2.6.5 Direct Mortality Population Modeling

To determine if population productivity would be at risk due to direct mortality resulting from either acute or chronic exposures to the criterion concentrations of the chemicals of concern, a series of modeling applications was undertaken. These assessed whether juvenile salmon during their freshwater residence encountering the established criterion concentrations would be impacted, and if those changes would be sufficient to produce a change in the population growth rate, *i.e.*, lambda (λ). Model Run I examined the potential lethal and sublethal effects of ammonia, cadmium and copper on salmon productivity. These compounds were chosen because they are more data rich for specific life stages of salmonids and could potentially parameterize population models assessing direct mortality and somatic growth. Specific details regarding model design and parameterization are described in detail in Appendix 3. Model Run II assessed direct mortality impacts on population productivity resulting from exposure to the acute criteria for compounds with limited data.

Model Run I uses the direct mortality population model to assess the impact of the acute and chronic freshwater criteria on population productivity using a taxa- and life stage-specific subset of the acute and chronic toxicity data for ammonia, copper, and cadmium, and uses data-specific calculated dose-response slopes for the toxicity model runs (Appendix 3). This included direct mortality from either acute or chronic exposures. The model applied a mortality factor to first-year survival of the respective life-history models to assess changes in λ.

Model Run II uses the direct mortality population model (Appendix 3) to assess the impact of the acute freshwater criteria on population productivity using the acute toxicity data (LC_{50}), and a default dose-response slope. To assess the impact of the acute freshwater criteria on population productivity, we used the direct mortality population models. To do this, the dose-response slope for each LC_{50} toxicity test is needed. The BE does not provide any dose-response information for the data used in the analysis. Many of toxicity studies we reviewed either did not report the slope or did not provide the information required to calculate the dose-response curve. Since the direct mortality population model requires an LC_{50} slope, we used a default slope (probit slope of 4.5 converted to a sigmoid slope of 3.6) as recommended by EPA:

> In the event that dose response information is not available to estimate a slope, a default slope assumption of 4.5 (lower and upper bounds of 2 to 9) (Urban and Cook 1986 as cited in EPA 2007) is used.

In the analysis for Model Run I and Model Run II we assess the potential for effects associated with chemical exposure during subyearling freshwater rearing on Pacific salmon and steelhead populations using quantitative methods; a direct mortality model linked to a life history population model and a somatic growth model linked to the life history population model. Both methods predict changes in the modeled population's intrinsic rate of growth, *i.e.*, λ. General life-history strategies were constructed and analyzed for coho salmon, sockeye salmon and ocean-type and stream-type Chinook salmon. The model assesses direct mortality to subyearling salmon and its impact on population productivity. Data was reviewed in an attempt to paramaterize a somatic growth population model that explicitly links impairments in the somatic growth of individual subyearling salmon to the productivity of salmon populations.

Available data was insufficent to parameterize the somatic growth model. Both models address impacts on first-year survival, and the results are incorporated into one of four life-history strategies in the model to quantify changes in population productivity (for a detailed description, see Appendix 3).

Primary differences between the four modeled life-history strategies are life span of the female, time to reproductive maturity, the number and relative contribution of the reproductive age classes and general demographic rates (Appendix 3). The models depict general populations representing each life-history strategy and were constructed based upon literature data described in Appendix 3. Specific populations were not modeled due to the difficulty in finding sufficient demographic data for single populations. Due to similarities in life-history strategies, the ocean-type Chinook model was used to estimate impacts on chum salmon and the stream-type Chinook model to estimate impacts on steelhead.

The endpoint used to assess population-level impacts for the direct mortality population model was the percent change in the intrinsic population growth rate (lambda, λ) resulting from chemical exposure. Change in λ is an accepted population parameter often used in evaluating population productivity, status, and viability. The NMFS uses changes in λ when estimating the status of species, conducting risk and viability assessments, developing ESA recovery plans, composing opinions, and communicating with other Federal, state and local agencies (McClure *et al.* 2003 as cited in Appendix 3). While values of $\lambda < 1.0$ indicate a declining population, in cases when an exposure causes the population growth rate to decrease more than natural variability, a loss of productivity will result even if lambda remains above 1.0. Decreases in response to chemical exposures can be a cause for concern since the impact could make a population more susceptible to decline (*i.e.*, λ dropping below 1.0) due to impacts from other stressors.

2.6.5.1 Direct Mortality Population Model Description

A direct mortality population model was constructed that estimated the population-level impacts of first-year mortality resulting from exposure to the criterion concentrations of aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc (Model Run II). For Model Run II, impacts of first-year mortality resulting from exposure to the criterion concentrations of ammonia, copper, and cadmium over various time frames and life stages of data. These models excluded sublethal and indirect effects of the chemical exposures and focused on the population-level outcomes resulting from an annual exposure of young-of-the-year to a chemical at the criterion concentrations. Scenarios were chosen to represent both the acute and chronic criteria. This was done by parameterizing the model with toxicity data (LC_{50}s) derived from short term (<96 hrs) and long term (>28 days, based on the available data, see Table A3 in Appendix 3) experiments. The lethal impact was implemented as a change in first-year survival for each of the salmon life-history strategies. In order to understand the relative impacts of a short-term exposure of a single chemical on exposed vs. unexposed fish, we used parameters for an idealized control population that exhibits an increasing population growth rate. Four life-history strategies were modeled: ocean-type and stream-type Chinook salmon, coho salmon and sockeye salmon. The details for

each general population model are provided in Appendix 3. Due to similarities in life-history strategies, the ocean-type Chinook model was used to estimate impacts on chum and the stream-type Chinook model to estimate impacts on steelhead.

Population model output consists of the percent change in λ from the unexposed control populations derived from the mean of one thousand calculations each of the unexposed control and the chemical exposed populations. The percent change in lambda (with standard deviation), representing alterations to the population productivity, was selected as the primary model output for reasons outlined previously. The percent change in lambda is considered different from the control when the difference is greater than the percent of one standard deviation of the control λ.

Model Run I: Direct mortality, somatic growth, and population modeling— ammonia, cadmium, and copper.

Model Toxicity Scenario Parameterization

Ammonia (acute criterion = 5.6 mg/L; chronic criterion = 1.7 mg/L): The documents identified by the first round of literature review applying to acute toxicity of ammonia to salmonids were further reviewed for data appropriate to parameterize the direct mortality population model. Data needed to conform to 96-hr LC50 values for subyearling salmonids (free-swimming, 1-4g fish preferred, but did include data on fish of less than 10 g when that was all that was available). The range of values identified for Chinook salmon, coho salmon, rainbow trout and cutthroat trout and are shown below in the units of mg NH_3-N/L, as N (total ammonia-nitrogen). All values were normalized to a pH of 8 using an un-ionized ammonia computer worksheet available from the American Fisheries Society, as cited in Appendix 3. Following the practice in the ammonia Ambient Water Quality Criteria documents (1999, 2009, all as cited in Appendix 1), the fish LC50 values were not normalized for temperature. The normalized species mean values were 26.8, 15.1, 26.2 and 29.4 mg NH_3-N/L for Chinook salmon, coho salmon, rainbow trout and cutthroat trout, respectively (Servizi and Gordon 1990; Buckley 1978; Thurston and Russo 1983; Thurston *et al.,* 1981, Table A3, all as cited in Appendix 3). The genus geometric mean from these data was 23.6 mg NH_3-N/L. A sigmoid dose-response slope was calculated as 6.4 (Broderius and Smith 1979; Buckley 1978, as cited in Appendix 3). Both the genus geometric means and minimum species mean values were used to parameterize the model as discussed above. To assess the chronic criterion, a chronic study was found that exposed cutthroat trout to ammonia for 29 days and reported an LC50 of 21.3 mg NH_3-N/L (Thurston *et al.,* 1978, as cited in Appendix 3). No slope was identified, so the 96-hr slope was used in the model.

Documents investigating the effects of ammonia on growth of fish were reviewed for data appropriate as input to the somatic growth model. No studies were found that could provide the appropriate data. Most studies on exposure of juvenile salmonids to ammonia found that any effects on growth or food intake were temporary and compensation occurred before the end of the exposure period (Lang *et al.*, 1987, Linton *et al.*, 1998, Beamish and Tandler 1990, Larmoyeux and Piper 1973 as cited in Appendix 3). Other studies have shown effects on growth, but exposure occurred over early developmental stages and also produced developmental delays and abnormalities, so differences in size may not have been attributable to direct impacts on

metabolism or growth (Brinkman *et al.*. 2009 as cited in Appendix 3). From a 90-day exposure (Brinkman *et al.*. 2009 as cited in Appendix 3) calculated an EC_{20} that includes hatch effects, delayed swimup, and sac-fry growth of 5.56 mg NH_3-N/L normalized to pH 8. In addition, Lazorchak and Smith (2007 as cited in Appendix 1) reported decreases in growth of rainbow trout (size range <0.2 g) after a 7 day exposure to ammonium chloride, but at concentrations that overlapped with those inducing mortality in the test population inhibition concentration (IC) IC_{25} ranged from 104-210 mg/L ammonium chloride and LC_{50} ranged from 163-271 mg/L ammonium chloride). Moreover, the study organisms used by Lazorchak and Smith (2007 as cited in Appendix 3) were too young to fit within the life stage criteria established for this modeling exercise. In addition, pH was not reported in this study, so accurate normalization was not possible. Broderius and Smith (1979 as cited in Appendix 3) also exposed small rainbow trout (0.18 g) to ammonia over a 30-day period. Significant reductions in growth were seen at 0.32 mg NH_3-N/L, but survival was 70% of that observed in the controls (60%), so the quality and usefulness of this data is suspect. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects if studies where significant mortality occurred were included. Since data for the appropriate life stages or time frames were unavailable, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium (acute criterion = 2.0 µg/L; chronic criterion = 0.25 µg/L): Studies identified by the first round of literature review as having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data for parameterizing the population models. All data were hardness adjusted to 100 mg $CaCO_3$/L and reported as dissolved cadmium in µg/L using the hardness equations found in Mebane (2006 as cited in Appendix 3). The acute toxicity focused on 96-h mortality data for swimup fry, parr and subyearling smolt. Species mean values (geometric means of LC_{50} values) were calculated for salmonid fishes, and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species means at 4.53 µg/L (Appendix 3, Table A3). Sigmoid slopes were calculated when dose-response data were available. The resulting geometric mean of the slopes was 6.4 and the range was 4.7-7.8 (Besser *et al.* 2007, Finlayson and Verrue 1982, Davies *et al.* 1993 as cited in Appendix 3). Besser *et al.*. (2007 as cited in Appendix 1) estimated a 28-day LC_{50} for rainbow trout of 5.5 µg/L (Appendix 1, Table A3). The normalized LC_{50} value of 5.36 µg/L and the acute slope of 6.4 were used to parameterize the chronic criteria scenario of the mortality model.

Chronic cadmium studies were examined for applicable input data for the somatic growth model. Studies on the effects of cadmium on the growth of subyearling salmonids supported the statement by Mebane (2006 as cited in Appendix 3) that growth is seldom a sensitive endpoint for cadmium. At concentrations that produced changes in somatic growth, increased mortality was also observed in most studies (Mebane *et al.*. 2008, Brinkman and Hansen 2007, Hansen *et al.*, 2002b). In 24- and 30-day exposures of Atlantic salmon (*Salmo salar*), a reduction in size was seen after alevins were exposed to 6.75-21.8 µg Cd/L but these concentrations also produced 80-90% mortality (Rombough and Garside 1982, Peterson *et al.*, 1983). Bull trout (*Salvelinus confluentus*) fry (0.2 g) exposed to 1.57 µg Cd/L for 55 days (hardness adjusted to 100 mg $CaCO_3$/L) showed a 28% reduction in growth at this single time point, along with a 37% reduction in survival (Hansen *et al.* 2002b as cited in Appendix 3). No dose response curve for

growth was generated by the study, so these data could not be used for extrapolation to other concentrations.

Brinkman and Hansen (2007 as cited in Appendix 3) exposed brown trout fry (*Salmo* trutta) to cadmium for 30 days under different water chemistries and calculated a range of IC_{20}s from 1.7-4.8 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for reduced growth in the surviving individuals. Mortality chronic values for the same tests ranged from 2.04 to 4.79 µg Cd/L. They also calculated LC_{50} values for the first 96 h of the exposures and these ranged from 3.27 to 6.75 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L). Possible size-selective mortality or growth compensation due to decreased density were not addressed in the study design. Rainbow trout fry exposed to cadmium for 28 days exhibited increased mortality and dry weight at concentrations above a calculated NOEC of 1.3 µg Cd/L (Besser *et al.* 2007 as cited in Appendix 3). This may be attributed to size-selective mortality or an increase in somatic growth. One rainbow trout early-life-stage exposure lasting 62 days determined an EC_{10} for growth of 0.31 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) without the increased mortality (Mebane *et al.* 2008 as cited in Appendix 3). Changes in growth at these life stages (embryos and alevins) are not compatible with the somatic growth model that assesses changes in free-swimming, feeding fry during the linear portion of their growth phase, and could not be used to parameterize the model. Similarly, brook trout (*Salvelinus fontinalis*) exposed to 0.36 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for 30 days showed reduced prey capture efficiencies and differences in prey selection in artificial stream channels (Riddell *et al.* 2005 as cited in Appendix 3), which may link to changes in somatic growth, but this link could not be translated into appropriate input parameters for the current growth model.

Copper (acute criterion = 13 µg/L; chronic criterion = 9 µg/L): Studies having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data needed to establish values for several parameters of the population models. All data was hardness adjusted to 100 mg $CaCO_3$/L using the acute and chronic hardness equations for copper (EPA 2002 as cited in Appendix 3). For studies with non-laboratory water that reported total instead of dissolved copper, total copper was adjusted by 80% to estimate the dissolved portion of copper in µg/L. The acute toxicity focused on 96-h mortality data for swim-up fry, parr and subyearling fish. Species mean values (geometric means of LC_{50} values) were calculated (Appendix 1, Table A3) and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species. For direct mortality, the genus mean LC_{50} was 86.8 µg/L with species means ranging from 48.3-190.6 µg/L, while for chronic toxicity (exposures of at least 30 days) the genus mean value was 98.9 µg/L with a range of 73.9-132.2 µg/L. Sigmoid slopes were calculated when dose-response data were available (Appendix 3, Table A3). The resulting geometric means (with ranges) of the slopes were 5.2 (4.1-7.6) for the 96-hr exposures and 4.2 (3.1-5.4) for the longer term mortality studies.

Growth studies on fry over 0.2 grams and under 6 grams produced EC_{50} values ranging from 20.33 µg/L to 112.43 µg/L (all values hardness adjusted, Appendix 3, Table A4). Exposures lasted 15 - 98 days. NOEC values ranged from 5.83 - 113.82µg/L. Mortality was often observed in these studies and ranged from none reported to well over 50% at similar concentrations to those that produced growth effects (Appendix 1, Table A4). For example, Besser *et al.*. (2005 as cited in Appendix 3) reported the lowest growth EC_{50} of 20.33µg/L for 0.2 g fry after a 30 day

exposure, but also reported a 30-day LC_{50} of 16.83µg/L with a slope of 5.4 (Appendix 3, Table A4). Therefore, similar to the results with cadmium exposures occurring to subyearling salmonids between 1 and 6g, growth effects often were confounded by mortality since most of the growth studies reported mortality assessment values (LC_{50}s, chronic values, NOECs) that overlapped with or were less than the growth assessment values (EC_{50}s, NOECs; Appendix 1, Table A4). Hansen *et al.*(2002c as cited in Appendix 3) used the IC_{20} as an endpoint for comparison since concentrations producing over 20% growth inhibition were often accompanied by significant mortality. Many other growth studies found in the literature search were excluded for reasons such as using too few exposure concentrations, using exposures beginning before swim-up (usually just after fertilization), or reporting no effect on growth for the concentrations tested. As mentioned above, in the remaining studies concentrations that produced effects on growth often also showed significant decreases in survival. For example, Mudge *et al.*. (1993 as cited in Appendix 3) reported that, for three of their five tests in coho, mortality was more sensitive than growth (Appendix 3, Table A4). Nonetheless, some limited scenarios were run in the somatic growth model that looked at whether growth alone would be affected by exposures at the chronic criteria value for copper. The time-to-effect and time-to-recovery values used for copper were both 0.5 days.

Model Output

Ammonia: Using the genus geometric mean LC_{50} and dose-response slope, with 100% of the population exposed to the criteria concentrations, the direct mortality population model output showed 0% mortality to subyearlings and a zero percent change in the population growth rate (lambda) for all four life-history models (Table 2.6.5.1.47). The lowest species mean value in the *Oncorhynchus* range was also tested at 15.1 mg NH_3-N/L, and resulted in zero percent mortality and zero percent change in λ. When the chronic criterion was assessed with a 29-d exposure, the direct mortality population model predicted no mortality or change in λ.

Studies on chronic exposures of juvenile salmonids to ammonia reported no or very little effects on somatic growth, but these were accompanied by mortality. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium: Direct mortality population model runs were conducted using exposures to the criteria concentrations and the genus mean value calculated for *Oncorhynchus* (Table 2.6.5.1.1). This value produced 1 percent mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC_{50} and slope values as opposed to the minimum end of the range for species mean values (Table 2.6.5.1.1). Only when the minimum species mean value and the minimum slope were used did mortality rise to a level that produced changes in lambda that were greater than the standard deviation of the control models (Table 2.6.5.1.47). Changes in population growth rates for the stream-type Chinook and coho salmon were larger than one standard deviation from the control models. An estimated 28-day exposure to the chronic criterion produced no mortality or change in lambda.

Studies on chronic cadmium toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model was not run for cadmium.

Copper: Direct mortality population model runs were conducted using exposures to the criteria concentrations and both the acute and chronic parameters calculated for *Oncorhynchus* (Table 2.6.5.1). The acute LC_{50} and slope produced 0% mortality and no changes in the population growth rate for any of the four life history population models. The chronic LC_{50} and slope produced 0 percent mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC_{50} and slope values as opposed to the minimum end of the range for species mean values, but no mortality was projected (Table 2.6.5.1.1).

Studies on copper toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. In spite of this, some growth model scenarios were run. When the maximum exposure period was used for the chronic criteria value in the growth model (140, 164 or 184 days depending on the life history), with an EC_{50} of 20.33, slope of 2.7 (Besser 2005 as cited in Appendix 3) and the chronic criterion value of 9 μg/L, the percent change in λ ranged from -1 to -4 percent (depending on life history). None of these reductions exceeded the control standard deviations. A 30-day exposure produced no decline in population growth rates. When a 30-day exposure for direct mortality was modeled using the minimum species values with a LC_{50} of 73.9 μg/L and a slope of 4.2, the chronic criterion (9 μg /L) produced no change in λ for the four life history models.

Table 2.6.5.1.1 Direct mortality population model scenarios for ammonia, cadmium and copper criteria. Standard scenarios used the genus mean values for the criteria. Since no effect resulted, the minimum species mean values were assessed. The numbers in parentheses are the natural variability in λ. Bold indicates a percent change in lambda greater than one standard deviation from the baseline population model. The direct mortality population model scenarios for ammonia, cadmium, and copper do not take into account sublethal responses, indirect effects, mixture toxicity, and baseline stressors.

		Mortality input parameters			**Output**	**Percent change in lambda**			
Chemical	Test length	LC_{50} (mg/L)	Sigmoid slope	Criteria Conc.	Percent mortality	Chinook ocean-type	Chinook stream-type	Sockeye	Coho
Ammonia	96-hr	23.6[1]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	96-hr	15.1[2]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	29-d	21.3	6.4[3]	1.7	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Cadmium	96-hr	4.53[1]	6.4[1]	2.0	1	0(13)	0(4)	0(8)	0(7)
Cadmium	96-hr	4.53[1]	4.7[2]	2.0	2	-1(13)	-1(4)	-1(8)	-1(7)
Cadmium	96-hr	2.67[2]	6.4[1]	2.0	14	-4(12)	-3(4)	-3(8)	-5(7)
Cadmium	96-hr	2.67[2]	4.7[2]	2.0	20	-7(12)	**-5(4)**	-5(8)	-7(7)
Cadmium	28-d	5.36[1]	6.4[3]	0.25	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Copper	96-hr	86.8[1]	5.2[1]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	96-hr	48.3[2]	4.1[2]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	98.9[1]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	73.9[2]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)

[1]Genus geometric mean for *Oncorhynchus* values
[2]Minimum species mean value from the range of *Oncorhynchus* values.
[3]Slope for chronic exposures not identified, used genus mean slope from 96-hr exposures.

Summary: The only scenarios producing direct mortality sufficient to decrease the population growth rates or productivity were those using the lowest species mean values for cadmium. The other scenarios assessing the direct mortality from exposure to the suggested criteria values for ammonia, cadmium and copper did not result in significant changes in population productivity greater than one standard deviation from baseline population model.

Model Run II: Acute toxicity exposure-response analysis and direct mortality population modeling—aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc.

The statistical inputs for the Model Run II are displayed in Table 2.6.5.1.2. Tables 2.6.5.1.3 through 2.6.5.1.243 provide the output of the direct mortality population modeling on the percent mortality and changes in λ for each freshwater compound and for each of the six

salmonid fishes life history strategies. The NMFS only used LC_{50} toxicity data for free-swimming juvenile life stages for the direct mortality population modeling. Each table provides information on the chemical, concentration (criterion), LC_{50}, the geometric mean and the minimum species mean value of the 96-hour LC_{50} for the respective acute toxicity data set; the default dose-response sigmoid slope; species; percent mortality resulting from the LC_{50} and slope; the percent of the population exposed; the percent change in λ and its standard deviation (impacted) measured against the baseline population model; the mean value of lambda and its standard deviation, the first-year survival rate (S1); and the significant change, which is the percent change in lambda that exceeds one standard deviation of the baseline model. The first table is for each life history type and provides the results of the model run based on the geometric mean of the 96-hour LC_{50}. The second table is for each life history type and provides the results of the model run based on the minimum species mean value of the 96-hour LC_{50}. For details regarding the model output information in Tables 2.6.5.3 through 2.6.5.1.243, refer to Appendix 3.

The direct mortality population model scenarios for aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc do not take into account sublethal responses, indirect effects, mixture toxicity, and baseline stressors.

Table 2.6.5.1.2 Freshwater toxicity data statistics used as inputs for the Model Run II.

Compound	Acute Criterion	Acute Data (Geometric Mean)	Acute Data Used in the Direct Mortality Population Model (the geometric mean and the minimum species mean values)
Aluminum	750	2247	2671—445
Ammonia	5.6	32	32—7.3
Arsenic	340	16698	34269—10
Lindane	0.95	22.7	19.7—1
Cadmium	2	9.1	9—1.16
Chromium (III)	570	9825	9825—7762
Chromium (VI)	16	74908	74908—12079
Copper	13	96	96—5.7
Dieldrin	0.24	27	24—0.56
Endosulfan-alpha	0.22	0.66	0.66—0.17
Endosulfan-beta	0.22	0.66	0.66—0.17
Endrin	0.086	1.1	0.6—0.089
Heptachlor Epoxide	0.52	13.6	13.6—6.7
Lead	65	14675	17042—320
Nickel	470	18793	17663—588
Pentachlorophenol	19	86.9	86.1—10
Selenium	190	2850	4268—0.4
Silver	3.2	63	63—1.28
Tributyltin	0.46	3.2	2.6—0.21
Zinc	120	1190	1188—238

Cadmium

Table 2.6.5.1.51 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	12.9
LC50	10.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.52 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-45
Concentration	2	% chg l std	-	7.0
LC50	1.16	lambda mean	1.09	0.60
LC50 slope	3.6	lambda std	0.10	0.05
species	chinook, ot	S1	5.62e-003	6.94e-004
% Mortality	88	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.53 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	4.3
LC50	10.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.42e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.54 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-40
Concentration	2	% chg 1 std	-	2.6
LC50	1.16	lambda mean	1.00	0.60
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	7.94e-003
% Mortality	88	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.55 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg 1 std	-	7.9
LC50	10.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.56e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.56 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-39
Concentration	2	% chg 1 std	-	4.8
LC50	1.16	lambda mean	1.01	0.62
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.57e-002	3.17e-003
% Mortality	88	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.57 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg 1 std	-	7.5
LC50	10.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.58 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-50
Concentration	2	% chg 1 std	-	3.7
LC50	1.16	lambda mean	1.03	0.51
LC50 slope	3.6	lambda std	0.05	0.03
species	coho	S1	2.97e-002	3.66e-003
% Mortality	88	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.59 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg 1 std	-	4.4
LC50	10.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.41e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.60 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-40
Concentration	2	% chg 1 std	-	2.5
LC50	1.16	lambda mean	1.00	0.60
LC50 slope	3.6	lambda std	0.03	0.02
species	steelhead	S1	6.43e-002	7.93e-003
% Mortality	88	Significant change		3.0
Percent Exposed	100	[]		

Table 2.6.5.1.61 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg 1 std	-	12.8
LC50	10.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.61e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.62 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-45
Concentration	2	% chg 1 std	-	7.0
LC50	1.16	lambda mean	1.09	0.60
LC50 slope	3.6	lambda std	0.10	0.05
species	chum	S1	5.63e-003	6.94e-004
% Mortality	88	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.243 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-3
Concentration	120	% chg 1 std	-	12.6
LC50	238	lambda mean	1.09	1.06
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.20e-003
% Mortality	8	Significant change		9.2
Percent Exposed	100	[]		

Summary. Based on the direct mortality population modeling results, juvenile salmon and steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc is predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) ranging from zero percent to -100 percent based on the exposure scenario. Direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for both modeling scenarios.

2.6.6. Case Study on Extrapolating Growth Reductions in Fish to Changes in Population Extinction Risks: Copper and Chinook Salmon

This section examines the potential consequences of reduced growth on the survival of juvenile Chinook salmon from exposure to low levels of copper that commence prior to hatching. Toxicological assays generally do not consider or attempt to link effects on growth to changes in population and to long-term extinction risks. However, Mebane and Arthaud (2010) suggested that size reductions from early-life stage chronic sublethal copper exposure could potentially reduce juvenile salmon survival and population recovery trajectories. This study is different from the direct mortality, somatic growth, and population modeling in section 2.6.5 in which the literature found that growth of fry, on the whole, was not a sensitive endpoint for the effect of copper on juvenile salmonids relative to mortality. In the case study by Mebane and Arthaud (2010) they conclude that growth resulting from early life stage exposure is usually a more sensitive endpoint than mortality to copper. This case study modeled responses of juvenile Chinook salmon exposed to sustained exposures of low levels of copper starting during early development and extrapolated growth reductions and changes in survival related to individual size. Most of the literature on copper and juvenile salmonid fry that examines reduced growth shows little mortality in laboratory toxicity tests, which tend to be short in exposure duration and do not look at relationships between reduced growth and size-dependant survival. Chapman (1994 as cited in Mebane and Arthaud 2010) exposed different life stages of steelhead (*Oncorhynchus mykiss*) for the same duration (3 months) to the same concentration of copper (13.4 µg/L at a hardness of 24 mg/L as $CaCO_3$). The survival of steelhead that were initially