelihood of adverse effects from stressors of the action at the population and species level. We use two tools to integrating exposure and response, Risk-plots and where applicable, population models. A weight-of-evidence approach which considers the limitations and uncertainties inherent in the available information is then applied to characterize risk. Whenever possible, most sensitive toxicological endpoints used in the Risk-plots are from those studies that were conducted on species with best fit as surrogates to Pacific Salmonids (e.g. rainbow trout).

The following risk hypotheses for the effects of 1,3-D and metolachlor on Pacific salmonids (chum, chinook, coho, sockeye, steelhead) are based on the life history, exposure, and response considerations described in the previous sections of this chapter.

11.5.1.1 Risk Hypotheses

Salmonid:

1. Exposure to the pesticide is sufficient to reduce abundance via acute lethality.
2. Exposure to the pesticide is sufficient to reduce abundance via reduction in prey availability.
3. Exposure to the pesticide is sufficient to reduce abundance via impacts to growth (direct toxicity).
4. Exposure to the pesticide is sufficient to reduce productivity via impairments to reproduction.
5. Exposure to the pesticide is sufficient to reduce abundance and productivity via impairments to ecologically significant behaviors.

Critical Habitat:

1. Exposure to the stressors of the action is sufficient to reduce the conservation value via reductions in prey in migration, and rearing sites.
2. Exposure to the stressors of the action is sufficient to reduce the conservation value via degradation of water quality in migration, spawning, and rearing sites.
3. Exposure to the stressors of the action is sufficient to reduce the conservation value via impacts to vegetative cover in migration, spawning, and rearing sites.

Mixtures:

1. Mixtures: Formulated products and tank mixtures containing the active ingredient are anticipated to increase the risk of effects to fish in freshwater habitats.

11.6 Weighing the uncertainties in the best commercial and scientific information

All estimates of exposure and response must rely on assumptions with associated uncertainties that may contribute to the possibility of overestimating or underestimating risk, or in some circumstances may do either. Uncertainties may be due to natural variability, lack of knowledge, measurement error, or model error. Accounting for uncertainty is critical when weighing model outputs and when applying outputs in risk conclusions. This section describes how we utilized a

variety of tools with different assumptions to increase our confidence in risk estimates, and how we weighed key assumptions and associated uncertainties of our risk assessment to reach conclusions consistent with the purpose of Section 7(a)(2)⁸. In Table 31, we identify key assumptions associated with estimates utilized in our assessment of the effects of the action. X's indicate if the assumption contributes to the possibility that risk will be underestimated or overestimated. In some cases, the assumption may contribute to the possibility of either underestimating or overestimating risk, depending on the specific circumstances being evaluated. In succeeding paragraphs below the table we discuss how these assumptions and associated uncertainties are factored into our weight-of-evidence approach presented in the risk characterization section below.

Table 31. Assessment assumptions and influence on risk estimates

Assumption (estimate)	Underestimate Risk	Overestimate Risk
1. Pesticide application rates- Pesticides will be applied at the highest labeled rate for the use site or crop grouping (EECs)		X
2. Treatment of authorized use sites- Pesticides may be applied on authorized use sites (Risk-plot)		X
3. Annual maximal exposures– the risk calculation only considers the likelihood of exposure to maximum annual values (e.g. 24-hr EEC). It does not account for effects over the full effective range of predicted exposures (Risk-plot)	X	
4. GIS data layers accurately represent the presence and absence of use sites (pesticide/species overlap analysis)	X	X
5. Exposure to multiple stressors do not increase risk – The risk estimates or information do not account for other real world stressors known to exacerbate response (e.g. temperature, other pesticides, etc.) (Risk-plot)	X	
6. Species surrogacy – The sensitivity of endangered species and their prey to pesticide exposure is comparable to that of available surrogate species (Risk-plot)	X	X
7. Exposure estimates accurately predict pesticide concentrations in habitats relevant to listed species (EECs, Risk-plot)	X	X

⁸ Section 7(a)(2) of the ESA requires consultation with the Services by a Federal agency to insure a Federal action authorized, funded, or carried out is not likely to jeopardize the continued existence of any endangered species or threatened species or result in the destruction or adverse modification of habitat of such a species.

Assumption (estimate)	Underestimate Risk	Overestimate Risk
8. Responses to pesticides that degrade over time in the environment can be accurately predicted using toxicity data generated under test conditions that maintain concentrations at relatively constant concentrations (EECs, Risk-plot, Population models).	X	X
9. Effects to essential behaviors are assumed to have fitness consequences regardless of the presence/absence of a quantitative link to an apical endpoint (mortality, reproduction, or growth).	X	X

- 1) Pesticide application rate assumptions tend to **overestimate** risk: Exposure estimates assumed the pesticides are applied at the highest labeled rate for a particular crop, crop grouping, or other use site. This assumption contributes to the possibility that exposure and risk will be overestimated because applications may occur at lower than maximum rates. However, EPA’s proposed action encompasses all uses authorized by approved product labels, so this assumption is needed to determine whether label requirements are likely to avoid jeopardy to listed species and adverse modification to designated critical habitat and to “ensure that no potentially unsafe pesticide applications are ignored” (NRC NAS 2013).

- 2) Treatment of authorized use sites assumptions tend to **overestimate** risk: Treatment of authorized use sites assumptions tend to overestimate risk: Risk-plots display exposure estimates for aquatic habitats adjacent to treated uses sites. In order to evaluate the full extent of EPA’s authorization of pesticide use, we assume that pesticide treatment may occur to any use site authorized by product labeling. This assumption contributes to the possibility that exposure and risk may be overestimated. However, we do not assume that usage will occur everywhere that an authorized use site exists, nor do we assume that all usage occurs at the same day and time. Instead, we consider that pesticides may be applied to any authorized use site/location during the 15-year action. This distinction, between “will be applied to every” and “may be applied to any”, is important in understanding the assumptions of our analysis. When we consider the extent of authorized use sites within a species range (e.g. acres of corn), we do not make the assumption that pesticides will be applied to every acre of corn. Instead, we assume that: 1) the pesticide may be applied to any acre of corn 2) the greater the extent of corn acres in the species range equates to a greater chance that application may occur in close proximity to species habitat. Our risk characterization incorporates a number of factors to characterize the likelihood of exposure to the concentrations predicted by modeling (e.g. spatial overlap of use sites with range of species, seasonal overlap in use and presence of species, persistence of the compound, number of applications, and the duration of the

species residency in areas where treatment may occur). Uncertainties associated with each of these factors are incorporated into the confidence rankings that qualify each risk estimate. For example, we consider usage data compiled by EPA to help characterize the uncertainty associated with the spatial overlap analysis. In this way, evidence that pesticide usage within a species range are probable represent one factor considered in the confidence rankings to evaluate each risk hypothesis (see Chapter 4 for details regarding the likelihood of exposure assessment).

- 3) Annual maximum exposures assumptions tend to **underestimate** risk: Risk-plots display annual time-weighted average concentrations for different durations (peak 1-day, 4-day, and 21-day EECs). However, exposure to lesser concentrations (submaximal) can also contribute to risk (Figure 4). While the maximum daily peak occurs one day a year, toxic residues may persist for days, weeks, or months, depending on the frequency of repeated applications and the persistence of the pesticide. The focus on annual maximum exposures de-emphasizes the range of submaximal exposures which may also be expected to cause mortality and other adverse effects, and thus contributes to the likelihood that risk will be underestimated. Therefore, to mitigate the impact of this assumption, chemical persistence and the number of applications allowed were adopted as factors in our analysis to weigh the likelihood of exposure.

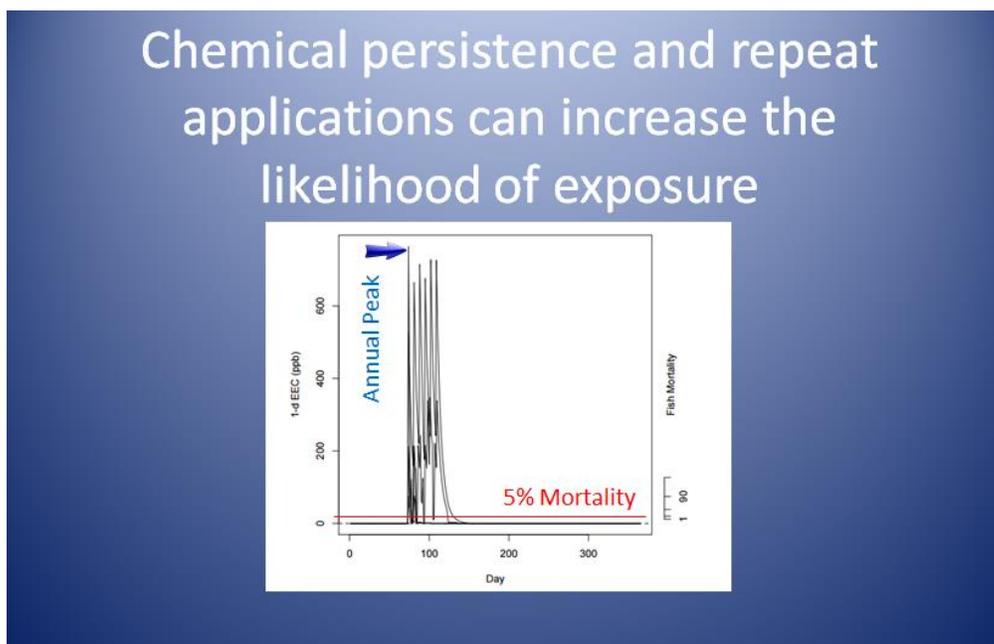


Figure 4. Conditions conducive to mortality and other adverse effects may persist for months due to the combinations of a chemical’s persistence and repeat applications. The

time series plot presented here is for illustrative purposes only and does not represent metolachlor or 1,3-D.

- 4) GIS data layer assumptions may **overestimate or underestimate** risk: Our analysis relies on GIS data layers representing land use classifications which we use as surrogates for locations where pesticides can be applied (pesticide use sites). Three issues arise that may contribute to an over- or under-estimate of risk.
 - a. Accuracy of data layers. The GIS data layers contain inaccuracies, for example, local knowledge suggests that land use type is sometimes misclassified. The extent of the inaccuracies is uncertain as information quantifying the level of inaccuracy is available for only a subset of the layers relied upon. The Cropland Data Layer (CDL) has over 100 different cultivated classes which were grouped by USEPA in order to reduce the likelihood of errors of omission and commission between similar crop categories. CDL groupings were designed to minimize uncertainties, however they also introduce the possibility that overlap percentages include uses for which metolachlor and/or 1,3-D have not been registered. Although we have confidence that registered use sites occur within the GIS layers, the extent and specific location of those use sites are somewhat less certain. We considered these uncertainties when evaluating the GIS layers as part of our “likelihood of exposure” analysis.
 - b. The estimates of acreage of use sites within a species range presented in Risk-plots rely on an assumption that recent land use (sampling from a 6-year data set) will represent future land use over the next 15 years. This assumption is uncertain as changes in cropping patterns and other land uses may contribute to assessment inaccuracies.
 - c. Data layer availability. In evaluating percent overlap we considered how well the available use-data-layer represented the labeled uses and, where feasible, made adjustments to the percent overlap value. Some 1,3-Dichloropropene labels approve applications to broadly defined use sites which required the evaluation of multiple GIS layers. For example, 1,3-Dichloropropene is approved for use on “field crops” which we assessed by evaluating 6 different CDL layers: corn, cotton, other grains, pasture, soybeans, and wheat. These GIS overlap layers are not always mutually exclusive of each other. This was taken into consideration when evaluating those labels which are represented by multiple GIS layers. Additionally, the overlap acreage and percent values associated with state-specific SLN labels represent the acreage within the species range overall, and are not specific to the state. Thus, in cases where species ranges crossed state boundaries, the state state-specific value includes acreage from outside the state. The uncertainties associated with acreage and percent overlap values were considered when making our risk and confidence characterizations. Overall, these different kinds of inaccuracy in GIS data would not tend to systematically over- or underestimate risk, and we assumed these sources of uncertainty could contribute equally to the likelihood of underestimating or overestimating exposure. When

data layers where not available to evaluate the presence/absence of use sites we expressed low confidence in risk estimates.

- 5) Assumption that exposure to multiple stressors will not increase risk may **underestimate** that risk: The risk summarized in the Risk-plots do not account for other real world stressors that may exacerbate responses to 1,3-D and metolachlor (i.e. temperature, exposure to other pesticides, etc.). This assumption contributes to the likelihood that risk will be underestimated. To account for potential increases in risk associated with multiple stressors, we evaluated the available information supporting the risk hypothesis that pesticide mixtures applied as multi-a.i. formulations or tank mixtures could increase risk from direct and indirect effects for the listed species. The mixtures' risk hypotheses were evaluated qualitatively by generating exposure and response estimates for examples of multi-a.i. pesticide formulations and tank mixtures as described in the Effects of the Action below. Exposure to other stressors, including temperature stress, was evaluated in the Environmental Baseline based on the occurrence of impaired water quality due to exceedance of temperature thresholds (Clean Water Act section 303(d) listings) in the habitat of the listed species.
- 6) Species surrogacy assumptions may **underestimate or overestimate** risk: In most instances, the sensitivity of endangered species and their prey to the stressors of the action have not been tested; their sensitivities are assumed to be comparable to surrogate species that have been tested. These assumptions may underestimate or overestimate risk, depending on the relative sensitivity among the species. Species surrogacy represents a large source of uncertainty because sensitivities among even closely related species can span several orders of magnitude. Endpoints lacked sufficient data to construct Species Sensitivity Distributions. When more than one study was available for a particular endpoint (e.g. growth) consideration was given to both the sensitivity of response as well as the surrogacy of the test species. Relevant studies with sensitive endpoints were emphasized in order to weight the analysis in a way that errors were more likely to be protective of the listed species yet consider all of the available data.
- 7) Exposure estimate assumptions may **underestimate or overestimate** risk: Exposure estimates were developed for the aquatic habitat bins with the PWC model (an integration of PRZM5 and the VVWM), as described above (11.3). The accuracy of the exposure estimates depends on how well model inputs represent site-specific conditions. We generated geographically-specific EECs for a variety of aquatic habitats (bins) for all HUC2 regions within the distribution of listed Pacific salmonids. A substantial amount of variability in environmental conditions occurs at the HUC2 scale that influences exposure. Input variables were selected to represent sites vulnerable to runoff within the region as described in EPAs organophosphate BEs (EPA 2017a; EPA 2017b; EPA 2017c). The models are designed to predict pesticide concentrations in aquatic habitats on the edge of a treated field. We expect the models to provide reasonable estimates of exposure in habitats located in close proximity to treated areas, particularly when the size of the assumed drainage area is comparable with the size of single spray applications (e.g. smaller drainages areas such as those represented by the flowing aquatic bin 2, and the static freshwater bins 5, 6, and 7). While inputs are weighted to generate estimates at the

higher end of the exposure range within the region, it's possible that exposure is underestimated for some sites (e.g. those that receive greater rainfall than assumed, or site with soil characteristics more conducive to runoff). However, overall we expect the EEC to provide reasonably accurate estimates with a tendency to overestimate exposure under most conditions. There is much greater uncertainty with regard to estimates generated for aquatic habitats represented by bin 3 and 4 with the PWC; unlike the other freshwater bin estimates which assume pesticide treatment of drainage areas consist with the size of single outdoor applications (<0.0001-600 acres), bins 3 and 4 assume drainage from much larger watersheds that would include multiple land uses, use sites, and areas where use may not be permitted (9,000-several million acres). The assumption that all of the use sites within these large watersheds are treated with pesticides tends to overestimate risk, while averaging concentrations over such large areas does not account for potential variation within the watershed and may underestimate risk when individuals are distributed in close proximity to use sites. We did not rely on EECs for bin 3 and 4 given the lack of confidence in these estimates. Even greater uncertainty exists for marine habitats where model estimates that account for complex currents and tidal exchange are not available. Consequently, we took a qualitative approach and assumed exposure in larger flowing freshwater habitats (streams and river) and marine habitats (bins 8, 9, and 10) would be something less than the concentrations predicted in runoff and in smaller streams (bin 2). We consider exposures both qualitatively and quantitatively in our conclusions.

- 8) The assumption that field and laboratory exposure result in comparable responses may **underestimate or overestimate** risk: Standardized laboratory toxicity tests typically require that pesticide concentrations be maintained at a relatively stable concentration for the duration of the exposure period. In the natural environment, pesticides continue to degrade and dissipate at varying rates depending on site-specific conditions and the pesticide's physical-chemical properties. The conventional approach for handling the uncertainty associated with the differing exposure patterns was assumed; exposure estimates using time-weighted average (TWA) concentrations that factor in degradation and dissipation were assumed to produce similar responses to toxicity test conducted under relatively constant exposure concentrations conducted with comparable exposure durations. TWA exposure estimated for acute durations (1d and 4d) were used to estimate responses based on acute toxicity studies and TWA estimates for chronic durations (21-d) were used to estimate responses using chronic studies. Utilizing average concentrations estimated under natural conditions can either underestimate or overestimate risk because response is a function of both exposure duration and concentration. Actual response may vary depending on site-specific dissipation pattern and toxicokinetic factors.
- 9) Assumptions on lack of information empirically linking effect endpoints with fitness level consequences may **underestimate or overestimate** risk: Sublethal effects to essential behaviors, such as impacts to a fish's ability to swim or a bird's ability to fly, can clearly translate to fitness level consequences by impairing an individual's ability to feed, escape predation, migrate, etc. If information is lacking to establish the degree to which impacts to a fish's ability to swim impact its ability to survive and reproduce, we can either assume the apical endpoints will not be impacted and likely underestimate the risk, or we can assume they will impact individual fitness which may overestimate risk.

To ensure protection of the species, we logically infer observed impacts to a species essential behaviors (e.g. effects on the ability of salmon to feed, escape predation, migrate, home, osmoregulate, etc.) and impacts to the availability of food are capable of producing fitness level consequences regardless of the presence of empirical studies quantitatively linking these assessment measure to an apical endpoint. The paucity of studies evaluating ecologically relevant endpoints contributes to the uncertainty and may increase the likelihood of underestimating risk.

References for the metolachlor ecological effects studies cited in this chapter can be found in EPA's Registration Review Problem Formulation for Metolachlor and S-Metolachlor as well as the Metolachlor/S-Metolachlor Draft Ecological Risk Assessment for Registration Review. These documents can be found at <https://www.regulations.gov/docket?D=EPA-HQ-OPP-2014-0772>.

References for the 1,3-D ecological effects studies cited in this chapter can be found in EPA's Problem Formulation for the Environmental Fate and Ecological Risk, Endangered Species, and Drinking Water Assessments in Support of the Registration Review of 1,3-Dichloropropene (Telone) as well as the 1,3-dichloropropene (1,3-D) Draft Risk Assessment (DRA) in Support of Registration Review. These documents can be found at <https://www.regulations.gov/docket?D=EPA-HQ-OPP-2013-0154>.

References for the chloropicrin ecological effects studies can be found at <https://www.regulations.gov/docket?D=EPA-HQ-OPP-2013-0153>.

Other references cited can be found in Chapter 19.