APPENDIX C

Screening-Level Ecological and Human Health Risk Assessment

SILVER KING CREEK
PAIUTE CUTTHROAT TROUT RESTORATION PROJECT

Health Risk Assessment for the Proposed Application of Rotenone

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FEBRUARY 2010

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C.1 SUMMARY

This screening-level assessment examines the ecological risks potentially associated with the proposed use of rotenone to eradicate non-native trout throughout an 11-mile reach of Silver King Creek and its tributaries in Alpine County, California. Chapter 3.0, Project Alternatives, of the EIS/EIR provides a detailed description of the study area. This assessment uses standard ecological risk assessment guidance and protocols (USEPA 1998, ASTM 1997, Cal/EPA 1992) and follows four steps or phases including:

- Problem formulation;
- Hazard assessment;
- Exposure assessment; and
- Risk characterization.

This screening-level assessment examines only the potential toxicological impacts on ecological receptors at Silver King Creek from the use of rotenone formulations. The accompanying EIS/EIR addresses other treatment alternatives and other potential environmental impacts, such as noise, recreation, economic impacts, and other analyses required by NEPA and CEQA. The findings of this assessment are integrated into Chapter 5.0, Environmental Consequences, of the EIS/EIR as they relate to potential impacts on aquatic and terrestrial biological resources, water quality, and human exposure.

For a "screening-level" assessment, no site-specific data or in-situ toxicity tests are conducted on site receptors. Rather, risks are characterized based on modeled doses and comparison with literature values. Specifically, risks were evaluated by estimating chemical uptake (i.e., dose) in ecological receptor populations from the maximum estimated exposure point concentrations of rotenone formulation constituents expected from each complete exposure pathway. These estimated doses were then compared to published toxicity reference values (TRV) from the literature for each significant formulation constituent. These comparisons were used to predict whether the formulation constituents would pose a hazard to the receptor populations.

Screening-level evaluations are designed to be conservative estimations of hazard that overestimate potential exposures and associated risks. This approach is consistent with regulatory guidance for risk assessment which emphasizes providing agency managers with information for protecting the environment. Because this screening-level assessment uses a conservative approach, actual exposures and risks would likely be lower than those presented below. The Agencies propose to continue monitoring after the proposed treatment to assess effects on ecological receptors and the effectiveness of mitigation measures presented in the EIS/EIR and to initiate adaptive management actions to reduce residual effects to acceptable levels.

C.2 PROBLEM FORMULATION

Problem formulation is the process of defining the goals, objectives, hypotheses and methods for evaluating ecological effects are developed (USEPA 1998). This requires development of (1) risk assessment endpoints that adequately reflect management goals within the ecosystem under study, (2) conceptual site models that illustrate the key relationships between a "stressor"

(i.e., the chemical(s) of potential concern) and the pathways through which selected ecological receptors in the study area could be exposed, and (3) the analysis plan (i.e., methods) by which effects from the stressor(s) will be examined. To initiate the process, risk assessors review existing information from the treatment area to scope the problem or question to be addressed, identify the receptors and potentially important exposure pathways, and develop an approach for assessing exposure risks.

C.2.1 Scope of Problem and Objective

The Paiute cutthroat trout is 1 of the 4 minor sub-species derived from the Lahontan cutthroat trout. The Paiute cutthroat trout was reclassified as threatened under the Endangered Species Act (ESA) of 1973 (USFWS 1975) and a special rule under ESA section 4 (d) was published in conjunction with the downlisting rule to facilitate management by the states and allow state permitted sport harvest ~~to facilitate management and allow regulated angling~~. Although a number of transplant populations have been established outside of the Silver King Creek Watershed, it currently occupies approximately 18.6 kilometers (11.5 miles) of historically fishless stream habitat within the upper Silver King drainage, above Llewellyn Falls (USFWS 2004). The entire historic range of Paiute cutthroat trout within Silver King Creek between Llewellyn Falls and Silver King Canyon (a total of 11 miles of mainstem and tributary habitat) is occupied by non-native trout (i.e., rainbow trout, Lahontan trout and golden trout) which also pose a threat to occupied habitat above Llewellyn Falls should non-natives move into that habitat.

Hybridization with non-native trout is the primary threat to the Paiute cutthroat trout (USFWS 2004). The fish present in reaches downstream from Llewellyn Falls to Silver King Canyon are a genetic mixture of introduced rainbow, Lahontan cutthroat, golden trout, and native Paiute cutthroat trout (Finger et al. 2008). When associated with Lahontan cutthroat trout or rainbow trout, Paiute cutthroat trout tend to lose their distinctiveness through hybridization (USFWS 1985). Llewellyn Falls (a complete barrier to upstream migration) currently separates hybridized trout and Paiute cutthroat trout. Llewellyn Falls is easily accessed by the public, which could lead to rogue or inadvertent transfer of hybridized fish to areas above the falls. Should this occur, they would hybridize with Paiute cutthroat trout and pose a significant threat to the survival of the sub-species.

Repatriating Paiute cutthroat trout into their historic range would isolate Paiute cutthroat trout from other trout species and greatly reduce the likelihood of an illegal introduction. There are 6 potential fish barriers in the Silver King Canyon, the 2 highest being 8 feet and 10 feet. The objective of the proposed Action is to remove all non-native trout from the Paiute cutthroat trout's historical native range. Once accomplished, the Agencies would restock the treatment area with Paiute cutthroat trout from genetically putative pure populations within the watershed.

C.2.2 Historical Efforts to Restore Paiute Cutthroat Trout

Since 1964, the Agencies have made multiple efforts to restore Paiute cutthroat trout populations to Silver King Creek and its tributaries. Initial chemical treatments were conducted on upper Silver King Creek, Corral Valley Creek, and Coyote Valley Creek during 1964. A repeated chemical treatment was conducted in upper Silver King Creek, Coyote Valley and Corral Valley Creeks during 1976 and 1977 to remove hybridized trout. Electrofishing surveys following the

1977 treatment were conducted to remove surviving hybridized trout; however, these efforts showed that the initial chemical treatments of Coyote Valley Creek had failed. A repeat treatment during 1987 and 1988 appeared successful as no hybridized trout have been observed during subsequent electrofishing surveys. These results were reconfirmed by allozyme and nuclear DNA analysis of tissue samples from all populations (Israel et al. 2002).

Subsequent efforts to restore putative pure Paiute cutthroat trout populations above Llewellyn Falls appear to have been successful following multiple chemical treatments between 1991 and 1993, combined with removal of non-native hybridized trout using electrofishing. The 3-year chemical treatment project successfully removed non-native hybrid trout from Silver King Creek in Upper Fish Valley upstream of Llewellyn Falls. Paiute cutthroat trout populations in Fly Valley Creek have remained isolated by a barrier falls and have never been treated. Additionally, hybridized trout were removed from Four Mile Canyon Creek by electrofishing and chemical treatment during 1991 through 1993. The upper headwater areas in Silver King Creek, Fly Valley Creek, and Four Mile Canyon Creek, have never been treated with rotenone.

Prior to CDFG's successful fish removal efforts in 1991–1993 in Silver King Creek above Llewellyn Falls, hybridized trout were removed from the creek and introduced into Tamarack Lake, a presumed fishless lake. Tamarack Lake's outlet flows into Silver King Creek within the proposed treatment area. Since the introductions, Tamarack Lake was gill netted during 2001–2008, and no fish were captured or observed. This lake was last stocked during 1991, but there is spawning habitat in a small stream entering the lake (Gerstung 1978 ~~Somer, pers. comm. 2003~~). This potential source of fish may require rotenone treatment to ensure that there is no downstream movement of hybridized fish into the treatment area. However, if further gill netting surveys of Tamarack Lake do not indicate the presence of hybridized trout, the Agencies would not implement this component of the proposed Action. As a result of extensive sampling in 2009 the Agencies have deemed Tamarack Lake to be fishless (Somer and Hanson 2009, Hanson 2009). The result of this determination is that Tamarack Lake will not be chemically treated and is no longer considered part of this project.

C.2.3 Overview of Proposed Action and Alternatives

The Agencies have considered a variety of options to remove non-native hybridized trout from Silver King Creek. After completion of the alternatives screening analysis, the Agencies selected the proposed Action and another action alternative. Chapter 3.0, Project Alternatives, of the EIS/EIR presents a detailed description of these alternatives, including the No Action alternative. This appendix evaluates only the potential ecological effects of rotenone and neutralizing agents. The Agencies selected the proposed Action to meet the following objectives:

- be completed quickly,
- use a method that has been proven to be effective in laboratory and field experiments,
- use a method that is technically feasible to implement,
- be in compliance with applicable laws,
- be implemented in a manner that, protects public health and safety, and
- minimize environmental impacts during and after application.

As described in detail in Chapter 3.0, Project Alternatives, of the EIS/EIR, each rotenone treatment alternative would require neutralization with potassium permanganate ($KMnO_4$). The Agencies propose to use a rotenone application of CFT Legumine™~~,~~ or Noxfish® ~~or Nusyn-Noxfish®~~ at a concentration up to 1.0 milligrams per liter (mg/L). The concentration of potassium permanganate (the oxidizing agent) shall be applied to Silver King Creek downstream of the study area at a concentration up to 2 to 4 mg/L in the receiving waters. This step would neutralize the rotenone and prevent the effects of rotenone in downstream areas. Potential impacts/risks from neutralization with potassium permanganate ~~$KMnO_4$~~ are assessed below.

C.2.4 Project Area and Land Use

C.2.4.1 Project Area Location

The Silver King Creek drainage is located on the eastern slope of the Sierra Nevada Range, in Alpine County, California. The drainage is a tributary of the East Fork of the Carson River, which drains into the Lahontan Basin. The proposed treatment area occurs within the Carson-Iceberg Wilderness on National Forest System lands administered by the Carson Ranger District, Humboldt-Toiyabe National Forest.

The treatment area includes the area that would be affected directly by the proposed rotenone treatment of CFT Legumine™~~,~~ or Noxfish® ~~or Nusyn-Noxfish®~~ at a concentration up to 1.0 mg/L and neutralization with potassium permanganate at a concentration up to 2 to 4 mg/L. This area includes Silver King Creek, its tributaries, and springs ~~and possibly Tamarack Lake.~~, depending on the results of gill netting surveys. Specifically, the treatment area includes the reach of Silver King Creek between Llewellyn Falls as the upstream boundary and the confluence with Snodgrass Creek at Silver King Canyon as the downstream boundary.

C.2.4.2 Land and Water Use in Project Area

The Carson-Iceberg Wilderness, within which the treatment area is located, grants permits for only certain activities, including hiking, fishing, and hunting. The USFS permitted grazing until 1995 when the grazing permit ended.

The Basin Plan defines the beneficial uses of Silver King Creek to include Municipal and Domestic Supply, Agricultural Supply; Groundwater Recharge; Water Contact Recreation; Non-contact Recreation; Commercial and Sport Fishing; Cold Freshwater Habitat; Wildlife Habitat; Rare, Threatened or Endangered Species; and Spawning, Reproduction, and Development.

C.2.5 Management Goals and Assessment Endpoints for Estimating Risk

C.2.5.1 Ecological Health

The Agencies' management goal for the proposed Action is to eradicate introduced species and reintroduce Paiute cutthroat trout to its native range while protecting the environment and non-target receptor populations from potentially adverse effects of the proposed rotenone application. This is consistent with the regulatory goals of the ~~f~~Federal Toxic Substances Control Act (TSCA

§2[b][1], Clean Water Act (304(a)CWA), and the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). It is also consistent with CDFG management goals as outlined in California Fish and Game Code Sections 1700 and 5501. The ecological goal or "assessment endpoint" for this exposure evaluation therefore is the continued existence of ecological receptor populations.

C.2.5.2 Human Health

The management goal for the human health assessment is to protect human populations from harmful exposure to rotenone formulation constituents during the proposed treatment by complying with all applicable and relevant regulatory standards, label use requirements, and site safety and health plan specifications. The human health risk assessment does not address potential worker exposure during chemical application. Worker exposure would be addressed by using protective equipment, following label use restrictions, and complying with a project-specific health and safety plan.

Human exposures to rotenone would be reduced by the following factors:

- The treatment area is located within a wilderness area (Carson-Iceberg Wilderness) located approximately 19 stream miles (or 10 air miles) from the nearest downstream human population in Markleeville, California.
- The rotenone would be neutralized chemically at the downstream end of the treatment area using potassium permanganate.
- Rotenone and its associated inert ingredients degrade rapidly in the environment.

In addition, the Agencies would (1) prevent the human consumption of fish killed by the rotenone treatment, (2) prevent the use of the treated water for irrigation purposes, and (3) prevent the release of the treated waters within one-half mile of a drinking water and/or irrigation water intake. Because the proposed Action is within a wilderness area located approximately 19 stream miles from the nearest downstream human population in Markleeville, California, this assessment assumes these goals would be satisfied.

C.2.6 Ecological Conceptual Model and Risk Hypothesis

The conceptual site model (CSM) represents the potentially complete ecological exposure pathways. It outlines: (1) all potential sources of chemical exposure; (2) chemical transport and release mechanisms; and (3) potential exposure pathways, including receptors.

Based on the description of the proposed Action and alternatives, the primary chemical exposure source would be the intentional release of rotenone formulations into Silver King Creek. Rotenone would be released at the upstream end of the study area and sprayed along the edge of the creek and tributary streams. Once released, the primary transport and release mechanisms would include:

- dissolution into surface water,
- adsorption onto sediments, and
- adsorption onto aquatic and riparian vegetation.

Thus, the "exposure points" through which non-target ecological receptors could be exposed to rotenone and its constituents would include: (1) treated surface water, (2) vegetation, (3) sediment contact and/or ingestion, (4) groundwater (drinking water), and (5) food chain bioaccumulation from consumption of dead fish. Based on these release and exposure mechanisms, risks to aquatic and terrestrial receptors are potentially significant, and the null and alternative hypotheses for this screening-level assessment follow:

- **Ho:** rotenone application from 0.5 mg-formulation/L rotenone-receiving water up to 1.0 mg-formulation/L rotenone-receiving water will result in significant exposure of non-target aquatic and terrestrial biota.
- **Ha:** rotenone application at up to 0.5 mg-formulation/L rotenone-receiving water up to 1.0 mg-formulation/L rotenone-receiving water will not result in significant exposure of non-target aquatic and terrestrial biota.

C.2.6.1 Potential Ecological Receptors

This section summarizes the species that could occur within the treatment area (also see Chapter 5.0, Environmental Consequences, of the EIS/EIR). Silver King Creek flows through a narrow valley that represents a mosaic of high elevation (7,000 to 8,000 feet) forest, upland brush communities, and a mix of riparian communities including aspen, willow, and wet meadow habitats. These habitats support a variety of wildlife, including special status species. Although few data specific to the treatment area are available, wildlife observations have been documented for the larger Humboldt-Toiyabe National Forest. Species that could inhabit the treatment area include over 13 species of birds, seven~~7~~ mammals, one~~1~~ reptile, and four~~2~~ amphibians (see EIS/EIR Section 5.2, Terrestrial Biological Resources). These include Forest Service Management Indicator Species (MIS), Forest Sensitive Species (FSS), and federally-listed species. Potential amphibians and reptiles include Sierra Nevada ~~Mountain~~ yellow-legged frog (*Rana sierrae* ~~*muscosa*~~), Yosemite toad (*Bufo canorus*), and northern sagebrush lizard (*Sceloporus graciosus graciosus*). Potential mammal species include bats (*Myotis, Euderma* and *Eumops* spp.), wolverine (*Gulo gulo luteus*), fisher (*Martes pennanti pacifica*), and Sierra Nevada red fox (*Vulpes vulpes necator*). Potential bird species include the Bald eagle (*Haliaeetus leucocephalus*), owls, (*Otus* and *Strix* spp.), Mountain quail (*Oreortyx pictus*) and the White-headed woodpecker (*Picoides albolarvatus*).

In addition to Paiute cutthroat trout, special status (~~f~~Federal, State, USFS, or Calfed conservation strategy) species that could occur in the treatment area include:

- Sierra Nevada ~~Mountain~~ yellow-legged frog (*Rana sierrae* ~~*muscosa*~~) (Sierra Nevada distinct population segment (DPS), candidate
- Yosemite Toad (*Bufo canorus*), candidate
- Fisher (*Martes pennanti*) (West Coast DPS), candidate
- Wolverine (*Gulo gulo luteus*), CA state threatened
- Bald eagle (*Haliaeetus leucocephalus*), CA state threatened

Fish species include Paiute cutthroat trout, Lahontan cutthroat trout, golden trout, rainbow trout, mountain whitefish, Paiute sculpin, and hybrids. An extensive list of benthic macroinvertebrates

includes stoneflies, mayflies, beetles, and caddisflies (see Appendix D and E). The study area supports no known special-status aquatic invertebrate species.

Chapter 5.2 of the EIS/EIR, Terrestrial Biological Resources, summarizes the plant communities in the treatment area, which include riparian, wetland, upland, and scrub-shrub mosaic of habitats found in the northern Sierra Nevada. USEPA recently concluded during registration of rotenone that plants are not sensitive to rotenone or its formulation constituents (USEPA 2006).

The exposure assessment below focuses on surrogate species selected to represent groups of similar species or guilds. Guilds are species groups with similar life histories or niches (e.g. insectivorous birds). Surrogate species within guilds were used to estimate exposure rather than estimating exposure for each individual species. The risk calculations for a single surrogate for which reliable life history information is available and whose exposure parameters represent a conservative estimate of exposure, can be extrapolated to the entire guild. The guild that includes western toad, for example, may also include special status species such as Yosemite toad.

Figure C-1 presents a conceptual model for the ecological exposures that could result from the proposed Action and represents the relevant receptor guilds for Silver King Creek. Several of these are special status species as summarized in Chapter 5.0 of the EIS/EIR, Environmental Consequences. Complete exposure pathways are identified based on the receptor's habitat, life history, and association with the treatment area. Exposure pathways include ingestion, dermal contact, and inhalation routes. When fisheries managers use rotenone as a piscicide, it is applied directly to the water body - in this case Silver King Creek and its tributaries. Once applied to the water, exposures would result for non-target receptors—essentially all aquatic non-fish organisms resident to the treated waters. Fish and other aquatic receptors would likely be exposed directly and receive the highest exposure. Exposures of terrestrial receptors, such as birds and mammals, would likely be through less direct pathways and thus insensitive to rotenone compared to aquatic receptors (Ling 2003).

Figure C-1 reflects the differences in exposure pathways between the selected receptors. Closed squares indicate complete exposure pathways. Open squares indicate incomplete exposure pathways. Closed circles represent potentially complete exposure pathways but for which exposure is likely insignificant. Direct contact exposure with the treated water is a complete pathway for all aquatic organisms as well as amphibians and reptiles. The route of uptake is direct contact and bioconcentration from the water.

Exposure of terrestrial biota through dermal contact is likely complete but insignificant because of skin barriers and minimal direct skin contact that would occur during the short treatment period (<24 hours). Similarly, inhalation exposure is complete but likely insignificant because exposure would be infrequent and of short duration. Ingestion of water and food would be a complete exposure pathway for terrestrial biota.

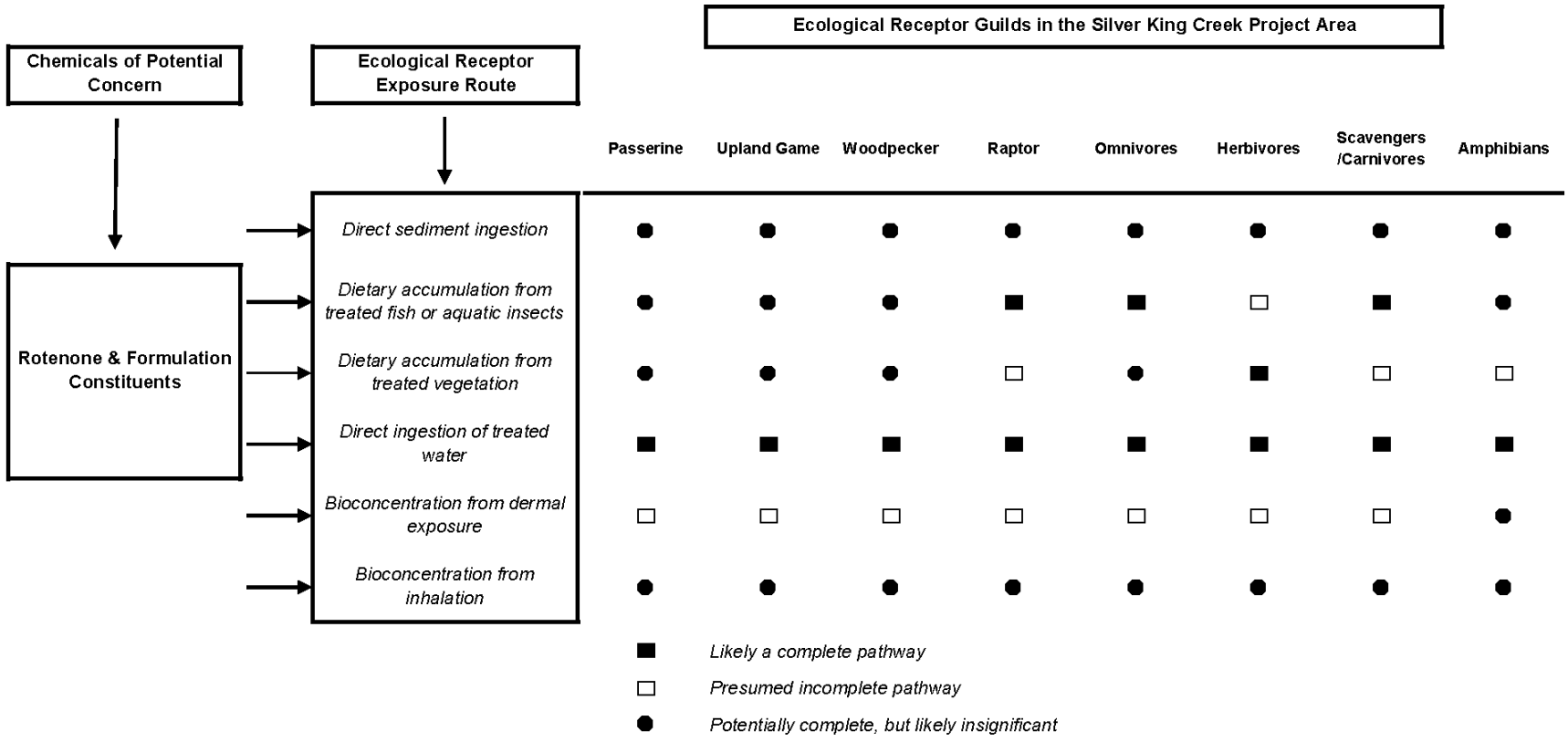


Figure C-1 Ecological Receptor Conceptual Site Model

C.2.6.2 Potential Human Receptor Populations

The proposed treatment area is within the Carson-Iceberg Wilderness on ~~f~~Federal lands administered by the USFS, Humboldt-Toiyabe National Forest. No residences or businesses occur within the treatment area. The nearest populated area downstream of this area is Markleeville, approximately 14~~0~~ miles from the neutralization station. Coleville, California, is located in Mono County approximately 5 miles from the northeastern corner of the treatment area. Coleville is upstream and has no direct access to the treatment area.

An unlikely but potential human exposure would be to an unauthorized visitor who could consume contaminated fish and or game during or after the treatment. However, this type of exposure would be minimized in the following ways. Fish killed during the rotenone application would be collected and buried to the extent practicable. The treatment area would not be restocked until any rotenone was dissipated. Fish would likely not be restocked until a year after the last treatment. Hence, newly stocked fish will not accumulate rotenone residues from the water. If dead fish were consumed, the primary health concern would be the acute illness associated with food poisoning, such as *Salmonella* sp. and other bacteria that could be present in fish tissue (Finlayson et al. 2000). Since the dead fish would have a strong foul odor, it is unlikely an unauthorized visitor would consume these dead fish.

Based on the remoteness of the treatment area, the distance to any downstream human population, and the controls that would be placed on human access during and for a period after the treatment, human exposure pathways are considered incomplete and human exposure is not addressed further in this assessment.

C.2.7 Ecological Risk Characterization Plan

This section outlines the specific methods employed to characterize ecological exposure of the receptors identified in the conceptual model. The methods focus on complete exposure pathways and ecological risks to receptors with potentially significant exposure to rotenone or rotenone formulation constituents.

C.2.7.1 Ecological Toxicity Risk Assessment Methods

The approach used in this ecological risk assessment follows the EPA guidance for conducting ecological risk assessments (USEPA 1998) as well as state guidance (Cal/EPA 1992). Briefly, the approach involves:

- identification of chemicals of potential concern (COPCs),
- selection of toxicity reference values (TRVs) for the COPCs,
- identification of habitats, biological communities, and biological receptors that could be exposed to the COPCs,
- identification of exposure parameters and exposure assessment methods (equations, calculations),

- estimation of exposures to COPCs, and
- comparison of estimated COPC doses to TRVs and estimation of risk.

Section C.3 of this appendix, the Hazard Assessment, identifies the COPCs and TRVs used. These values were developed based on a literature review of the substances that could be released from the rotenone treatment. Exposure parameters were based on review of species life histories and wildlife exposure parameter databases. Exposures were calculated using Equation [1].

EQUATION 1

Daily intake = CM * CR * FI * AF/BW

Where,

BW	=	Body Weight
CM	=	Concentration of contaminant in exposure media(s) of concern.
CR	=	Contact Rate—an estimate of the quantity of the medium consumed per day.
FI	=	Fractional Intake—The fraction of time (site use factor) spent in contact with the contaminated media (e.g., the proportion of the total diet obtained from the site, as extrapolated from information such as home range data or empirical findings).
AF	=	Absorption Fraction—the amount of contaminant contacted (e.g., consumed) that is actually assimilated into the receptor.

The contact rate may include the additive uptake from several exposure pathways (e.g. ingestion of prey tissue and aquatic sediments exposed to rotenone). The exposure assessment presented below (Section C.4) presents methods to account for exposure to multiple media as well as the exposure parameters used.

The Hazard Quotient (HQ) calculation characterizes the risks from the estimated exposure doses by dividing the dose by the TRV. For obligate aquatic species, risks were characterized by dividing the estimated concentration of rotenone and formulation constituents in the stream assuming complete mixing as the exposure point concentration (EPC) by effect concentrations from the literature – see Equation [2].

EQUATION 2

$HQ_1 = EPC/TRV$

Where:

EPC	=	Exposure Point Concentration (i.e., the concentration of contaminant in the exposure media), and
TRV	=	Toxicity Reference Value, as summarized by species in Section C.3.

The Risk Characterization, presented in section C.5 of this appendix, lists the resulting HQs by species and represents the combined consideration of the exposure and toxicity assessments.

C.3 TOXICITY ASSESSMENT

This section presents a review of the toxicological literature on rotenone and the most concentrated formulation constituents to identify the most appropriate TRVs from which to

characterize ecological risks. This section also summarizes the fate, transport and persistence of the formulation constituents and qualitatively assesses the potential for longer-term environmental exposures to formulation constituents or their breakdown products.

C.3.1 Rotenone Origin, Synthesis and Uses

Rotenone ({2R,6aS,12aS}-1,2,6,6a,12,12a-hexahydro-2-isopropenyl-8,9-dimethoxychromeno[3,4-b]furo[2,3-h]chromen-6-one) is a naturally occurring flavonoid derived from the roots of tropical plants in the pea and bean family (*Leguminosae*), including jewel vine (*Derris* spp.) and lacepod (*Lonchocarpus* spp.) found in Australia, Oceania, southern Asia, and South America (Finlayson *et al.*, 2000 cited in USEPA 2006). Resins extracted from these plants' roots with ether or acetone may contain between 2 and 40% rotenone (Ray 1991). Rotenone is a non-specific botanical insecticide, acaricide, and piscicide and was historically used as a fishing method by indigenous tribes of South America and Malaysia. Roots containing the compound were ground up and the pulp applied to water bodies.

The use of rotenone as a pesticide was first patented in Britain in 1912. Today, because of rotenone's natural origin, toxicity to pest organisms, relatively low toxicity to birds and mammals, rapid detoxification in warm water, and low environmental persistence has made it a popular and effective organic pest management tool. It is used by gardeners, for lice and tick control on pets, and for fishery management (USEPA 2006). In the United States, rotenone is classified as a General Use Pesticide (GUP), although uses on cranberries and for fish control are restricted (Extoxnet 1996).

Rotenone is a naturally occurring chemical obtained from the roots of several tropical and subtropical plant species belonging to the genus *Lonchocarpus* or *Derris*. Rotenone can be extracted with chloroform and determined by ultraviolet spectroscopy or analyzed using high performance liquid chromatography (HPLC) with UV detection. Liquid formulations of rotenone may contain petroleum hydrocarbons as solvents and emulsifiers to disperse rotenone in water (naphthalene, methylnaphthalenes, xylenes, etc.) (Washington Dept. of Fish and Wildlife [WDFW] 2002). The proportion of these carriers varies substantially by formulation, and formulations with synergists generally contain far less petroleum-based carrier products. The potential effects on ecological receptors associated with the adjuvants and carriers in the proposed formulations are discussed below.

Rotenone is the active ingredient in the commercially available piscicides Chem-Fish®, Cuberol®, Fish Nox®, Noxfire®, Nusyn-Noxfish®, Noxfish®, powder (Cube Powder Fish Toxicant®), and CFT Legumine™. Such formulations of rotenone include crystalline preparations (approximately 95% pure), emulsified solutions (approximately 50% pure), and dusts (approximately 0.75-5% pure) (Extoxnet 1996). This risk assessment compares the potential hazards and risks from the use of CFT Legumine™ , Noxfish® and Nusyn-Noxfish® formulations.

C.3.2 Mechanism of Action of Rotenone on Fish

Historically, rotenone was believed to suppress oxygen uptake across the gills, eventually leading to death by suffocation (Schnick 1974). Recent studies, however, demonstrated that rotenone increases blood oxygen concentrations in some fish species (Fajt and Grizzle 1998).

Rotenone interrupts aerobic cellular respiration by blocking electron transport in mitochondria through the inhibition of the enzyme NADH ubiquitone reductase (Singer and Ramsay 1994, Fukami et al. 1969, Lindahl and Oberg 1961) which prevents the availability of oxygen for cellular respiration. In other words, rotenone inhibits a biochemical process at the cellular level, making it impossible for fish to use the oxygen absorbed in the blood and needed for releasing of energy during respiration (Finlayson et al. 2000). In effect, rotenone causes death through tissue anoxia by blocking oxygen uptake at the cellular level and not at the water/blood interface at the gills (Ling 2003). The lack of cellular oxygen availability initiates anaerobic respiration in turn leading to increased lactic acid concentrations and dropping blood pH levels (Fajt and Grizzle 1998).

Rotenone is highly toxic to fish (Extoxnet 1996), and is ideal for the control of invasive or unwanted fish species. In the aquatic environment, rotenone is readily transmitted across the permeable membranes of the gills. Gills are highly evolved respiratory structures that maximize the uptake of oxygen (O_2) and excretion of carbon dioxide (CO_2) because of their large surface area, thin lamellar membrane, and efficient countercurrent exchange mechanism (Moyle and Cech 1988). Fish supplement this efficiency by actively ventilating water across the gills by controlled branchial pumping. These features make fish highly susceptible to low concentrations of rotenone. Variation in rotenone sensitivity exists between fish species; however, rotenone tolerance generally varies inversely with oxygen requirements, as would be expected for a respiratory poison (Engstrom-Heg et al. 1978).

C.3.2.1 Bioconcentration, Bioaccumulation and Metabolism

Persistence of chemicals in biological tissues is commonly characterized through bioconcentration or bioaccumulation. Bioconcentration of a chemical can occur in an organism when it accumulates chemicals in its tissues following direct exposure, at a concentration greater than that found in the exposure media (e.g. water, air). Bioaccumulation in the food chain results in higher concentrations in predators. Ney (1998) explains that bioaccumulation of organic chemicals in animals is a function of a chemical's solubility in fat. Fat-soluble (hydrophobic, non-polar) chemicals are more prone to bioaccumulate in fatty tissues and are more slowly metabolized. Chemicals that are insoluble in lipid, exhibit polarity and are readily metabolized.

Rotenone appears to bioconcentrate in aquatic organisms at acutely toxic concentrations but is detoxified and eliminated relatively fast when exposure concentrations do not result in mortality. Rach and Gingerlich (1986) examined concentrations of rotenone and rate of breakdown in tissues in common carp *(Cyprinus carpio*), bluegill (*Lepomis macrochirus*), and yellow perch (*Perca flavescens*) following treatment. Common carp (*Cyprinus carpio*) exhibited the greatest tolerance to rotenone and contained the highest concentrations (approximately 20 times that of the ambient water). Bluegill tissue contained eight times the water concentration and yellow perch contained four times the ambient water concentration. These bioconcentration factors (BCFs) are moderate to low relative to other organic compounds that exhibit BCFs orders of magnitude greater than rotenone.

Rach and Gingerlich (1986) also found that carp quickly eliminated rotenone with rotenoid metabolites accumulating in the bile. This confirmed results reported previously by Fukami et al. (1969), who examined the detoxification of radionuclide-labeled rotenone by liver enzymes in carp. Rach and Gingerlich (1986) found that rotenone was rapidly detoxified to a variety of

hydroxylated rotenoids and more water-soluble products with toxicities at least 1 to 2 orders of magnitude less than the parent rotenone. Thus the most likely route of detoxification and elimination is biliary excretion from the liver in the form of excretable metabolites.

Rotenone does not appear to bioconcentrate with prolonged exposure at sublethal doses. Rotenone is rapidly detoxified by the mixed function oxidase (MFO) system of the liver enzymes. Fish not killed by the treatment recover relatively quickly with residual effect, as shown in 30-day flow-through toxicity tests (Marking and Bills 1976).

Absorption of rotenone in the stomach and intestines in mammals is relatively slow and incomplete. If absorbed, rotenone is metabolized effectively by the liver to produce less toxic excretable metabolites (Ray 1991). Approximately 20% of the oral dose (and probably most of the absorbed dose) is excreted within 24 hours as water soluble products, with the remainder as hydroxylated rotenoids (Fukami et al. 1969). Large oral doses (200 mg/kg in pigeons and 10 mg/kg in dogs) usually stimulate vomiting (Haag 1931 *as cited in* Ling 2003). Based on a review of results from these papers and others, Ling (2003) concluded that rotenone is not easily absorbed in higher animals and does not accumulate in the body. These results also show that rotenone would not readily bioaccumulate in the food chain.

C.3.3 Environmental Fate and Chemistry

C.3.3.1 Physical Chemistry

Rotenone is a naturally occurring compound with empirical formula $C_{23}H_{22}O_6$ (Figure C-2) and a molecular weight of 394.43 (Extoxnet 1996, ~~FAO 1970~~). It is derived from the roots of tropical plants (*Derris spp., Lonchocarpus spp*., and *Tephrosia spp*.) found in S. America, Australia and parts of Southern Asia (USEPA 2009 ~~2002~~). Rotenone is highly soluble in organic solvents such as alcohol and acetone, but is only slightly soluble in water: 0.2 mg/L at 20°C, 15 mg/L at 100°C (Extoxnet 1996).

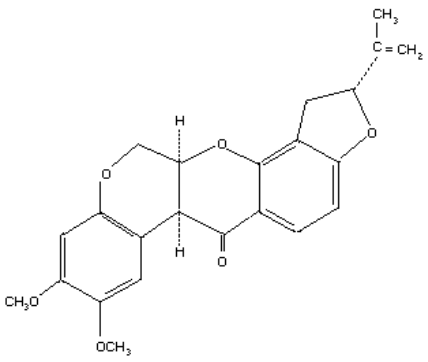


Figure C-2 Chemical Structure of Rotenone

C.3.3.2 Environmental Transport and Degradation of Rotenone

In mild temperatures, rotenone dissipates rapidly in both soil and water with a half-life between 1 and 3 days. It has a high tendency to adhere to soil particles and is unlikely to leach from soils; therefore, it is not likely to be a groundwater pollutant (Finlayson et al. 2001, Extoxnet 1996). Rotenone is considered as a "highly active but short-lived photosensitizer" (Extoxnet 1996), meaning any organism consuming rotenone and unable to metabolize it, will become highly sensitive to the sun for a short period.

Wildlife consumption of rotenone-killed fish can be a means of environmental transport into other portions of the food web. However, a literature search found no instances where birds or mammals suffered ill effects after consuming fish killed by rotenone treatment, or by drinking treated waters. As previously discussed, birds and mammals neutralize rotenone in their guts by enzymatic action, preventing adverse effects and bioaccumulation. These physiological

adaptations, coupled with the minute concentrations of rotenone generally found in dead fish, limits the extent to which rotenone exposure occurs through this pathway.

Rotenone is very sensitive to light and temperature and degrades rapidly in the presence of sunlight and warm temperatures (Extoxnet 1996). Rotenone persistence in natural water bodies may vary from a few days to several weeks depending on the season (Ling 2003, Finlayson et al. 2001). Water temperature, light intensity, depth, dissolved oxygen, pH, turbidity, aquatic vegetation, and the presence of a thermocline may all affect the persistence and efficacy of rotenone.

Finlayson et al. (2001) conducted laboratory tests to record the degradation of rotenone in water at 4°C in the absence of light (Table C-1). After 6 days, 4 out of 6 samples showed significant decreases in rotenone concentration. Water with higher alkalinity (>170 mg/L $CaCO_3$) and pH (>9.0) had higher degradation rates (-24% and -25%) than water with lower alkalinity (40 mg/L $CaCO_3$) and pH (7.7) (no change to –16%). As demonstrated in Table C-1, the combination of high alkalinity and high pH did not accelerate degradation. However, there was no test condition where high alkalinity and low pH were paired in this study.

Table C-1 Mean Rotenone Concentrations (µg/L) Before and After Six Days Storage at 4°C in the Absence of Light

Alkalinity (mg/L $CaCO_3$)	pH	Rotenone Before	Rotenone After	Percent change
40	7.8	91	93	+2
180	9.2	68	52	-24*
40	7.7	31.6	28.2	-11*
40	7.7	47.8	40	-16*
40	9.3	238	238	0
172	9.6	14	10.5	-25*

*Significant changes (p>0.05) using the Kruskal-Wallis test. (Source: Finlayson et al. 2001).

Gilderhus et al. (1986) conducted a study to determine the effect of water temperature on rotenone persistence (Table C-2). Rotenone degraded much quicker in warmer water—nearly 10 times faster at 23°C than at 1°C. Rotenone treatment at 100 parts per billion (ppb) in cold water remained toxic to rainbow trout 14 days after the initial treatment, even though the concentration measured was only 6 ppb. Similar findings were reported by Finlayson et al. (2001) after measuring the half-life of rotenone in several California reservoirs: Kaweah Reservoir (20-22°C), Frenchman Lake (10-22°C) and Lake Davis (5-12°C) had rotenone half-lives of 1.7, 3.5 and 7.7 days respectively (Table C-2).

Table C-2 Persistence of Rotenone in Ponds at Two Different Temperatures

Water Temperature	Initial Treatment: Rotenone Concentration	Time to Decay to 0.02 mg/L	Half-Life of Rotenone
1°C	0.10 mg/L	11 days	83.9 hours, (3.5 days)
23°C	0.15 mg/L	48 hours (2 days)	13.9 hours, (0.5 days)

Source: Gilderhus et al. 1986
Note: Rotenone concentrations were analyzed by high performance liquid chromatography [HPLC]

Dawson et al. (1991) conducted a similar experiment in 1986 to evaluate the effects of temperature and sediment adsorption on rotenone persistence. Persistence was compared between two ponds: one lined with cement, the other with an earthen-bottom. Studies with different water temperatures were completed during the spring, summer and fall (Table C-3). Similar to the results of Gilderhus et al. (1986), rotenone degradation rate was positively correlated with increasing water temperature. In addition, for every temperature tested, rotenone disappeared two to three times quicker in the earthen pond versus the concrete lined pond, supporting the claim that rotenone tends to adhere to particles. However, while high initial sorption to the sediments was to be expected, rotenone concentrations in the sediment decreased to below limits of detection within 3 days of treatment, with water temperatures that ranged from 15 to 22°C. Dawson et al. (1991) also discovered that filtered water samples contained significantly less rotenone than the unfiltered samples, suggesting that rotenone is also readily absorbed by suspended particles in the water column.

Table C-3 Effects of Temperature and Sediment Adsorption on the Half Life (in Days) of Rotenone

Pond Substrate	Half Life of Rotenone (days)		
	Spring (8°C)	Summer (22°C)	Fall (15°C)
Concrete	3.7	1.3	5.2
Earthen	1.8	0.7	1.8

Source: Dawson et al.1991

Rotenone aging studies conducted under laboratory conditions by Marking and Bills (1976) highlight rotenone's much shorter persistence when subjected to natural conditions. Half-lives for laboratory-aged solutions of rotenone in soft water were 13 days at 17°C and 22 days at 12°C, much longer than those of Dawson et al. (1991) and Gilderhus et al. (1986) in field experiments. Furthermore, the toxicity of rotenone solutions declines in parallel with chemical decay, indicating that the breakdown products are comparatively non-toxic (Marking and Bills 1976). Cheng et al. (1972) used photodegradation to identify the breakdown products of rotenone, identifying 20 separate products, most of which were rotenoids, only one of which (6αβ, 12αβ-rotenolone) is considered toxic (Cheng et al. 1972).

Recent field studies in California by Finlayson et al. (2001) support previous findings that rotenone breaks down rapidly in the environment. Finlayson found that the estimated half-life of rotenone ranged between 0.58 and 7.7 days (mean of 2.3 days) depending on the waterbody. Rotenone half-life values measured in four reservoir systems increased with increasing water depth, supporting the hypothesis that light is an important catalyst in rotenone degradation. Kaweah Reservoir, Success Reservoir, Lake Davis, and Frenchman Lake had half-life values measured at 1.7, 2.4, 7.7 and 3.5 days respectively (average depths of 8-12m) and Percolation Reservoir 12 and Meiss Lake had respective half-lives of 0.94 and 0.83 day (average depths of 0.8-1.0m) (Table C-4). Recently, rotenone had a half-life of 5.6 days in Lake Davis in 2007 following rotenone application to Lake Davis, California in 2007 (McMillin and Finlayson 2008).

Table C-4 Rotenone Concentrations (µg/L) and Corresponding Half-Life Values in Lakes of Varying Depths

Location (Year)	Rotenone Concentrations (µg/L)				Half-life (days)	Average Depth (m)
Kaweah Reservoir (1987)	76 (1)	55 (3)	43 (5)	<2 (12)	1.7	8-12
Bravo Reservoir (1987)	254 (1)	46 (2)	<2 (6)	---	0.65	---
Lonestar Pond (1987)	310 (1)	49 (2)	24 (6)	<2 (14)	1.8	---
Percolation Reservoir 5 (1987)	370 (1)	150 (3)	120 (8)	<2 (15)	1.7	---
Percolation Reservoir 12 (1987)	200 (1)	27 (3)	<2 (8)	---	0.94	0.8-1.0
Success Reservoir (1988)	122 (1)	39 (2)	22 (6)	<2 (30)	4.6	8-12
Meiss Lake (1988)	64 (0.13)	30 (1)	8.2 (3)	<2 (6.2)	0.96	0.8-1.0
Meiss Lake (1989)	47 (0.08)	41 (0.17)	30 (0.5)	18 (1)	0.96	0.8-1.0
Meiss Lake (1990)	11 (0.04)	5.9 (2.9)	3.8 (0.92)	<2 (1.9)	0.58	0.8-1.0
Frenchman Lake (1991)	90 (1)	39 (2)	28 (3)	6 (14)	3.5	8-12
Wolf Creek Lake (1992)	16 (8)	<2 (21)	<2 (28)	<2 (51)	2.9	---
Lake Davis (1997)	44 (1)	32 (3)	29 (7)	11 (21)	7.7	8-12

Source: Finlayson et al. 2001

Due to its low Henry's Law constant (1.1×10^{-13} atm-m^3/mol), rotenone is not expected to volatilize appreciably from surface water. The small amount of rotenone that may volatilize into the atmosphere would be degraded readily through reactions with photochemically produced hydroxyl radicals. The half-life for this reaction is approximately 1.2 hours (NLM 2006).

C.3.4 Rotenone Toxicity to Ecological Receptors

C.3.4.1 Toxicity to Fish

The efficacy of rotenone on various aquatic organisms has been examined in controlled aquatic toxicity tests. Such tests commonly determine the LC50 value (the median water concentration of the active ingredient that kills 50% of the animals) over specified periods of time (e.g. 24 hr, 96 hr, etc.). Marking and Bills (1976) summarized rotenone toxicity data for a variety of fish species (Table C-5). The tests used to establish these values were conducted with laboratory quality water lacking the colloid and sediment load typical of field settings. These organic loads consistently increase the amount of chemical required to elicit a toxic effect. Thus, these laboratory values provide a conservative estimate of the effect that could be observed in a lake environment. However, in flowing waters, rotenone dissipates relatively quickly (less than 24 hr) due to dilution and increased rates of hydrolysis (USEPA 2007~~Borriston Laboratories 1983~~) and photolysis (Cheng et al. 1972, USEPA 2007~~Biospherics 1982) (~~CDFG 1994).

Table C-5 Fish Toxicity of Noxfish®, Containing 5% Rotenone, in Standardized Laboratory Tests at 12°C

Species	Lethal Concentration of Noxfish®		Lethal Concentration of Rotenone (x 0.05)	
	LC50 24h. (µg/L)	LC50 96h. (µg/L)	LC50 24h. (µg/L)	LC50 96h. (µg/L)
Northern Pike	44.9	33.0	2.3	1.7
Atlantic salmon	35.0	21.5	1.8	1.1
Brook trout	47.0	44.3	2.4	2.2
Chinook salmon	49.0	36.9	2.5	1.9
Coho salmon	71.6	62.0	3.6	3.1
Lake trout	26.9	26.9	1.4	1.4
Rainbow trout	68.9	46.0	3.5	2.3
Goldfish	---	497.0	---	24.9
Common carp	84.0	50.0	4.2	2.5
Fathead minnow	400.0	142.0	20	7.1
Channel catfish	400.0	164.0	20	8.2
Black bullhead	665.0	389.0	33.3	19.5
Smallmouth bass	93.2	79.0	4.7	4.0
Largemouth bass	200.0	142.0	10	7.1
Green sunfish	218.0	141.0	10.9	7.1
Bluegill sunfish	149.0	141.0	7.5	7.1
Yellow perch	92.0	70.0	4.6	3.5
Longnose sucker	67.2	57.0	3.4	2.9
White sucker	71.9	68.0	3.6	3.4
Bowfin	57.5	30.0	2.9	1.5

Source: Marking and Bills 1976.

Rotenone applications of commercial formulations between 1 and 3 mg/L have generally proven sufficient to eliminate all fish in the treated water body (Ling 2003). Such formulations result in active ingredient (a.i.) concentrations of rotenone (i.e., rotenone) ranging from 50 to 150 µg/L. In such aquatic exposures, the water-borne chemical enters fish by simple diffusion across the gills. Marking and Bills (1976) recorded 24hr LC50 rotenone concentrations of 1.4 µg/L to 33.3 µg/L, and 96hr LC50 concentrations of a.i. ranging from 1.1 µg/L to 24.9 µg/L. Some of the most resistant species in field and lab applications have included black bullhead (*Ictalurus melas*), channel catfish (*I. punctatus*), and fathead minnow (*Pimephales promelas*) with 24 hr LC50 rotenone concentrations of 33.3 µg/L, 20 µg/L, and 20 µg/L, respectively.

Fishery managers have exploited this range in sensitivity among fish species to remove unwanted species selectively from mixed-species communities (Bills et al. 1996). Reasons for such marked differences may be a result of differences in tissue distribution, rates of uptake, and rates of detoxification based on differences in the levels of liver enzymes responsible for rotenone breakdown and elimination, or supplemental means for oxygen uptake from air. Another possible explanation is that certain species are biochemically more successful in using alternative pathways to generate adenosine triphosphate (ATP) (Rach and Gingerlich 1986) and are therefore still able to function at rotenone concentrations that would kill other species.

Omnivorous fish species generally demonstrate higher tolerance levels to rotenone than strict carnivores. One explanation for this elevated tolerance is that bottom-feeding omnivorous fish tend to have much higher concentrations of the mixed function oxidase (MFO) enzymes

responsible for metabolizing rotenone than strict carnivores (Moyle and Cech 1988). The MFO class of enzymes metabolize foreign compounds like rotenone, and accelerate their elimination, thus increasing the tolerance of such species with high rates of MFO induction to withstand otherwise lethal rotenone concentrations.

C.3.4.1.1 EFFECTS OF PHYSICAL AND BEHAVIORAL PARAMETERS ON ROTENONE TOXICITY TO FISH

Water-temperature and contact time are perhaps the two most important variables that modulate efficacy of rotenone treatments. G~~u~~ilderhus (1972) found that the time required to achieve 100% mortality (LC100) in various freshwater fish decreased approximately 2- to 3-fold for every 5-degree increase in water temperature. Additionally, fish mortality will not occur if there is inadequate contact time between the chemical and the fish. This is especially problematic for short-term exposures that typically occur in stream treatments lasting 4 to 8 hours. Some fish species demonstrate avoidance behaviors to rotenone, favoring areas with lower concentrations, or areas that are free of rotenone (Hogue 1999). Therefore, to achieve complete elimination of target species, rotenone must be dispersed throughout the fish inhabitable waters in the treatment area., ~~including the possible treatment of Tamarack Lake should fish be present.~~

Furthermore, fertilized fish eggs are less susceptible to rotenone poisoning than fishes themselves because their rate of toxicant uptake is much lower (Table C-6) (Ling 2003, Marking and Bills 1976). Programs aimed at eradicating a certain fish species must conduct the treatment before the spawning season or after all eggs have hatched.

Water hardness, pH, and rotenone formulation can also modulate rotenone toxicity. Generally, rotenone is reported to be more effective when the natural body of water is somewhat acidic, with low hardness (i.e., soft water). However, Marking and Bills (1976) noted that the toxicity of rotenone to fish was not affected significantly by hardness or pH. However, toxicity to newly fertilized fish eggs *decreased* with softer water (Table C-6), suggesting, somewhat counterintuitively, that rotenone permeability through the egg chorion is diminished by softer water.

Table C-6 Toxicity of Rotenone in 12°C Water at Various Degrees of Hardness to Rainbow Trout and Rainbow Trout Eggs

Species	Median 96h LC50 (µg/L)			
	Very Soft Water	Soft Water	Hard Water	Very Hard Water
Rainbow trout (*O. mykiss*)	2.7	2.8	2.75	2.65
Newly fertilized *O.mykiss* eggs	280	221	160	125

Source: Marking and Bills 1976.

Following rotenone treatment, fish exhibit certain characteristic behaviors. In the induction stage of treatment, observed behaviors include reduced opercular ventilation coupled with erratic swimming bursts. Surfacing and a 'gulping' behavior or skimming at the surface film may follow before fish experience a complete loss of equilibrium. Eventually, fish sink to the bottom and die (Ling 2003, Fajt and Grizzle 1998, Rach and Gingerlich 1986).

C.3.4.2 Rotenone Toxicity to Non-target Aquatic Organisms

C.3.4.2.1 AQUATIC MACROINVERTEBRATES

With their gill-like tracheae, aquatic invertebrates are theoretically as susceptible to the toxic effects of rotenone as fish or amphibian larvae (Bradbury 1986). However, laboratory tests conducted by Chandler and Marking (1982) concluded that apart from an Ostracod (*Cypridopsis* sp.), aquatic invertebrates are much more tolerant of rotenone than most fishes and amphibian larval stages. The most resistant organisms were a snail (*Helisoma* sp.) and the Asiatic clam (*Corbicula manilensis*) for which the LC50 96 hr concentrations were 50 times greater than those reported for the black bullhead (*Ictalurus melas*) (Marking and Bills 1976), one of their most resistant fishes. Sanders and Cope (1968) also conducted lab tests examining the effect of rotenone on the nymph or naiad stage of a stonefly (*Pteronarcys californica*). They found that the LC50 24 hr was 2,900 μg/L and the LC50 96 hr was 380 μg/L. These values are an order of magnitude greater than previous findings for black bullhead (*Ictalurus melas*) (Marking and Bills 1976), indicating that aquatic invertebrates are much less sensitive to rotenone than fish. Larger, later instar naiads were less susceptible to given concentrations of toxin than were smaller, earlier instars of the same species (Sanders and Cope 1968).

Field studies examining the effect of rotenone on aquatic macroinvertebrate communities have provided varied results. Whereas some workers noticed dramatic, long-term effects (Mangum and Madrigal 1999, Binns 1967), others observed rotenone has a negligible effect on most aquatic macroinvertebrates (Demong 2001, Melaas et al. 2001, Trumbo et al. 2000a, 2000b, Whelan 2002, Vinson and Vinson 2007). In general, the rotenone effects on benthic macroinvertebrates are less pronounced and more variable on macroinvertebrates than on zooplankton. Like the range of sensitivities demonstrated by various fish species to rotenone, different species of aquatic macroinvertebrates also exhibit a range of tolerances (Mangum and Madrigal 1999, Chandler and Marking 1982, Engstrom-Heg et al. 1978), again perhaps based on their oxygen requirements (Table C-7).

Rotenone treatments in streams and rivers also cause significant loss of invertebrate fauna but effects are usually more noticeable close to rotenone application stations. Not all invertebrate losses in stream treatments are due to the death of the animals because rotenone also causes increases in invertebrate drift downstream (Morrison 1977 as cited in Ling 2003). A 5 year study of the Strawberry River, Utah, following a 48 hour treatment to remove coarse fish showed that up to 33% of the benthic invertebrate species were unaffected by the treatment. Forty-six percent of the species had recovered after 1 year but a further 21% were still missing after 5 years. Most of the species that were most sensitive to rotenone and which failed to recover were mayflies, stoneflies and caddis flies, although some members of each of these groups were also resistant to rotenone treatment. Although some species that were present before the treatment were still missing 5 years later, other species not present before the rotenone treatment had appeared and were possibly filling vacated niches (Mangum and Madrigal 1999 as cited in Ling 2003). The variable response from invertebrates is due to differences in concentration and duration of rotenone used in the stream treatment (Vinson and Vinson 2007).

Table C-7 Rotenone Toxicity Reported in Some Aquatic Invertebrates

Species Guild	Test Species	Test Endpoint	Lethal Concentration (mg/L)
Flatworm	*Catenula* sp.	LC50 24h	5.1
	Planaria sp.	LC50 24h	<0.500
Annelid worms	Leech	LC50 48h	<0.100
Copepod	*Cyclops* sp.	LC100 72h	<0.100
Branchiura	*Argulus* sp.	LC50 24h	~0.025
Cladoceran	*Daphnia pulex*	LC50 24h	0.027
	D. pulex	LC50 24h	<0.025
	Diaptomus siciloides	LC50 24h	<0.025
Ostracod	*Cypridopsis sp.*	LC50 24h	0.490
Conchostracan	*Estheria* sp.	LC50 24h	~0.050
Freshwater prawn	*Palaemonetes kadiakensis*	LC50 24h	5.15
Crayfish	*Cambarus immunis*	LC50 72h	>0.500
Dragonfly naiad	Macromia sp.	LC50 24h	4.70
Stonefly naiad	*Pteronarcys californica*	LC50 24h	2.90
Backswimmer	*Notoncta* sp.	LC50 24h	3.42
	Notonecta sp.	LC50 24h	~0.100
Caddis fly larvae	*Hydropsychye* sp.	LC50 96h	0.605
Whirligig beetle	*Gyrinus* sp.	LC50 24h	3.55
Water mite	Hydrachnidae	LC50 96h	~0.050
Snail	*Physa pomilia*	LC50 24h	6.35
	Oxytrema catenaria	LC50 96h	1.75
	Lymnaea stagnalis	LC50 96h	>1.00
Bivalve Mollusc	*Dreissena polymorpha*	LC50 48h	0.219
	Obliquaria reflexa	LC50 48h	>1.00
	Elliptio buckleyi	LC50 96h	2.95
	Elliptio complanata	LC50 96h	2.00
	Corbicula manilensis	LC50 96h	7.50

Note: as summarized by Ling 2003, from a variety of sources

C.3.4.2.2 PLANKTON

Rotenone can have significant effects on abundance and structure of the plankton community, which can have subsequent effects on fish populations that depend on plankton either directly or indirectly for nutrition. From 1954 to 1955, Hoffman and Olive (1961) conducted an experiment to document the effect of rotenone on the zooplankton community in a Colorado reservoir. They observed a complete elimination of protozoans and Entomostracans and a major reduction in the Rotifer population following treatment. Their finding agreed with previous research (Hooper 1948, Brown and Ball 1943, Hamilton 1941) and more recent findings that rotenone is highly toxic to zooplankton (Melaas et al. 2001, Beal and Anderson 1993, Neves 1975, Anderson 1970, Kiser et al. 1963), especially in acidic conditions (Kiser et al. 1963). Unlike many benthic invertebrates, which may escape the immediate effects of rotenone by burrowing into sediment, zooplankton remain in the water column for the full duration of treatment. However, some populations may recover from resistant life-stages and or eggs (Kiser et al. 1963). A full recovery of the zooplankton community may take longer however. Beal and Anderson (1993) demonstrated that some populations make take up to 8 months to recover following rotenone treatment, while Anderson (1970) noted a 3-year recovery period in 2 mountain lakes. These

studies suggest that rotenone treatment and restocking of lakes must allow zooplankton communities to reestablish before restocking.

C.3.4.3 Toxicity to Terrestrial Wildlife Receptors

Rotenone can be toxic to both aquatic and terrestrial species depending on the dose, method of administration, duration of exposure, and sensitivity of the species and life stage. Table C-8 outlines chemical toxicity guidelines established by the USEPA that are used in assessments of rotenone toxicity to birds and mammals. Table C-8 lists two hazard categories: the acute oral or dermal LD50 and the acute inhalation LC50. The LD50 is the statistical derivation of a dietary or drinking water dose, predicted to cause 50% mortality. The LC50 is based on the concentration of a compound in air or water.

Table C-8 Chemical Hazard Classifications for Wildlife Risk

Hazard Category	Mammals		Avian	
	Acute Oral or Dermal LD50 (mg/kg)	Acute Inhalation LC50 (ppm)	Acute Oral or Dermal LD50 (mg/kg)	Acute Inhalation LC50 (ppm)
Very highly toxic	<10	<50	<10	<50
Highly toxic	10-50	51-500	10-50	51-500
Moderately toxic	51-500	501-1000	51-500	501-1000
Slightly toxic	501-2000	1001-5000	501-2000	1001-5000
Practically non-toxic	>2000	>5000	>2000	>5000

Source: USEPA 1998

C.3.4.3.1 ROTENONE TOXICITY TO MAMMALS

Mammalian acute oral toxicity LD50 values for rotenone range from 39.5 mg/kg for female rats to 1,500 mg/kg for rabbits. For most lab mammals, rotenone is much more toxic when administered intravenously or inhaled rather than taken orally. For example, the average oral LD50 for rats is 60 mg/kg compared with just 0.2 mg/kg for rotenone introduced directly into the bloodstream. Efficient breakdown of rotenone by the liver, oxidation of rotenone in the gut, and slow absorption in the stomach and intestines may account for this significant difference in toxicity (Narongchai et al. 2005, Ling 2003). This explanation may also account for the significant difference in rotenone sensitivity between mammals and fishes, and not from a difference in the primary site of action (Fukami et al. 1969). Indeed, USEPA considers rotenone safe to use in the presence of cattle (USEPA 2007~~1981~~).

C.3.4.3.2 ROTENONE TOXICITY TO BIRDS

Rotenone has a very low toxicity to wildfowl, and birds are extremely unlikely to be affected by fisheries management practices (Ling 2003). Avian acute toxicity LD50 values range from 130 mg/kg for the nestling English song sparrow (Cutcomp 1943) to 2,200 mg/kg for an adult mallard duck (USEPA 1988). In general, young birds are about 10 times more sensitive to rotenone poisoning (CDFG 1994) and, like mammals, birds have a much lower tolerance to rotenone when introduced intravenously. During rotenone treatments in California, fish-eating birds and mammals were observed foraging eradicated fish for several days following treatment. No sightings or dead birds or mammals followed (CDFG 1994).

Ling (2003) examined rotenone poisoning and sublethal toxicity in birds after consuming fish or even fish management baits. Ling concluded "rotenone is slightly toxic to wildfowl, and birds are extremely unlikely to be affected by normal fisheries management programs." For example, baits used to kill carp for management purposes have approximately 0.01 g of rotenone each. Ling calculated that a duck would need to consume approximately 200 baits to receive a fatal dose. Birds would be very unlikely to consume bait but could consume fish killed by rotenone. The concentration of rotenone in poisoned fish, however, is usually 25,000 times lower than that found in bait.

C.3.4.3.3 ROTENONE TOXICITY TO TERRESTRIAL INSECTS

Rotenone is extremely toxic to many species of insects in many different insect orders (caterpillars, beetles, flies, etc.) hence its wide popularity as an insecticide. However, the compound is considered non-toxic to bees unless used in combination with pyrethrum (Extoxnet 1996). Because rotenone would be used for fisheries management and would be applied strictly to an aquatic environment, only aquatic insects or aquatic stages of terrestrial insects would be significantly affected.

C.3.4.3.4 ROTENONE TOXICITY TO AMPHIBIANS

Rotenone is toxic to amphibians, but generally less toxic than to fish. Rotenone may be absorbed into both skin and respiratory membranes, but skin may present more of a barrier due to a greater distance for the chemical to diffuse across (Fontenot et al. 1994), and a smaller surface area relative to gill structures. Indeed, Fontenot et al. (1994) reported that amphibian larvae with gills are most sensitive to rotenone. In early 1974, African clawed frogs (*Xenopus laevis*) were discovered in some ponds located in the Santa Clara River drainage. An eradication program using rotenone to extirpate the exotic frogs was undertaken in the spring of 1974. Results indicated that all *X. laevis* tadpoles were killed but adults were unaffected and thus able to reproduce again later that spring (McCoid and Bettoli 1996).

In standard laboratory 24 hr and 96 hr aquatic rotenone toxicity tests, the LC50 values for tadpoles (*Rana sphenocephala*) and larval amphibians ranged between 5 μg/L and 580 μg/L in 24 hr tests and 25 μg/L to 500 μg/L in 96 hr tests (Fontenot et al. 1994, Chandler and Marking 1982). The adult Northern leopard frog demonstrated a much greater resistance with LC50 concentrations ranging from 240 μg/L and 1,580 μg/L (24 hr) and 240 μg/L and 920 μg/L (96 hr) (Table C-9). This suggests that tadpoles and other larval forms of amphibians that utilize gills for respiration are just as sensitive to rotenone as fishes while adult forms, which no longer utilize gills, are much less susceptible to rotenone. Larval amphibians appear to have resistance roughly equivalent to those of the most tolerant fish species.

Table C-9 Toxicity of Rotenone to Various Amphibians in Lakes

Species	Stage	Temp °C	24 hours LC50 (µg/L)	96 hours LC50 (µg/L)	Original Reference
N. Leopard frog (*Rana pipiens*)	Juvenile/ Adult	—	10	—	Haag 1931
	Tadpole	—	5	—	Hamilton 1941
	Adult	12	240	240	Farringer 1972
	Adult	12	1200	290	Farringer 1972
	Adult	12	1460	920	Farringer 1972
	Adult	12	1580	640	Farringer 1972
Tiger salamander (*Ambystoma tigrinum*)	Larvae	—	5	—	Hamilton 1941
S. Leopard frog (*Rana sphenocephala*)	Tadpole	15-17	30	25	Chandler and Marking 1982

C.3.4.3.5 ROTENONE TOXICITY TO REPTILES

Studies of rotenone toxicity to reptiles are particularly lacking (Fontenot et al. 1994). Carr (1952) and Dundee and Rossman (1989) suggested that soft-shelled turtles (*Apalone* spp.) may be affected by rotenone applications in fisheries, although neither provided supporting data. The adult green anole (*Anolis carolinensis*) was the only reptile species evaluated for acute toxicity in pre-registration testing of chemicals, including rotenone compounds (Fontenot et al. 1994). Aquatic turtle species with specialized respiratory mechanisms such as buccopharyngeal respiration (*Apalone spinifera* and *Kinosternon minor*), or modified skin and cloaca to enhance respiration (*Trachemys scripta* and *K. odoratum*) may be more susceptible to rotenone than other more terrestrial species. Turtle species in the Family Kinosternidae generally possess these special respiratory systems (Fontenot et al. 1994).

A fish population study using rotenone on Lake Conroe (Montgomery County, Texas) conducted between 1980 and 1986 indicated that aquatic turtles (*K. subrubrum*) were indeed susceptible to rotenone poisoning. At least 60 dead or dying individuals were observed around the periphery of the lake 24 to 48 hours after treatment, with the actual number of dead likely much higher because K. subrubrum tends to sink when dead (McCoid and Bettoli 1996). Freshwater aquatic snakes do not utilize aquatic respiration and absorption of rotenone through their thick skin is considered very unlikely (Fontenot et al. 1994). One study (Haque 1971), however, reported the death of an aquatic snake in a pond 48 hours after treating with rotenone, but also noted a second healthy-looking snake swimming in the same pond. The mechanism of action of uptake and toxicity of rotenone to reptiles requires further study.

C.3.4.4 Summary of Toxicity Reference Values (TRVs) Used for Ecological Risk Assessment

Table C-10 summarizes the range of acute and chronic TRVs identified for rotenone for vertebrates other than fish. Most mammal species are relatively resistant to rotenone.

The risk characterization (Section C5.1) uses these to calculate hazard quotients (HQ). Hazard quotients were evaluated using the methods presented in the USEPA ecological risk assessment for registration of rotenone (USEPA 2006). Hazard quotient standards were adjusted using several factors or "risk presumptions" to derive "Levels of Concern" (LOC) as listed in

Table C-11 and Table C-12. These values, similar to safety factors, are based on the endpoint used (i.e., acute versus chronic), frequency or duration of exposure (restricted or unrestricted site use), and the receptor's conservation status. For example, exposure of endangered species was evaluated using an LOC of 0.05 rather than a HQ of 1. If an acute toxicity value was used as the TRV, an LOC of 0.5 was used rather than an HQ of 1. In comparison, LOCs based on chronic exposure values were not adjusted.

Table C-10 Toxicity of Rotenone to Selected Mammalian and Avifauna

Animal Group	Test Endpoint	Lethal Concentration	Reference(s)
Mammals			
Human	Acute LD50 oral	300-500 mg/kg-body wt (Estimated)	Ray 1991; Gosselin et al. 1984
Rat	Acute LD50 oral	132-1500 mg/kg	Kidd and James 1991
	Acute LD50 oral	39.5 mg/kg (female)	USEPA 1988
	Acute LD50 oral	102 mg/kg (male)	USEPA 1988
	Acute LD50 I.V.	0.2 mg/kg	Hayes 1982
	Chronic LD50 oral	~10 mg/kg	Nat'l Research Council 1983
Mouse	Acute LD50 oral	350 mg/kg	Kidd and James 1991
Guinea pig	Acute LD50 oral	75 mg/kg	Haag 1931
	Acute LD50 I.P.	2 mg/kg	Haag 1931
	Acute LD50 I.M.	7 mg/kg	Haag 1931
	Acute LD50 S.C.	16 mg/kg	Haag 1931
Rabbit	Acute LD50 oral	~1.5 g/kg	Haag 1931
	Acute LD50 I.V.	~0.35 mg/kg	Haag 1931
	Acute LD50 I.M.	~5 mg/kg	Haag 1931
	Acute LD50 S.C.	~20 mg/kg	Haag 1931
Cat	Acute LD50 I.V.	~0.65 mg/kg	Haag 1931
Dog	Acute LD50 I.V.	~0.65 mg/kg	Haag 1931
	Chronic LD50 oral	~10 mg/kg (30d)	Haag 1931
	Chronic LD50 oral	>>10 mg/kg (180d)	Nat'l Research Council 1983
Birds			
Pigeon	Acute LD50 I.V.	1 mg/kg	Haag 1931
Japanese quail	Acute LD50 oral	1882 mg/kg	Hill et al. 1975
Mallard duck	Acute LD50 oral	2600-3568 mg/kg	Hill et al. 1975
Ring-necked pheasant	Acute LD50 oral	1608 mg/kg	Hill et al. 1975

Table C-11 Risk Presumptions for Aquatic Invertebrates Exposed to Rotenone Formulation Constituents from Silver King Creek Treatment

Toxicity Endpoint	Hazard Quotient (HQ) Calculation	Level of Concern (LOC) with Hazard Quotient
Acute Exposure	EPC[1]/LC50[2] *or* EC50[3]	0.5
Acute Restricted Use Exposure	EPC/LC50 or EC50	0.1
Acute Endangered Species Exposure	EPC/LC50 or EC50	0.05
Chronic Exposure	EEC/NOAEC[4]	1

Source: USEPA 1988

1. Exposure point concentration in primary media of exposure.
2. Median lethal concentration of chemical that kills 50% of the test organisms
3. Median effective concentration of chemical that elicits measurement of effect in 50% of the test organisms
4. No observable adverse effect concentration

Table C-12 Risk Presumptions for Non-Target Terrestrial Animals Exposed to Rotenone Formulation Constituents from Silver King Creek Treatment

Toxicity Endpoint	Hazard Quotient (HQ) Calculation	Level of Concern (LOL) with Hazard Quotient
Acute Exposure	EPC1/LC502 or EC503	0.5
Acute Restricted Use Exposure	EPC/LC50 or EC50	0.2
Acute Endangered Species Exposure	EPC/LC50 or EC50	0.1
Chronic Exposure	EEC/NOAEC4	1

Source: USEPA 2006

1. Exposure point concentration in primary media of exposure.
2. Median lethal concentration of chemical that kills 50% of the test organisms
3. Median effective concentration of chemical that elicits measurement of effect in 50% of the test organisms
4. No observable adverse effect concentration

C.3.5 Environmental Fate and Hazards from Formulation Ingredients and Potassium Permanganate Neutralizing Agent

Concern about risks to the environment include whether or not the chemical constituents in commercial rotenone formulations are toxic to wildlife, how rapidly they break down in the environment, and whether or not they build up in the food chain. Thus, these constituents constitute the chemicals of potential concern or COPCs for this assessment. This section also evaluates the fate and hazards of potassium permanganate, the compound used to neutralize rotenone and to protect downstream areas.

C.3.5.1 Physical and Chemical Properties of Carrier and Dispersant Ingredients in Rotenone Formulations

The manufacturer reports that formulations contain the same concentration of rotenone (5%). However, the concentrations and types of dispersant and carrier compounds in the 2 formulations differ substantially. Table C-13 summarizes some of the physical and chemical characteristics of rotenone compared to the various inert ingredients and carrier compounds present in CFT Legumine™, NoxFish®, and Nusyn-Noxfish®. The physical and chemical characteristics of a compound determine its fate in the environment. The rate and manner of the breakdown of each chemical is dependent on its solubility, volatility, tendency to adsorb to soil or sediment particles, and other factors shown in this table. As demonstrated in Table C-13, several of the components are common to both formulations, and others are unique.

C.3.5.1.1 CFT LEGUMINE™

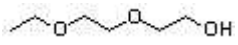
The CFT Legumine™ formulation contains approximately 5% rotenone, 10% methyl pyrrolidone (MP), 60% diethylene glycol monoethyl ether (DEGEE), 17% Fennodefo 99™ (Fennodefo), and 3% other compounds (CDFG 2007). The 2 primary inactive carrier components in CFT Legumine™ are MP and DEGEE, which comprise approximately 93% of the formulation by weight as determined by CDFG (Table C-13). Both of these chemicals are infinitely soluble in water and have an estimated organic carbon partition coefficient (i.e., the "K_{oc}") of 12, indicating their water solubility and tendency not to adsorb to sediment particles (NLM 2006). Based on their low Henry's Law constants, these chemicals do not readily

volatilize from surface water, and neither chemical is expected to undergo hydrolysis or direct photolysis (NLM 2006).

Aerobic biodegradation would be the most important mechanism for the removal of MP and DEGEE from aquatic systems (NLM 2006). The small amount of these chemicals that may volatilize into ambient air would be readily degraded by reaction with photochemically-produced hydroxyl radicals, with an atmospheric half-life of up to 12 hours (NLM 2006). The Fennodefo constituent in CFT Legumine™ facilitates emulsification and dispersion of the otherwise relatively insoluble rotenone. Two classes of constituents, polyethylene glycols (PEGs) and the solvent hexanol (alcohol), are part of the inert additive Fennodefo in CFT Legumine™, which also contains fatty acid esters. As stated in the "Screening Level Risk Analysis of Previously Unidentified Rotenone Formulation Constituents Associated with the Treatment of Lake Davis" (ENVIRON 2007), the fatty acid ester mixture in Fennodefo is likely derived from 'tall oil.' Tall oil has been independently reported as a mixture of naturally occurring fatty acids, resins and neutrals that are a by-product of wood pulp, and is a common constituent of soap formulations. The fatty acids in tall oil, principally oleic and linoleic acids, are naturally occurring constituents that are also part of the building blocks that make up fats and oils (triglycerides). Highly unsaturated fatty acids, like linoleic, are considered essential dietary constituents in humans, as they cannot be synthesized. Polyethylene glycols (e.g. propylene glycol) are common ingredients in a variety of consumer products, including soft drink syrups (as an antioxidant), in plasticizers, suntan lotions and antifreeze, among other uses (ENVIRON 2007).

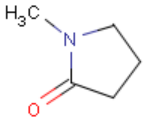
The structures and oral toxicities of the two most concentrated constituents in CFT Legumine™ are summarized below.

DIETHYLENE GLYCOL MONOETHYL ETHER



- Approximate concentration in formula: 569,000 mg/L
- Toxicology: RAT ORAL LD50: 4,700-9,740 mg/kg.
- Chemical formula: C6H14O3
- Chemical structure: C2H5OCH2CH2OCH2CH2OH

1-METHYL-2-PYRROLIDINONE



- Approximate concentration in formula: 90,000 mg/L
- Toxicology: RAT ORAL LD50: 3,914 mg/kg
- Chemical formula: C5H9NO

Table C-13 Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Concentration in Formulation (µg/g)	Concentration in 0.5 mg/L Treatment (µg/L)[1]	Concentration in 1.0 mg/L Treatment (µg/L)[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity (g/mL)	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
CFT Legumine™																	
Rotenone	50,900 as reported in lab analyses (Environ~~Fisher~~ 2007)	25.5	50.9	394.4	210-220 / 0.5 mm	0.2 mg/L (Re-registration doc and HSDB)	6.9 x 10^{-10}		1.1 x 10^{-13}	1.27 @ 20°C	4.10	Hydrolysis: 3.2 days @ pH=7, 2 days @ pH=9 Aqueous photolysis: 21 hrs (1 cm), 191 days (2 m, well mixed) Entire pond system (water + sediment): 20 days in cold water (5°C), 1.5 days in warm water (25-27°C) Air photooxidation: 0.05 days Soil: 3 days		TOC: 0.36 mg/kg			LD50 Mice (i.p.): 2.8 mg/kg Rats (oral): 132 mg/kg-bw; (i.v.): 6 mg/kg Human: ingestion or inhalation of large doses may lead to numbness of oral mucosa, respiratory paralysis at lethal doses, tremor, tachypnea, nausea, vomiting. Chronic exposure may produce fatty changes in liver and kidney. More toxic when inhaled than ingested. Skin irritation from direct contact.
Rotenolone	7,340 as reported in lab analyses (Environ~~Fisher~~ 2007)	3.67	7.34	412.42													Oral LD50 Mice: rotenolone I, 4.1 mg/kg rotenolone II, 25 mg/kg
1-Methyl-2-pyrrolidinone (Methyl pyrrolidone)	98,900 as reported in lab analyses (Environ~~Fisher~~ 2007)	49.5	98.9	99.13	202	infinitely soluble in water	0.345	3.4	4.46 x 10^{-8}	< 1.0	-0.54	Air photooxidation: 5 hrs Soil: 4 days in clay, 8.7 days in loam, 11.5 days in sand	1 mg/m³ = 0.24 ppm	mild amine odor		NOEL = 5 g/L in bacteria, algae (*Scenedesmus)* and protozoa (*Colpoda)*	
Diethylene glycol monoethyl ether (Diethylene glycol ethyl ether)	610,000 as reported in lab analyses (Environ~~Fisher~~ 2007)	305	610	134.2	202	infinitely soluble in water	0.13	4.62	4.86 x 10^{-8}	0.99 @ 20°C / 4°C	-0.08 (USEPA RAGS E and HSDB)	Air photooxidation: 12 hrs	1 mg/m³ = 0.188 ppm	Quality: sweet, musty Hedonic tone: unpleasant to pleasant; Abs.: 0.21 ppm 50% recog: 1.10 100% recog: 1.10 O.I. recog: 600 O.I. at 20°C = 120	BOD: 0.20 NEN 3235-5.4 COD: 1.85 NEN 3235-3.3	24 hr LC50: > 5,000 mg/L (goldfish, static); 96 hr LC_{50}: > 10,000 mg/L, (*Menidia beryllina*, static)	Oral LD50 (single dose): Rat = 8.69-9.74 g/kg Guinea pig: 3.67-4.97 g/kg Cat: 1 ml/kg (lethal) Rat NOEL: 0.49 g/kg (repeat oral dose) Rabbit, cat, guinea pig, mouse inhalation—no injury w/ 12 day exposure to saturated vapor.
1-Hexanol	4,239 as reported in lab analyses (Environ~~Fisher~~ 2007)	2.12	4.24	102.2	158	5,900 mg/L @ 20°C	0.98 mm @ 20°C	3.52		0.82			1 mg/cu m=0.24 ppm 1 ppm=4.25 mg/cu m	Odor: sweet alcohol	BOD: 28% of ThOD; COD: 94% of ThOD		LED50 orally in rats: 4.59 g/kg Toxicity threshold (cell multiplication inhibition test): bacteria (Pseudomonas putida): 62 mg/l; algae
sec-Butylbenzene	3.9 [0.00055% by wt]	0.00195	0.00390	134.21	173	17	1.1 (20°C)	4.62	0.019	0.862		Aqueous volatilization: est. 3.4 hrs for model river, 4.6 days for model lake, and 88 days for model pond (includes sediment adsorption) Air photooxidation: 1.9 days	Relative chemical reactivity [RCR]: 1.31	distinctive aromatic odor			Eye irritation reactivity [EIR] in man @ 1.8
1-Butylbenzene (n-Butylbenzene)	23.9 as reported in lab analyses (Environ~~Fisher~~ 2007)	0.0120	0.0239	134.21	183	14	1	4.62	0.0883	0.860	4.03	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake, and 16 days for model pond (includes sediment adsorption) Air photooxidation: 1.8 days	RCR: 1.03		ThOD: 3.22		EIR: 6.4 (man)
1,4-diethylbenzene	500 as reported in lab analyses (Environ~~Fisher~~ 2007)	0.250	0.500	134.2	183.7	17	.92	.006646	.00755		4.06	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake				

Table C-13 Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Concentration in Formulation (µg/g)	Concentration in 0.5 mg/L Treatment (µg/L)[1]	Concentration in 1.0 mg/L Treatment (µg/L)[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity (g/mL)	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
1,2,4-Trimethylbenzene	34.8 as reported in lab analyses (EnvironFisher 2007)	0.0174	0.0348	120.19	169	57	2.1	4.15	0.00616	0.8761	3.78	Aqueous volatilization: est. 3 hrs for model river, 4 days for model lake, Air photooxidation: 12 hrs	1 mg/m³ = 0.203 ppm				
1,3,5-Trimethylbenzene (*aka* mesitylene)	4 [0.00056% by wt]	0.00200	0.00400	120.19	164.7	48.2	2.4	1.006 @ 20°C	0.147	0.865	4.00	Aqueous volatilization: est. 3 hrs for model river, 4 days for model lake, and 5 days for model pond (includes sediment adsorption) Air photooxidation: 7 hrs	1 mg/m³ = 0.203 ppm; 0.4% of emitted hydrocarbons from diesel engines	Avg recog.: 0.027 mg/L Range: 0.00024-0.062 mg/L	BOD: 3% of Theoretical Oxygen Demand (ThOD) COD: 10% of ThOD	96 hr median threshold limit = 13 mg/L (goldfish, flow-through)	
1,2,4,5-Tetramethylbenzene	402 as reported in lab analyses (EnvironFisher 2007)	0.201	0.402	134.2	196.8	33.9	0.118	0.000852	.00799	.84	4.0	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake				
Toluene	222 as reported in lab analyses (EnvironFisher 2007)	0.111	0.222	92.13	110.6	56.2	30	3.1	0.00664	0.8636 @ 20°C / 4°C	2.75	Aqueous volatilization: est. 1 hr for model river and 4 days for model lake Water: 4 days (aerobic), 56 days (anaerobic) Uncontaminated estuarine: 90 days Soil biodegradation: several hrs to 71 days Air photooxidation: 3 days	1 mg/m³ = 0.265 ppm	water: 0.04 ppm air: 2.14 ppm			LD50 (rats) 7.53 g/kg
4-Isopropyltoluene (p-Isopropyltoluene)	5.1 [0.00072% by wt]	0.00255	0.00510	134	177	16.8	1.75	4.62	0.0183	0.8610 @ 20°C / 4°C	4.16	Aqueous volatilization: est. 1 hr for model river, 5 days for model lake, and 30 days for model pond (includes sediment adsorption) Air photooxidation: 1 day		sweet aromatic odor			
Methylnaphthalene	140 [0.0198% by wt]	0.0700	0.140	142.19	241	24.6	0.0677	4.91	5.17×10^{-4}	1.025	3.86	Aqueous volatilization: est. 5.5 hrs for model river, 5.3 days for model lake, and 78 days for model pond (includes sediment adsorption) Air photooxidation: 7.4 hrs	1 mg/m³ = 0.17 ppm;	water: 0.023 ppm (range = 0.0025-0.17 ppm) TOC (detection) = 0.0075 mg/kg		24, 48, 72, 96-hr LC50 = 39, 9, 9, 9 mg/L in FHM (static); 48-hr LC50 in brown trout yearlings = 8.4 mg/L (static); BCF: 20 to 130 in coho salmon muscle, depending on length of exposure.	

Table C-13 Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Concentration in Formulation (µg/g)	Concentration in 0.5 mg/L Treatment (µg/L)[1]	Concentration in 1.0 mg/L Treatment (µg/L)[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity (g/mL)	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
Naphthalene	253 as reported in lab analyses (EnvironFisher 2007)	0.127	0.253	128.6	217.9	31	0.23	4.42	4.83×10^{-4}		3.36	Aqueous volatilization: est. 3 hrs for model river and 5 days for model lake Aqueous photolysis: 71 hrs Aqueous biodegradation: 0.8-43 days Sediment: Degradation rates in sediment are 8-20 times higher than in the above water column. Biodegradation half-lives ranged from 2.4 weeks in sediments chronically exposed to petroleum hydrocarbons to 4.4 weeks in sediment from a pristine environment. Soil biodegradation: 2-18 days Air photooxidation: 18 hrs	1 mg/m³ = 0.191 ppm	water: 0.021 ppm air: 0.084 ppm			
Fennodefo 99™ ingredients (a mixture of tree resin components (polyethylene glycols, fatty acids and resin acids) that represents approximately 173,000 µg/g of CFT Legumine™)																	
Triethylene Glycol	326 as reported in lab analyses (EnvironFisher 2007)	0.163	0.326	150.2	285	Easily soluble in cold water	<0.001 mm @ 20 degrees C	5.17		1.1@20C/4 C			1 ppm-6.14 mg/cu m	Practically odorless	BOD5: 0.03 NEN 3235-5.4, 1.4% of ThOD; BOD10: 0.50 std.dil.sew.; 10 days: 3.7% of ThOD; 15 days: 11.5% of ThOD; 20 days; 17.0% of ThOD: COD: 1.57 NEN 3235-5.3	LC50/ 96 hr values for fish are between 10 and 100 mg/l. Therefore, this material is expected to be slightly toxic to aquatic life.	LD 50 Oral mice, rats (g/kg): 21, 15-22; Toxicity threshold (cell multiplication inhibition test) in mg/l: bacteria (Pseudomonas putida): 320; alage (Microcystis aeruginosa): 3600; protozoa (Entosiphon sulcatum). Goldfish: 24 hr LD50=>5,000 mg/l; guppy: 7 d LC50: 62.600 ppm. Single oral doses LD50: Guinea pig: 14.6 g/kg; 7.9 ml/kg. Rat (repeated oral dose): no effect@3-4 g.kg/day, 30 days; Man: very low acute and chronic toxicity
Tetraethylene Glycol	1,304 as reported in lab analyses (EnvironFisher 2007)	0.652	1.30	194.2	327	Fully miscible in water	0.001 mm @ 20 degrees C	6.7		1.12				Faint amine odor	BOD10: 0.50 std. dil.sew.		Rats: single oral LD50: 32.8 g/kg, and 28.9 ml/kg-1; Rabbit: skin LD 50>20,000 mg/kg
Pentaethylene Glycol	2,826 as reported in lab analyses (EnvironFisher 2007)	1.41	2.83	238.3	338-340					1.126							
Hexaethylene Glycol	5,109 as reported in lab analyses (EnvironFisher 2007)	2.55	5.11		217 @ 4 mm Hg	Fully miscible in water				1.127				Not determined			Oral Rat LD50: 32,000 mg/kg-1; Oral Guinea Pig: 20,000 mg/kg-1
"Tall Oil" is an byproduct of the Kraft process that is used to create pulp from wood and includes naturally-occurring fatty acids and resin acids that are widely used by the food, soap and other industries.	Unknown, butestimated to be ≤ 163,435 based on the Fennodefo 99™ content minus the summed concentration of ethyl,ene glycols	≤ 81.7	≤ 163		160-210 at 6.6 hPa	Virtually insoluble in water	Negligible at 25 deg C				4.89-5.98 at 25 deg C					Fish: Semistatic; 96 hour exposure; NOEC >=1000mg/L Invertebrates: (Crustacea); 48 hour exposure; NOEC>=1000mg/L Plants: (Algae); 72 hour exposure; NOEC>=1000mg/L	Oral: LD50, Rat @ 74000 mg/kg bw (Oleic) LD50 Rat @>3200 mg/kg bw (linoleic) LD50, Rat @ 7600 mg/kg bw (Rosin) Skin: Rabbit, Slight Irritant Eye: Rabbit, Slight irritant

Table C-13 Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Concentration in Formulation (µg/g)	Concentration in 0.5 mg/L Treatment (µg/L)[1]	Concentration in 1.0 mg/L Treatment (µg/L)[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity (g/mL)	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
Abietic Acid	unknown			302.4	250 @ 9 mm Hg	insoluble											LC50 values to crustaceans: 6.2 mg/l=96 hr, *Nitocra spinipes*; LC50 values in fish: 0.56 mg/l=96 hr, *Salmo gairdneri*; 0.41 mg/l=96 hr, *Oncorhynchus kisutch*.
Beta-Pinene	unknown			136.2	167		2 mm Hg @ 20 degrees	4.7	0.049 mol/kg*bar								
Isopimaric Acid	unknown			302.5		26 mg/mL										LC50=0.4 mg/l for rainbow trout for isopimaric acid in lodgepole pine sapwood (Wang et al. 1995).	
Oleic Acid (112-80-1) <Tall Oil Partition>	unknown			282.5	360 deg C	Insoluble	1 mm Hg @ 177 deg C	9.7 (air=1)		0.895 (water=1)				Rancid odor (Lard like)		Fish: Fathead Minnow: LC50 = 205 mg/L; 96 Hr.; Static condition	LD50/LC50: Draize test, rabbit, eye: 100 mg Mild; Oral, mouse: LD50 = 28 gm/kg; Oral, rat: LD50 = 25 gm/kg; Human Skin Draize 15 mg/3D intermittent; REACTION: Moderate.
Linoleic Acid (60-33-3) <Tall Oil Partition>	unknown			280.4	229-230 deg C @ 16.00mm Hg	Insoluble				0.9020g/cm 3					COD: 8.38% of ThOD BOD: 71% of ThOD	Invertebrate toxicity: EC50 (duration unspecified) purple sea urchin 0.28-1.07 mg/kg inhibited fertilization (Cherr et al. 1987).	Oral, mouse: LD50 = >50 gm/kg
Linolenic Acid (463-40-1) <Tall Oil Partition>	unknown			278.4	230-232 deg C @ 1 mm Hg	Insoluble		9.6									
Noxfish® and Nusyn Noxfish®																	
Rotenone	50,000 in Noxfish® and 25,000 in Nusyn Noxfish®	25.0	25.0														
Rotenolone	15,000	7.5	15														
Piperonyl butoxide	25,000 in Nusyn Noxfish®	Not applicable	25.0	338.45	180					1.509		Air: 3.4 hours; water 0.55 to 1.64 days; soil ≤ 4.3 days				Fish LC50 3.94 to 6.15 mg/L; Invertebrate LC50 0.23 to 0.51 mg/L	Rat oral LD50 4,570 to 12,800 mg/kg; mouse oral LD50 2,600 mg/kg; rabbit oral LD50 2,700 to 5,300 mg/kg
Trichloroethene (Trichloroethylene)	73	0.0365	0.0730	131	87	1,100	75	4.53	0.0103	1.4642 @ 20°C / 4°C	2.71	Aqueous volatilization: est. 3.5 hrs for model river, 5 days for model lake Aqueous hydrolysis: 10.7 months Air photooxidation: 7 hrs	1 mg/m³ = 0.186 ppm	water: 10 ppm air: 50 ppm, disagreeable above 200 ppm			
Toluene	1,800	0.900	1.80	92.13	110.6	56.2	30	3.1	0.00664	0.8636 @ 20°C / 4°C	2.75	Aqueous volatilization: est. 1 hr for model river and 4 days for model lake Water: 4 days (aerobic), 56 days (anaerobic) Uncontaminated estuarine: 90 days Soil biodegradation: several hrs to 71 days Air photooxidation: 3 days	1 mg/m³ = 0.265 ppm	water: 0.04 ppm air: 2.14 ppm			LD50 (rats) 7.53 g/kg

Table C-13 Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Concentration in Formulation (µg/g)	Concentration in 0.5 mg/L Treatment (µg/L)[1]	Concentration in 1.0 mg/L Treatment (µg/L)[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity (g/mL)	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
1,3- and/or 1,4-Xylene (m-/p-xylene)	610	0.305	0.610	106		185	9.5	3.7	0.00766	0.86104 @ 20°C / 4°C	3.20	*1,3-xylene* Aqueous volatilization: est. 3 hrs for model river and 4 days for model lake Air photooxidation: 16 hrs *1,4-xylene* Aqueous volatilization: est. 3 hrs for model river and 4.1 days for model lake Air photooxidation: 27 hrs	1 mg/m³ = 0.23 ppm	mixed isomers: water: 0.53 ppm air: 0.102 ppm			
1,2-Xylene (o-xylene)	76	0.0380	0.0760	106	144	178	7	3.7	0.00519	0.8801 @ 20°C / 4°C	3.13	Aqueous volatilization: est. 3.2 hrs for model river and 4.1 days for model lake Air photooxidation: 1.2 days	1 mg/m³ = 0.23 ppm	mixed isomers: water: 0.53 ppm air: 0.102 ppm			
Isopropylbenzene	52	0.0260	0.0520	120	153	61.3	4.6	4.1	0.0131	0.862 @ 20°C / 4°C	3.50	Aqueous volatilization: est. 1.2 hrs for model river and 4.4 days for model lake Air photooxidation: 2.5 days		detection: 0.008 ppm recognition: 0.047 ppm			
1-Propylbenzene (n-Propylbenzene)	310	0.155	0.310	120	158	23.4	2.5	4.14	0.00659	0.862 @ 20°C / 4°C	3.60	Aqueous volatilization: est. 1 hr for model river and 4 days for model lake Air photooxidation: 2 days					
1,3,5-Trimethylbenzene	860	0.430	0.860	120.19	164.7	48.2	2.4	1.006 @ 20°C	0.147	0.865	4.00	Aqueous volatilization: est. 3 hrs for model river, 4 days for model lake, and 5 days for model pond (includes sediment adsorption) Air photooxidation: 7 hrs	1 mg/m³ = 0.203 ppm; 0.4% of emitted hydrocarbons from diesel engines	Avg recog.: 0.027 mg/L Range: 0.00024-0.062 mg/L;	BOD: 3% of Theoretical Oxygen Demand (ThOD) COD: 10% of ThOD	96 hr median threshold limit = 13 mg/L (goldfish, flow-through)	
1,2,4-Trimethylbenzene	10,000	5.00	10.0	120	169	57	2.1	4.15	0.00616	0.8761 @ 20°C / 4°C	3.78	Aqueous volatilization: est. 3 hrs for model river and 4 days for model lake Air photooxidation: 12 hours	1 mg/m³ = 0.203 ppm				
1-Butylbenzene (n-Butylbenzene)	9,000	4.50	9.00	134	183	14	1	4.62	0.0883	0.860	4.03	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake, and 16 days for model pond (includes sediment adsorption) Air photooxidation: 1.8 days					

Table C-13 Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Concentration in Formulation (µg/g)	Concentration in 0.5 mg/L Treatment (µg/L)[1]	Concentration in 1.0 mg/L Treatment (µg/L)[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity (g/mL)	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
4-Isopropyltoluene (p-Isopropyltoluene)	1,000	0.500	1.00	134	177	16.8	1.75	4.62	0.0183	0.8610 @ 20°C / 4°C	4.16	Aqueous volatilization: est. 1 hr for model river, 5 days for model lake, and 30 days for model pond (includes sediment adsorption) Air photooxidation: 1 day		sweet aromatic odor			
Naphthalene	70,000 (EPA method 8260) 28,000 (EPA method 8270)	35.0 (EPA 8260)	70.0 (EPA 8260)	128.6	217.9	31	0.23	4.42	4.83×10^{-4}	1.162	3.36	Aqueous volatilization: est. 3 hrs for model river and 5 days for model lake Aqueous photolysis: 71 hrs Aqueous biodegradation: 0.8-43 days Sediment: Degradation rates in sediment are 8-20 times higher than in the above water column. Biodegradation half-lives ranged from 2.4 weeks in sediments chronically exposed to petroleum hydrocarbons to 4.4 weeks in sediment from a pristine environment. Soil biodegradation: 2-18 days Air photooxidation: 18 hrs	1 mg/m³ = 0.191 ppm	water: 0.021 ppm air: 0.084 ppm			
Potassium permanganate neutralizing compound for rotenone																	
Potassium permanganate	100% (applied at 4x rotenone concentration	2 mg/L	4 mg/L	158		64,000 (20°C)	Na	na	na	na				odorless		96-hr LC50: 3.6 mg/L (goldfish) 0.75 mg/L (channel catfish) 96-hr LD50: 2.7-3.6 mg/L (bluegill)	Oral LD50 (single dose): Guinea pig: 810 mg/kg Mouse: 750 mg/kg Rat: 750 mg/kg

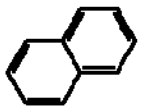
[1] CFT Legumine™ can be applied at either 0.5 mg/L or 1.0 mg/L; Noxfish® is applied only at 0.5 mg/L and Nusyn Noxfish® is applied only at 1.0 mg/L

C.3.5.1.2 NOXFISH®

In contrast to CFT Legumine™, the inert and carrier chemicals for Noxfish® consist of the polycyclic aromatic hydrocarbon (PAH) naphthalene, numerous alkylated benzenes, and trichloroethene. These chemicals are moderately soluble in water, with aqueous solubilities ranging from 14 to 1,100 mg/L (NLM 2006). K_{oc} values range from 94 to 3,200 L/kg, suggesting that these chemicals may also tend to adsorb to sediment particulates, thus increasing their half-lives in natural waterbodies (NLM 2006). The half-lives for these chemicals in surface water bodies range from several hours to several months, depending on the characteristics of the waterbody (i.e., temperature, flow velocity, turbulence, etc.), as well as the amount of sunlight on the water surface. With Henry's Law constants ranging from 0.00048 to 0.15 atm-m^3/mol, the primary removal mechanism from surface water for these carrier chemicals is volatilization, with direct photooxidation, hydrolysis and biodegradation contributing to a much smaller degree. Once in the ambient air, chemical vapors are readily degraded by reaction with photochemically-produced hydroxyl radicals. The chemical-specific half-lives for this reaction in air range from a few hours to a few days (NLM 2006). Naphthalene comprises slightly less than 50% of the NoxFish® formulation by weight (see Table C-13). This PAH, which gives moth balls their distinctive odor, has an odor threshold in air of 0.084 ppm, or 0.44 mg/m^3.

NAPHTHALENE

- Approximate concentration in Noxfish® formula: 70,000 mg/L
- Toxicology: MOUSE ORAL LD50: 533 mg/L
- Chemical formula: C10H8
- Chemical structure:

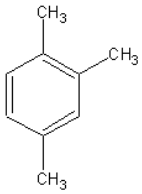


TOLUENE

- Approximate concentration in Noxfish® formula: 1,800 mg/L
- Toxicology: MOUSE ORAL LD50: 636 mg/kg
- Chemical formula: C_7H_8
- Chemical structure:

1, 2, 4-TRIMETHYLBENZENE

- Approximate concentration in Noxfish® formula: 10,000 mg/L
- Toxicology: MOUSE ORAL LD50: 5,000 mg/kg
- Chemical formula: C_9H_{12}
- Chemical structure:



C.3.5.2 Fate, Transport and Toxicity of Proposed Rotenone Formulation Constituents and Potassium Permanganate Neutralization Solution

C.3.5.2.1 REVIEW OF ROTENONE DISPERSANT FATE AND TOXICITY FROM FIELD STUDIES CONDUCTED OUTSIDE PROJECT AREA

Surface and groundwater near California lakes and streams treated with liquid and powdered rotenone formulations have been monitored after ten treatment projects since 1987 (Finlayson et al. 2001, McMillin and Finlayson 2008). They determined that all measured concentrations of dispersant ingredients were well below USEPA drinking water standards. For example, TCE concentrations never exceeded the USEPA drinking water standard (Maximum Contaminant Level [MCL]) of 5 μg/L. Similarly, xylene concentrations of xylene never exceeded the drinking water standard (Health Advisory) of 620 μg/L (WDFW 2002). No drinking water standards exist for naphthalene and methylnaphthalenes; however, these VOCs and semivolatile organic compounds (SVOCs) disappeared before rotenone dissipated, typically within one to three weeks.

The physico-chemical properties of the VOCs and SVOCs in the rotenone formulations do not promote accumulation or persistence in sediment. Finlayson et al. (2001) reported that rotenone, rotenolone and only two SVOCs (naphthalene and methylnaphthalene) were detected above detection limits (30 μg/kg-dry wt for rotenone and rotenolone, and 6 μg/kg for the VOCs and SVOCs. In standing water sediments from these nine study sites, rotenone and rotenolone were detected a maximum of 60 days, with maximum concentrations of 522 and 890 μg/kg-dry weight, respectively. No VOCs (e.g. xylene, TCE) were ever detected in either flowing or static water sediments. The only SVOCs detected in lake sediments were naphthalene, 1-methylnaphthalene, and 2-methylnaphthalene. Detectable concentrations of these SVOCs were measured up to 180 days after treatment in standing water sediments, with maximum concentrations of 91 and 231 μg/kg for naphthalene and methylnaphthalene, respectively.

The rotenone formulation used at Lake Davis, California in 1997, contained several VOCs and SVOCs (USEPA 2006). These chemicals included naphthalene, methyl naphthalene, toluene, and xylene. Additionally, TCE, a chemical used to extract rotenone from plant tissues has also been reported. In addition to these compounds, formulated end-products may also contain varying amounts of cube root resin (rotenoloids such as rotenololone) and the extent of their toxicity is uncertain. However, toxicity testing with formulated end products suggests that, in general, co-formulants do not substantially affect the toxicity of rotenone based on reported distributions of acute 96 hr LC50 values among different species (USEPA 2006). Based on these results, the distribution of species sensitivities observed in laboratory tests represents the distribution of sensitivities likely encountered in the environment.

The Minnesota Department of Health conducted a risk assessment of the inert ingredients in Nusyn-Noxfish® for the Minnesota Department of Natural Resources. Their assessment reported August 7, 1991, stated that "There is negligible risk to human health from the contaminants found in rotenone whether the exposure is from drinking, swimming or eating fish from treated waters (as cited in WDFW 2002). In addition, they determined that treatment with rotenone will introduce contaminants into the lake, but at concentrations considerably lower than the levels that would harm human health" (WDFW 2002).

As part of the re-registration process USEPA (2006) conducted a review of the available toxicity data on all formulated products of rotenone and the formulation ingredients typically evaluated. However, only limited toxicity data were available on the inert ingredients. The evaluation of both technical grade rotenone (>95% active ingredient) and formulated end-product determined that the technical grade active ingredient is generally more toxic than formulated end-product [corrected for active ingredient] by at least a factor of 2 (USEPA 2006). These data suggest that for the formulated products tested and the toxicity endpoints measured, the dispersant ingredients do not contribute substantially to the toxicity of the active ingredient and are effectively inert.

In addition, USEPA (2006) suggested that the similarly structured rotenolones of plant resins (cube root resins) contained in varying amounts in formulated end-products also do not contribute substantially to the toxicity of rotenone. Rotenolone persists longer than rotenone, especially in cold, alpine lakes; rotenolone has been detected for as long as 6 weeks in cool water temperatures (<10°C) at high elevations (>8,000 feet). In part, this occurs because rotenone may be more susceptible to photolysis than rotenolone (Finlayson et al. 2000). However, studies have indicated that rotenolone is approximately one-tenth as lethal as rotenone (Ott 2006 ~~CDFG 1991 as cited in~~ Finlayson et al. 2000, Gersdorff 1933). In those rare cases of rotenolone persistence, fish restocking would be delayed until both rotenone and rotenolone residues have declined to below detection limits (<2 ppb) to err on the side of safety (Finlayson et al. 2000). Table C-14 summarizes available toxicity information for the inert ingredients identified in the rotenone formulations proposed for Silver King Creek.

C.3.5.2.2 POTASSIUM PERMANGANATE NEUTRALIZING SOLUTION

Potassium permanganate ($KMnO_4$) is a strong oxidizing agent used in many industries and laboratories. It is used as a disinfectant in treating potable water. In fisheries and aquaculture, Potassium permanganate is used to treat some fish parasites. Under the proposed Action, Potassium permanganate would be used to neutralize rotenone (USEPA 2006, Ling 2003). Following rotenone application, Potassium permanganate is applied to the treated water at a ratio between two and four parts Potassium permanganate to each part of rotenone (USEPA 2006). Under the proposed Action, the potassium permanganate concentration may range from 2 to 4 mg/L depending on the organic load in the receiving water at the time of treatment.

Manganese is the principal element in the permanganate solution with potential toxicity. However, manganese is also an essential nutrient for plants and animals, and specific signs of manganese deficiency include a wide range of symptoms including nervous system disorders, bone fragility, and growth suppression (Browning 1961). Manganese comprises about 0.1% of the earth's crust and is ubiquitous in the environment (rock, soil, water). Potassium permanganate is produced by thermal oxidation of manganese dioxide (MnO_2) followed by electrolytic oxidation. The environmental chemistry and fate of manganese is controlled largely by pH. At pH values above 5.5 (approximately), colloidal manganese hydroxides generally form in water. Such colloidal forms are not generally bioavailable. As a strong oxidizing agent, permanganate is reduced when it oxidizes other substances (such as rotenone). Thus, in the process of oxidizing rotenone, Potassium permanganate is in turn reduced, liberating bioavailable oxygen in the process. This mechanism counters rotenone's respiratory toxicity. In the process, potassium ions are liberated (also an essential electrolyte), and manganese dioxide is formed. Manganese dioxide is insoluble, hence not bioavailable, and chemically similar to the MnO_2 found in the earth's crust (Vella 2006).

Table C-14 Aquatic and Terrestrial Toxicity Data for Inert Ingredients Present in Proposed Rotenone Formulations

Ingredient	Toxicity to Aquatic Receptors	Toxicity to Terrestrial Receptors			
		Acute ORAL LD50	IHL LC50 /IPR/IVN LD50	Acute Dermal LD50	Other
Rotenone	See rotenone information				
Rotenolone	Not Available	Not Available	Not Available	Not Available	Not Available
Piperonyl butoxide	Fish LC50s 3.94 to 6.12 mg/L;invertebrate LC50s 0.23 to 0.51 mg/L	Rat LD50s 4,570 to 12,800 mg/kg; Rabbit LD50s 2,700 to 5,300 mg/kg	Rat acute inhalation LD50 >5,900 mg/L	Rat LD50 7,960 to 13,500 mg/kg; rabbit LD50 2,650 to 5.300 mg/kg; mouse LD504,030 mg/kg	
Methyl pyrrolidone (*aka* n-methylpyrroli)		RAT: 3,914 mg/kg MUS: 7,725 mg/kg	IPR-RAT LD50: 2,472 mg/kg IVN-RAT LD50: 2,266 mg/kg	RBT: 8,000 mg/kg	Typical LTEL: 25 ppm. AIHA Workplace environmental exposure level: 10 ppm (8h).
Diethylene glycol ethyl ether	24h LC50: 5,000 mg/L (Goldfish, static). 96h LC50: >10,000 mg/L (*Menidia beryllina*, static)	RAT: 8,690-9,740 mg/kg GPIG: 3,670-4,970 mg/kg			CAT: 1 ml/kg (lethal) RAT NOEL: 490 mg/kg (repeat oral dose) RBT, CAT, GPIG, MUS inhalation: no injury with 12d exposure to saturated vapor.
Fennodefo 99™	As "tall oil" 96 Hr fish NOEC ≥ 1,000 mg/L; 48 hr crustacean NOEC ≥ 1,000 mg/L; algae 72 hr NOEC≥ 1,000 mg/L	Rat LD50 74,000 mg/kg (oleic acid); Rat LD50 3,200 mg/kg (linoleic acid); Rat LD50 7,600 mg/kg (rosin);		Rabbit, slight irritant	Slight irritant to rabbit eye
1,3,5 trimethylbenzene (aka mesitylene)			IHL-RAT: 24 mg/m^3 (4h)		Typical STEL: 35 ppm.
Sec-butylbenzene			IHL-RAT: >1,900 mg/kg	RBT: >13,000 mg/kg	Eye irritation reactivity [EIR] in MAN @ 1.8
n-butylbenzene	Unknown	Unknown	Unknown	Unknown	EIR in MAN: 6.4
p-isopropyltoluene (*aka* p-cymene)		RAT: 3,669-4,750 mg/kg	IHL-MUS: 19,500 mg/m^3		RBT (Moderate skin irritation): 500 mg (24h).
Methyl napthalene (*aka* 1-Methylnapthalene)	24, 48, 72, 96h LC50: 39, 9, 9, 9 mg/L in FHM (static). 48h LC50: 8.4 mg/L in B. trout yearlings (static). BCF: 20-130 in Coho salmon muscle, depending on exposure time.	RAT: 1,840 mg/kg			RBT-SKIN-LDLO (lowest recorded lethal dose): 7,500 mg/kg.

Table C-14 Aquatic and Terrestrial Toxicity Data for Inert Ingredients Present in Proposed Rotenone Formulations

Ingredient	Toxicity to Aquatic Receptors	Toxicity to Terrestrial Receptors			
		Acute ORAL LD50	IHL LC50 /IPR/IVN LD50	Acute Dermal LD50	Other
Napthalene	96h LC50: 305.2 ppm (Trout)	MUS: 533 mg/kg RBT: 3,000 mg/kg	IVN-MUS: 100 mg/kg		Rat LOAEL 10 mg/kg bw/day LDLO (lowest published lethal dose) for Child: 100 mg/kg (ORAL) LDLO for human: 29 mg/kg (unknown entry). Threshold Limit Value (TLV): 10 ppm. RBT (Mild skin irritation): 100 mg. RBT (Mild eye irritation): 495 mg.
n-methyl-2-pyrrolidone	See Toxicity data for Methyl Pyrrolidone				
Di ethyl ether		RAT: 1,215 mg/kg MAN-LDLO: 260 mg/kg	IHL-MUS: 31,000 ppm (0.5h)		Human eye irritation: 100 ppm. RBT (Mild Skin irritation): 360 mg GPIG (Severe skin irritation): 30 mg/24h.
Ethylene glycol		RAT: 4,700 mg/kg HUMAN-LDLO: 786 mg/kg	IPR-MUS: 5,614 mg/kg		
Trichloroethylene		RAT: 7,193 mg/kg HUMAN-LDLO: 7,000 mg/kg	IPR-DOG: 1,900 mg/kg IVN-MUS: 34 mg/kg IHL-HUMAN-TCLO: 6,900 mg/m^3 (10 mins) (Lowest Published Toxic Concentration). IHL-MAN-LCLO: 2,900 ppm		Typical STEL: 150 ppm Typical LTEL: 100 ppm
Toluene		RAT: 636 mg/kg RAT: 2,600-7500 mg/kg HUMAN-LDLO: 50 mg/kg	IPR-RAT: 1,332 mg/kg IPR-MUS: 59 mg/kg IHL-RAT: 8,000 ppm (4h) IHL-Unspecified Mammal species: 30 g/m^3		RBT (Mild Skin irritation): 435 mg. Human eye irritation: 300 ppm.
Ethylbenzene	LC50 (96h): Trout: 4.2 mg/L FHM: 12.1mg/L Guppy: 9.9 mg/L Bay Shrimp: 0.490 mg/L Crab: 13 mg/L	RAT: 3,500 mg/kg	IHL-GPIG-LCLO: 10,000 ppm.	RBT: 17800 mg/kg	RBT (Mild Skin irritation): 15 mg (24h).
M xylene		RAT: 5,000 mg/kg			Typical PEL (prolonged exposure limit): 100 ppm.

Table C-14 Aquatic and Terrestrial Toxicity Data for Inert Ingredients Present in Proposed Rotenone Formulations

Ingredient	Toxicity to Aquatic Receptors	Toxicity to Terrestrial Receptors			
		Acute ORAL LD50	IHL LC50 /IPR/IVN LD50	Acute Dermal LD50	Other
P xylene		RAT: 5,000 mg/kg	IPR-RAT-LDLO: 2,000 mg/kg		Typical PEL (prolonged exposure limit): 100 ppm
O xylene		RAT: 4,000 mg/kg	IPR-MUS: 1.5 ml/kg		Typical STEL: 150 ppm
Isopropyl benzene (*aka* cumene/cumol)		RAT: 1,400 mg/kg	IHL-RAT: 8,000 ppm (4h)	RBT: 12300 mg/kg	Typical TLV/TWA: 50 ppm
n-propylbenzene (*aka* propylbenzene)		RAT: 6,040 mg/kg			
1,2,4-trimethylbenzene		RAT: 5,000 mg/kg	IHL-MUS: 8,147 ppm IPN-RAT-LDLO: 2,000 mg/kg IPN-GPIG-LDLO: 1,566 mg/kg		

After treating rotenone, permanganate is reduced and does not persist in the environment. According to a recent American Water Works Association survey, permanganate is commonly used (second only to chlorine) as a pre-treatment method for removing organic contaminants such as naphthalene and tetrachloroethene (TCE) in potable groundwater wells (as cited in Vella 2006). In groundwater, it controls iron, manganese, sulfides and color, and it can also be used to reduce high concentrations of radionuclides and arsenic by forming insoluble colloids. Potassium permanganate is also used in drinking water treatment plants to control taste and odor problems.

Potassium permanganate is considered moderately to highly toxic to aquatic organisms. Like rotenone, its toxicity differs among species. It may present a hazard to aquatic vertebrates during application. USEPA (2006) reported toxicity at concentrations of 1 to 2 ppm. However, this range of concentrations is also within the therapeutic range for treating fish diseases. Indeed therapeutic doses range from 2 to 25 ppm depending on the time prescribed for treatment (i.e., prolonged bath versus dip treatments). A 4 ppm concentration is generally recommended for "permanent bath" treatments of external parasites (Cross and Needham 1988). In a permanent bath, concentrations would not be reduced by flushing and degradation would occur through natural oxidative processes—generally within 1 to 4 days. Marking and Bills (1976) demonstrated that its toxicity was inversely proportional to water temperature for both rainbow trout and channel catfish. It is more toxic in hard water, potentially due to precipitation of manganese dioxide on fish gills. Although not as well studied, potassium permanganate is also considered toxic to aquatic invertebrates and zooplankton although, as with vertebrates, there is likely to be a wide tolerance range between various freshwater invertebrates.

C.4 EXPOSURE ASSESSMENT

C.4.1 Estimated Exposure Point Concentrations (EPC)

The exposure point concentration (EPC) represents the concentration in the exposure media to which ecological receptors would be exposed in the treatment area. The EPC experienced by a receptor would differ depending on the exposure media (i.e., air, water, food, and sediment), habitat use, the amount of time spent in the available habitat, and by application rate. For the proposed Action and alternatives (excluding the No Action alternative), the Agencies propose to use a rotenone application of CFT Legumine™~~,~~ or Noxfish®~~, or Nusyn-Noxfish®~~ at a concentration from 0.5 mg/L up to 1 mg/L. The concentration of potassium permanganate (the oxidizing agent) shall be applied to Silver King Creek downstream of the study area at a concentration up to 2 to 4 mg/L in the receiving waters.

C.4.1.1 Surface Water

To estimate EPCs in surface water, this assessment assumed that Noxfish®, CFT Legumine™ and Nusyn-Noxfish® would be applied by 5 gallon drip cans with a certain amount of diluted product that would be fully mixed in the streamflow. Estimated water concentrations of each constituent are presented in Table C-13.

C.4.1.2 Air

Ambient air samples were collected before and during the application of rotenone to Lake Davis in 2007 for pike elimination. The sampling methods were constructed to monitor for rotenone (the active ingredient), MP (water soluble solvent for rotenone), and naphthalene (odiferous, but minor constituent of applied technical material). Background samples were collected prior to application of the rotenone to the lake. Results of the sampling indicated that no rotenone above the detection limit (3 ng/m^3) was found at any of the sample sites. No MP above the detection limit (150 ng/m^3) was found at any of the sites. Low levels of naphthalene were detected at the sample sites. Since naphthalene is a known byproduct of combustion, particularly diesel oil combustion and other petroleum based activities and is known to already exist in ambient air, measureable amounts would be expected. Although some of the naphthalene levels increased after rotenone application activities began, these slightly elevated levels could be attributed to the increase of motor vehicle and boat traffic in the area. Urban levels of naphthalene, as measured by USEPA, can range between 300 ng/m^3 and 700 ng/m^3. All naphthalene levels detected in the samples were below the 300 ng/m^3 level. The VOC results from the sample collected at the fire station site indicate a higher level of combustion products as compared to the other samples. The 1,2-dichloroethane and dichloromethane concentrations were also elevated at this site in comparison with the other sample sites (Cal/EPA, Air Resources Board 2007). Overall, the monitoring data collected indicate that no appreciable increase in rotenone, MP, naphthalene, and VOC levels were attributable to activities associated with the Lake Davie rotenone project.

C.4.1.3 Groundwater

Terrestrial ecological receptors are not exposed to groundwater, thus direct exposure to groundwater or ingestion were considered incomplete pathways. Benthic macroinvertebrates may be exposed to very shallow groundwater at the sediment-water interface. However, this assessment assumed that because rotenone would be applied to the overlying surface water, any exposure from groundwater would be insignificant.

C.4.2 Ecological Exposure Estimates

This section presents the ecological exposure parameters used to estimate doses of rotenone and formulation constituents. Exposures are based on the complete and potentially significant exposure pathways identified in the conceptual model (see Figure C-1). The species selected for the exposure assessment use the treatment area for all or a portion of their life history. For the initial screening of exposure and risks, average weights, surface areas, and daily consumption rates were used to represent exposure. If the calculated HQ equaled or exceeded a level of concern (LOC) (as outlined in Table C-8) then the initial screening would be considered positive (potentially significant exposure) and a more detailed risk characterization step completed.

C.4.2.1 Ecological Receptor Exposure Factors

Table C-15 summarizes the exposure factors used to calculate estimated doses for ecological receptors, such as body weight and food ingestion rate, for the selected surrogate species. These exposure factors were obtained from the Wildlife Exposure Handbook (USEPA 1993) or from Sample et al. (1996). When species-specific data for food and water intake were not found in

these compendia, allometric equations were utilized to estimate the rates of food and/or water ingestion for the receptor species in the same guild.

Allometric equations, used extensively in biological sciences, correlate food and water intake to body weight and are documented in Sample et al. (1996) and the USEPA Wildlife Exposure Factors Handbook (1993). Separate equations were used for mammals and birds, as documented below.

Food ingestion rate (mammals):

$$Y = 0.235(Wt)^{0.822}$$

Food ingestion rate (birds):

$$Y = 0.648(Wt)^{0.651}$$

Where:

Y = food ingestion rate (g/day)

Wt = representative body weight (g) of a mammalian/avian receptor.

Water ingestion rate (mammals):

$$WI = 0.099(Wt)^{0.90}$$

Water ingestion rate (birds):

$$WI = 0.059(Wt)^{0.67}$$

Where:

WI = water ingestion rate (L/d)

Wt = representative body weight (kg) of a mammalian/avian receptor.

Dosage estimates were developed in more detail by providing additional input parameters - see Equation [3].

EQUATION 3

$$Dose = (SUF(IR[food]*C[food]) + (IR[water]*C[water]) + (IR[sed]*C[sed]*AE))/BW \qquad \text{Equation [3]}$$

Where:

SUF = Site Use Factor of Habitat Area (percent); SUF = 1 for this assessment

IR = consumption (i.e., intake) rate of [media: food, water, or sediment]

C = concentration of contaminant in [media: food, water, sediment]

AE = assimilation efficiency of contaminants in consumed soil or sediment

BW = Body Weight

Table C-15 Exposure Factors for Wildlife Used to Assess Risks from Rotenone Use in Silver King Creek Project Area

Species	Adult Body Weight (g)	Daily Food Intake (g)	Daily Water Intake (ml)	Inhalation Rate (m^3/day)	Surface Area (cm^2)	Soil and Sediment intake (% of diet)	Relevant Life History Characteristics and Dietary Preference Relevant to Exposure	Conceptual Exposure Pathways (confirmed by uptake model results)
Northern bobwhite quail	190	19.5	19	F: 0.10 M: 0.11	F: 298 M: 320	9.3	Breeding in April-July; hatching May to August; Non-migratory; annual mortality rate of approx. 80% Diet: Plants and insects. Max insects 20% in summer	Unlikely for Rotenone application, but considered surrogate for non-water dependent bird species Primary: Food & Water Secondary: Incidental soil ingestion Tertiary: Inhalation of drift
Marsh wren	11.25	8	3	-	F: 45 M: 48	0	Breed in April; hatch in May; Migration in fall and spring. Diet: Insects, spiders, mollusks, and crustaceans.	Unlikely for Rotenone application, but considered surrogate for passerine bird species. Primary: Food – aquatic insects assumed Secondary: Water Tertiary: Inhalation of drift
Hairy woodpecker	60	9.2	9	-	-	0	Diet: Insects, fruits, berries and nuts.	Has the potential to occur within project area along with Williamson's sapsucker. Primary: Food & Water Secondary: Inhalation of drift
Bald eagle	3,750	450	139	F: 1.43 M: 1.19*	F: 2,970 M: 2,530*	5.9	Diet: Fishes, waterfowl, small mammals and carrion.	Has the potential to occur within project area. Primary: Food – fish assumed Secondary: Incidental soil ingestion Tertiary: Water
Mouse	21	2.8	7	F: .025 M: 023	F: 86 M: 91	2	Breed several times during the year. Diet: Mixture of nuts, seeds, and insects	Unlikely for Rotenone application, but considered surrogate for small mammal species. Primary: Water Secondary: Food & incidental soil ingestion Tertiary: Inhalation
Pygmy rabbit	450	49	48	-	-	6.3	Breed several times during the year Diet: Herbivorous: Grasses, shrubs, woody plants	Has the potential to occur within project area. Primary: Food & Water Secondary: Incidental soil ingestion Tertiary: Inhalation
California Wolverine	18,000	725	1,350	-	-	3.0	Large home range. Sighted in the project area. Breeding occurs during June-August. Diet: Carrion and intermediate sized vertebrates	Has the potential to occur within project area. Also used as surrogate for fisher. Primary: Food – fish assumed Secondary: Water & incidental soil ingestion Tertiary: Skin contact

Table C-15 Exposure Factors for Wildlife Used to Assess Risks from Rotenone Use in Silver King Creek Project Area

Species	Adult Body Weight (g)	Daily Food Intake (g)	Daily Water Intake (ml)	Inhalation Rate (m^3/day)	Surface Area (cm^2)	Soil and Sediment intake (% of diet)	Relevant Life History Characteristics and Dietary Preference Relevant to Exposure	Conceptual Exposure Pathways (confirmed by uptake model results)
Sierra Nevada Red Fox	4,530	237	428	F: 1.7 M: 2.0	F: 2760 M: 3220	2.8	Breeding in December – February Diet: Omnivorous: mostly small mammals, birds, insects, and fruit. Plant material is common in summer and fall diet.	Has the potential to occur within project area. Primary: Water Secondary: Food Tertiary: Incidental soil ingestion
Mule deer	75,470	2400	4,800	M: 30.05* F: 17.26	M: 28,468.5* F: 18,142.4	6.8	Breeding in June. Diet: Herbivorous: leaves and twigs of trees and shrubs. Acorns, legumes and fleshy fruits	Primary: Water Secondary: Food Tertiary: Incidental soil ingestion
Black bear	128,870	3900	7,800	M: 67.05* F: 43.19	M: 54,641.8* F:38,220.6	2.8	Hibernation period: 3-4 months during winter (January-April) Diet: Omnivorous: Grasses and forbes in spring, fruits in summer, nuts and acorns in fall, insects and beetles. Carrion.	Primary: Food — fish consumed Secondary: Water & incidental soil ingestion Tertiary: Inhalation
Yosemite Toad**	20	0.2	20	-	-	25	Aquatic habitat. Diet: Plankton and plant material as juveniles; insects as adults.	Primary: Water ingestion & dermal contact Secondary: Food & incidental soil ingestion Tertiary: Inhalation with drift
Mountain yellow-legged frog**	25	0.25	25	-	-	25	Aquatic habitat. Diet: Plankton and plant material as juveniles; insects as adults – adults may predate on Yosemite toad and its own young.	Primary: Water ingestion & dermal contact Secondary: Food & incidental soil ingestion Tertiary: Inhalation with drift

* Estimated

** Tadpole stages were not considered because they are unlikely to be present during late summer, the planned time period for the treatment

For Equation [4], the food concentration of contaminant was calculated using Equation [4]:

EQUATION 4

Concentration of Contaminant in Food:

For carnivores: $C[food] = C[water] * BAF_f) + C[sed] *$ (percent of food contaminated)

For herbivores and hairy woodpecker: $C[food] = C([water] * BAF_s$ of 1) + C[soil] * (percent of food contaminated)

Where:

Percent of food contaminated = 100%

C[water] = concentration calculated in Table C-13

$C[soil] = C[water] * BAF_s$ of 1 (no loss to atmosphere)

$BAF_f = K_{oc} * 0.05$ (general bioaccumulation model for nonpolar organic compounds into aquatic animals containing 5% lipid; Mackay 1982); For Rotenone a BAF_f of 20 was used (Rach and Gingerlich 1986)

$C[sed] = C[water] * K_{oc}*0.01$ (general equilibrium partitioning model for sediments containing 1% organic carbon Van Leeuwen et al 1992)

$K_{oc} = K_{ow} * 10^{-0.21}$ (Karikoff et al. 1979); K_{ow} values are listed in Table C-13

For this screening-level assessment, the following conservative exposure assumptions were used:

- The BAF (20 L/kg) published by Rach and Gingerlich (1986) was used for rotenone. For the inactive ingredients, BAFs for animal dietary matter were estimated based on a general equilibrium partitioning model for nonpolar inorganic compounds, assuming the aquatic animals contained 5% lipid (Mackay 1982). For vegetable dietary matter and soils that could inadvertently receive overspray during application, BAFs = 1 were used to estimate the concentrations of all ingredients.
- The site use factor (SUF) was assumed to be 100% (i.e., the receptor's home range was assumed to be the same as the treatment area). A very conservative assumption for animals with broad home ranges, such as birds and mammals).
- The assimilation efficiency of ingredients contained in food, water or adsorbed to sediment was assumed to be 100%.
- Assumed 100% of the food was contaminated for all wildlife receptors. This assumption places a high (conservative) bias into the assessment because wildlife would almost certainly eat a variety of food items, including uncontaminated food items.
- Assumed that 100% of the water consumed by all receptors was contaminated at the maximum estimated concentration in Table C-13. This assumption places a high (conservative) bias into the assessment because it ignores losses from volatilization, photodegradation and other pathways that would decrease the concentrations of ingredients in Silver King Creek.
- No additive dose from inhalation.

C.4.2.2 Mammalian Wildlife Exposures

Mammalian wildlife can be exposed to rotenone and other formulation constituents through dermal, oral (ingestion of food and/or water) or inhalation routes. For this assessment, only ingestion routes (diet, water, and soils/sediment) were considered complete and potentially significant. Dermal exposure was determined either incomplete or insignificant. Exposures were

modeled for 6 mammalian species: the Sierra Nevada red fox, California wolverine, pygmy rabbit, mouse, black bear, and mule deer. These wildlife species have been documented in the treatment area or have been the foundation for much of the toxicological effects literature (e.g. mouse).

C.4.2.3 Avian Exposure

Exposure for birds may occur via the same pathways as mammals: ingestion, dermal contact, and inhalation. To represent the range of dietary habits and life histories of birds occurring in the treatment area, ingestion exposure calculations were completed for Northern bobwhite quail, marsh wren, bald eagle, and hairy woodpecker. Direct contact was considered a potentially complete pathway, but an insignificant one because of protection from feathers. Of these species, the bald eagle and hairy woodpecker have the potential to occur in the treatment area. The Northern bobwhite quail was included based on availability of toxicity values while the marsh wren was included because it was considered representative of the life history of many passerines, has the potential to occur in the treatment area, and many toxicity data are available for the species.

C.4.2.4 Aquatic Animal Exposure

Exposure of fish and aquatic invertebrates to rotenone and formulation constituents in water would be a complete pathway. This exposure assessment assumed a maximum EPC to correspond to the concentration at full mixing in the stream (see Table C-13). Rotenone was assumed to be fully diluted to 25 ppb or 0.025 ppm (with a maximum concentration of 50 ppb or 0.05 ppm for the higher potential application rate). These concentrations were then compared to aquatic exposure TRVs. Given the sensitivity to rotenone of aquatic receptors, this exposure was considered "worst case" and exposure to other formulation constituents was not evaluated. In addition, because of the degree of direct exposure to water-borne rotenone, exposure to rotenone adsorbed to sediment was considered an insignificant exposure pathway.

C.4.2.5 Amphibian Exposure

Dermal contact is the most direct exposure pathway for amphibians and/or across the gills (i.e., for juvenile amphibians). Dietary uptake was also considered a complete pathway. Amphibians in the riparian and littoral zones could be sprayed directly if chemical is administered via backpack. However, because workers would not apply chemical to riparian and littoral vegetation and would avoid spraying amphibians, this exposure pathway was considered possible but insignificant. Because rotenone can elicit toxicity through dermal exposure and gill absorption, and because juveniles with gills are the most sensitive life stage of amphibians, exposure risks to amphibians were evaluated by comparing surface water EPCs to aquatic TRVs.

C.5 RISK CHARACTERIZATION

C.5.1 Wildlife Risks from Ingestion

Table C-16 presents estimated rotenone doses based on modeled food web exposure pathways and the most concentrated constituents in the rotenone formulations. HQ values below the LOC were considered to pose little or no risk, while values equal to or exceeding the LOC were considered to indicate a potential risk (refer to Table C-17 for the HQ values).

Table C-16 Estimated Ingestion Doses of Most Concentrated Rotenone Formulation Constituents from Combined Food, Water and Sediment Intake

		Rotenone	Diethylene Glycol Monoethyl Ether	1-Methyl-2-Pyrrolidinone	Fennodefo 99™	Rotenone	Naphthalene	Toluene	1,2,4 -Trimethylbenzene
Class	**Species**	*CFT-Legumine™ at 0.5 mg/L*				*Noxfish® at 0.5 mg/L*			
Avian	Bald Eagle	0.058	0.012	0.019	19.3	0.057	0.23	0.0015	0.087
	Bobwhite Quail	0.0026	0.031	0.0050	0.0087	0.0025	0.0035	0.000090	0.00050
	Marsh Wren	0.37	0.087	0.014	147	0.036	1.77	0.011	0.66
	Hairy Woodpecker	0.0038	0.046	0.0074	0.013	0.0038	0.0053	0.00014	0.00075
Mammalian	Red Fox	0.032	0.029	0.0047	10.9	0.031	0.13	0.00091	0.049
	California Wolverine	0.025	0.023	0.0037	8.4	0.024	0.10	0.00070	0.038
	Mule Deer	0.0016	0.019	0.032	0.0055	0.0016	0.0022	0.000057	0.00032
	Black Bear	0.019	0.019	0.0030	6.3	0.018	0.077	0.00053	0.029
	Mouse	0.0085	0.10	0.017	0.029	0.0083	0.012	0.00030	0.0017
	Pygmy Rabbit	0.0027	0.033	0.0053	0.0092	0.0027	0.0037	0.000096	0.00053
Amphibian	Yosemite Toad	0.035	0.31	0.050	2.3	0.035	0.061	0.0011	0.015
	Mountain Yellow-Legged Frog	0.035	0.31	0.050	2.3	0.035	0.061	0.0011	0.015

Class	Species	*CFT-Legumine™ at 1.0 mg/L (ppm)*				*Nusyn-Noxfish® at 1. 0 mg/L (ppm)*			
Avian	Bald Eagle	0.12	0.024	0.038	38.5	0.057	0.46	0.0030	0.17
	Bobwhite Quail	0.0051	0.061	0.0099	0.017	0.0025	0.0070	0.00018	0.0010
	Marsh Wren	0.74	0.17	0.027	294	0.36	3.5	0.023	1.3
	Hairy Woodpecker	0.0076	0.092	0.015	0.026	0.0038	0.011	0.00027	0.0015
Mammalian	Red Fox	0.064	0.059	0.0094	21.8	0.031	0.27	0.0018	0.099
	California Wolverine	0.050	0.046	0.0075	16.8	0.024	0.21	0.0014	0.076
	Mule Deer	0.0032	0.039	0.0063	0.011	0.0016	0.0045	0.00011	0.00064
	Black Bear	0.037	0.037	0.0060	12.6	0.018	0.155	0.0011	0.057
	Mouse	0.017	0.20	0.033	0.058	0.0083	0.023	0.00060	0.0033
	Pygmy Rabbit	0.0054	0.065	0.011	0.019	0.0027	0.0075	0.00019	0.0011
Amphibian	Yosemite Toad	0.071	0.61	0.099	4.5	0.035	0.12	0.0021	0.030
	Mountain Yellow-Legged Frog	0.071	0.61	0.099	4.5	0.035	0.12	0.0021	0.030

All doses as mg ingredient/kg body weight/day

Table C-17 Wildlife Hazard Quotients from Combined Food Water and Sediment Ingestion Exposure Pathways

Class	Species	Toxicity Text	Rotenone[a]	Diethylene Glycol Monoethyl Ether[b]	1-Methyl-2-Pyrrolidinone[c]	Fennodefo 99™ [g]	Rotenone [a]	Naphthalene [d]	Toluene[e]	1,2,4 Trimethylbenzene[f]	Level of Concern
			CFT-Legumine™ at 0.5 mg/L				*Noxfish® at 0.5 mg/L*				
Avian	Bald eagle	NOAEL	0.15	2.5×10^{-5}	1.9×10^{-6}		0.14	-	4.8×10^{-6}	-	1
		LOAEL	0.029	-	-		0.029	0.023	-	-	1
		LD50	0.00045	-	-	0.0060	0.00044	-	-	1.7×10^{-5}	0.1
	Bobwhite quail	NOAEL	0.013	1.3×10^{-4}	1.0×10^{-5}		0.0066	-	6.1×10^{-7}	-	1
		LOAEL	0.0027	-	-		0.0013	0.00074	-	-	1
		LD50	4.2×10^{-5}	-	-	1.0×10^{-5}	2.0×10^{-5}	-	-	2.1×10^{-7}	0.1
	Marsh wren	NOAEL	0.92	0.00018	1.3×10^{-5}		0.91	-	3.6×10^{-5}	-	1
		LOAEL	0.18	-	-		0.18	0.18	-	-	1
		LD50	0.0028	-	-	0.046	0.0028	-	-	0.00013	0.1
	Hairy Woodpecker	NOAEL	0.019	1.9×10^{-4}	1.5×10^{-5}		0.0094	-	8.8×10^{-7}	-	1
		LOAEL	0.0039	-	-		0.0019	0.0011	-	-	1
		LD50	5.9×10^{-5}	-	-	5.3×10^{-5}	2.9×10^{-5}	-	-	3.0×10^{-7}	0.1
Mammalian	Red Fox	NOAEL	0.080	6.0×10^{-5}	4.7×10^{-6}		0.078	-	5.8×10^{-6}	-	1
		LOAEL	0.016	-	-		0.016	0.027	-	-	1
		LD50	0.00081	7.9×10^{-6}	1.2×10^{-6}	0.0034	0.00079	0.00050	2.8×10^{-6}	2.0×10^{-5}	0.1
	Wolverine	NOAEL	0.062	4.7×10^{-5}	3.7×10^{-6}		0.061	-	4.5×10^{-6}	-	1
		LOAEL	0.012	-	-		0.012	0.21	-	-	1
		LD50	0.00063	6.3×10^{-6}	9.5×10^{-7}	0.0026	0.00062	0.00039	2.2×10^{-6}	1.5×10^{-5}	0.1
	Mule Deer	NOAEL	0.0062	6.1×10^{-5}	4.8×10^{-6}		0.0030	-	2.8×10^{-7}	-	1
		LOAEL	0.0012	-	-		0.00061	0.0034	-	-	1
		LD50	6.3×10^{-5}	8.1×10^{-6}	1.2×10^{-6}	1.7×10^{-6}	3.1×10^{-5}	6.4×10^{-6}	1.4×10^{-7}	9.7×10^{-8}	0.1
	Black Bear	NOAEL	0.047	3.8×10^{-5}	3.0×10^{-6}		0.046	-	1.7×10^{-6}	-	1
		LOAEL	0.0093	-	-		0.0091	0.0077	-	-	1
		LD50	0.00047	5.1×10^{-6}	5.7×10^{-7}	0.0020	0.00046	0.00015	8.3×10^{-7}	5.7×10^{-6}	0.1
	Mouse	NOAEL	0.030	0.00029	2.3×10^{-5}		0.015	-	1.4×10^{-6}	-	1
		LOAEL	0.0060	-	-		0.0029	0.0016	-	-	1
		LD50	3.4×10^{-5}	3.9×10^{-5}	5.9×10^{-6}	4.7×10^{-4}	1.7×10^{-5}	3.1×10^{-5}	6.6×10^{-7}	4.7×10^{-7}	0.1
	Pygmy Rabbit	NOAEL	0.014	1.4×10^{-4}	1.1×10^{-5}		0.070	-	6.4×10^{-7}	-	1
		LOAEL	0.0028	-	-		0.0014	0.00078	-	-	1
		LD50	3.8×10^{-5}	1.8×10^{-6}	2.8×10^{-6}	4.1×10^{-6}	1.9×10^{-6}	1.5×10^{-5}	3.1×10^{-7}	2.2×10^{-7}	0.1

Table C-17 Wildlife Hazard Quotients from Combined Food Water and Sediment Ingestion Exposure Pathways

Class	Species	Toxicity Text	Rotenone[a]	Diethylene Glycol Monoethyl Ether[b]	1-Methyl-2-Pyrrolidinone[c]	Fennodefo 99™ [g]	Rotenone [a]	Naphthalene [d]	Toluene[e]	1,2,4 Trimethylbenzene[f]	Level of Concern
Amphibian	Yosemite Toad	NOAEL	-	0.00062	4.9×10^{-5}		-	-	3.4×10^{-6}	-	1
		LOAEL	-	-	-		-	0.0061	-	-	1
		LD50	0.061	-	-	0.00071	0.60	-	2.8×10^{-6}	3.0×10^{-6}	0.1
	Mountain Yellow-Legged Frog	NOAEL	-	0.00062	4.9×10^{-5}		-	-	3.4×10^{-6}	-	1
		LOAEL	-	-	-		-	0.0061	-	-	1
		LD50	0.061	-	-	0.00071	0.60	-	2.8×10^{-6}	3.0×10^{-6}	0.1

			CFT-Legumine™ at 1.0 mg/L				*Nusyn-Noxfish® at 1.0 mg/L*				
Avian	Bald eagle	NOAEL	0.29	4.9×10^{-5}	3.7×10^{-6}		0.14	-	9.5×10^{-6}	-	1
		LOAEL	0.058	-	-		0.029	0.046	-	-	1
		LD50	0.00090	-	-	0.012	0.00044	-	-	3.5×10^{-5}	0.1
	Bobwhite quail	NOAEL	0.027	2.6×10^{-4}	2.1×10^{-5}		0.0013	-	1.2×10^{-6}	-	1
		LOAEL	0.0054	-	-		0.0027	0.0015	-	-	1
		LD50	8.3×10^{-5}	-	-	2.1×10^{-5}	4.1×10^{-5}	-		4.2×10^{-7}	0.1
	Marsh wren	NOAEL	1.8	0.0012	0.0001		0.92	-	7.3×10^{-5}	-	1
		LOAEL	0.37	0.00035	2.7×10^{-5}-		0.18	0.35	-	-	1
		LD50	0.0057	-	-	0.092	0.0028	-	-	0.00026	0.1
	Hairy Woodpecker	NOAEL	0.039	0.00038	3.0×10^{-5}		0.0019	-	1.8×10^{-6}	-	1
		LOAEL	0.0077	-	-		0.0038	0.0021	-	-	1
		LD50	1.2×10^{-4}	-	-	1.1×10^{-4}	5.8×10^{-5}	-	-	6.1×10^{-7}	0.1
Mammalian	Red Fox	NOAEL	0.16	0.00012	9.4×10^{-6}		0.078	-	5.8×10^{-6}	-	1
		LOAEL	0.032	-	-		0.016	0.027	-	-	1
		LD50	0.0016	1.6×10^{-5}-	2.4×10^{-6}	0.0068	0.00079	0.00050	2.8×10^{-6}	2.0×10^{-5}	0.1
	Wolverine	NOAEL	0.12	9.5×10^{-5}	7.5×10^{-6}		0.061	-	4.5×10^{-6}	-	1
		LOAEL	0.025	-	-		0.012	0.21	-	-	1
		LD50	0.0013	1.3×10^{-5}	1.9×10^{-6}-	0.0052	0.00062	0.00039	2.2×10^{-6}	1.5×10^{-5}	0.1
	Mule Deer	NOAEL	0.012	1.2×10^{-4}	9.6×10^{-6}		0.0061	-	5.6×10^{-7}	-	1
		LOAEL	0.0025	-	-		0.0012	0.00068	-	-	1
		LD50	1.3×10^{-4}	1.6×10^{-5}	2.5×10^{-6}	3.4×10^{-6}	6.2×10^{-5}	1.3×10^{-5}	2.8×10^{-7}	1.9×10^{-7}	0.1
	Black Bear	NOAEL	0.093	7.6×10^{-5}	6.0×10^{-6}		0.046	-	3.4×10^{-6}	-	1
		LOAEL	0.019	-	-		0.0091	0.015	-	-	1

Table C-17 Wildlife Hazard Quotients from Combined Food Water and Sediment Ingestion Exposure Pathways

Class	Species	Toxicity Text	Rotenone[a]	Diethylene Glycol Monoethyl Ether[b]	1-Methyl-2-Pyrrolidinone[c]	Fennodefo 99™ [g]	Rotenone [a]	Naphthalene [d]	Toluene[e]	1,2,4 Trimethylbenzene[f]	Level of Concern
		LD50	0.00094	1.0×10^{-5}	1.5×10^{-6}	0.0039	0.00046	0.00029	1.7×10^{-6}	1.1×10^{-5}	0.1
	Mouse	NOAEL	0.060	0.00058	4.6×10^{-5}		0.029	-	2.7×10^{-6}	-	1
		LOAEL	0.012	-	-		0.00059	0.0033	-	-	1
		LD50	6.8×10^{-5}	7.8×10^{-5}	1.2×10^{-5}	9.4×10^{-4}	3.4×10^{-5}	6.2×10^{-5}	1.3×10^{-6}	9.4×10^{-7}	0.1
	Pygmy Rabbit	NOAEL	0.028	2.8×10^{-4}	2.2×10^{-5}		0.0014	-	1.3×10^{-6}	-	1
		LOAEL	0.0057	-	-		0.0028	0.0016	-	-	1
		LD50	7.5×10^{-6}	3.7×10^{-5}	5.6×10^{-6}	8.2×10^{-6}	3.7×10^{-6}	2.9×10^{-5}	6.3×10^{-7}	4.4×10^{-7}	0.1
Amphibian	Yosemite Toad	NOAEL	-	0.0013	9.9×10^{-5}		-	-	6.8×10^{-6}	-	1
		LOAEL	-	-	-		-	0.012	-	-	1
		LD50	0.12	-	-	0.0014	0.060	-	-	5.9×10^{-6}	0.1
	Mountain Yellow-Legged Frog	NOAEL	-	0.0013	9.9×10^{-5}		-	-	6.8×10^{-6}	-	1
		LOAEL	-	-	-		-	0.012	-	-	1
		LD50	0.12	-	-	0.0014	0.060	-	-	5.9×10^{-6}	0.1

NOAEL: No observable adverse effect level.

LOAEL: Lowest observable adverse effect level.

LD50: The concentration of chemical leading to a 50% mortality of the test animals within a given time period.

- No data available.

Footnotes on Toxicity Reference Values (TRVs):

[a]The rotenone NOAEL value for all mammal and bird species was 0.4 mg/kg-bw/day. This value represents the lowest NOAEL value available for separate lab-based studies on rats and dogs. The rotenone LOAEL of 2/0 mg/kg bw/day is also based on a laboratory study for rats (USEPA 1988, USFWS 1980). The rotenone LD50 of 130 mf/kg bw/day for birds is based on nestling English sparrows (Cutcomp 1943). The LD50 for mammals of 39.5 mg/kg bw/day is based on a rat study. The LD50 for mice of 350 mg/kg bw/day is based on a mouse study (Kenaga et al. 1985 ~~and Allison 1971~~). The LD50 for rabbits of 1500 mg/kg bw/day is based on a rabbit study. The rotenone LD50 value for all amphibian species was 0.58 mg/kg. This value represents the lowest LD50 value available for lab-based studies on adult and larval amphibians.

[b]The Diethylene Glycol Monoethyl Ether NOAEL value for all species was 490 mg/kg-bw/day. This value represents the lowest NOAEL value available for lab-based studies on rats (see Table C-15). No reports on studies using different animal classes were available.

[c]The 1-Methyl-2-Pyrrolidinone NOAEL value for the Norway rat was 3000 mg/kg-bw/day based on lab rats. The 1-Methyl-2-Pyrrolidinone NOAEL value for all other species was 1000 mg/kg-bw/day. This value represents the lowest available NOAEL obtained from lab-based studies on mice (MSDS Number: B&J 0304, 2001). No data was available for amphibians.

[d]The Naphthalene LOAEL value for all mammal and bird species was 10 mg/kg bw/day (NTP 1992). This value represents the lowest TRV available for lab-based studies on rats. An LD50 value of 533 mg/kg bw/day from a mouse study was used for mammalian receptors.

[e]The Toluene NOAEL value for all mammal and bird species was 312 mg/kg-bw/day (NTP 1990). This value represents the lowest TRV value available and refers to a lab-based rat study. The lowest available LD50 of 636 mg/kg bw/day from a rat study was used for mammalian receptors. No data was available for amphibians.

[f]The 1, 2, 4–Trimethylbenzene LD50 value for all mammal and bird species was 5000 mg/kg-bw. This represents the acute 24 hr LD50 value for lab-based studies on rats. No data was available for amphibians.

[g]The Fennfodefo LD50 value of 3,200 mg/kg bw/day is based on the toxicity of linoleic acid on rats. This is the lowest LD50 for a "tall oil" component.

C.5.1.1.1 CFT LEGUMINE™

ROTENONE

Risks for rotenone were based on NOAELs and LOAELs for rats and dogs, the only sublethal literature values available for terrestrial species. These values were based on chronic (6-month) studies, which are very conservative for the brief exposures proposed. The NOAEL and LOAELs from mammals were applied across all species and were more conservative than species-specific LD50 values because they represented a more protective endpoint and a lower exposure concentration.

MAMMAL RISK

As demonstrated by the Hazard Quotient (HQ) summary (refer to Table C-17), none of the doses calculated for mammals exceeded LOCs. Most of the HQs, which are based on the NOAEL, were far less than 1. This indicates that risks to mammalian receptors from rotenone or insignificant.

AVIAN RISK

Rotenone is considered slightly to non-toxic to adult birds, based on the USEPA criteria outlined above. However, some studies indicate rotenone may be moderately toxic to nestlings (Cutcomp 1943). Most HQs for birds were all below LOCs (refer to Table C-17). The NOAEL-based HQ for wrens was exceeded for CFT Legumine™ applied at the 1.0 mg/L application rate; however, the LOAEL-based HQs were all far less than 1 for the avian species. All LD50-based HQs were far less than one. These results indicate that adverse affects to birds from the proposed Action are very unlikely.

AMPHIBIAN RISK

Rotenone is considered highly toxic to amphibians, based on USEPA criteria, particularly for juveniles. If present at the time of application, juveniles could be killed by the rotenone application through exposure across the skin and gills. Adult amphibians are more mobile and would be more capable of avoiding the treatment area. Modeled doses relative to the larval LD50 value resulted in HQs just above 0.1 for both amphibian species if CFT Legumine™ were applied at the 1.0 mg/L application rate, indicating risks are bordering on significant. Because amphibians are particularly sensitive to rotenone and the uncertainty inherent in the screening-level risk assessment approach (e.g. using LD50 values), and the potential for presence of sensitive life stages, risks to amphibians could be significant.

DIETHYLENE GLYCOL MONOETHYL ETHER (DEGEE)

As with rotenone, toxicology data for DEGEE were available for only a few mammalian species. Therefore, the lowest NOAEL (490 mg/kg-bw/day for mice) was applied to all receptors.

MAMMAL RISK

DEGEE is nearly non-toxic to mammals based on the USEPA criteria. All studies show mammals with LD50s >5,000 mg/kg (IUCLID 2000). None of the HQs for DEGEE exceeded LOCs (refer to Table C-17), indicating that risks from DEGEE are insignificant.

AVIAN RISK

No data were available to demonstrate the toxicity of DEGEE to birds. Using the mammalian NOAEL value, none of the calculated exposure doses exceeded an LOC of 1 (refer to Table C-17) relative to the NOAEL, indicating risks to birds from DEGEE would be insignificant.

AMPHIBIAN RISK

No amphibian toxicity data were available for amphibians. Using the mammalian NOAEL, none of the HQs exceeded an LOC of 0.1, indicating risks to amphibians from DEGEE are insignificant.

1-METHYL-2-PYRROLIDINONE (MP)

Toxicology data for MP were limited to a few mammalian species. Therefore, the lowest NOAEL value (1,000 mg/kg-bw/day for mice) for mammals was applied to all receptors.

MAMMAL RISK

MP is considered as being slightly toxic to mammals, based on the USEPA criteria, with studies showing LD50s <2,000 mg/kg (B&J 0304, 2001). None of the calculated HQs exceeded LOCs relative to the NOAEL (refer to Table C-17), indicating risks to mammals would be insignificant.

AVIAN RISK

No data were available to demonstrate the toxicity of MP to birds. Using the NOAEL value for mice, none of the HQs exceeded LOCs for any of the avian species modeled (refer to Table C-17). Therefore, risks to avian species would be insignificant risk from the proposed Action.

AMPHIBIAN RISK

No amphibian toxicity data were available for MP. Using the NOAEL value for mice, none of the HQs exceeded an LOC of 0.1 (refer to Table C-17), indicating risks to amphibians would not be significant.

FENNODEFO 99™ (FENNODEFO)

Each of the chemicals of potential concern that make up Fennodefo constituent in the CFT Legumine™ were evaluated by Jeff Fisher (ENVIRON 2007) to determine to what extent these chemicals are recognized in state and ~~f~~Federal statutes as hazardous materials, and, if so, their regulatory criteria. In summary, no California-specific or ~~f~~Federal regulatory screening values were identified for the protection of human or ecological health for these new chemical constituents.

Acute LD50 values from laboratory studies using rats were available for 3 constituents of Fennodefo (Table C-14). The most conservative LD50 of 3,200 mg/kg for linoleic acid was applied to all receptors.

MAMMAL RISK

As demonstrated by the HQ summary (Table C-17), none of the doses calculated for mammals exceeded LOCs. The HQs, which are based on the LD50, were far less than 0.1. This indicates that risks to mammalian receptors from Fennodefo are insignificant.

AVIAN RISK

None of the doses calculated for birds exceeded LOCs. The HQs, which are based on the LD50, were far less than 0.1. This indicates that risks to avian receptors from Fennodefo are insignificant.

AMPHIBIAN RISK

None of the doses calculated for amphibians exceeded LOCs. The HQs, which are based on the LD50, were far less than 0.1. This indicates that risks to amphibian receptors from Fennodefo are insignificant.

C.5.1.1.2 NOXFISH® AND NUSYN-NOXFISH®

ROTENONE

The concentration of rotenone in Noxfish® is the same as that of CFT Legumine™. Therefore, the HQ results are the same as those presented above for CFT Legumine™ at the 0.5 mg/L application rate, which is used for Noxfish®. Nusyn- Noxfish® is applied at 1.0 mg/L; however it contains half the rotenone of Noxfish® (or CFT Legumine™), so the exposure to rotenone is the same for both formulations. At the application rates used for Noxfish® and Nusyn- Noxfish®, rotenone risks to receptors are less-than-significant.

NAPHTHALENE

Toxicology data for naphthalene were limited to a few mammalian species. Although a NOAEL for mice was available (100 mg/kg-bw/day), a lower LOAEL for rats (10 mg/kg-bw/day) was used as the TRV for all receptors. The concentration of naphthalene is significantly higher in the Noxfish® formulations than in CFT Legumine™; however, all HQs calculated for all species were well below the LOC of 1.

MAMMAL RISK

Naphthalene is considered moderately toxic to mammals based on the USEPA criteria with studies showing LD50s < 501 mg/kg. None of the HQs exceeded LOCs, indicating risks to mammals from naphthalene exposure would be insignificant.

AVIAN RISK

Because no data were available for naphthalene toxicity in birds, the LOAEL value for rats was used to assess risks to birds. None of the HQs exceeded LOCs, indicating naphthalene exposure risks for birds would be less-than-significant.

AMPHIBIAN RISK

Because no data were available for naphthalene toxicity in amphibians, the LOAEL value for rats was used to assess amphibian risk. None of the HQs exceeded an LOC of 0.1, indicating naphthalene exposure risks for amphibians would be less-than-significant.

TOLUENE

Toxicity data for toluene were only available for rats (NOAEL of 312 mg/kg-bw/day). Therefore, the HQs for assessing toluene were based on this value.

MAMMAL RISK

Toluene is considered moderately toxic to mammals based on the USEPA criteria listed in Table C-11, with studies showing LD50s < 501 mg/kg (~~Neurotoxicology. Vol. 2, Pg. 567,~~ Benignus1981). However, none of the calculated HQs for selected mammal species exceeded the LOC of 1 relative to the NOAEL (refer to Table C-17). This result indicates risks to mammals from toluene would be insignificant.

BIRD RISK

Adopting the NOAEL value for rats as the TRV, none of the HQs exceeded LOCs, indicating risks to birds from toluene exposures are insignificant.

AMPHIBIAN RISK

Adopting the NOAEL value for rats as the TRV for amphibians, none of the HQs exceeded an LOC of 0.1, indicating risks to amphibians from toluene exposures would be insignificant for the proposed Action.

1, 2, 4-TRIMETHYLBENZENE

Toxicity data for 1, 2, 4-Trimethylbenzene were limited to an acute value for rats (LD50 of 5,000 mg/kg). Therefore, this value was used as the ingestion TRV for all species modeled.

MAMMAL RISK

1, 2, 4-trimethylbenzene is considered practically non-toxic to mammals based on USEPA criteria. Laboratory studies have derived LD50 values > 2,000 mg/kg. None of the HQs exceeded LOCs for any of the mammal species modeled relative to the LD50 (refer to Table C-17), indicating risks to mammals from 1, 2, 4-trimethyltoluene would be insignificant.

BIRD RISK

No toxicity data were available for 1, 2, 4-trimethylbenzene exposure for birds. Using the LD50 value for rats, none of the HQs exceeded LOCs for any of the avian species modeled (refer to Table C-17). This indicates risks from the proposed Action to birds would be insignificant.

AMPHIBIAN RISK

No toxicity data were available for 1, 2, 4-trimethylbenzene exposure for amphibians. Using the LD50 value for rats, none of the HQs exceeded LOCs for any of the amphibian species modeled (refer to Table C-17). This indicates risks from the proposed Action to amphibians would be insignificant.

C.5.1.2 Wildlife Ecological Receptor Risks

Because of the low volume of chemical required for the proposed Action to treat Silver King Creek during the fall with low-flow conditions and the water's limited surface area, risks to wildlife (mainly via inhalation) were considered negligible as summarized in Table C-17.

Table C-18 Terrestrial Toxicity Hazard Quotients to Rotenone

Class	Species	Surrogate Species	Rotenone TRV		HQ CFT Legumine™ at 0.5 mg/L	HQ CFT Legumine™ at 1.0 mg/L	HQ Noxfish® at 0.5 mg/L	HQ Nusyn-Noxfish® at 1.0 mg/L	Reference
			Test End Point	TRV Value (mg/kg bw/day)					
Avian	Bald Eagle	Bald Eagle	NOAELChronic 6 Month	0.4	0.15	0.29	0.14	0.14	1
			LOAELChronic 6 Month	2	0.029	0.058	0.029	0.029	1
	Great Grey Owl	Bald Eagle	NOAELChronic 6 Month	0.4	0.15	0.29	0.14	0.14	1
			LOAELChronic 6 Month	2	0.029	0.058	0.029	0.029	1
	Mountain Quail	N. Bobwhite Quail	NOAELChronic 6 Month	0.4	0.013	0.027	0.013	0.013	1
			LOAELChronic 6 Month	2	0.0027	0.0054	0.0027	0.0027	1
		Japanese Quail	LD50 5 Day	1882	2.9×10^{-6}	5.8×10^{-6}	2.9×10^{-6}	2.9×10^{-6}	2
	Willow Flycatcher	Marsh Wren	NOAELChronic 6 Month	0.4	0.92	0.013	0.91	0.91	1
			LOAELChronic 6 Month	2	0.18	0.0025	0.18	0.18	1
		English Song Sparrow (nestlings)	LD50 24h	130	0.0028	3.9×10^{-5}	0.0028	0.0028	3
	Yellow Warbler	Marsh Wren	NOAELChronic 6 Month	0.4	0.92	0.013	0.91	0.91	1
			LOAELChronic 6 Month	2	0.18	0.0025	0.18	0.18	1
		English Song Sparrow (nestlings)	LD50 24h	130	0.0028	3.9×10^{-5}	0.0028	0.0028	3
	Hairy Woodpecker	Hairy Woodpecker	NOAELChronic 6 Month	0.4	0.019	0.039	0.019	0.019	1
			LOAELChronic 6 Month	2	0.0039	0.0077	0.0038	0.0038	1
		English Song Sparrow (nestlings)	LD50 24h	130	5.9×10^{-5}	1.2×10^{-4}	5.8×10^{-5}	5.8×10^{-5}	3
	Williamson's Sapsucker	Hairy Woodpecker	NOAELChronic 6 Month	0.4	0.019	0.039	0.019	0.019	1
			LOAELChronic 6 Month	2	0.0039	0.0077	0.0038	0.0038	1
		English Song Sparrow (nestlings)	LD50 24h	130	5.9×10^{-5}	1.2×10^{-4}	5.8×10^{-5}	5.8×10^{-5}	3

Table C-18 Terrestrial Toxicity Hazard Quotients to Rotenone

Class	Species	Surrogate Species	Rotenone TRV		HQ CFT Legumine™ at 0.5 mg/L	HQ CFT Legumine™ at 1.0 mg/L	HQ Noxfish® at 0.5 mg/L	HQ Nusyn-Noxfish® at 1.0 mg/L	Reference
			Test End Point	TRV Value (mg/kg bw/day)					
Mammalian	Sierra Nevada Red Fox	Red Fox	NOAELChronic 6 Month	0.4	0.080	0.16	0.078	0.078	1
			LOAELChronic 6 Month	2	0.016	0.032	0.016	0.016	1
		Rat	LD50 24h	39.5	0.00081	0.0016	0.00079	0.00079	4
	Californian Wolverine	Californian Wolverine	NOAELChronic 6 Month	0.4	0.062	0.12	0.0061	0.0061	1
			LOAELChronic 6 Month	2	0.012	0.025	0.012	0.012	1
		Rat	LD50 24h	39.5	0.00063	0.0013	0.00062	0.00062	4
	American Marten	Californian Wolverine	NOAELChronic 6 Month	0.4	0.062	0.12	0.0061	0.0061	1
			LOAELChronic 6 Month	2	0.012	0.025	0.012	0.012	1
		Rat	LD50 24h	39.5	0.00063	0.0013	0.00062	0.00062	4
	Small Mammal	Mouse	NOAELChronic 6 Month	0.4	0.030	0.060	0.029	0.029	1
			LOAELChronic 6 Month	2	0.0060	0.012	0.0059	0.0059	1
			LD50 24h	350	3.4×10^{-5}	6.8×10^{-5}	3.4×10^{-5}	3.4×10^{-5}	5
	Small Herbivorous Mammal	Pygmy Rabbit	NOAELChronic 6 Month	0.4	0.014	0.028	0.014	0.014	1
			LOAELChronic 6 Month	2	0.0028	0.0057	0.0028	0.0028	1
			LD50 24h	1500	3.8×10^{-6}	7.5×10^{-6}	3.7×10^{-6}	3.7×10^{-6}	Unknown
	Ungulate	Mule Deer	NOAELChronic 6 Month	0.4	0.0062	0.012	0.0061	0.0061	1
			LOAELChronic 6 Month	2	0.00012	0.025	0.0012	0.0012	1
	Black Bear	Black Bear	NOAELChronic 6 Month	0.4	0.047	0.093	0.046	0.046	1
			LOAELChronic 6 Month	2	0.0093	0.0186	0.0091	0.0091	1
			LD50 24h	39.5	0.00047	0.00094	0.00046	0.00046	4

C.5.1.3 Aquatic Ecological Receptor Risks

Table C-19presents the calculated HQs based on surface water EPCs identified in Table C-13 and the aquatic TRVs identified in Table C-5 and Table C-9. As anticipated, based on their direct exposure to the treated water and/or potential presence of sensitive life stages, HQ values for larval frogs and toads and rainbow trout exceeded LOCs. However, at the proposed treatment concentrations, the proposed Action would not expose most aquatic invertebrate taxa to lethal concentrations of rotenone. Cladocerans and several other invertebrate species could be affected by the treatment.

Table C-19 Aquatic Toxicity Hazard Quotients to Rotenone

Class	Species	Surrogate Species	Rotenone TRV		HQ CFT Legumine™ at 0.5 mg/L	HQ CFT Legumine™ at 1.0 mg/L	HQ Noxfish® at 0.5 mg/L	HQ Nusyn-Noxfish® at 1.0 mg/L	Reference
			Test End Point	TRV Value (mg/kg bw/day)					
Amphibian	Mountain yellow-legged frog (adult)	Northern leopard frog (adult)	LC50 24h	240	0.11	0.21	0.10	0.10	1
	Mountain yellow-legged frog (larvae)	Northern leopard frog (tadpole)	LC50 24h	5	501	10	5.0	5.0	2
	Yosemite toad (adult)	Northern leopard frog (adult)	LC50 24h	240	0.11	0.21	0.10	0.10	1
	Yosemite toad (larvae)	Northern leopard frog (tadpole)	LC50 24h	5	5.1	10	5.0	5.0	2
Fish	Rainbow trout		LC50 24h	3.5	7.3	15	7.1	7.1	3
Macroinvertebrate	Flatworm	*Catenula* sp.	LC50 24h	5100	0.0050	0.010	0.0049	0.0049	4
		Planaria sp.	LC50 24h	<500	~0.051	~0.10	~0.050	~0.050	4
	Annelid worms	Leech	LC50 48h	<100	~0.26	~0.51	~0.25	~0.25	4
	Copepod	*Cyclops* sp.	LC100 72h	<100	~0.26	~0.51	~0.25	~0.25	4
	Branchiura	*Argulus* sp.	LC50 24h	~25	~1	~2	~1	~2	4
	Cladoceran	*Daphnia pulex*	LC50 24h	27	0.94	1.9	0.93	0.93	4
		D. pulex	LC50 24h	<25	~1	~2	~1	~2	4
		Diaptomus siciloides	LC50 24h	<25	~1	~2	~1	~2	4
	Conchostracan	*Estheria* sp.	LC50 24h	~50	~0.5	~1	~0.5	~0.5	4
	Freshwater prawn	*Palaemonetes kadiakensis*	LC50 24h	5150	0.0050	0.0099	0.0049	0.0049	4
	Crayfish	*Cambarus immunis*	LC50 72h	>500	<0.051	<0.10	<0.50	<0.50	4
	Dragonfly naiad	*Macromia* sp.	LC50 24h	4700	0.0054	0.011	0.0053	0.0053	4
	Stonefly naiad	*Pteronarcys californica*	LC50 24h	2900	0.0088	0.0176	0.0086	0.0086	4
	Backswimmer	*Notoncta* sp.	LC50 24h	3420	0.0075	0.0149	0.0073	0.0073	4
		Notoncta sp.	LC50 24h	~100	~0.26	~0.51	~0.25	~0.25	4
	Caddis fly larvae	*Hydropsychye* sp.	LC50 96h	605	0.042	0.084	0.041	0.041	4
	Whirligig	*Gyrinus* sp.	LC50 24h	3550	0.0072	0.0143	0.0070	0.0070	4
	Water mite	*Hydrachnidae*	LC50 96h	~50	~0.5	~1	~0.5	~0.5	4
	Snail	*Physa pomilia*	LC50 24h	6350	0.0040	0.0080	0.0039	0.0039	4
		Oxytrema catenaria	LC50 96h	1750	0.015	0.029	0.014	0.014	4
		Lymnaea stagnalis	LC50 96h	>1000	<0.026	<0.051	<0.025	<0.025	4
	Bivalave mollusk	*Dreissena polymorpha*	LC50 48h	2190	0.012	0.023	0.011	0.011	4
		Obliquaria reflexa	LC50 48h	>1000	<0.026	<0.051	<0.025	<0.025	4
		Elliptio buckleyi	LC50 96h	2950	0.0086	0.017	0.0085	0.0085	4
		Elliptio complanata	LC50 96h	2000	0.013	0.025	0.013	0.013	4
		Corbicula manilensis	LC50 96h	7500	0.0034	0.0068	0.0033	0.0033	4
	Ostracod	*Cypridopsis*	LC50 24h	490	0.052	0.10	0.051	0.051	4

C.5.2 Risk Assessment Uncertainties, Assumptions, and Data Gaps

C.5.2.1 Environmental Fate and Toxicity Assessment

Toxicity data are frequently unavailable for chemicals that are the subject of ecological risk assessments. This is the case for many of the selected receptors and several of the chemicals present in the rotenone formulations proposed for use under the proposed Action. In some cases, toxicity data were available for certain exposure routes (e.g. intravenous) but not for more significant exposure routes such as ingestion and dermal contact, or inhalation. When toxicity information was available for relevant exposure routes, they were not available for the receptors found near Silver King Creek. Therefore, TRVs from typical laboratory species were extrapolated to the ecological receptors selected for this assessment.

The following bullets highlight the specific data gaps identified in literature review for this assessment, and qualitatively characterize the significance of the uncertainties created by these data gaps:

1. Essentially no information was found on the toxicity of rotenone to aquatic or terrestrial plants. Given rotenone is used as an organic pesticide approved for use on over 90 organic food crops (USEPA 2006) at application rates far greater than what would be applied under the proposed Action, plant toxicity is considered extremely unlikely.

2. Chronic rotenone toxicity data for birds was lacking in the literature. Because the proposed Action includes a single, short-term treatment, and because rotenone breaks down quickly in the environment, chronic exposures were considered insignificant.

3. Essentially no information was found on the photo-degradation rate of rotenone in soil. These data could be useful in predicting wildlife exposure through incidental consumption of soils at the water's edge. The uncertainty created by this data gap in estimating dose, however, is considered minor given the chemical would be applied directly to the stream and any application to soil would be inadvertent.

4. Toxicity data for reptiles and amphibians are few for rotenone and its formulation constituents. Standard practice is to use avian toxicity data as a surrogate for these species. However, given rotenone's respiratory toxicity mechanism, such data were not considered useful.

5. Toxicity and empirical fate data for several formulant dispersants were incomplete in the literature. For example, no inhalation toxicity values for DEGEE, degradation rates for permanganate (as a covariate of organic matter), or dermal toxicity values were found for most formulation constituents. Although such data would be useful in the exposure assessment, formulation constituents and degradation products are less toxic than rotenone by at least a factor of 2 (USEPA 2006). Such results indicate that the dispersants in the end-product formulations do not contribute to rotenone's toxicity (and may actually reduce it).

C.5.2.2 Ecological Exposure Assessment

Exposure point concentrations were estimated by assuming full mixing of all chemicals in the creek. Exposure doses were calculated using the following assumptions that tend to overestimate ingestion risk, which is appropriate for this screening-level risk assessment.

1. The ~~Site Use Factor (~~SUF~~)~~ was assumed to be 100% for all receptors. While this assumption may be accurate for species with small home ranges, it is a very conservative assumption for larger mammals and birds.

2. Bioavailability of contaminants was assumed to be 100%. Unless a chemical is delivered intravenously, bioavailability is likely less than 100% because contaminants may adhere to food items or not be completely absorbed. Because rotenone tends to adhere to sediments and water-borne particles, this assumption is conservative. In addition, bioavailability may be affected by environmental parameters such as oxygen levels, pH, and temperature.

3. The bioaccumulation factor (BAF_f) for rotenone in fish was 20, which reflects the maximum bioaccumulation factor determined by Rach and Gingerlich (1986). The BAFs for the inert ingredients in fish were estimated based on the organic carbon partition coefficient (Koc) and an assumed lipid (organic carbon) content of 5% (Mackay 1982). The BAFs for all ingredients in sediment were estimated based on Koc and an assumed sediment organic carbon content of 1% (Van Leeuwen et al. 1992). Because of the volatility and degradability of the ingredients comprising rotenone formulations, these chemicals were considered highly unlikely to bioaccumulate in upland areas incidentally exposed to overspray and were assumed to have a BAF of 1 for vegetative matter and soil.

4. The percent of contaminated food was always assumed to be 100%, which assumed all food sources were contaminated. This is a conservative assumption as most organisms would have diverse diets.

5. Where species-specific data relating to food and water intake were not available, intake rates of food, water and air as well as surface area were estimated for each receptor using allometric equations from the Wildlife Exposure Handbook (USEPA 1993) and Sample et al. (1996). These equations us the species' average weight to determine intake rates. These values can vary by population; however, data specific to the Silver King Creek area were not available.

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