

Risk Assessment for Piscicidal Formulations of Rotenone

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TABLE OF CONTENTS

1.	Introduction.....	8
1.1	Background.....	8
1.2	Objectives.....	9
1.3	Methods.....	9
2.	Problem Formulation.....	9
2.1	Objectives of use of rotenone by WDFW.....	10
2.2	Types of sites where rotenone may be used.....	11
2.3	Nature of rotenone as a stressor.....	11
2.4	Ecological receptors that may be exposed to rotenone use.....	12
2.5	Considerations of human exposure.....	13
2.6	Special considerations.....	13
2.6.1	Potential of rotenone to cause Parkinson's disease.....	13
2.6.2	Mobility and Potential Ground Water Contamination by Rotenone in Areas of Fractured Basalt.....	14
3.	Label Description and History.....	15
3.1	Rotenone registered products.....	15
3.1.1	For use as piscicides (labels and MSDS sheets to be attached).....	15
3.1.2	Other uses.....	15
3.2	Application methods and rates.....	15
3.3	Efficacy and selectivity of rotenone products.....	17
3.4	Expected changes in rotenone labeling.....	18
3.5	Additional requirements of the RED other than labeling.....	19
4.	Chemical Characteristics.....	19
4.1	Composition of the various rotenone end-use products.....	19
4.1.1	Active ingredients.....	19
4.1.2	Impurities.....	20
4.1.3	Added inert ingredients.....	21
4.1.4	Added synergists.....	23
4.1.5	Nature of formulation (e.g., powder, emulsifiable concentrate).....	23
4.2	Color.....	23
4.3	Physical State.....	23
4.4	Odor.....	24
4.5	Melting Point.....	24
4.6	Boiling Point.....	24
4.7	Density, Bulk Density or Specific Gravity.....	24
4.8	Solubility.....	25
4.9	Vapor Pressure.....	25
4.10	Disassociation Constant.....	25
4.11	Octanol/Water Partition Coefficient.....	26
4.12	pH.....	26
4.13	Stability.....	26
4.14	Oxidizing or Reducing Action.....	26
4.15	Flammability.....	26
4.16	Explosibility.....	27
4.17	Storage Stability.....	27

4.18	Viscosity	27
4.19	Miscibility	27
4.20	Corrosion Characteristics.....	27
4.21	Dielectric Breakdown Voltage.....	28
5.	Environmental Fate.....	28
5.1	Volatilization.....	28
5.2	Hydrolysis.....	28
	5.2.1 Half-life.....	28
	5.2.2 Degradation products	28
5.3	Aqueous photolysis.....	29
	5.3.1 Half-life.....	29
	5.3.2 Degradation products	29
5.4	Soil photolysis.....	29
	5.4.1 Half-life.....	30
	5.4.2 Degradation products	30
5.5	Summary of photolysis	30
5.6	Degradation and Persistence - soil.....	31
	5.6.1 Half-life.....	31
	5.6.2 Degradation Products.....	31
5.7	Degradation and persistence - aquatic systems.....	31
	5.7.1 Half-life and Disappearance Time	31
	5.7.2 Degradation Products.....	32
	5.7.3 Physical and Chemical Factors affecting aquatic persistence.....	32
5.8	Microbial Degradation	34
5.9	Mobility.....	34
	5.9.1 Soil 34	
	5.9.2 Sediment	35
	5.9.3 Groundwater	35
6.	Environmental Effects Assessment.....	35
6.1	Objectives	35
6.2	Sources of Information	36
6.3	Aquatic Toxicity information	36
	6.3.1 Microbes	36
	6.3.2 Algae 36	
	6.3.3 Aquatic macrophytes	36
	6.3.4 Aquatic invertebrates	36
	6.3.4.1 Acute.....	37
	6.3.4.2 Chronic.....	37
	6.3.5 Amphibians (aquatic stages).....	37
	6.3.6 Fish 37	
	6.3.6.1 Acute.....	38
	6.3.6.2 Chronic.....	45
	6.3.7 Sediment organisms.....	45
6.4	Terrestrial Toxicity Data.....	45
	6.4.1 Mammals.....	46
	6.4.1.1 Acute.....	46

	6.4.1.2 Chronic.....	46
6.4.2	Birds	46
	6.4.2.1 Acute.....	46
	6.4.2.2 Chronic.....	46
6.4.3	Reptiles	47
6.4.4	Amphibians (adult/terrestrial stages)	47
6.4.5	Insects	47
6.4.6	Plants	48
7.	Ecological Exposure Assessment	48
7.1	Routes of exposure.....	48
	7.1.1 Aquatic plants and algae	49
	7.1.2 Fish and other aquatic vertebrates; aquatic invertebrates	49
	7.1.3 Terrestrial organisms	49
7.2	Estimated concentrations of rotenone.....	49
	7.2.1 Water column – lentic.....	50
	7.2.2 Water column – lotic.....	50
	7.2.3 Sediments.....	50
	7.2.4 Adjacent terrestrial areas.....	50
7.3	Persistence and duration of residues	51
	7.3.1 Water	51
	7.3.2 Sediment	52
	7.3.3 Soil and vegetation.....	53
	7.3.4 Detoxification of rotenone	53
7.4	Bioconcentration and Bioaccumulation.....	53
	7.4.1 Within organisms.....	54
	7.4.2 Accumulation and other food chain transfer.....	54
7.5	Ground and well water considerations.....	54
	7.5.1 General aspects of groundwater and wells.....	54
	7.5.2 Mobility of rotenone and considerations for use in fractured basaltic areas.	55
8.	Risk Assessment and Characterization for Ecological Effects	57
8.1	Direct Effects	59
	8.1.1 Fish	59
	8.1.2 8.1.2 Other aquatic biota.....	60
	8.1.3 Terrestrial biota.....	62
	8.1.4 Endangered and threatened species	62
8.2	Effects on water quality	66
8.3	Effects from interactions with other pesticides.....	66
8.4	Effects on pristine and contaminated sites.....	66
8.5	Indirect effects	66
	8.5.1 From removal of fish and other aquatic biota.....	66
	8.5.2 Potential for increased erosion and resuspension of soils and sediments resulting from effects on plants.....	67
	8.5.3 Effects on aquatic habitats	67
	8.5.4 Potential effects upon agriculture	67
	8.5.5 Indirect effects on endangered and threatened species.....	68

8.6	Impacts of multiple applications.....	68
8.7	Impacts on terrestrial organisms and environments.....	68
8.8	Impacts on wetlands other than target application sites.....	68
8.9	Uncertainty analysis.....	68
8.10	Additional needs for information.....	69
	8.10.1 Soil and sediment.....	69
	8.10.2 Water	69
	8.10.3 Plants	70
	8.10.4 Acute toxicity studies.....	70
	8.10.5 Chronic toxicity studies	70
8.11	Mitigation measures.....	70
8.12	Conclusions and recommendations.....	70
9.	Human Health Effects.....	70
9.1	Toxicity information and sources	71
	9.1.1 Acute toxicity.....	71
	9.1.1.1 Oral	71
	9.1.1.2 Dermal.....	71
	9.1.1.3 Inhalation	71
	9.1.1.4 Irritation and sensitization.....	71
	9.1.1.5 FIFRA toxicity categories for various exposure routes	72
	9.1.2 Pharmacokinetics	72
	9.1.3 Subchronic toxicity	73
	9.1.4 Chronic and reproductive toxicity	73
	9.1.5 Developmental toxicity.....	74
	9.1.6 Mutagenicity and carcinogenicity.....	74
	9.1.7 Neurotoxicity	75
	9.1.7.1 Guideline considerations of neurotoxicity	75
	9.1.7.2 Potential of rotenone to cause Parkinson’s disease.....	76
	9.1.8 Epidemiology.....	77
	9.1.9 Incident reports	77
9.2	Exposure assessment.....	78
	9.2.1 Exposure routes.....	78
	9.2.1.1 Swimming.....	78
	9.2.1.2 Drinking water	78
	9.2.1.3 Occupational exposure.....	78
	9.2.1.4 Other	79
10.	Risk Assessment and Characterization for Health Effects	79
	10.1 Drinking water	79
	10.2 Fish consumption.....	80
	10.3 Rotenone exposure from swimming.....	80
	10.4 Exposure during applications.....	80
	10.5 Chronic exposure	81
	10.6 Uncertainties	81
	10.7 Conclusions.....	82
11.	References.....	83

Risk Assessment for Piscicidal Formulations of Rotenone

Executive Summary

Rotenone is a piscicide which has long been used by the Washington Department of Fish and Wildlife (WDFW) as a fisheries management tool to rehabilitate lakes, ponds, streams, and other waters to enhance recreational fishing and native fish populations. Rotenone is a natural plant alkaloid extracted from the roots of tropical plants in the pea family. Among the various products registered by the U. S. Environmental Protection Agency (USEPA) are four basic kinds of formulations: powdered extracts, emulsifiable liquids, emulsifiable liquids with the synergist piperonyl butoxide, and baits. WDFW uses rotenone powders, which also contain other alkaloids extracted with the rotenone.

Rotenone acts by blocking oxidative phosphorylation in the electron transport system at complex I within the mitochondrion. It affects cellular respiration in the cells of a wide variety of animals where it can reach the mitochondria. It is selective in its toxicity to whole organisms based upon its ability to reach the electron transport system in these organisms. It is highly toxic to fish, insects and aquatic organisms relying on gills though which it is readily absorbed, but much less toxic when ingested or through dermal contact. Among terrestrial vertebrates, the most significant route of exposure is through inhalation. It is not toxic to plants.

Rotenone may applied by a variety of aerial or ground application methods. Most use by WDFW is through direct application into water as a slurry, via backpack sprayer, or by drip systems directly into water although any labeled method might be used. Application rates are based upon achieving a specific concentration in the water; 250 parts per billion (ppb) or $\mu\text{g/L}$ is the maximum amount allowed on current rotenone labels. Lower application rates are frequent depending upon the type of use and the fish species to be controlled. Most use of rotenone by WDFW has been to treat lakes, ponds, and reservoirs, but it has been use to treat streams. Where transport of rotenone away from the intended treatment sites is a concern, such as streams, potassium permanganate may be used to deactivate the rotenone.

Rotenone has very recently been reviewed by USEPA in conjunction with its reregistration process. It has been used as an “organic” insecticide on crops, livestock, and pets, but these uses have been voluntarily withdrawn by rotenone registrants, leaving the piscicidal use as the only registered use. As a result of its analysis, USEPA is proposing some label changes, primarily to reduce the potential exposure to applicators and other persons, but also to ensure more consistent applications such as by requiring certified applicator training and following the use of a “Standard Operating Procedure” manual.

Rotenone is relatively insoluble in water and has low volatility. It degrades primarily through hydrolysis, with photolysis also being important in clear, shallow water. The primary degradation product is rotenolone. Rotenone degrades fairly rapidly in the environment, with half-lives ranging from a few hours to several weeks. Persistence is longer at lower temperatures and higher pHs. Bioavailability of rotenone is reduced as a result of strong adsorption to sediments, plants, and particulate matter in treated waters. As a result, rotenone should not leach

into groundwater; no rotenone has ever been detected in groundwater, even in test areas associated with rotenone treatments. Typically, fish toxicity is reduced in treated waters by either degradation or partitioning out of the water column to the extent that fish may be restocked within 2-4 weeks. The potential for bioconcentration is low in aquatic organisms and food chain transfer should be minimal, if any at all.

As would be expected from its use as a piscicide, rotenone is highly toxic to fish. There are extensive fish toxicity data which indicate median lethal (96-hour LC₅₀) values as low as 0.84 µg/L, or in studies used by USEPA in its risk assessment, as low as 1.94 µg/L, both for rainbow trout. Bullheads, catfish, and goldfish are considerably less sensitive than most other tested fish. Rotenone formulations containing the synergist, piperonyl butoxide, appear to be about six times as toxic as formulations without the synergist. Chronic toxicity data for fish are limited, but suggest that toxicity is not substantially greater than in acute tests; a 32-day test for rainbow trout had a no-adverse-effect-concentration of 1.01 µg/L.

Limited laboratory data indicate that toxicity to aquatic invertebrates is quite variable. Among aquatic arthropods, cladocerans appear to be the most sensitive with LC₅₀ values as low as 3.7 µg/L for *Daphnia magna*. Benthic invertebrates including amphipods, crayfish, and mollusks are much less sensitive. As with fish, chronic toxicity is not substantially lower than acute toxicity. Data from field observations following treatments indicate typically serious, but fairly short term, effects on zooplankton, with recovery occurring in several months to as long as several years. Even in the observations where zooplankton are considered to be eradicated, overall zooplankton recovery occurs, although not necessarily with the same species diversity. Some benthic organisms are affected.

Gilled stages of amphibians are moderately sensitive to rotenone. LC₅₀ values have been determined to be as low as µg/L for a 5% EC formulation for larval Southern leopard frogs (*Rana sphenoccephala*). Adult stages of frogs tested in water were much less sensitive, with LC₅₀ values as low as 3.2 mg/L. Observations following rotenone treatments have generally not noted effects on amphibians.

Among terrestrial organisms, for which exposure is expected to be minimal, rotenone has low toxicity. Mammals are more sensitive, with the rat acute oral LD₅₀ of 39.5 mg/Kg. Rotenone is considered practically non-toxic in contact studies with honeybees; however, rotenone has long been used as an insecticide, indicating that the honeybee or the contact route of exposure is comparatively insensitive.

Indirect effects may occur for organisms that rely on fish or zooplankton, resulting from loss of a food supply. Typically, terrestrial organisms can find other locations or types of food sources. For aquatic organisms, such effects will typically be of limited duration. It is important to the purposes of using piscicides that the food sources for restocked fish be available. Experience indicates that this will typically occur no later than the year following treatment.

Effects on threatened and endangered (T&E) species are not expected. While T&E fish are quite susceptible, rotenone would not be used where they occur except in conjunction with permits from the National Marine Fisheries Service and the Fish and Wildlife Service. Exposure to

terrestrial T&E species is highly unlikely. Aquatic T&E plants would not be sensitive to rotenone.

The toxicity data base with respect to humans is somewhat incomplete. On an acute basis, inhalation toxicity is of most concern, with oral toxicity also being classified as highly toxic. Females are more sensitive than males in all studies where effects were classified according to gender. Chronic toxicity data for rotenone are limited to oral exposure. In a two generation rat study, a no-observed-adverse-effect-level was 7.5 mg/Kg diet based upon weight gain in pups. Chronic inhalation data were waived by USEPA because no chronic inhalation exposure is expected after cancellation of non-piscicidal uses. There is no evidence of any carcinogenicity, mutagenicity, or teratogenicity. An older study that found tumors at low doses did not find them at higher doses, which contradicts dose-response theory; newer, more thorough studies found no evidence of carcinogenicity at all.

Rotenone has been found to mimic an effect associated with Parkinson's syndrome, and has been used to assess the effects of chemical agents that may inhibit the development of the syndrome. This is not considered a concern for any piscicidal uses of rotenone because it required chronic injection of rotenone into the jugular vein of rats to produce the effect. No such exposure would even remotely approximate exposure from fish control uses, but proposed new labeling precludes any exposure through the use of respirators, protective clothing and closed application systems.

Although there are some uncertainties, the available data and the extensive use experience with rotenone for fish control purposes indicate that it can be used safely. Beyond the intended target fish, some direct effects are expected on certain aquatic invertebrates, primarily zooplankton, and indirect effects can be expected on other organisms as a result of the direct effects on fish and invertebrates. Such effects should be of limited duration without serious consequences to the environment.

1. Introduction

The Washington State Department of Fish and Wildlife (WDFW) is responsible for management activities relating to recreational fishing within the State of Washington. Maintaining a high quality fishery sometimes requires intervention on the part of fisheries managers to enhance habitat for threatened, endangered, and other desirable species, to remove introduced fish that may compete with or prey upon native fish species, to control diseases, to sample fish populations, or for other reasons. Rotenone has been used as a piscicide (i.e., to kill fish) since 1934 (Bradbury, 1986) as a safe and effective fisheries management tool. Rotenone is generally used to eliminate or significantly reduce entire fish populations in lakes, ponds, reservoirs, and streams. Rotenone may also be used in streams and rivers flowing into lakes, ponds, and reservoirs as a barrier treatment to keep fish from escaping into those lotic waters. (J. Anderson, WDFW, personal communication, May 7, 2007).

1.1 Background

WDFW has been using rotenone as a fisheries management tool since 1940 (WDFW, 2002). In 1976, WDFW first developed an Environmental Impact Statement (EIS) for use of rotenone to

rehabilitate lakes and streams. Supplements to this EIS have been developed periodically; major supplements were issued in 1992 and 2002 (WDFW, 2002) and a literature review was completed in 1988 (WDFW, 1988).

1.2 Objectives

The objective of this analysis is to provide an up-to-date ecological and human health assessment of rotenone for use by WDFW. Much of the older information on rotenone was considered in earlier WDFW assessments. However, some new information is available and there are new tools now available to assess some of the older information previously considered. It is expected that this analysis will be used to support an application by WDFW for renewal of an existing NPDES permit for their piscicidal use of rotenone.

1.3 Methods

This assessment draws heavily upon documents developed by the U. S. Environmental Protection Agency (EPA) as part of EPA's reregistration process for pesticides. A "Reregistration Eligibility Document" (RED) for rotenone was issued in March, 2007, and there are numerous supporting documents on EPA's rotenone docket at <http://www.regulations.gov/fdmspublic/component/main>, (search for docket # EPA-HQ-OPP-2005-0494) visited March 20, 2007. Additional fish toxicity data, along with a history of the WDFW fish rehabilitation program, were obtained from previous assessments by WDFW, EPA's ECOTOX data base (<http://cfpub.epa.gov/ecotox/>, accessed April and May 2007), and other sources. Because rotenone is a piscicide, it was considered important to capture and present as much toxicity data as possible for fish native to or introduced into U. S. waters, and to present information on the effects upon aquatic ecosystems and human health. Specific searches for literature were made to address the two special topics indicated by WDFW to be of importance in Washington. The first relates to the ability of rotenone to induce Parkinson's disease-like symptoms in the laboratory setting. The second addresses the potential for groundwater contamination in areas underlain by fractured basaltic rocks. Other open literature sources were used, as needed and available. Much information on rotenone is from the older literature and could only be obtained through its inclusions in more recent summaries in the time frame available for this assessment.

2. Problem Formulation

An analysis of piscicidal uses of rotenone in Washington state first requires a problem formulation such as that described in EPA's Framework for Ecological Risk Assessment (USEPA, 1992), and updated in the Guidelines for Ecological Risk Assessment (USEPA 1998). A problem formulation describes the nature of the stressor agent, rotenone in this case, considerations of the intentional and unintentional receptors of that stressor, and the effects of the stressor on those receptors. This section defines the scope of the assessment in terms of the stressor, the receptors, and the methods and models used to quantify and characterize the effects of the stressor on the receptors.

The purpose of this assessment is to provide updated information on rotenone and to support decision-making for the Washington Department of Fisheries and Wildlife's application to the

Washington Department of Ecology for an NPDES permit that is required for the use of potential pollutants in navigable waters.

Rotenone is a pesticide currently registered for use as a piscicide. The reigning paradigm for pesticides is that, for each type of receptor organism, there will be doses or concentrations of that pesticide that will affect those organisms and lower doses or concentrations that will not affect those organisms. Theoretically, there is a continuum, or dose-response, where increasing doses will result in increasing effects ranging from “no effect” to 100% effect on various types of receptors. This dose-response concept is well accepted in toxicology for the greatest part. However, there are debates regarding the theory as one approaches either the no effect dose or concentration and the 100% effect dose or concentration. For example, Calabrese (e.g., Calabrese and Baldwin, 2003) has long maintained that some low doses of what are normally considered toxins in human toxicology studies may actually be beneficial, and Chapman (e.g., Chapman, 2001) has applied the same concepts to ecological toxicology and risk assessment.

A typical risk assessment includes the nature and quantity of exposure of receptors to the stressor, the toxicology of the stressor to the various receptors, or surrogate organisms for those receptors, and a characterization of the effects. In the case of rotenone used as a piscicide, there are two areas of special concern to the Washington Department of Fisheries and Wildlife. The first area relates to the potential that rotenone may cause Parkinson’s disease or related symptoms in mammals that may be relevant to human exposure, which could occur to persons handling or applying rotenone, or to those who might be exposed in their food or water. The second area involves the potential for rotenone used in lakes to move through fractured basaltic substrates into groundwater that may supply drinking water wells. This current assessment will give special emphasis on these two concerns, as well as the overall risks of rotenone used as a piscicide.

The U.S. EPA has recently issued a Reregistration Eligibility Decision Document (USEPA, 2007). Based upon the findings and current regulations, EPA is requiring changes in registered uses and labeling that will affect both the use and the risks of rotenone as a piscicide. The requirements are subject to a public comment period. These changes will be presented and assessed for their risk reduction potential.

2.1 Objectives of use of rotenone by WDFW

Among its responsibilities, the Washington Department of Fish and Wildlife is charged with maintaining a viable recreational fishery in waters under its jurisdiction. For many such waters, the introduction of non-native species has occurred widely, leading to impaired fisheries as a result of competition, predation, or alteration of key parts of ecosystems. There have also been significant perturbations that have resulted in changed environmental conditions that may affect native fish populations. To fulfill their legislative mandate regarding fisheries, WDFW may use piscicides to control introduced or native fish whose populations may have become unbalanced and may have impaired the fishery. WDFW may also use rotenone to aid in the recovery of threatened and endangered fish species in Washington (J. Anderson, WDFW, personal communication, May 7, 2007).

Rotenone is normally used when all fish in a body of water are to be eliminated, with subsequent stocking of desired fish to rehabilitate the fishery. Rotenone is typically used to help establish or re-establish a cold water fishery with trout. In some situations, rotenone may be used to enhance a fishery of warm water species, such as bass, bluegill, or crappie. WDFW may also use rotenone to control native or non-native fish, such as the snakehead or northern pike, that pose a significant threat of becoming invasive and severely damaging natural aquatic ecosystems. Finally, piscicides may be used to eliminate non-native fish that may compete with, prey upon, or hybridize with salmon and trout that are protected under the Endangered Species Act. (WDFW, 2002)

Rotenone has also played a role for fisheries researchers in determining the abundance, density, and nature of fish populations. In many situations, it is not feasible or possible to obtain such information without taking a sample of the fish that are present. Such sampling may be done with electroshocking or with a piscicide such as rotenone, when the survival of individual fish, such as representatives of threatened or endangered species, is not an issue. WDFW does not use rotenone for sampling or other fisheries research (J. Anderson, WDFW, personal communication, May 7, 2007).

2.2 Types of sites where rotenone may be used

According to the 2002 WDFW revised plan for using rotenone (WDFW, 2002), most rotenone is used to treat lakes, ponds, and reservoirs. Inlet tributaries and lake outlet streams may regularly be included as part of a lake treatment project. WDFW has also used rotenone in a few situations involving sloughs or streams; six streams in eastern Washington have been treated to enhance resident trout. The first rotenone use in Washington was in 1940, and from that time through 2006, 514 water bodies have been treated. Rotenone was the primary piscicide used in the first 30 years, and since the late 1960s has been the only piscicide used by WDFW. Of the 514 lakes treated through 2006, 56.8% have been treated more than once, with an average time between treatments of 7.74 years.

Detailed information is not clear on where treated lakes are located; it is clear that treated lakes are typically at low elevations. Many, if not most, of early treatments were in western Washington. Between 1992 and 2006, all treatments except one were in eastern Washington. Lack of public support for the use of chemicals and changes in label restrictions that EPA imposed to eliminate use near irrigation or potable water intakes were cited as the primary reasons for not treating western Washington lakes, along with the potential for anadromous threatened and endangered salmonid stocks to be present in western Washington waters (WDFW, 2002).

2.3 Nature of rotenone as a stressor

Rotenone is a naturally occurring plant alkaloid obtained from plants in the Malaysian genus *Derris*, the South American genus *Lonchocarpus*, and the East African genus *Tephrosia*, all members of the pea family, Leguminosae (EPA, 2006a). Bradbury (1986) provided a solid overview of the rotenone literature at that time. Rotenone was recently reviewed under EPA's Reregistration program. A "Reregistration Eligibility Decision" (RED) document has been completed (EPA, 2007); this document and the supporting reports that contributed to the RED provide a fairly recent and detailed analysis of rotenone from a regulatory perspective. These

documents primarily, along with others as appropriate, can be used to frame the stressor characteristics of rotenone and to identify the types of receptors likely to be susceptible to rotenone.

Rotenone acts by blocking oxidative phosphorylation in the electron transport system at complex I within the mitochondrion (EPA 2006a). Bradbury (1986) summarizes literature demonstrating that rotenone affects cellular respiration in the cells of mammals, fish, insects, amphibians, and plants. However, in whole organisms, rotenone is very highly toxic to fish and insects, relatively non-toxic in mammals and others, and essentially not toxic to plants, which may be expected from its widespread use on garden plants to control insects. The selective toxicity of rotenone has been attributed to the ability of rotenone to reach the cellular electron transport system and to do so without detoxification. Bradbury cites various literature indicating that mammals are relatively insensitive because they absorb rotenone inefficiently from the intestine. However, in a laboratory setting, solvents such as acetone or ethanol are used to enable rotenone to reach the electron transport system. Under those circumstances, rotenone has been shown to be as toxic to mammals as to fish. The lack of phytotoxicity appears to be a result of the multiplicity of NAD⁺ or NADP⁺ electron carriers in plants (MacKenzie and McIntosh, 1999) and the existence of an NADH-dehydrogenase in plant mitochondria that is insensitive to rotenone (Menz and Day, 1996).

2.4 Ecological receptors that may be exposed to rotenone use

A wide array of ecological receptors could have been exposed to rotenone use prior to the cancellation of garden and pet uses. The piscicide use, however, would occur in a manner that would expose primarily aquatic organisms of all taxa, along with human applicators. While potential exposure of terrestrial organisms as a result of spray drift cannot be completely ruled out, it is unlikely. Rotenone products are generally introduced directly into or immediately over the water and typically have limited amounts of drift. Aerial applications are conducted by licensed commercial pesticide applicators using best management practices to reduce the drift of the product away from the treatment area.

Based upon the lack of phytotoxicity, and the relative lack of exposure of terrestrial animals, the focus of the risk among ecological receptors is primarily oriented towards aquatic animals. However, terrestrial organisms that feed upon aquatic animals may be exposed to rotenone in their food or may be indirectly affected by a loss of their food base. Terrestrial organisms that feed upon plants would not have a reduction in their food base, but could be exposed to rotenone from feeding on plants that have rotenone residues.

Aquatic animals and ecosystems will be exposed to rotenone when it is used as a piscicide. Fish are the intended receptors, but exposure of all types of aquatic organisms is unavoidable from this use. Rotenone labels specify the amount of exposure in the water column that would occur in accordance with label directions for using rotenone; 250 part per billion (ppb or µg/L) of active ingredient (a.i.) is the maximum amount of exposure specified on labels. Thus, species in the water column, such as fish, amphibians, aquatic arthropods, zooplankton, phytoplankton, and aquatic macrophytes could be exposed to this concentration. Uptake will be primarily through gill tissues for fish, amphibian larvae, and aquatic invertebrates that have gills. Adult amphibians

and some aquatic invertebrates will be exposed through dermal absorption, which is less efficient than gills, and which would result in less sensitivity.

Rotenone adsorbs to sediments or particulate matter, including plants, in the water column. The concentrations of rotenone in sediments are typically higher than in the water column in standing waters; however, rotenone residues are rarely detected in treated streams, based upon experience in California (Finlayson et al., 2001). Where there are large quantities of sediments and plants, rotenone is adsorbed to the extent that the amounts of rotenone applied may have to be higher to achieve the same target concentration in the water (Bradbury, 1986).

Rotenone may be ingested as drinking water or as residues in aquatic food sources for certain types of birds and mammals. Rotenone may also dermally expose birds, mammals, and reptiles while in treated waters. Neither route of exposure is very efficient for a water-insoluble chemical like rotenone. For many such species, such exposure would be transient. It is assumed that species that might continue to feed regularly in the water following an application of rotenone, such as piscivorous birds, ducks, muskrats, beavers, otters, snakes, and others, would be most at risk from the use of rotenone, and that the risk from transient exposure would be relatively insignificant.

2.5 Considerations of human exposure

Humans may be exposed to rotenone in several ways. The highest potential exposure would be from the preparation and application of rotenone. Dermal and inhalation exposure would be the primary routes of exposure for applicators. Rotenone labels currently allow for swimming in treated waters after the rotenone has been well mixed into the water to achieve the desired label concentrations. Thus, swimmers could be exposed dermally to concentrations as high as allowed on the labels, 250 ppb. Oral exposure of humans to rotenone could occur from ingestion of water while swimming, ingestion of treated fish or other organisms. Human exposure could theoretically also result from ingestion of crops that have been irrigated with rotenone-treated water or ingestion of water where rotenone reaches a potable water intake. However, label directions dictate that treated fish not be used as food or feed, and that no use of rotenone should occur within ½ mile (upstream in rivers or streams) of irrigation or potable water intakes.

2.6 Special considerations

2.6.1 Potential of rotenone to cause Parkinson's disease

In a research study, Betarbet, et al. (2000) conducted a study that demonstrated that rotenone produced Parkinson's-like anatomical, neurochemical, and behavioral symptoms in laboratory rats when administered chronically and intravenously. In this study, 25 rats were continuously exposed for 5 weeks to 2 to 3 mg rotenone (dissolved in dimethyl sulfoxide [DMSO] and polyethylene glycol [PEG]) per kg body weight per day. The exposure was accomplished by injecting the mixture directly into the right jugular vein of the rats using an osmotic pump. Twelve of the 25 rats developed lesions characteristic of Parkinson's disease. Structures similar to Lewy bodies (microscopic protein deposits) in the neurons of the substantia nigra in the brain (characteristic of Parkinson's disease) were produced in several of the rotenone-exposed rats. Although the route and duration of exposure was atypical of what humans or wildlife might

experience, this finding provoked concern because exposure via inhalation likewise bypasses the inefficient gut absorption of rotenone along with the detoxification mechanisms. A large body of literature has subsequently addressed this effect. (e.g., Scherer, et. al., 2002; Scherer, et. al., 2003a; Scherer, et. al., 2003b; Scherer, et. al., 2003c; Betarbet, et. al., 2006; Riederer, et. al., 2006; Hirsch et. al., 2003; Gao et. al., 2003; Yang et. al., 2003). See section 9.2.7.2.

Applicators, mixers, and loaders of rotenone for piscicidal use may be exposed via inhalation, especially to powder formulations, frequently over extended periods of time during the application seasons, primarily fall and spring. There appears to be no concern for the Parkinson's effect from other routes of exposure (oral and dermal), nor for relatively brief exposures. Therefore, the primary concern for the effect would be for persons using rotenone regularly. An analysis of the potential risks to applicators and others handling rotenone will be based upon the potential routes of exposure and a comparison with the studies demonstrating a link to Parkinson's disease. Considerations of required protective clothing and equipment will also be addressed.

2.6.2 Mobility and Potential Ground Water Contamination by Rotenone in Areas of Fractured Basalt

The typical route by which a pesticide reaches groundwater is through leaching, which can be fairly readily predicted based upon the physical-chemical characteristics of a pesticide and the nature of the soils through which it must pass to reach ground water. However, much of the Pacific Northwest has a highly volcanic history. Numerous layers of basalt flows, individually averaging about 100 feet thick, and collectively up to 15,000 feet thick, underlay the surface. As the lava flows cool, they tend to shrink, resulting in cracks or fissures through which liquids may permeate. Subsequent folding and faulting can also lead to openings in the layers. The tops and bottoms of these layers are particularly permeable because of fractures, vesicles and rubble zones. Unconsolidated, sedimentary soils between basalt layers may be even more permeable (USGS, 1994). At the same time, unfractured basalt layers are not permeable, and water would move laterally across these layers rather than vertically through them.

The potential movement of chemicals through fractured basaltic rocks and associated soils has become an issue in Washington as a result of studies at the Hanford site near Yakima, where radiologically and chemically contaminated water plumes are approaching the Columbia River (Williams, et al, 2000). Extensive studies by Williams, et al. (2000) and Spane and coworkers (Spane and Raymond, 1993; Spane and Vermeul, 1994; Spane and Webber, 1995; Spane, et al., 2001) have shown some aquifers are connected, while others are not, and lateral movement is as likely or more likely than vertical movement. These studies have also demonstrated that the hydrological characteristics of such basaltic soils vary significantly. Understanding the potential movement of substances in the ground water requires a detailed analysis of an individual site, and the extensive amount of research done to characterize the Hanford site is highly unusual.

Because the potential exists for movement through fractured basaltic soils, and because there is insufficient characterization of the hydrology for sites other than Hanford, it is necessary to analyze the potential movement of rotenone into groundwater from indirect means. Consequently, the analysis will involve an evaluation of rotenone monitoring and the physical-chemical characteristics of rotenone relative to the types of sites where it would be used. This

will be supplemented with information on ground water detections and well inventories, monitoring projects in Washington and other volcanic areas, and by an analysis of the mobility of rotenone through lake beds.

3. Label Description and History

3.1 Rotenone registered products

EPA issued a Reregistration Eligibility Decision (RED) document for rotenone in March, 2007 (USEPA, 2007). EPA concluded that rotenone is eligible for reregistration, but included requirements in this RED for registrants to change their labels, along with a data requirement to address drinking water concerns (see section 3.5), to be satisfied before rotenone products can actually be reregistered. The description of rotenone labels in this document, and the proposed labeling requirements in Appendix 2 are current, to the best of our knowledge, as of March, 2007. There will be a 60-day public comment period, most likely beginning the week of May 21, 2007. Some of the labeling requirements could change as a result of public comment.

3.1.1 For use as piscicides (labels and MSDS sheets to be attached)

There are currently 15 end-use product labels for rotenone as a fish toxicant. (USEPA, Pesticide Product Label System, accessed on-line at <http://www.epa.gov/pesticides/pestlabels/>, March 20, 2007). Rotenone end-use piscicidal products may be powders/dusts, emulsifiable liquids, or pellets/baits used for feeding. All end-use products for piscicidal use include associated cube resins, as well as rotenone, in the product in amounts equal to or greater than the rotenone, itself. Some products, including all dusts or powders, have only rotenone and cube resins as the active ingredients. Other emulsifiable liquids and both bait products have piperonyl butoxide added as a synergist.

The details of these labels, including product identification, applications methods, and warning statements, are presented in Appendix 1.

3.1.2 Other uses

Rotenone has been used widely as an “organic” insecticide on pets, livestock, garden and agricultural crops. The three registrants of manufacturing use rotenone products have requested voluntary cancellation of these uses (USEPA, 2006b); the cancellation of these products means that all end use products are also cancelled. Although no cancellation document was found, the RED makes it clear that all uses, other than piscicidal uses, have been or will be cancelled; the RED considers only the piscicidal uses. As a result of these cancellations, certain data requirements recommended to assess human health effects and terrestrial fate and transport have been put “in reserve” and are very unlikely to ever be required for piscicide uses (USEPA, 2007).

3.2 Application methods and rates

All rotenone end-use products, except for baits, base the application rates on the objectives for using the products and the size and nature of the water to be treated. The concentrations of rotenone active ingredient needed to achieve various kinds of control objectives are presented in

Table 3.1. The concentrations of active ingredient are the same for all labels, except baits, and are stated to be adapted from “Kinney, Edward. 1965. Rotenone in Fish Pond Management. USDI, Washington, D.C. Leaflet FL-576.” Each label also indicates the amount of that product necessary to achieve these concentrations of active rotenone, as well as the number of acre-feet of water to be treated by one gallon or one pound of product. The labels provide the applicator with methods to compute the number of acre-feet in a pond or, as applicable, a stream.

Table 3.1. Amount of active ingredient of rotenone for various treatments	
Type of Use	Ppb of rotenone active ingredient
Selective treatment	5-7
Normal pond use	25-50
Remove bullheads or carp	5-100
Remove bullheads or carp in rich organic ponds	100-200
Preimpoundment treatment above dam	150-250

Labeled application methods vary by the type of product, as well as the nature of the water to be treated. In general, for treatment in lakes, ponds, and reservoirs (hereafter, collectively referred to as “lakes”), the product is mixed with up to 10 gallons of water and then is to be uniformly applied over the surface or bubbled through underwater lines. The label for Sure Guard Powdered Cube adds suggestions on how to mix their surface-applied product into the water by use of the boat in several ways.

In conjunction with treatment of lakes, the water flowing into these reservoirs may need to be treated to prevent fish in the lake from moving upstream into untreated areas. As with lakes, labels specify how to determine the number of acre-feet to be treated, based on the size and flow of the stream, so that the concentration in the inflowing streams will be the same as in the lake. Applications to inflowing streams should be made before and during treatment to the lakes to provide an adequate barrier to fish movement from the lake, and they should be made as close to the lake as can be reached, but far enough to provide a barrier; these distances are not specified, but fisheries managers (i.e., experienced applicators) are generally knowledgeable regarding how such distances differ for various types of waters and fish treatments. Some labels indicate the application methods for inflowing streams; the several labels that include this information indicate that the premixed or undiluted rotenone be poured into the center of the stream. Other labels indicated that directions for treatment of outflowing streams be followed, while still other labels refer to the directions for outflowing streams, but those directions are not included in the available labels. (See Appendix 1 for details on individual labels) Again, there seems to be a presumption that fisheries managers will be experienced and knowledgeable applicators.

Those labels which allow for use in streams and rivers (other than the barrier treatments for streams flowing into lakes) generally have a monograph on such treatments. Calculations of amounts needed are necessary and essentially the same as for inflowing streams. Specified application methods include drip stations, sprays, and underwater application. Enough sites should be treated to ensure a minimum 2 hour exposure to the rotenone; typically application sites will be spaced 1-2 miles from each other, and the duration of the treatment would be 4-8 hours. Some labels for streams and rivers refer to the directions for lakes for “slow moving rivers” and provide directions only for “flowing rivers and streams;” other labels provide

directions for both. Some labels discuss the need for additional spraying, such as by backpack sprayer, of stagnant areas or backwaters. Again, see Appendix 1 for details according to individual labels.

WDFW has indicated that they may use any application method allowed by the label because there is a wide diversity of sites that may be treated (J. Anderson, WDFW, personal communication, May 7, 2007). Various application methods with boats are typical. Aerial application of emulsifiable concentrate formulations, typically by helicopter, but also by fixed wing aircraft, may be used especially in large shallow areas where access by boats is not feasible. Hand or backpack spray applications may be used for other areas. The goal is to ensure that there are no untreated areas to provide refugia for the target fish.

WDFW has also indicated that they want to consider all types of products and treatments. Historical use has been conducted with both powdered and liquid formulations. The bulk of the treatment has been done with the powdered formulation based on cost-effectiveness considerations. However, objectives of fisheries managers may warrant the use of liquid (emulsifiable concentrate) products, possibly including those products with piperonyl butoxide added as a synergist. Most previous treatments in Washington have been to restore or enhance trout fisheries, but treatments to enhance warm water fisheries are also done. In more recent years, treatments have been proposed or used in other jurisdictions to aid in the protection and recovery of threatened and endangered fish species. Having a maximum array of available tools will allow WDFW to be more responsive to the public, to Fish and Wildlife Commissioners, to common goals with federal agencies, and to legislators.

Two rotenone products are formulated as baits, one for carp and one for grass carp. These are used by setting up feeding stations where target fish are “trained” to feed on rotenone-free pellets for one to three weeks prior to using pellets with rotenone in them. These feeding stations may kill all of the target species that have been attracted to the station. Stations are moved periodically to expose different populations of target fish. The feasible goal of such treatment is a reduction in target fish numbers, rather than complete removal, unless the whole body of water can be treated at one time to prevent movement of target fish to areas away from an active feeding station.

3.3 Efficacy and selectivity of rotenone products

Most uses of rotenone by WDFW are for the purpose of removing all fish in a body of water. Subsequent restocking of salmonids (usually) or warm water fish (occasionally) is done once the rotenone is no longer present in concentrations high enough to kill fish. However, it is unlikely that rotenone treatments in accordance with labels will kill all fish for toxicological and ecological reasons. Biological variability in responses to toxicants is sufficient among naturally heterogeneous organisms that it is very rare that all organisms in a population will be killed. In addition, many treated waters have freshwater inflow at springs, isolated pockets or other areas in which the rotenone may be diluted below effective concentrations, or may not be distributed in sufficient quantities to have an effect.

In looking at differential sensitivity of various fish species to rotenone, it can be seen that rainbow trout are more sensitive than bluegill sunfish in standardized tests (Table 6.1). In

general, it appears that cold water fish, especially salmonids, tend to be more sensitive than warm water fish; however, variable test conditions for toxicity values in Table 6.2 preclude a good comparison.

3.4 Expected changes in rotenone labeling

The rotenone RED (EPA, 2007) specifies a number of label changes necessary for the end-use rotenone products to be reregistered. These are reproduced completely in Appendix 2. While these are technically “final,” there will be a public comment period for 60 days, and there is a potential for some of these to be changed. Key features are:

- A Standard Operating Procedure (SOP) manual, approved by EPA, must accompany the product and is considered labeling and is therefore mandatory. One SOP manual is apparently being developed by several federal agencies. The National Park Service may have the lead in developing the manual. EPA has not yet seen a draft of the manual (T. Steeger, EFED, telephone communication, April 8, 2007).
- The RED requires that all end-use formulations of rotenone be classified as restricted use, requiring certified applicators to be trained. The basis for this requirement was acute oral and acute inhalation toxicity to humans and toxicity to fish and other organisms. The piscicidal end-use formulations of rotenone were already classified for restricted use due to aquatic toxicity, but some labels did not include oral or inhalation toxicity in mammals as a basis.
- The Personal Protective Equipment (PPE) requirements have been strengthened. Previous labels were variable, but the only PPE specified for applicators and other handlers was goggles or safety glasses if using diluted material. An approved respirator was required only for those working with undiluted material. Now applicators and other handlers will be required to use a respirator, full face protection, and other protective clothing even with diluted material, except that boat pilots and others that are in an adequately enclosed area on the boat need only wear long sleeves, long pants, shoes and socks. See Appendix 2 for additional details.
- Closed cockpits are required for aerial applications.
- Mixing and loading is to be done in closed systems which vary for the formulation and stage of operation.
- Applications using a boom or other mechanized equipment must release the product below the water’s surface. Applications made with aircraft or with a backpack sprayer (for liquid formulations only) or other hand-held nozzle or equipment may release the product above the water’s surface. Application of wettable powders by backpack sprayers is prohibited.
- Persons re-entering the treated area within 72 hours must wear long sleeves and pants and chemical resistant gloves and footwear.
- Swimming or wading in the water is prohibited for 72 hours
- Maximum treatment concentrations will be reduced to 200 ppb for lakes, ponds, and reservoirs, and 50 ppb for streams and rivers.
- Water leaving the treatment area must be deactivated with potassium permanganate to prevent exposure beyond the defined treatment area. Instructions are in the Rotenone SOP Manual.

3.5 Additional requirements of the RED other than labeling.

The one requirement to develop new data is for a study to verify EPA's assumption that deactivation of rotenone by potassium permanganate or an oxidative water treatment will eliminate rotenone in drinking water. Additional data requirements are now being held in reserve because of the cancellation of agricultural uses, meaning that they will not be required for the piscicidal uses.

4. Chemical Characteristics

The physical/chemical data in the following sections are those required by USEPA when a product is registered for use in the U.S. as a pesticide. These characteristics assist in the basic understanding of the molecule and are later used in predicting environmental behavior or are considered when higher tiered studies are designed or requested. Pure active ingredient or technical grade active ingredient refers to the active compound(s), which cause the desired biological effect when applied to a target system. The technical grade active ingredient is typically formulated into end-use products, also known as formulated products. However, powdered cube root fish toxicants typically consist of the ground roots of tropical plants in the bean family (Leguminosae) including jewel vine (*Derris* spp.) and lacepod (*Lonchocarpus* spp.) and do not contain added technical-grade rotenone.

The end-use products consist of a known percentage active ingredient plus a solvent or solid carrier and may include surface active components to aid in dissolution, emulsification, suspension, etc., of the active ingredient. Technical products such as rotenone are normally the most highly purified preparation of the active ingredient and are rarely the desired form in the end-use product. One method used to produce a useful end-use product is to combine the technical grade active ingredient with suitable EPA approved solvent(s) and surface active ingredients such as emulsifiers, spreaders and stickers to produce an end use product which can be mixed with water or oil to be applied to the desired target object or organism. Alternately, the active ingredient can be applied to a solid substrate such as clay to produce a solid product. Such a product could be used as a dust, or it might be pelletized or tableted to produce a product which can be distributed with a reduced exposure to dust or other small particles which could be irritating to the applicator.

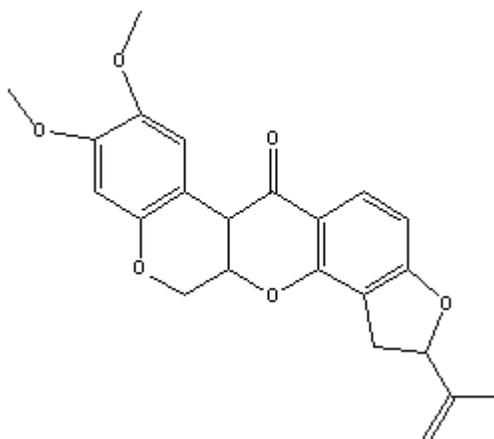
4.1 Composition of the various rotenone end-use products

4.1.1 Active ingredients

Rotenone {[2R-(2 α ,6 α ,12 α)]1,2,12,12a-tetrahydro-8,9-dimethoxy-2-(1-methylethenyl)-[1]benzopyranol[3,4-b[furo[2,3h][1]benzopyran-6(6ah)-one]} is the active component in several terrestrial insecticides and for use in the aquatic environment as a piscicide in static and flowing water to kill fish. Rotenone is a naturally occurring plant flavonoid that exhibits its pesticidal action by uncoupling oxidative phosphorylation in the cell mitochondria by blocking electron transport at complex I. (Finlayson, 2000).

Rotenone is obtained from the extracts of roots, leaves and seeds of several plants that are members of the pea or bean family (Leguminosae) including the jewel vine (*Derris* spp.) (EXTOXNET, 1996; Finlayson, 2000). The rotenone active ingredient is extracted from the plant and formulated as either a crystalline or liquid product, or the plant is ground to form a powder. Examples of rotenone preparations are Prentox Cube Powder (for manufacturing use only) (EPA Reg. No. 655-3), Prentox Cube Resins (for manufacturing use only) (EPA Reg. No. 655-69), Prentox Prenfish Toxicant (EPA Reg. No. 655-422), Noxfish Fish Toxicant Liquid-Emulsifiable (EPA Reg. No. 655-805). The Prentox® products are used predominantly for lake and static water treatments to eliminate all fish prior to restocking with desired species.

Common name: Rotenone
CAS Registry No.: 83-79-4
Chemical name: [2R-(2 α ,6 α ,12 α)]1,2,12,12a-tetrahydro-8,9-dimethoxy-2-(1-methylethenyl)-[1]benzopyranol[3,4-b[furo[2,3h][1]benzopyran-6(6ah)-one
Empirical formula: C₂₃H₂₂O₅
Molecular weight: 394.42
Structure:



4.1.2 Impurities

Information on impurities, other than the associated active ingredient, “cube resins,” is part of the Confidential Statement of Formulation, and because it is Confidential Business Information, it is not generally available. Some non-quantitative information in EPA documents is discussed below.

There are no impurities identified by the manufacturers or the US EPA which are known to be of toxicological or environmental concern. The majority of the products, both MUP and end-use also contain cube resins related to rotenone. While these compounds have not been investigated individually for their toxicological properties, the testing of the products includes the contributions of these related materials, therefore the toxicity stated for the commercial product includes their potential effects. In the case of the ground plant products, normal plant constituents will also be present in the final preparation. While considered “impurities”, plant

structural and cellular components other than the cube resins represent normal and natural constituents and should not be cause for concern. The US EPA has established guidelines that require that impurities of concern, such as N-nitrosoamines and chlorinated dioxins and furans must be disclosed. No such compounds are present in the Rotenone products.

4.1.3 Added inert ingredients

Information on added inert ingredients is part of the Confidential Statement of Formulation, and because it is Confidential Business Information, it is not generally available. Some non-quantitative information in EPA documents is discussed below.

The USEPA has established a category listing system for the “other” (inert) compounds used in pesticide formulations. The lists are designated 1, 2, 3, 4a and 4b. Compounds are assigned to the various lists according to their toxicological concern and to the extent their safety has been reviewed by the Agency. In the case of each list, if USEPA determines that a compound is no longer used in any pesticide formulation, it will be removed from the list.

List 1 contains eight compounds, which, due to their toxicological profile, require special labeling if used in a pesticide formulation. These compounds are generally not used in pesticidal formulations any longer. There are no List 1 compounds in the Rotenone formulations used in the State of Washington.

List 2 compounds are those for which USEPA has not yet determined a full profile but is reviewing existing information. At the completion of their evaluation, it is expected that the compounds still in use in pesticide formulations will be moved to List 1 or to List 4. There are no List 2 compounds in the Rotenone formulations used in the State of Washington.

List 3 contains those compounds which have not been fully evaluated, but which have profiles of lesser concern in the USEPA evaluation scheme. It is expected that most of these compounds will be moved to List 4 once their evaluation by the Agency is complete. There are some List 3 inert compounds in the Rotenone products.

List 4 is divided into two categories. List 4A contains compounds generally regarded as safe for use in pesticide formulations and includes such compounds as corn cobs and attapulgitic clay. List 4B contains those compounds that have sufficient data on file at EPA to substantiate that they can be used safely in pesticide products.

There are compounds from Inerts List 4 in several of the Rotenone formulations. The levels of these compounds include grain and vegetable products incorporated into the Carp and Grass Carp bait products.

In addition to the above-mentioned review by the USEPA, all registered pesticidal end-use products (the products actually applied to the environment to control weeds or pests) must undergo a series of toxicological tests to establish their safety. Because these tests are performed on the actual end-use formulation, the effects of the “other” ingredients are effectively tested simultaneously. This toxicological screen of the “other” compounds affords an additional

opportunity to examine comparative data on the active ingredient versus the end-use product to determine if there is a need to test each of them in a complete testing battery.

Intentionally added inert or “other” ingredients identified in rotenone formulations include grain and vegetable products in the fish bait products. Any formulations made from manufacturing use products prepared from dried plant will include the expected plant tissue residues which are not expected to pose any incremental risk in the typical use scenarios.

Table 4.1 lists the percentage active ingredients and other ingredients (inert ingredients) in several of the rotenone containing products labeled for aquatics use.

Table 4.1 Ingredients of selected rotenone products				
Product (form)	Ingredient (active ingredients)	Description (other ingredients)	Weight %	Purpose
Prentox Prenfish Toxicant (Emulsifiable Concentrate)	Rotenone		5	Active
	Cube Resins		10	Active
	Aromatic Petroleum Solvent	Naphthalene 9.9 % 1,2,4-trimethylbenzene 1.7% Mixed solvents 88.4%	≤80	Solvent
	Acetone		≤7.5	Solvent
	Emulsifier 1		1.5	Surfactant
Prentox Synpren-Fish Toxicant (Emulsifiable Concentrate)	Rotenone		2.5	Active
	Cube Resins		5	Active
	Piperonyl Butoxide Technical		2.5	Synergist
	Xylene Range Aromatic Solvent	1,2,4-trimethylbenzene 32% Mixed xylenes 3.0% Cumene 1.5% Ethyl Benzene 0.5%	≤90	Solvent
	Emulsifier 1			Surfactant
Prentox Cube Resins (Powder)	Emulsifier 2			Surfactant
	Rotenone		44.2	Active
Prentox Cube Powder (Wettable powder)	Cube Resins		44.2	Active
	Other Ingredients	Not Specified	11.6	
	Rotenone		7.4	Active
Powdered Cube Root (Powder)	Cube Resins		11.1	Active
	Other Ingredients	Not specified	81.5	
	Rotenone		5	Active
CFT Legumine (Emulsifiable Concentrate)	Cube Resins		5	Active
	Other Ingredients	Includes N-methylpyrrolidone	≤90	Solvent

4.1.4 Added synergists

Synergists are chemicals which lack pesticidal effects of their own, but which enhance the effects of other products. The synergist most frequently used with rotenone is piperonyl butoxide. Piperonyl butoxide is a cytochrome P450 inhibitor. Since the cytochrome P450 group of enzymes is one of the primary detoxification pathways for many pesticides, its inhibition allows the pesticidal product to remain intact in the target organism for a longer period of time and therefore increase its efficacy. NPTN, 2000 (National Pesticide Telecommunication Network, Oregon State University.)

4.1.5 Nature of formulation (e.g., powder, emulsifiable concentrate)

The nature of the various formulations may be found in Table 4.1 above. Powdered products are typically the solid form of the active ingredient ground to a fine powder which may be mixed with a diluent such as clay or an organic substrate to achieve the desired concentration. In the case of the Prentox Cube Resins it is likely that “other” ingredients consist primarily of plant extracts and ground plant structural components (cellulose, etc.) other than the resins. Emulsifiable concentrates are typically formulated as the active ingredient dissolved in a suitable solvent with surface active agents added to facilitate their dispersion (emulsification) in water allowing them to be readily introduced into the target waters. Wettable powders are formulated by mixing the solid form of the active ingredient with solid diluents such as clay along with surface active agents to facilitate the dispersion of the product in water in preparation for application.

4.2 Color

Color is an end-point observation of the product used to assist in identification.

Rotenone formulation	Color	Citation
Rotenone	White to off White	(Merck, 1989)
Prentox Cube Powder	Tan	Prentiss, 2000a
Prentox Cube Resins	Tan	Prentiss, 2000b
Prentox Synpren-Fish Toxicant	Amber	Prentiss, 2000c
Prentox Prenfish Toxicant	Amber	Prentiss, 2000d
Powdered Cube Root	Tan	Foreign Domestic, 2004
CFT Legumine	Orange	Prentiss, 2005b

4.3 Physical State

Physical state is an end-point observation of the product, solid, liquid or gaseous used to assist in identification.

Rotenone formulation	Physical State	Citation
Rotenone	Solid	(Merck, 1989)
Prentox Cube Powder	Solid powder	Prentiss, 2000a

Prentox Cube Resins	Solid Powder	Prentiss, 2000b
Prentox Synpren-Fish Toxicant	Liquid	Prentiss, 2000c
Prentox Prenfish Toxicant	Liquid	Prentiss, 2000d
Powdered Cube Root	Solid powder	Foreign Domestic, 2004
CFT Legumine	Liquid	Prentiss, 2005b

4.4 Odor

Odor is an end-point observation of the product used to assist in identification. Odor may also serve as a warning in cases where odorants are added as a safety factor.

Table 4.4 Odor of Rotenone and Formulations		
Rotenone formulation	Odor	Citation
Rotenone	Slight to none	(Merck, 1989)
Prentox Cube Powder	Wet Chalk	Prentiss, 2000a
Prentox Cube Resins	Wet dirt	Prentiss, 2000b
Prentox Synpren-Fish Toxicant	Aromatic	Prentiss, 2000c
Prentox Prenfish Toxicant	Aromatic	Prentiss, 2000d
Powdered Cube Root	Wet chalk or dirt	Foreign Domestic, 2004
CFT Legumine	Light solvent	Prentiss, 2005b

4.5 Melting Point

The melting point is a physical end point observation used for identification of pure compounds and may provide some indication of thermal stability. For the pure acid active ingredient (Rotenone), the melting point has been reported as 165-166°C (Merck, 1989) and 163-181°C (Tomlin, 1994 in USEPA, 2006c). Melting point is not applicable to the formulations because they are either liquids or mixed solids.

4.6 Boiling Point

The boiling point is a physical end point observation for identification of pure compounds. The boiling point for the pure active ingredient of rotenone is undefined because it is a solid at room temperature.

4.7 Density, Bulk Density or Specific Gravity

Bulk density is a measure of the weight per unit volume of the product and is useful for physical identification or differentiation of two similar products. The value may also be needed to calculate application rates in some instances. Density is typically reported as grams per cubic centimeter at 25°C.

Table 4.5 Density of Rotenone and Formulations		
Rotenone formulation	Density (g/cc)	Citation
Rotenone	0.24 (fluffed) 0.45 (packed)	Barnes, 2005 in USEPA 2006a
Prentox Cube Powder	0.24 (fluffed) 0.45 (packed)	Prentiss, 2000a
Prentox Cube Resins	0.67 (fluffed)	Prentiss, 2000b

	0.78 (packed)	
Prentox Synpren-Fish Toxicant	0.8964	Prentiss, 2000c
Prentox Prenfish Toxicant	0.9226	Prentiss, 2000d
Foreign Domestic Powdered Cube Root	0.024 (fluffed) 0.45 (packed)	Foreign Domestic, 2004
CFT Legumine	1.019	Prentiss, 2005b

4.8 Solubility

Solubility is a physical end point useful for understanding potential environmental impact. High water solubility is frequently associated with mobility and affects distribution in water and soil. This endpoint is determined for the active ingredient in a product and is typically reported as grams per 100 ml water at 25°C. Additionally, pure rotenone is known to be soluble in alcohol, acetone, chloroform, carbon tetrachloride, ether and other organic solvents.

Rotenone formulation	Solubility in Water @ 25°C (g/100 ml)	Citation
Rotenone	0.0002	Augustijn-Beckers, 1994 in USEPA 2006c
Rotenone	0.0000142 @ 20°C	Barnes, 2005 in USEPA 2006a
Powdered Cube Root	Insoluble	Foreign Domestic, 2004
Prentox Cube Powder	Insoluble	Prentiss, 2000a
Prentox Cube Resins	Insoluble	Prentiss, 2000b
Prentox Synpren-Fish Toxicant	Emulsifies	Prentiss, 2000c
Prentox Prenfish Toxicant	Emulsifies	Prentiss, 2000d

4.9 Vapor Pressure

Vapor pressure is a physical end point useful for understanding the distribution of the active ingredient between water/soil and air. High volatility is an indication of potential impact in the air compartment. This endpoint is determined for the active ingredient in a product and is typically reported Torr at a specified temperature. The vapor pressure is not required for end-use products.

	Vapor Pressure @ 25°C (Torr)	Citation
Rotenone	6.9 X 10 ⁻¹⁰ *	EPIWIN, 2004 in USEPA 2006c
Rotenone	< 1 x 10 ⁻⁵	MP Biochemicals, 2006

* Estimated

4.10 Disassociation Constant

Disassociation constant is a physical end point used to assess the distribution of the product in aqueous media. The reported values indicate the environmental pH at which the active ingredient molecule could dissociate to an ionic form. In the case of Rotenone, there are no dissociable

groups therefore no values are reported in the range of 2-12 pH (Barnes, 2005 in USEPA, 2006c).

4.11 Octanol/Water Partition Coefficient

Octanol/Water partition coefficient (K_{ow}) is a physical end point used to assess the potential of a compound to bioaccumulate in the environment. The value represents the ratio of product in octanol versus water at equilibrium at 25°C. Log values of K_{ow} less than 5 indicate reduced likelihood of bioaccumulation. Direct bioaccumulation studies (Gingerich and Rach, 1985 in USEPA, 2006c) indicate a low potential for accumulation in aquatic organisms. The partition coefficient is not required for end-use products.

Table 4.8 Octanol-water Partition Coefficient of Rotenone		
	Octanol/Water Coefficient (log Kow)	Citation
Rotenone	4.10	Hansch, 1995 in USEPA 2006c
Rotenone	4.16	Barnes, 2005 in USEPA 2006a

4.12 pH

pH is a physical end point used to identify the product and to assess the potential effect of the equilibrium in the environment. The measurement of pH was waived for the rotenone products (Barnes, 2005 in USEPA, 2006a).

4.13 Stability

Stability is a chemical evaluation of the product to assess the potential effect of heat, light, metals and metal ions on the active ingredient. In the case of Rotenone exposure to light and air will lead to degradation (Merck, 1989). In a study of product stability it was determined that there was no change in appearance on exposure to elevated temperatures and to metals. In each exposure there was less than 5% loss of parent rotenone (Barnes, 2005 in USEPA, 2006a).

4.14 Oxidizing or Reducing Action

Oxidizing or reducing action is an assessment of the potential for a compound to react with common oxidizers or reducers. In the case of Rotenone and its formulated products, there is little likelihood of such reactions occurring.

4.15 Flammability

Determination of flammability is measurement of the temperature that will sustain a flame and is used to classify the product for hazard in storage and shipping. Determination of flammability is not required for technical grade products. The emulsifiable concentrate products may contain aromatic hydrocarbons and have a defined flash point.

Table 4.9 Flash Point of Rotenone and Formulations		
Rotenone formulation	Flash Point (°F)	Citation

Table 4.9 Flash Point of Rotenone and Formulations		
Rotenone formulation	Flash Point (°F)	Citation
Rotenone	N/A	
Prentox Prenfish Toxicant	60	Prentiss, 2000d
Prentox Synpren-Fish Toxicant	105	Prentiss, 2000c
Prenfish Toxicant	45	Prentiss, 2002
Nusyn-Noxfish Fish Toxicant	115	Prentiss, 2001b
Noxfish Fish Toxicant Liquid Emulsifiable	45	Prentiss, 2005a
CFT Legumine Fish Toxicant	192	CWE, 2005

4.16 Explodability

Determination of explodability is measurement of the potential for a compound to explode when exposed to physical or thermal shock. Determination of explodability is not required for technical grade products. The formulated products are either aqueous or clay based and are not explosion hazards. Additionally, the Rotenone molecule contains no explodable functional groups. The measurement of explodability was determined to be not applicable for the rotenone (Barnes, 2005 in USEPA, 2006a)

4.17 Storage Stability

Storage stability is the physical determination of the stability of the active ingredient when stored in its commercial packaging over extended time periods, usually one to two years or more. Rotenone products have been shown to be stable under normal storage conditions for periods of at least one year in poly bags at 22°C and for 0.5 years in poly bags at 40°C. (Barnes, 2005 in USEPA, 2006a).

4.18 Viscosity

Viscosity is a physical end-point measurement used to identify the product and to assess the ability of the product to be poured or pumped. The measurement is not required on technical grade products or on solid products. The viscosity is reported in centipoise. The measurement of viscosity was waived for the Rotenone products (Barnes, 2005 in USEPA, 2006a).

4.19 Miscibility

Miscibility is a physical assessment of the ability of a formulated product to mix with spray oils for use during application. Since the Rotenone aquatic products are not labeled for application in oil, this data requirement is not applicable (Barnes, 2005 in USEPA, 2006a).

4.20 Corrosion Characteristics

Corrosion characteristics requires the physical observation/measurement of the effects of the product on the commercial packaging. Measurements of the weight, deformation and strength of the packaging are reported. A study of the corrosion characteristics for rotenone showed no effects on the packaging (Barnes, 2005 in USEPA, 2006a).

4.21 Dielectric Breakdown Voltage

Dielectric breakdown voltage is the physical measurement of the effect of an electric arc on the stability of the formulated product. This requirement applies only to formulations that are applied around electrical equipment or apparatus. As there is no likelihood of open electrical apparatus in the aquatic environment, this test is not applicable (Barnes, 2005 in USEPA, 2006a).

5. Environmental Fate

5.1 Volatilization

Rotenone is not persistent in the environment and its low vapor pressure (6.9×10^{-10} torr) and Henry's Law constant ($1.1 \times 10^{-13} \text{ atm-m}^3 \text{ mol}^{-1}$) limit its volatility (USEPA, 2006a).

5.2 Hydrolysis

Hydrolysis refers to the chemical interaction of the agrochemical with water as a mechanism of agrochemical breakdown. While aqueous or aquatic (the terms are synonymous) persistence studies are sometimes conducted in natural water bodies, true hydrolysis studies are conducted in laboratories using sterile distilled or deionized water so that the chemical effects of an aqueous environment can be isolated from biological, sunlight, or sediment interactions.

Laboratory hydrolysis studies for EPA submission are typically performed with radioactive ^{14}C pure compound at three pH values (pH 5, pH 7, pH 9) corresponding to slightly acid, neutral, and mildly alkaline, respectively) in sterile water for a period of 30 days at 25°C. Sampling for breakdown products and the remaining concentration of parent material occurs at frequent intervals.

5.2.1 Half-life

Because laboratory hydrolysis studies are normally only conducted to fulfill EPA registration requirements, only one such study was found. In this study (Thomas, 1983, in USEPA 2006c), hydrolysis testing was conducted at 3 nominal pH values and 25°C. Rotenone was relatively rapidly degraded with calculated half-lives of 3.2 days at pH 7, 12.6 days at pH 5 and 2.0 days at pH 9. As natural lakes would likely have a pH of approximately 7 or slightly higher in the State of Washington (WDOE, 2007) the half-life of rotenone as a function of hydrolysis would be expected to be less than 3-4 days.

5.2.2 Degradation products

In the above cited study, $6\alpha\beta$ - $12\alpha\beta$ -rotenolone was identified as the major hydrolysis degradation product, but the levels were not quantified (Thomas, 1983, in USEPA 2006c). The overall mass balance for the study was not fully detailed. No other degradates were identified. Portions of the loss may have been through volatilized carbon dioxide which would have indicated mineralization of the residues or complete breakdown of the parent and any degradates. No further discussion of degradation products was available for this study.

5.3 Aqueous photolysis

As with hydrolysis, photolysis testing is carried out in a laboratory. Vessels containing solutions of the test substance in sterile distilled or deionized water are irradiated with either a mercury vapor lamp or with natural sunlight. Identical vessels are kept in the dark for the duration of the study and also sampled in order to compensate for the effects of any hydrolysis occurring. Testing is usually carried out at 25°C, at pH 5, 7 and 9, but this is not always the case, particularly with very early studies. Other photolysis testing, such as photolysis of a pesticide on the surface of a soil, is also required by the EPA for products that might be incidentally applied to soil, as is the case for rotenone.

The purpose of photolysis experiments is to isolate the effect of sunlight, specifically the ultraviolet and near-ultraviolet part of the spectrum, on the degradation of a pesticide without biological or chemical interactions. Natural sunlight's visible spectrum covers wavelengths from about 800 nm (deep red) to about 300 nm (deep violet). Generally speaking, only light in the violet and ultraviolet end of the spectrum has enough energy to initiate or influence chemical reactions ("photochemical reactions"). Air, as well as ozone, strongly filters near-ultraviolet and ultraviolet radiation, and cuts off nearly all radiation below 290 nm wavelength. Water is transparent to radiation down to approximately 180 nm (far ultraviolet), assuming that there are no suspended solids or dissolved colored material such as humic acids to impair passage of the light.

As with hydrolysis, laboratory photolysis testing is generally conducted only in response to specific EPA registration requirements. One aqueous photolysis "registration" study was found for rotenone (Draper, 2002 in USEPA 2006c). No soil photolysis studies were identified that were related to rotenone.

5.3.1 Half-life

Draper et al. (2002, in USEPA, 2006c) conducted aqueous photolysis testing at pH 5, 7, and 9. The source, time and intensity of irradiation were not reported. The study was run for 30 days. They found rotenone half-lives to be 21 hours in surface (1 cm depth) and 191 days at a depth of 2 meters in a well mixed sample. The data were generated in a photoreactor and then subjected to the calculation methods of Zepp and Cline (1977, in USEPA, 2006c) to obtain the reported values. Details of specific pH and sample times were not reported.

5.3.2 Degradation products

In the data evaluated by USEPA (2006c), there was no discussion of the formation or identification of degradates from aqueous photolysis. No other information has been found.

5.4 Soil photolysis

Soil photolysis is carried out in the laboratory by exposing a thin layer of soil containing the target chemical to either artificial or natural sunlight. The exposed soil is usually extracted to determine the amount of parent compound and any degradates that are extractable. Additional effort is typically made to do an exhaustive extraction to remove as much of the residue as practicable, especially in the case of compounds such as rotenone which bind strongly to soil.

The soil extracts are examined to determine qualitatively and quantitatively the nature and amount of remaining parent and degradates.

No direct studies of rotenone on soil were located, however, a study of the effect of sunlight on foliar residues gives some insight as to the nature of the photolytic behavior of the compound. Rotenone was applied to the surface of bean leaves and exposed to natural sunlight (Cheng et al., 1972 in USEPA, 2006c). The residues were monitored and a half-life was determined.

5.4.1 Half-life

In the above cited study, a half-life of 1.4 hours was calculated by the authors (Cheng et al., 1972 in USEPA, 2006c). $6\alpha\beta$ - $12\alpha\beta$ -rotenolone was identified as the major degradation product, but the levels were not quantified. The overall mass balance for the study was not fully detailed. Several other degradates were separated chromatographically but were not qualitatively identified. Portions of the loss may have been through volatilization. When corrected for the apparent volatilization effects, the photolytic half-life on bean leaves was calculated to be 2.9 hours. No further discussion of degradation products was available for this study.

In a separate study Newsome, et al (1980, in USEPA, 2006c). applied two different rotenone formulations to lettuce and tomatoes and studied the residues on the leaf surfaces. The formulations were a 1% a.i. dust and a 5% a.i. wettable powder. Applications were made 7 days apart and sampling was initiated immediately following the final application. Parent rotenone and its $6\alpha'$, $12\alpha'$ -rotenolone (presumably $6\alpha\beta$ - $12\alpha\beta$ -rotenolone, but unclear from the paper) were found at their highest levels at the first sampling. The half lives for parent rotenone on lettuce were 2.9 days for the dust and 3.6 days for the wettable powder. The half life for the rotenolone degradate was 4.5 days for the dust and 5 days for the wettable powder. For the tomatoes, the half lives were 2.7 days for the dust and 0.9 days for the wettable powder. No half lives could be calculated for rotenolone, however there were no detectable residues at three days regardless of treatment. No data was reported for other plant parts or for the soil.

5.4.2 Degradation products

Cheng et. al., 1972 in USEPA, 2006c) applied rotenone to the leaves of pinto beans exposed to natural sunlight for four (4) hours. At sampling, parent rotenone accounted for just over 13% of the residue while $6\alpha\beta$ - $12\alpha\beta$ -rotenolone contributed an additional 11%. No other degradate exceeded 10% of the nominal residue [four (4) distinct chromatographic peaks totaling about 20% of the total residue were observed but not identified]. The authors ascribed some loss, about 25%, to volatilization, however there was no time zero sample, and no dark control making it impossible to confirm loss via this pathway. The low vapor pressure and low estimated Henry's Law Constant suggest that volatilization would not be an important loss pathway.

5.5 Summary of photolysis

The experiments reported here indicate that rotenone is likely to be degraded by exposure to sunlight at the range of pH values normally found in natural water bodies, from pH 7 to pH 9. And, that photolysis also contributes significantly to the degradation of rotenone.

Table 5.1 Photolysis of Rotenone

Matrix	pH	Temp (°C)	Half life (DT50)	Reference
Water	NR	NR	191 days	Draper, 2002 with Zepp and Cline, 1977 calculations both in USEPA, 2006c
Soil (Leaf surface)	NR	NR	ca2 49 days	Cheng et al., 1972 in USEPA 2006c

NR = Not reported

5.6 Degradation and Persistence - soil

Although only the aquatic uses of rotenone are considered in this document, the chemical has been registered as an insecticide and acaricide for terrestrial uses. Data regarding rotenone's persistence in soil is therefore required to be submitted to the EPA. This information also has relevance to accidental terrestrial overspray on lake or stream shorelines, and peripherally as an indication of possible fate on near-shore lake bottoms exposed by drought or drawdown following a rotenone application.

No data were located to address the degradation and persistence of rotenone in the soil environment. The registrants opted not to support the rotenone uses that would have required the submission of such data.

5.6.1 Half-life

No half lives have been calculated as there are no soil degradation data from which to draw conclusions.

5.6.2 Degradation Products

No degradation products have been identified as the result of metabolism or degradation in the soil environment.

5.7 Degradation and persistence - aquatic systems

The disappearance of rotenone from a lake or other natural water body is influenced by a number of factors. Various water chemistry conditions, physical conditions such as temperature, adsorption to the sediment, and the extent of water currents and dilution can all have very pronounced effects on the persistence of rotenone. This section reviews the disappearance times reported for natural water bodies and for artificial laboratory studies and looks at the reported factors that can influence such times.

5.7.1 Half-life and Disappearance Time

Table 3.1.4 summarizes the half-lives of rotenone reported in research papers, as well as the time to non-detection or very low levels as specified in the table. Depending on the intent of the reference, either one or the other parameter may not be reported. A half-life is the time required for rotenone to reach half of its initial concentration immediately following application. Depending on the type of study and the data collected, a half-life may be mathematically calculated using several analyses over time, or they may be interpolated from tabular data or figures given in a cited paper as was frequently necessary in this review.

Time to disappearance is the time necessary for a pesticide concentration to drop below the lower limit of analytical detection. Because of the variety of analytical techniques used over time (chemical analysis, bioassay), the Limit of Detection (LOD), the lowest reliable pesticide concentration that can be reliably quantified, has varied over time.

Half-life values are important for calculations, but can be misleading if the pesticide remains in the environment at significant concentrations after the half-life time. Times to disappearance are a useful tool for predicting impacts on biota and wildlife, particularly when used with calculated or estimated half-lives. The persistence of rotenone varies somewhat depending on the conditions of the system being tested which contributes to the range of half-life and disappearance times that has been reported in the literature.

Location	Temp (°C)	Half life (in water column)	Half life (water + sediment)	Reference
Wisconsin	5	23 hours	20 days	Gilderhus, 1986 in USEPA, 2006c
Wisconsin	23-27	10.6 hours	1.5 days	Gilderhus, 1986 in USEPA, 2006c
California	9	ca 49 days	NR	CDFG, 1999 in USEPA 2006c

NR = Not reported

5.7.2 Degradation Products

Few of the available studies were intended to produce data regarding the identity of specific rotenone degradates. The Gilderhus study conducted outdoors in Wisconsin assessed only the parent compound in assigning a half life of 1.5 days in warm (23-27°C) water and 20 days in cold (5°C) water (Gilderhus, 1986 in USEPA, 2006c). In the Lake Davis study only rotenolone was identified as a degradate/metabolite. The data indicate that rotenolone concentration increased between days 1 and 13 and then began to dissipate by day 20 post treatment (CDFG, 1999).

5.7.3 Physical and Chemical Factors affecting aquatic persistence

This section discusses the potential impact of various physical and chemical factors on rotenone persistence individually. It is difficult to separate the effects of the numerous water and sediment chemical and physical parameters on rotenone persistence. Temperature obviously will have an effect, as will pH. The aerobicity (presence or lack of dissolved oxygen) of a system, the trophic state and consequent microbial population present, adsorption to suspended solids and sediment and sediment characteristics, and transport/dilution can also influence the breakdown of rotenone. While most references do not address factors in isolated experiments, conclusions can be drawn from inference when the data are viewed as a whole.

Temperature. Temperature has a pronounced effect on the rate of chemical reactions and metabolic processes. In the case of rotenone, temperature appears to be at least somewhat important in that cooler temperatures appear to cause the half life of the product to increase as

compared to warmer water. In a small artificial pond study conducted in Wisconsin, rotenone was applied at a rate of 0.25 parts per million (ppm) of active ingredient (a.i.) to each of two ponds held at different temperatures (Gilderhus, 1986 in USEPA, 2006c). In the cooler pond (5°C) rotenone dissipated from the water column with a half life of 23 hours while in the warmer pond (23-27°C) the measured half life was 10.6 hours. Dissipation half lives from the system (water plus sediment) were 20 days from the cold water pond and only 1.5 days from the warm water pond. Similarly, the Lake Davis study amplifies this characteristic as the water temperature in this lake treatment was relatively cold (9°C) (CDFG, 1999). Under these conditions, the half life was extended to about 10.3 days. The data were based on a large number of data points, 9 sampling sites over 6 sampling dates.

In deeper lakes a thermocline can form during summer months wherein there is a sharp boundary between the warmer surface water and cold deeper water. Thermoclines can increase rotenone persistence in two ways. As there is little exchange of water across the thermocline, there is less water volume to dilute the product, particularly in lakes treated over a large percentage of their surface, and any rotenone that does penetrate the thermocline encounters a cold environment where the half life is extended.

Laboratory studies, typically conducted at 20°C to 25°C (68°F to 77°F) may yield rotenone half-lives that are somewhat shorter than studies in ponds or lakes. In addition, the latitude of the lake, with varying temperature regimes make comparisons difficult.

pH. Based on the hydrolysis data discussed above (Thomas, 1983 in USEPA, 2006c) it is expected that waters with a pH of 7 or greater will facilitate a more rapid degradation of parent rotenone. This phenomenon is tempered somewhat by the temperature data, especially from Lake Davis (CDFG, 1999) which showed that even at elevated pH, the cooler water appeared to extend the half life of the active ingredient beyond that seen in the laboratory studies, or in artificial ponds exposed to normal climatic conditions other than the control of the temperature.

Aerobic state. There are no available data to indicate the effect of local microbial populations, therefore the effects of differing oxygen levels in the water cannot be fully assessed. Based on the relatively short half lives seen in the hydrolysis and aqueous photodegradation studies, it is not anticipated that oxygen levels play an important role in the degradation of rotenone.

Transport and dilution. Probably the most important and obvious physical processes affecting rotenone concentration in larger water bodies are dispersion or transport from the treated site by water currents and dilution by untreated water. In spite of its low water solubility, rotenone is expected to be transported within water currents in a lake or stream, and by lateral dispersal. Obviously, the larger the area of a lake that is treated, the more water will be needed to dilute and disperse the product, with the extreme case occurring in whole-lake treatment.

In lakes without significant inflow or outflow, most dilution of rotenone-treated water will occur through vertical movement in the water column. Solar heating is not as important to water movement in these lakes as the effects of wind. While sunlight can heat the surface depths, the warmer water tends to stay at the surface and little vertical circulation occurs. Wind can induce mixing between water depths even at low velocities. Surface water driven against a shoreline is

driven downward and mixes with lower depth water, diluting the rotenone concentration of the surface water.

In summary, rotenone is frequently dispersed away from the treated areas of a lake and consequently diluted by water currents. Such currents can be caused by wind action or inlet and outlet streams or rivers. Vertical dispersion is the mechanism of dilution in whole-treated lakes. WDFW does not conduct "partial treatments" of lakes. Although dilution may occur through water flowing into another lake that, while not treated itself, is part of the overall treatment area.

5.8 Microbial Degradation

There were no studies available to address either aerobic or anaerobic microbial degradation of rotenone.

5.9 Mobility

When a chemical is applied to soil, a potential exists for the chemical to be carried down into the soil with water movement from rain and irrigation. Pesticides exhibit a wide range of leaching potential, from those that adsorb strongly to soil particles and are not released before they break down, to those that do not adsorb significantly (or adsorb, then desorb) and will travel considerable distances down through the soil, sometimes as far as the ground water table. Different chemicals are affected in different ways by various soil parameters such as organic matter, clay content and type, and pH.

Soil mobility data point out expected behavior of rotenone oversprayed on the shoreline and to some extent indicates what may happen if a lake level drops, exposing shoreline sediment to drying, soon after treatment. The data also give at least an indication of rotenone's adsorption potential on sediment. Sediment will usually have a higher organic material content than typical soils except for muck soils and therefore soil tests may underestimate the potential for rotenone adsorption to high-organic matter sediments.

5.9.1 Soil

Controlled laboratory "batch equilibrium" studies are designed to measure the adsorptive properties of the active ingredient of a pesticidal product. In the case rotenone, such a study has not been submitted. In the absence of this study, Hansch et al. (1995 in USEPA, 2006c) used the available chemical characteristics and the EPIWIN program from the USEPA to estimate the adsorption characteristics of rotenone. The sorption parameters indicate how well rotenone is adsorbed and released on that typical soil and hence will give one measure of leaching potential. The K_d values calculated from EPIWIN range from 4.2 to 122 L/kg leading to the conclusion that rotenone is expected to be sorbed to soil and sediment surfaces and not be likely to move through the soil/sediment compartment. Although there is some disagreement as to exact classification values, generally K_{dads} values greater than 5 are characteristic of compounds that are not appreciably mobile, values from about 1 to 5 indicate a potential for greater mobility, while values under 1 denote considerable mobility potential. In a similar manner, high K_{d} values indicate that a compound will stay bound to soil and resist being carried downward.

It should be emphasized that all of the "K" parameters discussed above are specific to a particular soil or sediment, and to the initial concentration of a chemical applied to the soil or in water over sediment. Where "K" values are given without the soil type and chemical concentration being specified, care should be exercised in using those values for evaluation of leaching potential. Unless specified otherwise, "K" parameters reported in published literature are for adsorption; measurement of desorption values is rare.

5.9.2 Sediment

There are few studies available that directly assess the behavior of rotenone in aquatic sediments. It is likely that the behavior of rotenone may be inferred from the K_d values obtained by Hansch et al., (1995). This reiterates that high K_d values indicate that rotenone is strongly bound to sediment and vegetation, rendering rotenone unlikely to move through the sediment. Rotenone in the aqueous phase of a lake or stream that is not degraded by either hydrolysis or photolysis is likely to be adsorbed to the surface of sediment particles when contacted. Such sorption will limit both the movement of the residue and its availability to the flora and fauna in the water body. In the case of Lake Davis, California, the sediment levels of rotenone peaked at 14 days post treatment and declined to less than detectable levels by 33 days. Similarly, rotenolone declined to non-detectable levels by 33 days post treatment (CDFG, 1999).

5.9.3 Groundwater

From the above data, it is clear that rotenone does not pose a significant threat to groundwater. Rotenone is not significantly mobile in most soils and sediments and is readily adsorbed to the high organic content sediments to be expected in lakes. Because rotenone is so readily degraded, with half-lives typically less than 14 days and usually less, it is gone from lake sediments before it can be transported into surrounding soil. Overspray onto lake shores, or exposure of treated shallow lake sediments is expected to be negligible. Even if those situations occur, rotenone is not significantly mobile in less-than-saturated soil situations to move beyond the immediate subsurface layers.

Data from the Pesticides in Ground Water Database indicate that for the number of wells sampled and limited numbers that have been analyzed for rotenone and related compounds there have been no detections (Barbash and Resek, 1996). Barbash indicates in Table 3.2 page 169 that there have been 12 random well samples analyzed for rotenone with no detections. Additionally, four (4) wells were analyzed for rotenolone and eight (8) wells for "other rotenone metabolites" with no detections in any of the wells. (See also section 7.5)

6. Environmental Effects Assessment

6.1 Objectives

The objective of this section is to present an overview of what ecological toxicity data are available and to present relevant data. Subject areas to be emphasized are those related to the piscicidal uses of rotenone. All higher taxa (e.g., birds, insects) will be addressed, but it is not the intent to be comprehensive for species only marginally related to piscicidal uses.

6.2 Sources of Information

One primary source of information is the U.S. EPA, which has developed a number of documents related to the re-registration of rotenone. The Environmental Fate and Effects Division's final chapter (USEPA, 2006c) has included data from a number of studies not available in the open literature. This chapter has also included some data from the open literature, but it does not attempt to be exhaustive in this area. Not all acute fish toxicity data reported to EPA are included in the EFED chapter. However, the endpoint and some additional supporting data are included in EFED's "one-liner" data base, available on-line at <http://www.ipmcenters.org/Ecotox/index.cfm>. All data have been validated by EPA, and all aquatic data not classified as "invalid" are included below, whether or not they are relied upon to support registration.

Rotenone has been used as a piscicide for over 60 years. Much of the older literature is not available or at least not readily available. A variety of documents have reviewed this literature. Bradbury (1986) has a particularly good summary, especially for fish toxicity, and data have been extracted from this publication.

The EPA's ECOTOX data base (on-line at <http://cfpub.epa.gov/ecotox/>, accessed April and May 2007) contains a substantial amount of fish toxicity data. Most of the 96-hour LC₅₀ rotenone data in this database were extracted if they were for fish native to or introduced into the United States. In a few situations, such as where an investigator conducted numerous tests with somewhat varying conditions, only representative data were extracted or data are lumped into one row.

6.3 Aquatic Toxicity information

6.3.1 Microbes

There are no data available to assess the acute or chronic toxicity of rotenone to microbes.

6.3.2 Algae

There are no standard laboratory data available to assess the acute or chronic toxicity of rotenone to algae. Bradbury (1986) cites anecdotal data indicating that "phytoplankton" increased in 6 of 9 lakes following rotenone treatment. He could not find a consistent reason, but suggested that loss of zooplankton feeding on the phytoplankton or an increase in phosphorus released from dead fish were two possible causes.

6.3.3 Aquatic macrophytes

There are no data available to assess the acute or chronic toxicity of rotenone to aquatic macrophytes.

6.3.4 Aquatic invertebrates

Standardized toxicity test data on aquatic invertebrates is quite limited. Most invertebrate data have been from field observations following rotenone treatments. These data largely relate to changes in populations and/or diversity after rotenone applications. Bradbury (1986) provides

substantial data on benthic and zooplanktonic invertebrates by species, and on zooplankton communities. The conclusions from various studies indicate that zooplankton, especially cladocerans and copepods, are that the most severely affected group of aquatic invertebrates, with many studies reporting 95-100% loss. “Complete recovery” required 17 weeks to 4 years in various studies.

6.3.4.1 Acute

USEPA (2006c) reported a 48-hour EC₅₀ for technical grade rotenone (96.5%) of 3.7 µg a.i./L for the water flea (*Daphnia magna*). Some additional data are listed in Table 6.2. A comparative study by Waller et al. (1993) indicated that zebra mussels were 20-30x less sensitive than fish (rainbow trout and channel catfish) and about 5x more sensitive than a native unionid mussel. There is insufficient information on other invertebrate tests to make comparisons, but they do indicate that cladocerans are typically the most sensitive of tested invertebrates..

The available acute data on the toxicity of rotenone to estuarine or marine invertebrates are from a variety of tests that do not meet EPA requirements, and which were therefore not used in the EFED chapter (USEPA, 2006c). However, the EFED one-liner database includes an LC₅₀ of 220 µg/L for eastern oysters (*Crassostrea virginica*) and an LC₅₀ greater than 1000 µg/L for brown shrimp (*Penaeus aztecus*).

6.3.4.2 Chronic

USEPA (2006c) reported a 21-day chronic toxicity No Observable Adverse Effect Concentration (NOAEC) of 1.25 µg a.i./L for technical grade rotenone (96.5%) for the water flea (*Daphnia magna*). The LOAEC was 2.5 µg a.i./L. No additional chronic invertebrate data were located.

6.3.5 Amphibians (aquatic stages)

Rotenone data on aquatic stages of amphibians is limited. One test with larval Southern leopard frogs (*Rana sphenoccephala*) is in the EPA one-liner data base (Table 6.2). The 96-hour LC₅₀ was 500 µg/L for a 5% EC formulation. No data were found in the ECOTOX database. In a study cited by Bradbury (1986), there was 100% mortality of leopard frog (*Rana pipiens*) tadpoles and metamorphosed tiger salamanders (*Ambystoma tigrinum*) after exposure of 8-24 hours in 100 µg/L rotenone; gilled stages of the salamander were affected, but not necessarily killed at 17 µg/L. The test material was the 5% EC formulation, however, it is not known if the results are based upon active ingredient or the formulation.

6.3.6 Fish

Toxicity data on fish are generally available for pesticides. Among freshwater fish, data to support pesticide registration are usually done for both cold water and warm water fish. Standardized protocols for developing fish toxicity data have been available for several decades, and as a result, comparisons can be made among species, chemicals, and aquatic characteristics (e.g., temperature, hardness). The rainbow trout, *Oncorhynchus mykiss*, and the bluegill sunfish, *Lepomis macrochirus*, are EPA’s preferred species for acute toxicity tests because they are sensitive indicator species. A large set of data exists particularly for these species. For subchronic and chronic toxicity tests, there is also a moderate amount of data for fathead minnows, as well as rainbow trout and bluegill.

Although there are data available for freshwater fish, there are very few acute and no chronic data on the toxicity of rotenone to estuarine or marine fish; none were noted in the EFED chapter (USEPA, 2006c). The EFED one-liner database includes a 48-hour LC₅₀ for the longnose killifish (*Fundulus similis*) of 36 µg/L. The ECOTOX database includes several short term LT₅₀ studies where the time to 50% mortality at a single dose was determined. Although these cannot be considered comparable in any way, the two-spotted goby was found to reach 50% mortality to concentrations of 0.5 to 5 mg/L of a synergized rotenone formulation after 5-36 hours; however the dose-response was very erratic.

6.3.6.1 Acute

Data presented below in Table 6.1 are all of the acute fish toxicity data included in USEPA, 2006EFED. A number of additional acute toxicity studies using various fish species are also available from the EFED one-liner database, and the open literature, primarily as obtained from ECOTOX. These data are included in a separate Table 6.2. However it should be noted that no valid fish toxicity values obtained from the open literature were lower than the registrant submitted 96-h LC₅₀ (1.94 µg a.i./L).

Species	% a.i.	96 h LC ₅₀ µg a.i./L ¹	Toxicity Category	MRID/Accession No.
Bluegill	98	4.9	Very highly toxic	439751-01
Bluegill	98.6	5.5	Very highly toxic	443829-02
Bluegill	2.5	138	Highly toxic	89909
Bluegill	5	157	Highly toxic	90425
Bluegill	5	56	Very highly toxic	121874
Bluegill	5	90	Very highly toxic	121877
Bluegill	5	72	Very highly toxic	121880
Bluegill	5	105	Highly toxic	121881
Bluegill	5	157	Highly toxic	121883
Bluegill	5	165	Highly toxic	121885
Bluegill	5	127	Highly toxic	90288
Bluegill	7.65	80	Very highly toxic	90366
Rainbow Trout	5	46.5	Very highly toxic	89905
Rainbow Trout	5	38.5	Very highly toxic	89906
Rainbow Trout	5	29	Very highly toxic	89908
Rainbow Trout	5	22	Very highly toxic	90367
Rainbow Trout	5	35	Very highly toxic	90420
Rainbow Trout	5	35	Very highly toxic	90421
Rainbow Trout	5	48	Very highly toxic	121882
Rainbow Trout	5	52	Very highly toxic	121884
Rainbow Trout	5	38	Very highly toxic	121886
Rainbow Trout	98	1.9	Very highly toxic	439751-02
Rainbow Trout	98.6	2.8	Very highly toxic	443829-01
Rainbow Trout	5	35	Very highly toxic	89907

Rainbow Trout	5	11.5	Very highly toxic	121783
Rainbow Trout	5	1.8	Very highly toxic	121875
Rainbow Trout	5	0.84	Very highly toxic	121876
Rainbow Trout	96.47	2.82	Very highly toxic	400633-01
Rainbow Trout	5	45	Very highly toxic	400633-01
Rainbow Trout	2.5	36.2	Very highly toxic	400633-01
Rainbow Trout	6.8	45	Very highly toxic	89904

¹ The EFED table containing these data states that these endpoint results were all based upon the amount of active ingredient. However, Brian Montague, who manages the EFED one-liner database stated that the instructions for entering data into the one-liner database are for toxicity values to be entered for the test material; they are not corrected for the percent active ingredient. (Brian Montague, EFED/EPA, email communication, May 17, 2007)

Table 6.2. Fish and aquatic invertebrate data not included in USEPA, 2006EFED.					
Species	Age/ Size	Test Type/ duration	Test material/ % Active ingredient	Toxicity value (LC₅₀)¹	Reference
Amphibians					
Southern leopard frog (<i>Rana sphenoccephala</i>)	larvae	S 96 hr	EC 5%	500 µg/L	EPA one-liners 10211 Chandler
Leopard frog (<i>Rana pipiens</i>)	adult	NR 96 hr	NR	3.2 mg/L	Farringer, 1972 in Bradbury, 1986
Leopard frog (<i>Rana pipiens</i>)	adult	NR 96 hr	NR	4.6 mg/L	Farringer, 1972 in Bradbury, 1986
Toad (<i>Bufo bufo japonicus</i>)	NR	NR 48 hr	tech	330 µg/L	ECOTOX 5761 Hashimoto
Toad (<i>Bufo vulgaris formosus</i>) 3 tests	NR	NR 24 hr	NR	52-92 µg/L	ECOTOX 6701 Nishiuchi
African clawed frog (<i>Xenopus laevis</i>)	NR	NR 96 hr	NR	>40 µg/L	ECOTOX 12665 Holcombe
Fish					
American Eel (<i>Anguilla rostrata</i>)	97 mm	NR 96 hr	EC 5%	50.49 µg/L	ECOTOX 592 Hinton
American Eel (<i>Anguilla rostrata</i>)	55 mm	NR 96 hr	EC 5%	15.25 µg/L	ECOTOX 593 Hinton
Atlantic salmon (<i>Salmo salar</i>)	1-1.5g	S 96 hr	EC 5%	21.5 µg/L	Marking & Bills, 1976
Black bullhead (<i>Ameiurus melas</i>)	1-1.5g	S 96 hr	EC 5%	389 µg/L	Marking & Bills, 1976
Black bullhead (<i>Ictalurus melas</i>)	NR	S 96 hr	NR 5%	260 µg/L	EPA one-liners
Black bullhead (<i>Ictalurus melas</i>)	NR	S 96 hr	WP 20%	173 µg/L	EPA one-liners
Bluegill (<i>Lepomis macrochirus</i>)	1-1.5g	S 96hr	EC 5%	141 µg/L	Marking & Bills, 1976

Species	Age/ Size	Test Type/ duration	Test material/ % Active ingredient	Toxicity value (LC₅₀)¹	Reference
Bluegill (<i>Lepomis macrochirus</i>) 10 tests ²	1-1.5g	S 96hr	EC 5%	122-141 µg/L	Marking & Bills, 1976
Bowfin (<i>Amia calva</i>)	1-1.5g	S 96 hr	EC 5%	30 µg/L	Marking & Bills, 1976
Brook trout (<i>Salvelinus fontinalis</i>)	fingerling	S 96 hr	EC 5%	47.0 µg/L	ECOTOX 525 Olson
Brook trout (<i>Salvelinus fontinalis</i>)	1-1.5 g	S 96 hr	EC 5%	44.3 µg/L	Marking & Bills, 1976
Brook trout (<i>Salvelinus fontinalis</i>)	eggs (green)	S 96 hr	EC 5%	3.4 µg/L	ECOTOX 525 Olson
Carp (<i>Cyprinus carpio</i>)	NR	S 96 hr	WP 20%	243 µg/L	EPA one-liners
Carp (<i>Cyprinus carpio</i>)	1-1.5g	S 96 hr	EC 5%	19 µg/L	Marking & Bills, 1976
Carp (<i>Cyprinus carpio</i>)	NR	S 96 hr	WP 20%	70 µg/L	EPA one-liners
Carp (<i>Cyprinus carpio</i>)	1-1.5g	S 96 hr	EC 5%	50 µg/L	Marking & Bills, 1976
Carp (<i>Cyprinus carpio</i>)	1-1.5g	F 96 hr	EC 5%	142 µg/L	Marking & Bills, 1976
Carp (<i>Cyprinus carpio</i>)	1-1.5g	F 30 days	EC 5%	68 µg/L	Marking & Bills, 1976
Carp (<i>Hypophthalmichthys nobilis</i>)	1-1.5	S 96 hr	EC 5%	43.7 µg/L	Marking & Bills, 1976
Channel catfish (<i>Ictalurus punctatus</i>)	0.5g	96 hr	“tech”	2.8 µg/L	Mayer & Ellersieck (1986)
Channel catfish (<i>Ictalurus punctatus</i>)	NR	S 96 hr	WP 20%	80 µg/L	EPA one-liners
Channel catfish (<i>Ictalurus punctatus</i>)	NR	96 hr	NR 5%	470 µg/L	ECOTOX 934 Clemens
Channel catfish (<i>Ictalurus punctatus</i>)	1-1.5g	S 96 hr	EC 5%	164 µg/L	Marking & Bills, 1976
Channel catfish (<i>Ictalurus punctatus</i>)	1-1.5g	96 hr	WP 4.85%	28 µg/L	ECOTOX 8048 Bridges
Channel catfish (<i>Ictalurus punctatus</i>)	0.5g	S 96 hr	NR 44%	2.6 µg/L	EPA one-liners
Channel catfish (<i>Ictalurus punctatus</i>)	NR	S 96 hr	NR 5%	115 µg/L	EPA one-liners
Channel catfish (<i>Ictalurus punctatus</i>) 10 tests ¹	NR	S 96 hr	EC 5%	164-328 µg/L	Marking & Bills, 1976
Chinook salmon (<i>Oncorhynchus tshawytscha</i>)	1-1.5g	S 96 hr	EC 5%	36.9 µg/L	Marking & Bills, 1976

Species	Age/ Size	Test Type/ duration	Test material/ % Active ingredient	Toxicity value (LC₅₀)¹	Reference
Chinook salmon (<i>Oncorhynchus tshawytscha</i>)	1-1.5g	S 96 hr	EC 5%	34.7 µg/L	Marking & Bills, 1976
Chinook salmon (<i>Oncorhynchus tshawytscha</i>)	1-1.5g	F 96 hr	EC 5%	71 µg/L	Marking & Bills, 1976
Chinook salmon (<i>Oncorhynchus tshawytscha</i>)	1-1.5g	F 20 days	EC 5%	59 µg/L	Marking & Bills, 1976
Coho salmon (<i>Oncorhynchus kisutch</i>)	1-1.5g	S 96 hr	EC 5%	62 µg/L	Marking & Bills, 1976
Fathead minnow (<i>Pimephales promelas</i>)	35-40d	F 96 hr	Tech 97%	6 µg/L	ECOTOX 3217 Geiger
Fathead minnow (<i>Pimephales promelas</i>)	0.09g	S 96 hr	Tech 97%	4 µg/L	ECOTOX 12858 Geiger
Fathead minnow (<i>Pimephales promelas</i>)	0.2g	S 96 hr	NR	6 µg/L	ECOTOX 12665 Holcombe
Fathead minnow (<i>Pimephales promelas</i>)	NR	S 96 hr	NR	3.4 µg/L	Gilderhus, 1982
Fathead minnow (<i>Pimephales promelas</i>)	1-1.5g	S 96 hr	EC 5%	16.9 µg/L	Marking & Bills, 1976
Goldfish (<i>Carrasius auratus</i>)	1-1.5g	S 96 hr	EC 5%	497 µg/L	Marking & Bills, 1976
Grass carp, white amur (<i>Ctenopharyngodon idella</i>)	1-1.5	S 96 hr	EC 5%	63 µg/L	Marking & Bills, 1976
Green sunfish (<i>Lepomis cyanella</i>)	NR	S 96 hr	NR 5%	74 µg/L	EPA one-liners
Green sunfish (<i>Lepomis cyanella</i>)	1-1.5g	S 96 hr	EC 5%	141 µg/L	Marking & Bills, 1976
Green sunfish (<i>Lepomis cyanella</i>)	NR	S 96 hr	WP 20%	58 µg/L	EPA one-liners
Green sunfish (<i>Lepomis cyanella</i>) 7 tests ³	NR	S 96 hr	EC 5%	158-378 µg/L	Marking & Bills, 1976
Lake trout (<i>Salvelinus namaycush</i>)	Eggs	S 96 hr	NR	>1000 µg/L	ECOTOX 525 Olson
Lake trout (<i>Salvelinus namaycush</i>)	1-1.5g	S 96 hr	EC 5%	26.9 µg/L	Marking & Bills, 1976
Largemouth bass (<i>Micropterus salmoides</i>)	1-1.5g	S 96 hr	EC 5%	142 µg/L	Marking & Bills, 1976
Longnose killifish (<i>Fundulus similis</i>)	Juv	F 96 hr	NR 5%	36 µg/L	EPA one-liners
Longnose sucker (<i>Catostomus catostomus</i>)	1-1.5g	S 96 hr	EC 5%	57 µg/L	Marking & Bills, 1976
Mozambique tilapia (<i>Tilapia mossambica</i>)	36g	S 96 hr	Tech 95- 98%	80 µg/L	ECOTOX 18762 Cruz-Lacierda

Species	Age/ Size	Test Type/ duration	Test material/ % Active ingredient	Toxicity value (LC₅₀)¹	Reference
Northern Pike (<i>Esox lucius</i>)	1-1.5	S 96 hr	EC 5%	33 µg/L	Marking & Bills, 1976
Rainbow trout (<i>Oncorhynchus mykiss</i>)	1-1.5g	S 96 hr	EC 5%	46 µg/L	Marking & Bills, 1976
Rainbow trout (<i>Oncorhynchus mykiss</i>)	1-1.5g	S 96 hr	Synergized EC 2.5%	1.02 µg/L	Marking & Bills, 1976
Rainbow trout (<i>Oncorhynchus mykiss</i>)	1-1.5g	S 96 hr	EC 5%	3.05 µg/L	Marking & Bills, 1976
Rainbow trout (<i>Oncorhynchus mykiss</i>)	1-1.5g	S 96 hr	Powder 33%	3.2 µg/L	Marking & Bills, 1976
Rainbow trout (<i>Oncorhynchus mykiss</i>) 10 tests ¹	1-1.5g	S 96 hr	EC 5%	43.4-70 µg/L	Marking & Bills, 1976
Silver carp (<i>Hypophthalmichthys molitrix</i>)	1-1.5g	S 96 hr	EC 5%	55.8 µg/L	Marking & Bills, 1976
Smallmouth bass (<i>Micropterus dolomieu</i>)	1-1.5g	S 96 hr	EC 5%	79 µg/L	Marking & Bills, 1976
Walleye (<i>Stizostedion vitreum vitreum</i>)	1-1.5g	S 72 hr	EC 5%	16.5 µg/L (72 hr)	Marking & Bills, 1976
Western mosquitofish (<i>Gambusia affinis</i>)	NR	NR 96 hr	NR	17 µg/L	ECOTOX 9036 Fabacher
Western mosquitofish (<i>Gambusia affinis</i>)	NR	NR 96 hr	NR	31 µg/L	ECOTOX 9036 Fabacher
White sucker (<i>Catostomus commersoni</i>)	1-1.5g	S 96 hr	EC 5%	68 µg/L	Marking & Bills, 1976
White sucker (<i>Catostomus commersoni</i>)	NR	S 96 hr	NR 5%	48 µg/L	EPA one-liners
White sucker (<i>Catostomus commersoni</i>)	NR	S 96 hr	WP 20%	47 µg/L	EPA one-liners
White sucker (<i>Catostomus commersoni</i>)	4.1g	NR 96 hr	NR	11 µg/L	ECOTOX 12665 Holcombe
White sucker (<i>Catostomus commersoni</i>)	1-1.5g	S 96 hr	EC 5%	17.9 µg/L	Marking & Bills, 1976
White sucker (<i>Catostomus commersoni</i>)	1-1.5g	F 96 hr	EC 5%	144 µg/L	Marking & Bills, 1976
White sucker (<i>Catostomus commersoni</i>)	1-1.5g	F 30 days	EC 5%	112 µg/L	Marking & Bills, 1976
Yellow perch (<i>Perca flavescens</i>)	1-1.5g	S 96 hr	EC 5%	30 µg/L	Marking & Bills, 1976
Yellow perch (<i>Perca flavescens</i>)	1-1.5g	S 96 hr	EC 5%	70 µg/L	Marking & Bills, 1976
Yellow perch (<i>Perca flavescens</i>)	1-1.5g	F 96 hr	EC 5%	60 µg/L	Marking & Bills, 1976

Table 6.2. Fish and aquatic invertebrate data not included in USEPA, 2006EFED.					
Species	Age/ Size	Test Type/ duration	Test material/ % Active ingredient	Toxicity value (LC₅₀)¹	Reference
Yellow perch (<i>Perca flavescens</i>)	1-1.5g	F 20 days	EC 5%	46 µg/L	Marking & Bills, 1976
Aquatic Arthropods					
Seed shrimp (<i>Cyridopsis</i> sp.)	NR	S 96	EC 5 %	340 µg/L	EPA one-liners
Whirligig beetle (<i>Gyrinus</i> sp.)	NR	S 96	EC 5 %	700 µg/L	EPA one-liners
Water flea (<i>Daphnia pulex</i>)	1 st instar	S 48 hr	NR 44%	100 µg/L	EPA one-liners
Daphnid (<i>Simocephalus serrulatus</i>)	1 st instar	S 48 hr	NR 44%	310 µg/L	EPA one-liners
Scud (<i>Gammarus fasciatus</i>)	juv	S 96 hr	NR 44 %	2600 µg/L	EPA one-liners
Scud (<i>Gammarus lacustris</i>)	NR	96 hr	Tech	2600 µg/L	Sanders, 1970
Scud (<i>Gammarus lacustris</i>)	NR	96 hr	EC 5%	3520 µg/L	ECOTOX 2094 Nebeker
Brown shrimp (<i>Penaeus aztecus</i>)	juv	F 48 hr	EC 5 %	>1000 µg/L	EPA one-liners
Water flea (<i>Daphnia magna</i>)	< 24 hr	S 24 hr	NR 5 %	27.5 µg/L	EPA one-liners
Glass shrimp (<i>Palaemonetes kadiakensis</i>)	NR	S 96 hr	EC 5 %	1120 µg/L	EPA one-liners
Dragonfly (<i>Macromia</i> sp.)	Larvae	S 96 hr	EC 5 %	1000 µg/L	EPA one-liners
Backswimmer (<i>Notonecta</i> sp.)	NR	S 96 hr	EC 5 %	1580 µg/L	EPA one-liners
Caddisfly (<i>Hydropsyche</i> sp.)	NR	S 96 hr	EC 5 %	605 µg/L	EPA one-liners
White River crayfish (<i>Procambarus acutus acutus</i>) 2 tests	60-66 mm total length	96 hr	EC 5%	3000-4000 µg/L	ECOTOX 11432 Wujtewicz
Molluscs					
Asiatic clam (<i>Corbicula manilensis</i>)	NR	S 96 hr	EC 5 %	7500 µg/L	EPA one-liners
Freshwater clam (<i>Elliptio complanata</i>)	NR	S 96 hr	EC 5 %	200 µg/L	EPA one-liners
Buckley's filter clam (<i>Elliptio buckleyi</i>)	NR	S 96 hr	EC 5 %	2950 µg/L	EPA one-liners
Zebra mussel (<i>Dreissena polymorpha</i>)	5-8 mm	S 48 hr	EC 5%	165 µg a.i./L	Waller et al., 1993
Zebra mussel (<i>Dreissena polymorpha</i>)	20-25 mm	S 48 hr	EC 5%	219 µg a.i./L	Waller et al., 1993

Species	Age/ Size	Test Type/ duration	Test material/ % Active ingredient	Toxicity value (LC₅₀)¹	Reference
Threehorn wartyback (<i>Obliquaria reflexa</i>)	20-45 mm	S 48 hr	EC 5%	> 1 mg a.i./L	Waller et al., 1993
Snail (<i>Helisoma</i> sp.)	NR	S 96 hr	EC 5 %	7950 µg/L	EPA one-liners
Snail (<i>Physa pomilia</i>)	NR	S 96 hr	EC 5 %	4000 µg/L	EPA one-liners
Snail (<i>Oxytrema catenaria</i>)	NR	S 96 hr	EC 5 %	1750 µg/L	EPA one-liners
Eastern oyster (<i>Crassostrea virginica</i>)	juv	F 96 hr	EC 5 %	220 µg/L	EPA one-liners
Other invertebrates					
Flatworm (<i>Catenula</i> sp.)	NR	S 96 hr	EC 5%	1720 µg/L	EPA one-liners

¹ All results are based upon test material, except those that specifically state µg a.i./L

² Marking and Bills (1976) tested rainbow trout, catfish, and bluegill at various temperatures (12-22° except rainbow 7-17°), water hardnesses (very soft to very hard), and pHs (6.5-9.5). Although a few “significant” differences were found a result of varying these parameters, none were more than two-fold different which is within the range of expected variation among multiple tests with the same fish.

³ Marking and Bills (1976) tested green sunfish at different pHs from 6.5-9.5. There were no consistent differences at various pH values.

Caution should be used in making comparisons of these fish toxicity data. Test conditions and size of fish varied among the different reports; these can contribute to differences on 2-5x or more between different testing laboratories. Marking and Bills (1976) did provide a comprehensive set of data using the same conditions and size of fish, or by varying one or two parameters in specific ways; these data can be used to assess comparative sensitivity. They found that pH, temperature, and hardness may play a small role in sensitivity. Some of the differences were statistically significant, but the magnitude of variation was rather small for these conditions. They did determine that salmonids are the most sensitive taxon tested, with a few other species, such as the bowfin and northern pike, also being sensitive. Ictalurids and goldfish were the least sensitive species tested. Centrarchids had more variable sensitivity, but were generally intermediate between the salmonids and ictalurids.

Formulation testing by Marking and Bills (1976) showed that the synergized rotenone formulation was about 3 times more toxic to rainbow trout for a material with half of the rotenone percent active ingredient. Although the data are limited, they suggest that the synergized formulation they tested was about 6 times more toxic than the rotenone formulation without piperonyl butoxide. Marking and Bills (1976) also found that fish in static tests showed consistently lower LC₅₀ values than those in flow-through tests. This suggests that degradation products, which would be carried away in flow-through tests, may be contributing to the toxicity.

Marking and Bills (1976) made one additional observation of importance. The fish toxicity data are based upon median lethal levels. But control of fish populations requires close to 100% mortality. For fish species with significant genetic variability, there would be a greater difference between the median lethal levels and the levels needed for complete control, than there would be for fish that are largely

homogenetic. The slope of the concentration-response line can provide good information on doses necessary to achieve complete control, but the slope is seldom published. Confidence limits can be used, with less accuracy, to indicate likely genetic variability of a population. It should be noted that 100% control may not be feasible, except with excessive concentrations, because there are often one or several individuals in a population that are resistant.

6.3.6.2 Chronic

There is one available standard study on chronic toxicity to fish. A 32-day early life stage test with rainbow trout resulted in a NOAEC of 1.01 µg/L. At the LOAEC of 2.2 µg/L, there was a 20% reduction in growth of the young fish.

Marking and Bills (1976) conducted a number of tests on rotenone, including a few lethality tests that had exposures for 20 or 30 days under flow-through conditions. The 20-day LC₅₀s for yellow perch and chinook salmon were 46 µg/L and 59 µg/L, respectively. The 30-day LC₅₀s for common carp and white sucker were 68 µg/L and 112 µg/L, respectively.

6.3.7 Sediment organisms

The EFED analysis for rotenone (USEPA, 2006c) did not include any data to assess the acute or chronic toxicity of rotenone to sediment-dwelling organisms. Several of the aquatic arthropods and all of the mollusks in Table 6.2, from the ECOTOX data base, are benthic organisms, but it is unclear whether they were tested with sediments included in the test system.

Other studies have looked at an assemblage of benthic organisms, typically field observations following rotenone treatments. Most of these look at overall abundance of organisms without regard to species; a few look at species diversity, as well. Bradbury (1986) summarized the results of 13 studies on 23 lakes and ponds. Techniques used in these studies were quite variable. From these data, he concluded that the effect on benthic organisms varies, but is generally less than effects on zooplankton in the water column. Immediate reductions in total numbers of benthic organisms ranged from 0% to 71%, with a mean value of 25%. He did note that, in other, non-quantitative studies, several authors reported drastic reductions in certain or all benthic organisms. For 11 studies that monitored benthic populations over a several month or longer period, recovery in terms of abundance was often 1-2 months; in several studies the abundance of benthic organisms increased substantially relative to pre-treatment levels. It is hard to draw any conclusions from these studies because of the considerable variation in both the specific environments treated and the techniques used to monitor the benthos. It does appear that rotenone does not have strong effects where bottom sediments and plant material are abundant, and that in most situations, effects on sediment-dwelling invertebrates are not long-lasting.

Most applications of rotenone will cause significant declines in zooplankton, and some benthic invertebrates (Ling, 2003). Among benthic invertebrates, certain amphipods are very sensitive (USEPA, 2006c).

6.4 Terrestrial Toxicity Data

6.4.1 Mammals

Generally, pesticide toxicity data on mammals are obtained from health effects studies on laboratory mammals often developed for pesticide registration. No data were found on wild or wild-type mammals. The following is a brief presentation of the mammalian toxicity data. Because females were more sensitive, only data on females are included below; see section 9.2 for more details, including results for males.

6.4.1.1 Acute

The mammalian LD₅₀ for laboratory rats is 39.5 mg/Kg for females and 102 mg/Kg for males, using the technical grade of rotenone. Formulated product toxicity is similar when adjusted for the amount of active ingredient and considering the presence of synergists or cube resins or both in these formulated products.

6.4.1.2 Chronic

With respect to ecological effects, the relevant mammalian chronic data indicate that long term exposure to rotenone may affect female rats at oral exposures of 2.4 mg/kg/day, (estimated 48 ppm in the diet) with a no-observed-adverse-effect-level (NOAEL) of 0.5 mg/kg/day (estimated 10 ppm in the diet). The effect was on body weight. No mortality or clinical signs of toxicity were observed even at the highest does of 3.75 mg/kg/day (75 ppm in the diet).

6.4.2 Birds

Avian toxicity data are typically available on northern bobwhite, *Colinus virginianus*, mallard duck, *Anas platyrhynchos*, ring-necked pheasant, *Phasianus colchicus*, or Japanese quail, *Coturnix japonica*, for acute data, and on the bobwhite and mallard for reproductive toxicity testing. EPA uses these data to assess effects not only to birds, but also to reptiles and terrestrial stage amphibians. All avian data are from USEPA (2006c) unless otherwise indicated.

6.4.2.1 Acute

Avian acute oral toxicity data are available for the mallard duck and ring-necked pheasant (Table 6.3). In these studies only female birds were tested. The LD₅₀ values for the mallard duck and ring-necked pheasant, based on formulated product (34.5% a.i. rotenone), were 2200 mg/kg and 1680 mg/kg, respectively (MRID 143250). Regurgitation occurred at concentrations above 1500 mg/kg. Based on these data, rotenone is classified as slightly toxic to birds and the taxa for which they serve as surrogates (reptiles and terrestrial phase amphibians) on an acute oral exposure basis.

Subacute dietary toxicity studies on formulated product (34.5% rotenone) have been conducted using ring-necked pheasants (*Phasianus colchicus*), Japanese quail (*Coturnix japonica*), and mallard ducks (*Anas platyrhynchos*). Toxicity (LC₅₀) values for 5-day subacute dietary toxicity studies in the three species are 1608, 1882, and 2600 ppm, respectively. Based on the most sensitive species tested, i.e., ring-necked pheasants, rotenone is classified as slightly toxic to birds on a subacute dietary exposure basis.

6.4.2.2 Chronic

No chronic toxicity data were available to assess the chronic effects of rotenone on birds.

6.4.3 Reptiles

No acute or chronic toxicity data were available to assess the chronic effects of rotenone on reptiles. EPA bases their assessment of reptile toxicity on avian data. One report was found where dead turtles, *Kinosternon subrubum*, were seen following rotenone treatment to a shallow reservoir. No other turtle or amphibian mortalities were observed (McCoid and Bettoli, 1996)

6.4.4 Amphibians (adult/terrestrial stages)

No data could be found to address amphibians exposed out of the water. Terrestrial exposure to such organisms would be very limited, if any, and would result from drift from applications made over water. The slurry system typically used by WFDW will minimize drift and thus terrestrial exposure.

A limited amount of data was found on adult stages of frogs exposed in water. Haag (1931) tested adult *Rana pipiens* by dosing with water covering all but the tops of the frogs. He found that 2 mg/L was the minimum lethal dose for this route of exposure and calculated that this concentration was equivalent to 4.0 mg/Kg of body weight. Frogs tested at 1 mg/L survived. Farringer determined that 96-hr LC₅₀ values for adult leopard frogs, *Rana pipiens*, were 3.2 mg/L in hard water and 4.2 mg/L in soft water (Bradbury, 1986). No information could be found on the test compound, Dri-noxfish. It apparently is no longer registered; the name suggests a powdered, piscicidal formulation of rotenone.

6.4.5 Insects

The purpose of the honeybee contact toxicity study was originally to determine the potential effects on pollinators visiting plants that had been sprayed with pesticides. The contact exposure of the study renders it less useful for evaluating other types of exposure, such as to sprays or films of pesticide, and the endpoint of micrograms/bee is not readily comparable to other expressions of toxicity. Nevertheless, there is a substantial body of comparative data on this type of study, and EPA uses it to represent all terrestrial insects.

The acute contact study in honey bees (*Apis mellifera*) using technical grade rotenone (95% a.i) yielded LD₅₀ value of greater than 60 µg a.i./bee (MRID 05001991). Based on these results, rotenone is classified as practically non-toxic to honey bees on an acute contact basis. An additional oral toxicity study has also been conducted, with the LD₅₀ found to be greater than 30 µg a.i./bee (MRID 05001991). This type of study is unusual and there are no ways to classify the toxicity of rotenone to insects on a comparative basis.

Table 6.3 Rotenone Toxicity to Terrestrial Animals			
Species	% a.i.	Toxicity Value	MRID/Accession No.
Avian Acute Toxicity-Oral			
Mallard duck	32.4	LD ₅₀ =2200 mg/kg	MRID 143250
Ring-neck pheasant	32.4	LD ₅₀ =1680 mg/kg	MRID 143250
Avian Sub-Acute Dietary			

Table 6.3 Rotenone Toxicity to Terrestrial Animals			
Species	% a.i.	Toxicity Value	MRID/Accession No.
Mallard duck	34.5	LC ₅₀ =2600 ppm	248788 (Hill et al., 1975)
Ring-neck pheasant	34.5	LC ₅₀ =1608 ppm	248788 (Hill et al., 1975)
Japanese quail	34.5	LC ₅₀ =1882 ppm	248788 (Hill et al., 1975)
Honeybee Acute Toxicity			
Honeybee	>95	LD ₅₀ =>60 µg a.i./bee	05001991 (Stevenson, JH 1978)
Honeybee	Technical	2.4 µg a.i./bee elicited 12% mortality	00036935 (Atkins et al., 1975)

6.4.6 Plants

There are no phytotoxicity data available to address terrestrial plants. A lack of phytotoxicity may be inferred from the wide variety of crops on which rotenone was formerly registered and a long history of use without any apparent incidents.

7. Ecological Exposure Assessment

7.1 Routes of exposure

The routes of exposure considered in this assessment are those related only to the piscicidal uses of rotenone that result from direct application to water. Non-piscicidal uses are not a consideration for this analysis, and are now no longer registered.

Rotenone applications may be made by aerial or ground methods. Aerial applications of only the emulsifiable concentrate products may be either by helicopter or fixed-wing aircraft, but WDFW typically uses helicopters. The most common method of ground application is by boat. WDFW uses the aspirator system developed by the Utah Division of Wildlife Resources in the 1990s. Please refer to pp. 99-101 in the AFS Rotenone Use Manual (Finlayson et al. 2000) for the description of this methodology. WDFW may use any of the methodologies described in Chapter 3 of this manual, or as otherwise allowed by current labels.

Labels suggest subsurface application by boat to allow the wash of the motor to aid in mixing, or application by a boom sprayer low over the water. Backpack or ATV-mounted sprayers are often used in areas not accessible by boat. Drip or other injection methods are used especially in lotic waters. When applied according to label directions, the ground applications should not result in off-site exposure. Aerial applications are normally done by applicators contracted by WDFW. Best management practices suggest that the application of as large a droplet size as possible will minimize drift. Some exposure of the terrestrial edges of treated waters may occur with aerial applications.

7.1.1 Aquatic plants and algae

Applications are made directly to water. Thus, the primary route of exposure for aquatic plants and algae would be through rotenone concentrations in the water. For applications made above the water, exposure to emergent plants could result from direct application or drift. Based upon the K_d (the soil-water adsorption coefficient) of rotenone, it would be expected that rotenone would adsorb not only to soils and sediments, but also to algae and plants in the water. While data are lacking to support any definitive conclusions, the likelihood that rotenone would penetrate into plant cells and tissues seems low, except that it may be possible for root uptake from sediments where rotenone has adsorbed to particulate matter. There are no data, even for terrestrial plants, to assess the potential uptake of rotenone through roots. Gilderhus (1982) found that toxicity of rotenone to fish was reduced by the presence of bentonite clay particles or by the presence of *Elodea canadensis*, a rooted aquatic macrophyte. The reductions seem most likely to be from reduced bioavailability resulting from adsorption.

7.1.2 Fish and other aquatic vertebrates; aquatic invertebrates

Applications are made directly to water. As with algae and aquatic plants, the primary route of exposure would be from rotenone in the water column, or for benthic organisms, from rotenone adsorbed in sediments. Fish, mollusks, and aquatic arthropods would take up rotenone fairly easily through their gills. Some dermal or oral uptake could also occur, but would be considerably less than would occur through the gills. The same routes would apply to gilled larval or neotenic stages of amphibians. Concentrations to which fish and water column invertebrates would be exposed would be determined by the objectives of the treatment project; models to predict exposure are unnecessary.

Other vertebrates that may occur in or on the water would be exposed to rotenone either through dermal uptake, which is expected to be low (USEPA, 2006a), or more likely, through ingestion of treated water or food items with rotenone residues. Samples of fish killed in Wisconsin field studies following a 250 ppb treatment had rotenone residues ranging from 171-329 ppb in edible portions of the fish and 392-696 ppb in the inedible parts (USEPA, 2006c)

7.1.3 Terrestrial organisms

Terrestrial organisms that would be exposed to rotenone are those that consume treated water, aquatic and riparian vegetation, or prey organisms from that water. Some exposure could occur through dermal contact with the water, but this would be relatively minor for terrestrial animals. Additional, but likely minimal, exposure could come from ingestion of terrestrial plants or other organisms exposed as a result of drift of rotenone from aerial applications.

7.2 Estimated concentrations of rotenone

Rotenone concentrations in the water are estimated by EPA to be up to 200 ppb, the apparent solubility limit for rotenone, although label directions allow applications to be made to achieve 250 ppb of rotenone (EPA, 2006c). With the proposed label requirements, the maximum application rate of 200 ppb will match the solubility of rotenone. During applications, there would be areas with higher amounts of rotenone until mixing is complete, but, given the solubility, it is questionable that concentrations of rotenone actually dissolved in the water would

be higher than after mixing is complete. Uptake of undissolved rotenone through fish or invertebrate gills would be limited if there is any at all because undissolved material would be particulate and too large to pass through gill pores.¹

7.2.1 Water column – lentic

Concentrations in the water column of lakes, ponds, and reservoirs may currently be a maximum of 250 ppb, although if only 200 ppb can be dissolved in water, the remaining 20% of that would likely be in the form of an emulsion. Under the proposed requirements, the maximum concentration will be 200 ppb. WDFW uses the aspirator system for spreading the powdered slurry to reduce the amount of airborne dust exposures to applicators and the environment. The action of the slurry mixture entering the water at high pressure results in much better mixing than would occur from non-pressurized application approaches. Mixing may be slower in deeper or stratified lakes or lakes with highly variable bottom topography. Applications made above the water would be expected to mix more slowly than subsurface applications.

7.2.2 Water column – lotic

At present, 250 ppb treatments are allowed in streams and rivers. Under the proposed requirements, a maximum treatment for streams and rivers will be 50 ppb. This would appear to apply to barrier treatments for inflowing streams and rivers also, but is not explicit, since the proposed labeling requirements do not distinguish between such barrier treatments and other stream and river treatments where treatment of the stream is the goal. Mixing in flowing water would likely be quick for the main channels. Side channels and backwaters would likely be mixed more slowly; some labels indicate that such areas should be treated, such as with a backpack sprayer, to ensure full coverage.

7.2.3 Sediments

After mixing is complete, concentrations of rotenone in the sediments would be expected to be higher than in the water column in lentic waters, due to the tendency of rotenone to adsorb to sediments. (Finlayson et al., 2001) found that residues in three lentic water projects were 310, 180, and 522 µg/Kg. In two of these, concentrations dropped below detection limits in 14 days. In the cold Lake Davis, concentrations were not detectable after 60 days. Data from stream applications indicate that these sediments are likely to have no or very low rotenone residues (Finlayson, et al., 2001).

7.2.4 Adjacent terrestrial areas

Broadcast sprays, especially aerially broadcast rotenone, may result in some exposure of land adjacent to the treated water as a result of spray drift. Because the amount applied depends upon the quantity of water to be treated, it is necessary to make some assumptions. For the purpose of this analysis, the EPA model of a one-acre pond, six feet deep (Urban and Cook, 1986) is used. (In recent times this has been expressed in metric units, but since label calculations for

¹ Based upon unpublished EPA data showing that hexachlorobenzene will pass through fish gill pores that are approximately 50 angstrom units in cross section, but that hexabromobenzene, with a cross section of approximately 60 angstrom units is not taken up through fish gills.

applications are based upon acre-feet, the older English units are used here.) To achieve the labeled maximum concentration of 250 ppm, it would require application of 13.5 lb of a 5% product per acre-foot or 81 pounds of product for six acre-feet. The latter would be 4.05 lb ai of rotenone, which would then be applied to a surface acre of water. For a direct application to plants, that would amount to plant residues on short grass of 972 ppm, based upon the 95% confidence limits findings of Fletcher et al., (1994). Since EPA models for terrestrial applications estimate 10% drift from the application site to adjacent water, it seems reasonable to assume that 10% of the material applied to water would drift to adjacent land. This would result in a maximum exposure on short grass of 97.2 ppm.

Residues on vegetation would be of short duration. The photolysis half-life of technical rotenone on leaves is 2.9 hours (adjusted for volatilization) on bean leaves. The dust and a powder formulation had photolysis half-lives on lettuce leaves of 2.9 and 3.6 days, respectively. For tomato leaves, the respective half-lives were 2.7 days for the dust and 0.9 days for the powder. (See section 5.4.1)

7.3 Persistence and duration of residues

EPA (2006c) reports that the fate and transport of rotenone are not well known, and that past characterization of rotenone as immobile and non-persistent in aquatic environments is only partly true, based on actual use of rotenone as a piscicide.

7.3.1 Water

The persistence of rotenone in water is dependent upon several factors. Specific data are not available to address all degradation parameters under actual use conditions. As discussed in section 5.3 above, rotenone undergoes aqueous photolysis rapidly in shallow water in the laboratory, with a half-life of 21 hours in the top 1 cm of water, but with a half-life of 191 days in 2 m of water (Draper, 2002 in EPA, 2006c). Hydrolysis is expected to be rapid. Hydrolysis occurs quickly in alkaline water ($T_{1/2} = 2$ days) or neutral waters ($T_{1/2} = 3.2$ days) at 25°C. It is more persistent in acidic water ($T_{1/2} = 12.6$ days) and based upon field data, can be more persistent at colder temperatures.

In studies in Wisconsin, rotenone applied to a warm water lake at 23-27°C had a half-life of 10.6 hours in the water column, and when applied to a cold lake at 5°C, the half life was 23 hours in the water column. For the whole aquatic system of both sediment and water column, the half lives were 20 days for the cold water system and 1.5 days for the warm water system. In these ponds, fathead minnows were restocked until 9 of 10 fish survived for 24 hours; this occurred 4 days after treatment in the warm water pond and 30 days in the cold water pond. (USEPA, 2006c)

In many treatment projects, rotenone residues have been monitored following applications. Bradbury (1986) summarizes the duration of toxicity for rotenone treatments for 113 Washington lakes from 1977-1984. In 59 eastern Washington lakes, periodic bioassays with caged fish conducted to determine when fish could be restocked showed that the maximum time that treated lakes were toxic to these caged fish was typically 4.55 weeks, with a range of 0.5 to 10 weeks. In 44 western Washington lakes, the mean time that lakes were found to be non-toxic was similar

at 4.8 weeks, and with a similar range of 1-11 weeks. Bradbury does not attempt to provide reasons for the variability.

The American Fisheries Society manual on rotenone treatments (Finlayson, et al., 2000) suggests that fish toxicity, as determined from bioassays on caged fish, typically will last from 1 day to 4 weeks after applications.

The well studied Lake Davis, California treatment was made to cold water (9°C). As reported by EPA (USEPA, 2006c), the half life for parent rotenone was 10.3 days, and rotenone fell to below levels of detection (2 ppb) in 39 days, or 5.5 weeks, not much different than the meantime to non-toxicity in Washington lakes. Similarly, in Diamond Lake, OR, rotenone was non-detectable (detection limit 2 ppb) in the water column after 35 days (David Loomis, Oregon Department of Fish and Wildlife project manager, telephone communication, May 15, 2007).

In streams and rivers, the duration of both residues and toxicity to fish would be considerably lower due to the flow of the river. USEPA (2006c) calculated the time that rotenone would be toxic to fish in streams and rivers, based upon dissipation data from lakes. The application of lake dissipation data to calculating toxicity in lotic waters does not provide a realistic estimate, primarily because of physical transport downstream. In studies of Silver King Creek in California (USEPA, 2006c, Appendix K), rotenone applied at target rate of 25 ppb (actual measured rate of 50 ppb) to the creek (water temperature 7-12°C) remained above the detection limits of 2 ppb for 18 hours above the detoxification station.

Finlayson et al., (2001) reported on the residues of rotenone in California lakes and streams following rotenone applications. Lentic waters sampled on the day of application had water column residues from 11-370 ppb for twelve sampling stations. No explanation was provided on why residues in one treatment project were above both the application rate and the solubility of rotenone. The effective half-life of rotenone was from 0.65 to 7.7 days. All stream treatments were detoxified with potassium permanganate, and residue samples taken downstream from detoxification stations indicated no rotenone residues.

7.3.2 Sediment

The Wisconsin study by Gilderhus (1986, in EPA, 2006c) showed that the half life of rotenone in water plus sediment was longer than in the water column. There was a larger difference in this parameter in the sediments for different temperatures than in the water column. The water plus sediment half life in the warm water pond was 1.5 days, approximately 3 times of the water column half life. But the water plus sediment half life was about 20 days for the cold water pond or approximately 20 times the half life duration in the water column.

Finlayson et al., (2001) report that sediments in flowing waters had no rotenone residues 7 days after treatment, and standing waters did not have rotenone residues in sediments after 60 days. Only one of six stream samples had rotenone detected in the sediments. The concentration was 37 µg /Kg (level of detection in sediment was 30 µg /Kg). Two stream samples had rotenolone residues in the sediments, but these were considered analytical anomalies.

In a 2006 rotenone treatment of Diamond Lake, OR, residue sampling started 3 days after treatment. No residues were detected in sediments above the detection limit of 2 ppb. (David Loomis, Oregon Department of Fish and Wildlife project manager, telephone communication, May 15, 2007)

7.3.3 Soil and vegetation

Residues of rotenone in soil would only occur from piscicidal uses as a result of drift from aerial applications. EPA (2006c) used photolysis data on leaves of bean plants to represent soil photolysis. The half-life in one report was estimated to be 2.9 hours when volatilization losses were included. Based upon 12-hours of comparable sunlight in a day, terrestrial residues would diminish by approximately 90% in one day. In another study, the photolysis half-life was estimated at 2.9 and 3.6 days on lettuce leaves; 90% loss would occur in 10-15 days at this rate.

7.3.4 Detoxification of rotenone

Potassium permanganate, a strong oxidizing agent, may be used to deactivate or detoxify rotenone. Nearly all end-use product labels indicate that rotenone will be detoxified naturally within a week to a month, but that potassium permanganate can be added to hasten detoxification. Labels that include use in streams and rivers (other than upstream barrier treatments) all indicate that, to limit downstream effects of rotenone treatment, potassium permanganate can be used at a rate of 2-4 ppm in streams and rivers, with the further recommendation that caged fish bioassays can be used to determine the need for detoxification and when detoxification is complete.

The American Fisheries Society's Rotenone Manual (Finlayson et al., 2000) indicates that potassium permanganate is the agent of choice for detoxification, although chlorine is also mentioned on labels. In a survey of state and federal agencies in the U. S. and Canada, it was reported in the Manual that 72% of the respondents used potassium permanganate to detoxify treated waters, but no information was reported on how many and what types of treated waters were detoxified. The Manual stated that "It may be necessary" to detoxify to limit the impacts on downstream users.

The proposed labeling requirements in the RED (See appendix 2), require deactivation of effluent water to prevent exposure beyond the defined treatment area. This would include both effluent streams from treated lakes and areas downstream of treated portions of rivers and streams. Detailed instructions are to be contained in the Rotenone SOP Manual under development, and have not been seen.

7.4 Bioconcentration and Bioaccumulation

Bioconcentration factors in fish are 10.8 for viscera, 27.9 for the head, and 27.6 for the carcass, when corrected for parent rotenone (EPA, 2006c). These data indicate a low potential for bioconcentration.

In the Wisconsin field study by Gilderhus (1986, in EPA, 2006c), residues of fish that were killed by the treatment were analyzed for parent rotenone residues, including fish reintroduced

after the application. These residues were up to 696 ppb, or less than three times the intended aquatic concentration of 0.25 ppm rotenone. Although this kind of study is not readily comparable with standard bioconcentration studies, the results do support the laboratory findings that rotenone is not a concern for bioconcentration within organisms or bioaccumulation across trophic levels.

The Wisconsin findings also suggest a potential exposure level for piscivorous birds and mammals. Residue analysis was split into edible and non-edible portions of fish, so there were no “whole fish” measurements that would relate more directly to ingestion by piscivorous birds and mammals. But taking the highest levels would conservatively suggest a maximum exposure concentration of about 0.7 ppm.

7.4.1 Within organisms

Surprisingly limited data were available on the residues that might occur in fish killed by rotenone poisoning. As noted immediately above, fish residues were found to be a maximum of 0.696 ppm in the Wisconsin study. No data were found relating to the duration of residues within exposed organisms. Generally, the aquatic organisms are killed and therefore not available for depuration studies. Ling (2003) reported that residues of rotenone in fish fillets are generally below 1 ppm, although higher concentrations of the material are found in non-edible parts.

7.4.2 Accumulation and other food chain transfer

No quantitative data were found on bioaccumulation and food chain transfer for rotenone in either aquatic or terrestrial systems. Fish residue data indicate that body burdens are so low as to not be of significance in food chains.

7.5 Ground and well water considerations

7.5.1 General aspects of groundwater and wells.

Rotenone does not create a ground water concern. The strong tendency of rotenone to adsorb to soils, sediments, and other particulate matter precludes leaching almost entirely. The soil-water partition coefficients, K_d , range from 4.2 to 122 Kg/L for a variety of soil types. There is some potential for leaching only when rotenone reaches the most vulnerable soils, i.e., “very sandy soils with low organic content” (USEPA, 2006c); even then, mobility should be limited, and hydrolysis should degrade any rotenone that does reach water.

There has been only limited ground water monitoring for rotenone, probably because of its low propensity to leach. Because of the expense of sampling and analysis, most groundwater monitoring has been targeted towards chemicals expected or known to leach. Some non-targeted monitoring may be done to search for a wide range of chemicals in surface water, but not in groundwater. The U. S. Geological Survey’s (USGS) National Water Quality Assessment (NAWQA) program has done extensive sampling for both ground and surface water for more than 2000 constituent chemicals or water parameters. Over 30,000 pesticide samples have been taken from about 7600 surface water sites and 8100 wells (Bell, R. W. and A. K. Williamson. 2006. Data Delivery and Mapping over the Web, USGS fact sheet, accessed on-line at

http://pubs.usgs.gov/fs/2006/3101/pdf/fs06-3101_508.pdf, May 16, 2007). In the NAWQA “data warehouse” (accessed on-line at <http://water.usgs.gov/nawqa/data>, May 8, 2007) rotenone was not listed as a pesticide for which USGS sampled, suggesting that even in this extensive program, rotenone is not considered a contaminant of concern for which sampling should be done.

California’s Department of Pesticide Regulation has an active sampling program for chemicals in surface and groundwater. Their on-line surface water database (<http://www.cdpr.ca.gov/docs/sw/surfcont.htm>, accessed May 15, 2007) has over 183,000 records from 7000 samples in California, but contains no sampling data for rotenone in either surface waters or sediments. However, in their well inventory database, sampling for rotenone was done in 14 wells in three different counties and no rotenone was found (CDPR, 2003).

As a result of the rotenone treatment of Lake Davis, California, a substantial amount of sampling was done for rotenone in Plumas County (Ridley, 2006). As of June, 2006, 1224 analyses had been done for 78 groundwater wells. No evidence of rotenone has been found. Four compounds were found, and none of these were considered related to the Lake Davis treatment. Brian Finlayson (California Department of Fish and Game, telephone communication, May 9, 2007, (916)358-2950) reported rotenone has not been detected in either ground water or surface water in California. As senior author of the American Fisheries Society’s Rotenone Use Manual, Finlayson has been involved with fisheries managers from all over the United States and Canada. He has never heard of any rotenone groundwater detections in North America. In a recent (2006) treatment of Diamond Lake, Oregon, groundwater samples have been taken in three wells, and no rotenone has been found at the detection limit of 2 ppb. (David Loomis, Project Manager, Oregon Department of Fish and Wildlife, telephone communication, May 14, 2007). All of this information and data support the conclusion that rotenone is not a concern in groundwater.

7.5.2 Mobility of rotenone and considerations for use in fractured basaltic areas.

No information on groundwater sampling for rotenone was located for Washington state. Despite the lack of detection anywhere that sampling has been done, the geology of eastern Washington has large expanses of fractured basalt substrate similar to volcanic areas of the Pacific Northwest, California and the Great Basin. Specifically concerns have been raised about the potential migration of rotenone through the fractured basalts of the Columbia plateau.

As noted in the Problem Formulation (section 2.6.2), much of the Pacific Northwest has a highly volcanic history. Numerous layers of basalt flows, individually averaging about 100 feet thick, and collectively up to 15,000 feet thick, underlay the surface. As the lava flows cool, they tend to shrink, resulting in cracks or fissures through which liquids may permeate. Subsequent folding and faulting can also lead to openings in the layers. The tops and bottoms of these layers are particularly permeable because of fractures, vesicles and rubble zones. Unconsolidated, sedimentary soils between basalt layers may be even more permeable (USGS, 1994). At the same time, unfractured basalt layers are not permeable, and water would move laterally across these layers rather than vertically through them.

The potential movement of chemicals through fractured basaltic rocks and associated soils has become an issue in Washington as a result of studies at the Hanford site near Yakima, where radiologically and chemically contaminated water plumes are approaching the Columbia River (Williams, et al, 2000). Extensive studies by Williams, et al. (2000) and Spane and coworkers (Spane and Raymond, 1993; Spane and Vermeul, 1994; Spane and Webber, 1995; Spane, et al., 2001) have shown some of the Hanford aquifers are connected, while others are not, and lateral movement is as or more likely than vertical movement. These studies have also demonstrated that the hydrological characteristics of such basaltic soils vary significantly. Understanding the potential movement of substances in the ground water requires a detailed analysis of an individual site, and the amount of research done to characterize the Hanford site is highly unusual.

Because the potential exists for movement through fractured basaltic soils, and because there is insufficient characterization of the hydrology for sites other than Hanford, indirect means are necessary to analyze the potential movement of rotenone into groundwater in this geological environment. Two general aspects are important: the availability of rotenone and the nature of the treated lake or stream and its underlying features.

As noted above (section 7.5.1), rotenone is not considered mobile through soils, based upon its physical and chemical characteristics and a lack of any detection in wells or groundwater. The evidence of mobility of water soluble chemicals in the Hanford area does not apply to fairly insoluble chemicals such as rotenone. The characterization of the geological environment of Hanford is indicative that a potential concern should be analyzed, but the situation is confounded by the wide variation on soil profiles and underlying structure in differing localities, even in close proximity.

The first consideration related to the use of rotenone as a piscicide. The application sites of concern will be lakes, ponds, and reservoirs. Applications of rotenone in streams and rivers may occur, but the material would move down the stream rapidly enough to have little opportunity to even get into sediments. Finlayson et al., (2001) found no rotenone residues above or below detoxification stations in stream sediment samples in Silver King Creek in California. The lack of rotenone residues in sediments above these stations is indicative of the transient nature of rotenone in stream situations.

Rotenone may persist in treated lentic waters for up to 4-5 weeks under normal circumstances at moderate temperatures. However, in the colder waters of Lake Davis (9°C) rotenone did not drop to below detection levels for almost 6 weeks and in experimental ponds in Wisconsin at 5°C, the hydrolysis half-life was found to be 20 days (USEPA, 2006c). Based upon an application rate of 200 ppb, rotenone could persist above detection levels for up to 140 days in the cold Wisconsin lake at 5°C .

To enter the fractured basaltic geologic system, rotenone would have to move through the lake bed into the fractured basalt area. Once it entered the fractured basalt area, it could move either laterally or vertically through openings, fissures and cracks in the rocks. However, the potential for that movement is expected to be zero because of adsorption to sediments in the lake bottom, and the immobility of rotenone.

Lake bottoms are not simply underwater soils. Lakes have some level of algae and aquatic macrophytes. Decaying plant material and waste materials from aquatic animals, accumulate over time and most go to the bottom of the lake creating a lake sediment that is typically rich in organic material. Even a thin sediment layer would create a barrier for rotenone movement since it binds to particulate matter and does not leach. This factor alone negates any movement into ground water, even in fractured basaltic areas.

As noted in section 7.5.1 above, no ground water detections were noted near Lake Davis, California or Diamond Lake, Oregon. Both of these are in areas where there is a significant layer of volcanic material underlying the treated lakes. These areas may not be quite the same as areas with the Columbia River Basalt Group of formations, but both are in areas where layers of lava flows occurred and where cooling would cause shrinking and cracking.

Eastern Washington, however, now has another feature that would further prevent movement through lake bottoms. In 1980, Mount St. Helens erupted and spread 540 million tons of ash over a 22,000 square mile area, covering nearly all of eastern Washington, except along parts of the Canadian border. Ash was 4-5 inches deep in Yakima and ½ deep in Spokane (Wikipedia entry written by Lyn Topinka, USGS, accessed online at http://en.wikipedia.org/wiki/1980_eruption_of_Mount_St._Helens, May 16, 2007). The coarser particles that fell nearer Mount St. Helens, such as those in Yakima, would not adsorb rotenone as much as the finer particles that traveled further, such as those in Spokane. However, the larger quantity in Yakima would substitute for the finer particles. A study in Lake Williams, near Cheney, Washington showed that ash layer was suspended for several months at the water-sediment interface before breaking up and sinking into uncompacted sediments (Anderson, et al., 1984). Presumably, a similar event would have occurred at lakes throughout eastern Washington. The fine nature of a small amount of ash, or the larger quantity of a coarser ash, either as a layer or in the uncompacted sediments, would adsorb rotenone to the extent that none would be expected to permeate the sedimentary layer and move into the underlying strata.

While it may be possible to extract water containing rotenone residues from the water column of a treated lake, the possibility that rotenone would move into groundwater through a lake bottom is non-existent. It should be further noted that the Lake Davis well monitoring that detected no rotenone residues in 78 different wells is located in an area of fractured volcanic rock.

8. Risk Assessment and Characterization for Ecological Effects

Risk characterization is the integration of exposure and effects characterizations. From an ecological perspective, there is no risk without a combination of both toxicity and exposure. Even relatively benign or nontoxic substance can be a risk if there is sufficiently high exposure, and even the most toxic substances are not a risk if there is no exposure. In this context, risk is a measure of the actual effects that may occur in those environments where a stressor reaches an ecological receptor in sufficient quantity. The variation in the amount and compartmentalization of a stressor and the differential sensitivity of receptors of different species, life stages, location, health, and other factors combine to result in uncertainties. There are never enough data to

eliminate all uncertainties, although large quantities of data may reduce the uncertainties to levels where conclusions about risk may be predicted within certain limits.

With fewer data, more assumptions are required to assess risk. USEPA requirements, for example, include a good breadth of data to address a wide variety of risk factors. However, their data requirements do not provide a great deal of depth of information, and considerable assumptions need to be made to assess risk. From the basic toxicity requirements, for example, all avian risk projections are based upon two bird species, and likewise, all fish risk projections are based upon data for two fish species. An assumption is necessary that these birds and fish are representative of all birds and fish, or at least that they can be used as a basis for modeling for all birds and fish. Similarly, a model for an estimated environmental exposure is typically based on one or a few sites for a given a use, with the assumption that those sites used are representative of all sites.

Benchmarks are useful in this context. Based on comparable data for large numbers of chemical substances, one can look at a quantitative combination of effects and exposure, such as a risk quotient. For example, the risk quotients for a new chemical can be determined and then compared with benchmark chemicals where there is sufficient information under actual use conditions to have a reasonably good idea of what will happen. A risk quotient (RQ) is derived by dividing the environmental concentration, usually the estimated environmental concentration (EEC), of a chemical by the toxicity value, such as an LC₅₀ or a no observed effect concentration (NOEC). A Level of Concern (LOC) is established to achieve certain results, such as protection of populations or protection of individuals, and the RQ is compared with the LOC. Risk is presumed for the type of risk in column 1 when the RQ in column 2 exceeds the Level of Concern in column 3 (Table 8.1). This is considered a “deterministic” approach, and is normally the method used unless there are extensive data available for a more refined “probabilistic assessment.” For rotenone, both EPA and this assessment use a deterministic approach.

Table 8.1. Risk presumptions used by US EPA		
Risk presumption¹	RQ²	LOC³
Acute risk - aquatic & terrestrial	EEC/LC ₅₀ or LD ₅₀ /ft ²	0.5
Acute restricted use - aquatic	EEC/LC ₅₀ or LD ₅₀ /ft ²	0.1
Acute restricted use - terrestrial	EEC/LC ₅₀ or LD ₅₀ /ft ²	0.2
Acute endangered species risk - aquatic	EEC/LC ₅₀ or LD ₅₀ /ft ²	0.05
Acute endangered species risk - terrestrial	EEC/LC ₅₀ or LD ₅₀ /ft ²	0.1
Chronic risk - aquatic & terrestrial	EEC/NOEC	1

¹Acute risk at this level relates to effects on populations of non-target organisms
 Acute restricted use relates to classification of a pesticide to be used only by certified applicators
 Acute endangered species relates to effects on individuals of a T&E species
²EEC= estimated environmental concentration; NOEC= no observed effect concentration
 The EC₅₀ may substitute for the LC₅₀, especially with aquatic invertebrates

³LOC = Level of Concern established by US EPA as a basis for regulatory concern. Specific numbers are derived from historical information and theoretical models (Urban and Cook, 1986).

Rotenone is somewhat unusual. The very nature of its use as a piscicide requires a “field” assessment after use to determine when rehabilitation of a water body can proceed to the next stage. That is, it is not effective to restock a treated body of water until the rotenone has dissipated to nontoxic levels. As a result, the time it takes to reach a non-toxic environment after treatment has been determined repeatedly, but only for each specific site and typically for only one or two species of fish. Additional monitoring is done to assess other conditions ranging from invertebrate populations to the concentrations of rotenone in various compartments. Since each treatment site is different in the natural environment, it is difficult to extrapolate to other environments. The best field-level predictions of risk, or indeed, even efficacy, seem most likely when a project is using rotenone in a water body that has been treated before. But the extensive use of rotenone over many decades does provide a considerable amount of information that can be used to assess risk to reasonably typical water bodies. There have been over 500 treatments of lakes in Washington. Surely, the vast majority of these lakes are closer to “average” in most characteristics than they are to extremes of these characteristics. Thus, the knowledge and data obtained from all of these treatments has to be applicable to a majority of future treatments within certain limits. Caution is needed, however, when a treatment is proposed for an “unusual” lake, i.e., one that has substantially different features than those upon which the body of knowledge has been developed.

One principle of toxicology is that within the limits of genetic variability, with some consideration of factors like life stage or health, toxicity does not change for a species. What may change the risk is variation in exposure and bioavailability. On that basis, risk characterization is much more a function of environmental exposure than environmental toxicology.

8.1 Direct Effects

8.1.1 Fish

Consistent with rotenone’s intended use as a piscicide, it is not only expected that fish will be killed from labeled use, it is intended that fish will be killed. There are considerable fish toxicity data for rotenone (Tables 6.1 and 6.2). The results show moderate variation even within a species. Bullheads, channel catfish, and goldfish are generally less sensitive than other tested species and salmonids are generally the most sensitive. When the reported LC₅₀ values are adjusted to reflect the toxicity of the active ingredient, all fall below 50 µg/L. Making such adjustments is confounded by the lack of knowledge of the toxicity of the cube resin component and all end-use formulations and the inclusion of the synergist, piperonyl butoxide, in some formulations.

Fish recover readily from sublethal doses of rotenone when placed in clean water. Rotenone is detoxified in the liver by mixed function oxidases (MFO). MFOs can be stimulated by repeated exposure to rotenone. As a result, breakdown of rotenone in the liver is enhanced and fish can become resistant (Ling, 2003).

WDFW does not use selective treatments in standing waters (J. Anderson, WDFW, personal communication, May 7, 2007). Since all fish in a treated lake are targets of the application, non-target fish are those downstream from the treated lake. Potential effects on these non-target fish would occur if treated water moved downstream in sufficient quantity. Thus, the potential effects on non-target fish can be avoided by precluding the downstream movement of treated water, or they can be avoided or reduced by lowering the concentration of rotenone in any treated water that does move downstream.

Reservoirs have a barrier that can be used to prevent movement of water downstream, at least when water levels are low enough. It may be impractical, however, to eliminate water flow into these streams even for the period of time necessary for rotenone to be degraded. Lakes and ponds may have an effluent stream. Thus, there is a risk from treatment of standing waters for non-target fish in streams flowing out of the treated waters. If treated water cannot be held until the rotenone has degraded, then detoxification is necessary to protect fish in the downstream waters. Detoxification is also necessary in stream applications.

Potassium permanganate is frequently used to detoxify rotenone. Current labels recommend, but do not require, the use of potassium permanganate in streams and rivers to detoxify rotenone leaving the treatment area. The rotenone manual (Finlayson, et al., 2000) states, "If the discharge [of treated water] cannot be stopped without impacts to downstream users, neutralization is necessary." The proposed labeling requirements in the rotenone RED (see Appendix 2) will state that neutralization is required for effluent water leaving the treatment area, with details on the procedure to be included in the Rotenone Standard Operating Procedure manual currently under development.

Caution is required when using potassium permanganate to ensure that concentrations are balanced with the amount of rotenone to be detoxified, since potassium permanganate does have moderate fish toxicity, with LC50 values reported by USEPA (2006c) of 750 µg/L to 4920 µg/L for various fish species. It is much more toxic in alkaline water than soft water (Marking and Bills 1975). Potassium permanganate breaks down in the natural environment quite rapidly and is a much-preferred alternative to the dispersion of a toxic plume of rotenone many miles downstream of the target area. If potassium permanganate concentrations are in balance with rotenone concentrations then toxic levels of potassium permanganate should be quickly reduced through the oxidation of organic components and rotenone in the water, and there would be, at most, a short plume of toxic potassium permanganate immediately below the target zone..

8.1.2 Other aquatic biota

Rotenone can be highly toxic to aquatic invertebrates (section 6.3.4 and Table 6.2). However, the toxicity is quite variable. Ling (2003) reported that zooplankton usually decline substantially following rotenone treatments and a few benthic invertebrates are also affected. Because *Daphnia*, a zooplanktonic cladoceran sensitive to rotenone, is a standard test species, it is often considered that aquatic arthropods are sensitive in general. However, benthic invertebrates, to a great degree are not sensitive at the labeled rates of rotenone use. Aquatic arthropods are often considered more sensitive than mollusks, but Table 6.2 shows that a modest number of tested arthropods are not very sensitive, and two of the four bivalve mollusks are as sensitive as many arthropods. In reports (e.g., Bradbury, 1986; Ling, 2003; Hanson, et al., 2006) on various

rotenone treatments in lakes, it has generally been found that there is a significant impact on zooplankton and a few other invertebrates, but that these recover by the following year, often to a greater degree initially than before rotenone treatment because the predators on the zooplankton have been removed and subsequent stocked fish take less zooplankton. Phytoplankton are less affected, if at all, by rotenone and tend to increase initially because of the loss of the zooplankton feeding on them, but then become markedly reduced the next spring when the zooplankton recover. Aquatic macrophytes are not affected directly by rotenone, and the increased clarity of the water due to the zooplankton feeding on the phytoplankton allows macrophytes to flourish and spread. Havens (1980) reported that recovery of 1-2 years were required for recovery of zooplankton and 3 years for macroinvertebrates in the colder areas near Cook's inlet in Alaska.

In a study designed specifically to evaluate effects on invertebrates, Blakely, et al., (2005) reported that there were few differences in species richness of invertebrates in rotenone-treated ponds versus untreated ponds in an orchard area six months, one year, and three years after treatment. The study was retrospective on already treated ponds; the ponds were not studied over a period of time. There were subtle differences in the zooplankton and macroinvertebrate community structures. Certain chironomids were more abundant in the treated ponds, and flatworms and diving beetles were more abundant in the untreated ponds. Among 5 taxa of zooplankton (two copepods, two cladocerans, and an ostracod), there was considerable variation that may have hidden significant differences. The highest species richness was in untreated ponds, but the lowest species richness was also in an untreated pond along with one treated 6 months previously. Cladocerans were more abundant than copepods in treated ponds, whereas the two taxa were comparable in untreated ponds; ostracods were most abundant in ponds treated 3 years previously. Although they did not observe ponds immediately after treatment, they assumed, on the basis of much literature, that zooplankton and some benthic invertebrates would have been affected, but that by 6 months after treatment, populations had recovered. Changes in community structure occurred, but were not pronounced; these changes may have been due to lack of fish to prey preferentially on some taxa, rates of recolonization, or the use of other agricultural chemicals, as well as possibly due to rotenone.

The toxicity of rotenone to gilled stages of amphibians, e.g., tadpoles and larval or neotenic salamanders, is approximately similar to fish and aquatic invertebrates. The southern leopard frog (*Rana sphenocephala*) is only slightly less sensitive than tested fish, based upon EPA one-liner data. It is unclear if the leopard frogs (*Rana pipiens*) tested by Farringer (in Bradbury, 1986) are gilled or adult, although it is clear that this was an aquatic test. These frogs were much less sensitive than fish and aquatic invertebrates. If they were the gilled tadpole stage, then the lower sensitivity might be relevant, but if they were simply adults in the water, then valid comparisons cannot be made. Nevertheless, toxicity occurred to these leopard frogs at much higher concentrations than would be used for fish control; thus, at least adults, and possibly larval frogs, should not be affected by rotenone at labeled treatment rates, based on this work. From comparisons made between Sanders (1970) and others' work on larval amphibians and data submitted over many years to EPA, EFED staff consider that tadpoles are slightly less sensitive than fish to most pesticides. USEPA (2006c) uses fish as a surrogate for the aquatic stages of amphibians. This is probably a valid assumption for rotenone for the gilled stages of amphibians whether larval or neotenic, but is quite conservative for non-gilled amphibians that are primarily aquatic.

Two additional publications warrant discussion because they have been widely cited. Fontenot et al. (1994) summarized the available data on rotenone and herpetofauna. They developed no new data, made an erroneous conclusion regarding formulations and active ingredients, and speculated broadly without sufficient data. However, they basically did conclude, as have others, that rotenone may impact larval stages of amphibians. They were unclear in other areas discussing concentrations. They did note that in a questionnaire sent to states, 83% of 174 respondents reported no or unknown effects on non-target organisms; 4% reported some dead tadpoles, 1% reported some dead sirens, and <1% reported some dead salamanders. They also noted that areas of one treated water body were protected from rotenone exposure by using Hessian sacking treated with potassium permanganate between the main treatment area and the part where the tadpoles were abundant. McCoid and Bettoli (1996) supplemented this summary in noting that, in three of six coves of a reservoir treated with 3 mg/L (150 µg a.i./L) of Noxfish, an estimated 10 dead turtles, *Kinosternon subrubum*, were observed after treatment in each of the three coves. They observed no deaths of amphibians or other turtle species.

8.1.3 Terrestrial biota

The most likely terrestrial animals that would be exposed to rotenone are those that are associated with aquatic environments (see section 7.1 and 7.2). Residues in dead fish have been found to be up to 0.696 ppm. For birds, such as osprey, feeding on fish from rotenone-treated waters, the low acute avian toxicity (LD₅₀=1680 mg/kg; LC₅₀=1608 ppm for most sensitive bird) the RQ is 0.00043. There is essentially no possibility of a risk to birds. Mammals are more sensitive than birds, with acute LD₅₀ values of 39.5 mg/kg for female rats. But this is still relatively non-toxic considering the exposure. For example, a mink may weigh 0.6 kg and ingest 0.124 kg of food per day (Canadian Environmental Quality Guidelines, online, undated, accessed May 13, 2007 at http://www.ccme.ca/assets/pdf/trg_protocol.pdf). A 0.6 Kg mink ingesting 124 gm of treated fish would receive a dose of 0.086 mg or 0.143 mg/Kg, far less than the most sensitive mammal LD₅₀. The RQ would be 0.0036, which is well below any LOCs.

8.1.4 Endangered and threatened species

The potential for effects on threatened and endangered (T&E) species must be considered in any fish rehabilitation project where such species may be in the vicinity. Table 8.1 presents the federal and state listed T&E species in Washington state.

Table 8.2 Endangered and Threatened Species in Washington		
Common name	Scientific name	Status¹
Mammals		
Bear, grizzly	<i>Ursus arctos horribilis</i>	T
Caribou, woodland	<i>Rangifer tarandus caribou</i>	E
Deer, Columbian white-tailed	<i>Odocoileus virginianus leucurus</i>	E
Fisher	<i>Martes pennanti</i>	SE
Gopher, Mazama (western) pocket	<i>Thomomys mazama</i>	ST
Lynx, Canada	<i>Lynx canadensis</i>	T
Otter, sea	<i>Enhydra lutris</i>	SE

Table 8.2 Endangered and Threatened Species in Washington		
Common name	Scientific name	Status¹
Rabbit, pygmy	<i>Brachylagus idahoensis</i>	E
Sea-lion, Steller	<i>Eumetopias jubatus</i>	T
Squirrel, western gray	<i>Sciurus griseus</i>	ST
Whale, humpback	<i>Megaptera novaeangliae</i>	E
Whale, Sei	<i>Balaenoptera borealis</i>	E
Whale, Fin	<i>Balaenoptera physalus</i>	E
Whale, blue	<i>Balaenoptera musculus</i>	E
Whale, black right	<i>Balaena glacialis</i>	E
Whale, Killer	<i>Orcinus orca</i>	E
Whale, sperm	<i>Physeter macrocephalus</i>	E
Wolf, gray	<i>Canis lupus</i>	E
Birds		
Albatross, short-tailed	<i>Phoebastria albatrus</i>	E
Crane, sandhill	<i>Grus canadensis</i>	SE
Eagle, bald	<i>Haliaeetus leucocephalus</i>	T
Grouse, sage	<i>Centrocercus urophasianus</i>	ST
Grouse, sharp-tailed	<i>Tympanuchus phasianellus</i>	ST
Hawk, ferruginous	<i>Buteo regalis</i>	ST
Horned lark, streaked	<i>Eremophila alpestris strigata</i>	SE
Murrelet, marbled	<i>Brachyramphus marmoratus marmoratus</i>	T
Owl, northern spotted	<i>Strix occidentalis caurina</i>	T
Pelican, American white	<i>Pelecanus erythrorhynchos</i>	SE
Pelican, brown	<i>Pelecanus occidentalis</i>	E
Plover, western snowy	<i>Charadrius alexandrinus nivosus</i>	T
Amphibians		
Frog, northern leopard	<i>Rana pipiens</i>	SE
Frog, Oregon spotted	<i>Rana pretiosa</i>	SE
Reptiles		
Sea turtle, green	<i>Chelonia mydas</i>	T
Sea turtle, leatherback	<i>Dermochelys coriacea</i>	E
Sea turtle, loggerhead	<i>Caretta caretta</i>	ST
Turtle, western pond	<i>Clemmys marmorata</i>	SE
Fish		
Salmon, chinook (Upper Columbia River spring run)	<i>Oncorhynchus tshawytscha</i>	E
Salmon, chinook (Snake River spring/summer run)	<i>Oncorhynchus tshawytscha</i>	T
Salmon, chinook (Lower Columbia River)	<i>Oncorhynchus tshawytscha</i>	T

Table 8.2 Endangered and Threatened Species in Washington		
Common name	Scientific name	Status¹
Salmon, chinook (Puget Sound)	<i>Oncorhynchus tshawytscha</i>	T
Salmon, chinook (Snake River fall run)	<i>Oncorhynchus tshawytscha</i>	T
Salmon, chum (Columbia River)	<i>Oncorhynchus keta</i>	T
Salmon, chum (Hood Canal summer run)	<i>Oncorhynchus keta</i>	T
Salmon, coho (Lower Columbia River)	<i>Oncorhynchus kisutch</i>	T
Salmon, sockeye (Snake River)	<i>Oncorhynchus nerka</i>	E
Salmon, sockeye (Ozette Lake)	<i>Oncorhynchus nerka</i>	T
Steelhead (Upper Columbia River Basin)	<i>Oncorhynchus mykiss</i>	T
Steelhead (Middle Columbia River)	<i>Oncorhynchus mykiss</i>	T
Steelhead (Snake River Basin)	<i>Oncorhynchus mykiss</i>	T
Steelhead (Upper Willamette River)	<i>Oncorhynchus mykiss</i>	T
Steelhead (Lower Columbia River)	<i>Oncorhynchus mykiss</i>	T
Steelhead (Puget Sound)	<i>Oncorhynchus mykiss</i>	T
Trout, bull	<i>Salvelinus confluentus</i>	T
Insects		
Butterfly, Oregon silverspot	<i>Speyeria zerene hippolyta</i>	T
Checkerspot, Taylor's	<i>Euphydryas editha taylori</i>	SE
Skipper, Mardon	<i>Polites mardon</i>	SE
Plants		
Sandwort, Marsh	<i>Arenaria paludicola</i>	E
Paintbrush, golden	<i>Castilleja levisecta</i>	T
Howellia, water	<i>Howellia aquatilis</i>	T
Lomatium, Bradshaw's	<i>Lomatium bradshawii</i>	E
Lupine, Kincaid's	<i>Lupinus sulphureus kincaidii</i>	T
Checker-mallow, Nelson's	<i>Sidalcea nelsoniana</i>	T
Checker-mallow, Wenatchee Mountains	<i>Sidalcea oregana calva</i>	E
Ladies'-tresses, Ute	<i>Spiranthes diluvialis</i>	T

¹ Status is federal status, if listed. Federally listed species accessed May 13, 2007 at <http://www.fws.gov/endangered/wildlife.html#Species> and <http://www.nmfs.noaa.gov/pr/species/esa/>. If not listed federally, status is state status i.e., SE (state endangered) and ST (state threatened), accessed at <http://wdfw.wa.gov/wlm/diversty/soc/soc.htm>.

Based upon the RQs above for birds and mammals, there is no risk for these taxa. Because avian toxicity data are used as a surrogate taxon to determine risks to reptiles, the lack of avian toxicity

indicates no risk to reptiles. Based upon an inferred lack of phytotoxicity (section 6.4.6), there is no risk to T&E plants.

The Mardon Skipper (Potter et al., 1999), the Taylor's Checkerspot (Stinson, 2005), and the Oregon Silverspot (USFWS, 2001) are all grassland species and in small, isolated populations. The likelihood of exposure of these insects to piscicidal uses of rotenone is remote. However, they would likely be sensitive to rotenone if exposed. If there is a possibility of rotenone use in the vicinity of any of these butterflies, avoiding the use of aerial applications should preclude exposure.

The Oregon spotted frog and the northern leopard frog are definitely associated with aquatic habitats, but may not occur where piscicide treatments are done. Any use of rotenone in the vicinity of these two species should only be done after conferring with state experts on the locations of these species.

Rotenone is likely to affect any fish species that is exposed. Most monitoring for rotenone residues has been with a detection limit of 2 ppb. The most sensitive species used by USEPA (2006c) for risk assessment has an LC₅₀ of 1.94 ppb. EPA would use an endangered species LOC of 0.097 ppb to assess risk to T&E fish. Therefore, it can be assumed that, without FWS permits, any exposure to rotenone in or downstream of a treatment site, whether detectable or not, would trigger EPA's required Endangered Species Act section 7 consultation with the U.S. Fish and Wildlife Service (FWS). However, WDFW lake and stream rehabilitation projects using rotenone products purchased with US Fish and Wildlife Service Federal Aid in Sport Fish Restoration funding undergo consultation under Section 7 of the Endangered Species Act. All projects are reviewed annually by WDFW Fish, Wildlife and Habitat program staff for potential impacts to threatened and endangered species, as well as other fish and wildlife species of concern. It should be noted that not all downstream waters would have rotenone residues in the water because it degrades. In addition, detoxification can remove rotenone to below detectable levels within 30 minutes. (See section 7.3.4 on detoxification.)

Rotenone may be used in conjunction with a program to help recover T&E fish, typically to control other fish that may prey upon or compete with these T&E fish. Even these beneficial actions will trigger the consultation requirements if the project "may affect" a T&E fish. Currently, EPA alone is responsible for a section 7 consultation, although it may request help from a state or other entities. A provision of the Section 7 regulations (50CFR402.08) allows EPA, as the action agency, to name a "designated non-federal representative" to conduct a biological assessment or an informal consultation with the FWS. If a finding is expected to result in a "may affect" determination, but that the use of rotenone is "not likely to adversely affect" the listed species, then the consultation requirement may be completed at the local level. At least one state agency, California's Department of Pesticide Regulation" has been named as a designated non-federal representative by the EPA. WDFW may find it advantageous to request be similarly "designated" by EPA, especially considering the number of T&E species and their broad locations within Washington.

WDFW does not treat waters with threatened or endangered species. According to Washington Fish and Wildlife Commission Policy C3010, "waters will not be treated in ways which would

cause significant negative impacts to fish or wildlife which are state or federally listed as Threatened, Endangered, Sensitive or Candidate Species”. An exception may be granted in the case of a biological emergency. Any treatment that would “take” a federally-listed species would require a permit from the U.S. Fish and Wildlife Service or the National Marine Fisheries Service.

8.2 Effects on water quality

There are no direct data available to assess the effects of rotenone applications to water quality parameters such as pH, dissolved oxygen, nitrate, nitrite, and ammonia production, and the release of phosphates. There are no phosphorous or nitrogen components of rotenone that could be released into treated water. However, Bradbury (1986) has discussed the effects of rotenone on water quality. He suggested that algal blooms following rotenone treatments may be due, in part, to the release of phosphorus from decaying fish. Bradbury further indicated that rotenone treatments would have negligible effect on dissolved oxygen, pH, temperature, alkalinity, or carbon dioxide. Effects on taste and odor of drinking water may occur as a result of the hydrocarbon solvents in some emulsifiable concentrate formulations of rotenone; these odors lasted for only a few days. Fishy odors have occurred as a result of decaying fish for up to 17 days. Both kinds of odors can be removed from drinking water by the use of activated carbon. This should not be a concern because label requirements state that rotenone shall not be used within ½ mile of potable water intakes. WDFW does not treat lakes unless the agency has received written assurance from legal water rights holders that they will not withdraw water during the period of toxicity.

8.3 Effects from interactions with other pesticides

There are no available data on the interaction of rotenone products with other pesticides, and no expectation that other pesticides would occur in treated waters in meaningful quantities. There is a synergist, piperonyl butoxide, in some rotenone products which is considered under acute toxicity of these products.

8.4 Effects on pristine and contaminated sites

There are no available data that address the differential effects that would occur in pristine versus contaminated sites. Toxicity data are generated in what could be considered pristine waters and should therefore apply to pristine sites. In the event that notably contaminated sites are treated, which seems unlikely, the effects would depend upon the nature and quantity of contaminants. However, data are lacking on the interactions of rotenone with other chemicals, except that potassium permanganate and chlorine may be used to detoxify rotenone; presumably other strong oxidizing agents would have a similar effect.

8.5 Indirect effects

8.5.1 From removal of fish and other aquatic biota

The loss of fish in a water body would potentially have an effect on piscivorous animals. Similarly, the loss of other aquatic biota could have an effect on predators of those biota. The mobility of birds, such as osprey, herons, shorebirds, and kingfishers, should be sufficient to

allow them to move to non-treated areas. Some effect may occur if birds are feeding young at the time of treatment. Most treatments seem to be done in the fall when water is low, and breeding birds would not be any more affected than non-breeding birds. Mammals may not be able to move as far as birds, but there are very few mammals that rely exclusively on fish or aquatic invertebrates as sources of food.

Dead fish would provide an immediate food source for crawfish, amphipods, insects and other aquatic species. WDFW rehabilitation planning usually includes the provision that dead fish remain in the lake (rather than be removed from the water), specifically to provide nutrients for plankton growth following the rotenone treatment. These plankton provide a food base for fish that are re-stocked into the waters following the treatments. Mink, otters, and other piscivorous birds and mammals will be deprived of food sources for a short time; there are generally nearby waters with remaining fish to which predatory birds and mammals may change foraging sites. Also, most waters will be re-stocked with fish soon after treatment; the loss of forage fish is temporary.

8.5.2 Potential for increased erosion and resuspension of soils and sediments resulting from effects on plants

Based upon the application directly to aquatic sites and the lack of any apparent phytotoxicity, as based upon use as an insecticide to various crops and garden plants, there is no potential for erosion and no expectation of resuspension of soils and sediments.

8.5.3 Effects on aquatic habitats

Lasting effects on aquatic habitats would not be expected from rotenone, with some uncertainty, because there is no evidence that rotenone would affect the plants or the water chemistry except for the potential for algal blooms resulting from loss of zooplanktonic grazers on algae and release of phosphorus from decaying fish. Of the 9 lakes analyzed by Bradbury (1986) only three had what were termed “major blooms” of algae, and the duration of these three “generally lasted 1-2 months.” Ling (2003) noted that algal blooms occur more often when dead fish are not removed, but also that after recovery of the invertebrate fauna, clarity and other water quality conditions typically improve. The likelihood of blooms is variable depending upon the timing of the treatment and other factors. Spring treatments reduce zooplankton, which allows phytoplankton to multiply rapidly. But fall treatment allows the zooplanktonic grazers to recover before the phytoplankton populations get too large. Hanson, et al. (2006) found that growth of zooplankton in the early spring following a treatment resulted in a significant reduction of phytoplankton which concomitantly cleared the water and enhanced the habitat for macrophyte growth.

8.5.4 Potential effects upon agriculture

There are no data upon which to assess effects of piscicidal use of rotenone on agriculture. No effects upon agriculture would be expected except a potential for use that could reach an irrigation water intake, and the level of effects would depend upon the concentration in the irrigation water. Some insecticidal activity could occur. However, labels prohibit the use within ½ mile of irrigation water uptakes. The proposed label requirements include a requirement for

the deactivation of rotenone “beyond the defined treatment area”, which would preclude any exposure of crops through irrigation water.

8.5.5 Indirect effects on endangered and threatened species

Indirect effects on T&E species would be those that would affect the food or habitat of a species. Effects on food would be the most likely and piscivorous species would be the most likely ones affected, especially if treatments occur when the adults are feeding their young. There could be a reduction in the fish food supply for Bald eagles following a rotenone treatment. American white pelicans could be similarly affected, but there are few areas in eastern Washington where they breed. Non-breeding pelicans or eagles could also be affected by a loss of fish food, but not to the extent of breeding individuals.

8.6 Impacts of multiple applications

Rotenone does not persist long in treated waters from a seasonal or annual perspective. It is possible that a lake might be retreated in the years following an initial treatment, but there would be no remaining rotenone from the previous year’s treatment.

8.7 Impacts on terrestrial organisms and environments

Based upon the application directly to aquatic sites and the low toxicity to terrestrial birds, mammals, and plants, the last based upon use as an insecticide to various crops and garden plants, there is negligible potential for impacts on terrestrial biota other than possible effects on insects in immediately adjacent areas subject to spray or powder drift.

8.8 Impacts on wetlands other than target application sites

Except for wetlands downstream from treatment sites, there would be no exposure of rotenone to untreated wetlands. Under the proposed label requirements, rotenone would have to be deactivated as it left the treated water, thus precluding an exposure of downstream wetlands of any type. Under current labels, it is possible for rotenone to reach downstream wetlands if it is not deactivated. Wetlands are subject to different definitions. Generally, wetlands are thought of as standing or very slow moving areas of shallow water. In such environments, any rotenone that did reach the wetland would likely be degraded fairly rapidly in the shallowest parts through aqueous photolysis ($T_{1/2}=21$ hours in 1 cm of water). Except in acidic waters, hydrolysis ($T_{1/2}=2$ days at pH 9 and 3.2 days at pH 7) would act to degrade the rotenone in a few days to two weeks. A characteristic of this type of shallow wetland includes substantial vegetation to which rotenone would be likely to adsorb, thus effectively removing it from the system.

In a broader context of wetlands, rotenone could enter a downstream lake, where there would be substantial dilution as well as degradation primary through hydrolysis. Or the downstream area could be a river, where there would be little residence time for the rotenone to have impact. Again hydrolysis would degrade much of the rotenone that might reach such an environment.

8.9 Uncertainty analysis

There are a number of uncertainties in this analysis. The toxicity profile for rotenone is good for fish, but not very broad for aquatic invertebrates, and non-existent for algae and aquatic

macrophytes. Extensive anecdotal information exists on the latter two groups. Toxicity data on amphibians are limited and not well characterized. No data were located on the aquatic toxicity or effects of rotenolone or other degradates.

The environmental fate profile does not include standard kinds of aquatic dissipation studies that can be used for comparative purposes. There is a modest amount of anecdotal information on the loss of rotenone from treated waters, but there has been little distinction between transport away from the treatment site and degradation. This makes it difficult to project what might happen in other lakes or treated areas except in the broadest sense. The lack of mass balance in degradation studies also contributes to this difficulty.

There is uncertainty on the use of potassium permanganate to deactivate rotenone. It is known to work, but Finlayson et al., (2000) noted that there have been a number of situations where fish have been killed as a result of an imbalance between the concentrations of rotenone and the potassium permanganate used to deactivate the rotenone. A better determination of the rotenone concentrations at the potassium permanganate deactivation points would limit or eliminate the problem, but is complicated by the nature of stream flow characteristics between the rotenone application points and the deactivation points.

There is uncertainty about the inert products in rotenone formulations. USEPA (2006c) indicated that the fish toxicity of formulations is less than the rotenone technical material when adjusted for the percentage of active ingredient. So the inert ingredients do not contribute to the toxicity. But there are uncertainties related to the transport and degradation of these inert ingredients

There is also uncertainty on what changes may occur with the rotenone labeling or other requirements. There will be a 60-day public comment period for the RED starting approximately May 22, 2007 (Lance Wormell, SRRD/OPP, personal communication May 15, 2007). There may be alterations in the requirements as a result of this comment. In addition, the Rotenone Standard Operating Procedure manual has not yet been available for review or comment, so its contents and requirements are not known. As currently proposed, it will be a mandatory requirement to follow this manual.

8.10 Additional needs for information

8.10.1 Soil and sediment

Data on the fate of sediment-associated rotenone would provide a better measure of how long it is likely to persist in sediments at toxic levels.

8.10.2 Water

Standardized aquatic dissipation studies would enhance knowledge of the fate and transport of rotenone. Dissipation studies that include the rotenolone degradates would be particularly useful.

8.10.3 Plants

Toxicity data on aquatic macrophytes are lacking. Such data would be useful for assessing the impacts, or lack thereof, of rotenone. More importantly, they would provide a basis for assessing the risks to T&E plant species, such as the Water Howellia.

8.10.4 Acute toxicity studies

For piscicidal uses of rotenone, the acute toxicity data base is reasonably complete on the rotenone itself. Additional breadth of toxicity data on aquatic invertebrates would help to better characterize the impacts on this taxa and those organisms that rely on them for food. Acute toxicity studies on rotenolone would be valuable to assessing the risk to this important degradates. In studies of Silver King Creek in California (Finlayson, et al., 2001), the amount of rotenolone found exceeded the amount of rotenone found in 15 of 47 water samples.

8.10.5 Chronic toxicity studies

Chronic aquatic toxicity data barely meet the minimum EPA requirements with one fish-early-life-stage test and a Daphnia reproduction test. Given the moderately short persistence of rotenone, the addition of intermediate term toxicity data on several more species, especially aquatic invertebrates is desirable.

8.11 Mitigation measures

There are very few mitigation measures that would reduce the risks of rotenone used as piscicide beyond the extensive ones already presented in the RED. The major mitigation measure would be site characterization for treatment projects, and this may already be being done to some extent. Certainly, the locations of both federal- and state-listed T&E species should be determined, along with their proximity to treated areas.

8.12 Conclusions and recommendations

Based upon this analysis and the proposed labeling and other requirements in the U. S. Environmental Protection Agency's Reregistration Eligibility Decision document, the potential adverse environmental effects of rotenone used as a piscicide in Washington state should be limited to the sites specifically treated and should last for only a few months. Adverse effects are most likely to organisms that would absorb rotenone through gills and that are in the water column, although effects are also likely on a few types of benthic invertebrates. Applications made in the fall, after amphibian larval stages have lost their gills, should reduce or eliminate effects on frogs and other amphibians. Fall applications also should permit more rapid recovery of affected invertebrates.

9. Human Health Effects

There are a number of data gaps reported by EPA/OPP's Health Effects Division (USEPA, 2006a). However, there are at least some data to address most human health effects, even if the data profile is incomplete.

9.1 Toxicity information and sources

9.1.1 Acute toxicity

9.1.1.1 Oral

Acute Oral Toxicity to Rats

The acute toxicity test is designed to quantify the lethal level (LD₅₀) of a compound. In addition, the incidence and severity of all abnormalities, including behavioral and clinical abnormalities, the reversibility of observed abnormalities, gross lesions, body weight changes, and any other toxic effects for the various dosing levels of the test.

Four acute oral studies were conducted with technical rotenone and three rotenone end-use formulations; results are given in table 9.1. Technical grade rotenone was more toxic to female rats (LD₅₀ = 39.5 mg/kg) compared to male rats (LD₅₀ = 102 mg/kg), based on an acute oral exposure (MRID 00145496). All formulated products were similarly more toxic to females than males. Although direct comparisons of technical versus end-use formulations are confounded by the presence of “cube resins” and/or the synergist, piperonyl butoxide, there does not seem to be a significant toxicity difference between the technical material and the formulated products on the basis of mg of active ingredients per kg of body weight.

Formulation	% Rotenone	Toxicity	MRID
Technical	99.2%	Males: LD ₅₀ =102 mg a.i./kg Females: LD ₅₀ =39.5 mg a.i./kg	00145496
Prentox Grass Carp Management Bait	2.6% rotenone 0.5% piperonyl butoxide	Males=1550 mg/kg bw Females=970 mg/kg bw	429817-01
Chem Sect Chem Fish Regular	5% rotenone 5% cube root extractables	Males=294.8 mg/kg bw Females=130.3 mg/kg bw	431270-01
Chem Sect Cube Root Powder Toxicant	8.08% rotenone	Males>1049 mg/kg bw Females>209 mg/kg bw	448492-01

9.1.1.2 Dermal

Technical grade rotenone exhibited negligible acute toxicity from dermal exposure (Table 9.2).

9.1.1.3 Inhalation

Technical grade rotenone is considered highly toxic, on an acute basis, from exposure via inhalation (Table 9.2). Acute inhalation toxicity was one of the bases for classifying certain rotenone products as Restricted Use in the past and for classifying all rotenone end-use products as Restricted Use in the future.

9.1.1.4 Irritation and sensitization

Technical grade rotenone exhibited little tendency for eye irritation and skin sensitization (Table 9.2).

Study Type	% Rotenone	Toxicity	MRID Reference
Acute Dermal – rabbit	97.9%	LD ₅₀ >5000 mg/kg	43907501
Acute Inhalation – rat	98%	LC ₅₀ =0.0212 mg/L combined Males: LC ₅₀ = 0.0235 mg/L Females: LC ₅₀ =0.0194	43882601
Acute Eye Irritation – rabbit	97.9%	Minimal, conjunctival irritation in unwashed eyes; PIS 3.3 at 1 hr, cleared in 24 hrs	43907503
Acute dermal irritation - rabbit	97.9%	PIS 0.08 at 1 hr, decreasing to 0 in 24 hrs	43907504
Acute Skin Sensitization – guinea pig	98%	Not a dermal sensitizer	43817903

9.1.1.5 FIFRA toxicity categories for various exposure routes

All pesticide products are placed in toxicity categories based on acute toxicity data to laboratory mammals. Based upon these categories, a “human hazard signal word” is placed on product labels. The signal word for highly toxic pesticides is “Danger” when any route of exposure is in Category I. In addition, if the pesticide is in Category I because of oral, inhalation, or dermal toxicity (as distinct from skin and eye local effects), then the word “Poison” and a skull and crossbones are added to the label. Category II pesticides have a signal word of “Warning” and Category III and IV pesticides have a signal word of “Caution.” Rotenone exhibits high acute toxicity by both inhalation and oral exposure to warrant the most severe signal words. The categories for different routes of exposure are in Table 9.3.

Route of Exposure	Toxicity Category	Signal Word(s)
Oral	I	Danger, Poison
Dermal	IV	Caution
Inhalation	I	Danger, Poison
Dermal irritation	IV	Caution
Eye irritation	IV	Caution

9.1.2 Pharmacokinetics

Pharmacokinetics is the study of what the body does to a drug that is ingested or taken in by other routes of exposure. Pharmacokinetics includes absorption, distribution, metabolism, and excretion. The rotenone data used by EPA did not include a metabolism study according to guidelines, but one non-guideline study (MRID #00145496) was considered adequate to address key features. USEPA (2006a) makes the following statement about pharmacokinetics:

“In this study, the primary route of excretion was in the feces with polar metabolites being identified in the feces. Metabolic profiles for the seven metabolites found in the feces were not

obtained. In conjunction with fecal elimination, rotenone underwent extensive enterohepatic circulation. Tissue accumulation was low, typically less than 1% of the administered dose. A definitive target organ has not been identified although the mechanism of action is well known. Rotenone uncouples oxidative phosphorylation by blocking electron transport at complex I within the mitochondria. Numerous published literature studies conducted over the past ten years indicate rotenone inhibits the activity of complex I of the mitochondrial electron transfer chain.”

9.1.3 Subchronic toxicity

EPA reported “minimal systemic toxicity” in subchronic and chronic studies (USEPA, 2006a). In studies from oral exposure of rotenone, the end-point effects were reduced body weight or reductions in gain of body weight. The no observed adverse effect levels (NOAEL) was 0.4 mg/kg/day, and the lowest observed adverse effect level (LOAEL) was 2 mg/kg/day in the 90-day dog study. No subchronic oral toxicity studies on rodents were completed for rotenone; the data needed from the 90-day rat study were obtained from the chronic/oncogenicity rat feeding study. See Table 9.4 for a summary of subchronic and chronic studies on rotenone.

There are no available subchronic data on inhalation toxicity, nor are there data on subchronic dermal toxicity. Data on both topics are considered data gaps by USEPA/HED. Of particular concern in the HED review (USEPA, 2006a) was a 21/28-day inhalation study with neurological parameters to be assessed. This study was required through a “Data Call-In” in 2004 (USEPA, 2007). But with the cancellation of all non-piscicidal uses of rotenone, the requirement is now being held in reserve because the concern for inhalation toxicity was based on homeowner and agricultural uses of rotenone, not the piscicidal uses.

9.1.4 Chronic and reproductive toxicity

The two-generation reproduction test is designed to provide information concerning the effects of a test substance on the male and female reproductive systems, and on the development, maturation, and subsequent reproductive capacity of the offspring. In the case of rotenone, as with many other compounds, some of the results may substitute for shorter toxicity tests by the same route of exposure.

In the two-generation rat reproductive study with rotenone, adult and offspring toxicity was indicated by decreased body weight (MRID 00141408). An NOAEL of 7.5mg/kg diet (0.5 and 0.6 mg/kg/day for male and female, respectively) was determined based on decreased F1 and F2 pup body weight and body weight gain. The offspring toxicity LOAEL for rotenone in male and female rats was 35.7 ppm (2.4 and 3.0 mg/kg/day for male and females, respectively), based on decreased bodyweight (10 - 50%) and body weight gain (20 - 60%) in both generations.

There are no available chronic data on inhalation or dermal toxicity of rotenone. These data are not required by EPA for rotenone. A chronic inhalation test had been required, but was waived for the piscicidal uses of rotenone when the home and agricultural uses were cancelled. Because of the potential for multiple applications for these uses, chronic inhalation exposure was likely.

9.1.5 Developmental toxicity

The primary effects noted in developmental studies of rotenone were reductions in maternal body weight gain and lower fetal weights and an increase in resorption of embryos. Rats were more sensitive than mice. Some rats exhibited clinical signs of toxicity (salivation and rubbing the face and paws after treatment) at maternal doses as low as 0.75 mg/kg/day. Rats also had a 24% reduction in live fetuses/litter when dams were exposed to 24 mg/kg/day. No treatment related structural, external, visceral, or skeletal abnormalities were found in fetuses from treated dams.

9.1.6 Mutagenicity and carcinogenicity

Bradbury (1986) presented an analysis of rotenone carcinogenicity studies done through 1983. At that time, most, but not all, studies had been negative for tumors. At least two studies found tumors at lower doses, but not at higher doses as would be expected. EPA convened their Science Advisory Panel in 1988 and presented the available evidence that rotenone might be carcinogenic. The Science Advisory Panel endorsed EPA's classification of rotenone in Group E (evidence of non-carcinogenicity for humans) because of lack of evidence of carcinogenicity in lifetime studies in rats and mice. The EPA Cancer Assessment Review Committee agreed with the Science Advisory Panel and rotenone has been classified as non-carcinogenic since that time (USEPA, 2006a).

In their analysis, the Health Effects Division found no evidence for carcinogenicity in mice or rats, based upon available carcinogenicity studies. Administration of rotenone to both species for up to two years did not result in an increase in overall tumor incidence or increase the incidence of any specific type of tumor. The chemical was negative for gene mutation in two studies with *Salmonella typhimurium* and for mitotic gene conversion with *Saccharomyces cerevisiae*. Micronucleus formation was not induced in the bone marrow of mice. Rotenone also did not cause chromosomal aberrations in CHO cells *in vitro* with or without activation or in bone marrow cells from rats administered up to 7 mg/kg orally. Positive results for gene mutation were obtained only in mouse lymphoma cells, without metabolic activation, at concentrations equal to and below those which also caused significant cytotoxicity. Taken together, the chronic toxicity and mutagenicity studies provide a sufficient basis for classifying rotenone as non-carcinogenic in humans.

Study Type	% Rotenone	Toxicity	MRID Reference
90-day oral toxicity – rat (based on results of two generation study)	97.9%	NOAEL (M/F)=0.5/0.6 mg/kg/day LOAEL (M/F)=2.4/3.0 mg/kg/day	00141408
90-day oral toxicity – dog	>99%	NOAEL = 0.4 mg/kg/day LOAEL = 2 mg/kg/day	00141406
Development toxicity – rat	97-98%	Maternal NOAEL = not identified LOAEL = 0.75 mg/kg/day, Developmental NOAEL = 3 mg/kg/day LOAEL = 6 mg/kg/day	00144294

Table 9.4 Subchronic, Chronic, and Reproductive and Other Toxicity of Rotenone			
Study Type	% Rotenone	Toxicity	MRID Reference
Development toxicity - mouse	98.2%	Maternal NOAEL = 15 mg/kg/day LOAEL = 24 mg/kg/day Developmental NOAEL = 15 mg/kg/day LOAEL = 24 mg/kg/day	00141707
Reproduction - rat	97.9%	Parental systemic: NOAEL (M/F)=0.5/0.6 mg/kg/day LOAEL (M/F)=2.4/3.0 mg/kg/day Reproductive: NOAEL (M/F) 2.4/3.0 mg/kg/day LOAEL (M/F) 4.8/6.2 mg/kg/day Offspring: NOAEL (M/F) 0.5/0.6 mg/kg/day LOAEL (M/F) 2.4/3.0 mg/kg/day	00141408
Carcinogenicity studies (rat)	95% (00143257), NR, NR, NR, 95% (143256)	No evidence of carcinogenicity in 3 rat, 1 mouse, and 1 hamster studies	40179801a&b, 46274301, 00143257, 00143256, 00156739, 41657101
Gene Mutation Salmonella typhimurium)	>98%, >95%	No evidence of mutations	40170506 40170502
Gene Mutation 84-2 870.5100 Mouse lymphoma cells	>98%	Evidence of a positive response of at 0.25-8.0 µg/mL without metabolic activation; significant cytotoxicity noted at 4 and 8 µg/mL.	40170505
Cytogenetics	NR	(1) No evidence of chromosome aberrations, of induced chromatid/chromosome aberrations, no significant increase in frequency of micronuclei.	40179801c 00093702
Micronucleus	NR	Negative	00093702
Mitotic gene conversion	>97%	No evidence of induced mutant colonies	00144292

9.1.7 Neurotoxicity

9.1.7.1 Guideline considerations of neurotoxicity

None of the results from the available studies, except clinical signs in the acute oral toxicity study, showed evidence of neurotoxicity. In acute oral studies, clinical signs included tremors, prostration, labored breathing, and soft feces. Decreased activity, gasping, piloerection, and sensitivity to touch after inhalation exposure were noted. These clinical signs of toxicity are likely the result of the known mechanism of action of rotenone, which is the uncoupling of

oxidative phosphorylation via blocking electron transport at complex I within the mitochondrion. No clinical signs of toxicity were noted in subchronic or chronic studies in dogs, rats, mice, or hamsters.

9.1.7.2 Potential of rotenone to cause Parkinson's disease

The EPA review of rotenone for assessing its eligibility for reregistration (EPA, 2006a) has raised a concern because the extensive research on Parkinson's disease includes a paper that shows a Parkinson's disease-like effect resulting from rotenone exposure (Betarbet et al., 2000). These researchers exposed Lewis rats to rotenone by continuously infusing the rotenone, dissolved in equal volumes of dimethylsulfoxide (DMSO) and polyethylene glycol (PEG), directly into the jugular vein at a rate of 2-3 mg/kg/day for 7 days to over 5 weeks. Of the 25 rats so exposed, 12 developed clear nigrostriatal dopaminergic lesions. The authors do not indicate the relative exposure duration for those rats that exhibited the Parkinson's disease syndrome and those that did not. However, at least some rats exhibited partial lesions after a 7-day exposure.

It is important to understand that researchers of Parkinson's disease had been working with another compound that produced lesions similar to Parkinson's disease, Methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), and 6-hydroxydopamine before that (Hirsch et al., 2003). Since MPTP did not produce all of the effects noted in Parkinson's disease, Ferrante et al. (1997) was apparently the first to try rotenone, by intravenous injection, because of its effects on the complex 1 system. They were able to produce selective damage in the striatum and the globus pallidus, but not in the substantia nigra. Betarbet et al. (2000) also noted that the MPTP similarity was incomplete because MPTP is highly selective for dopaminergic neurons rather than causing a systemic complex 1 defect. Their goal was to develop a better Parkinson's model and rotenone was selected because it was a widely used insecticide and it affected the complex 1 electron transport process. Subsequently, these and many other researchers have published numerous papers using this particular model for Parkinson's disease (e.g., Scherer, et. al., 2002; Scherer, et. al., 2003a; Scherer, et. al., 2003b; Scherer, et. al., 2003c; Betarbet, et. al., 2006; Hirsch et. al., 2003; Gao et. al., 2003; Yang et. al., 2003).

Hirsch et al. (2003) and Riederer et al. (2005) questioned that the rotenone effect was an appropriate Parkinson's model. Hirsch et al. (2003) was unable to reproduce the selective results only on the dopaminergic neurons reported by Betarbet et al. (2000). Rather, they also found effects on non-dopaminergic neurons in several, but not all parts of the brain, concluding that, while MPTP reproduced certain aspects of early idiopathic Parkinson's disease, rotenone caused complex forms of parkinsonism as are observed in multiple system degeneration. In consideration of genetic susceptibility, Hirsch et al. (2003) also raised the issue of variability in responses of different rats. Betarbet et al. (2000) had noted that Sprague-Dawley rats showed more variable and less consistent lesions than Lewis rats; their second stage experiments used only Lewis rats. Hirsch et al. (2003) found that Lewis rats obtained from a CERJ facility in France consistently exhibited neuronal lesions, whereas Lewis rats obtained from a Charles River facility in Italy never did. Although neither MPTP nor rotenone reproduced all of the features of Parkinson's disease, Hirsch et al (2003) concluded that rotenone-induced parkinsonism was a promising model, even with the atypical lesions, and could be used to test neuroprotective strategies for both dopaminergic and non-dopaminergic neurons.

That rotenone-induced parkinsonism is a useful model has been confirmed by such findings as those of Testa, et al., (2005) who determined that vitamin E and presumably other anti-oxidants block the effects of rotenone. Similarly, Jiang, et al. (2006) determined that L-(+)-2-amino-4-phosphonobutyric acid (L-AP4) can reduce rotenone toxicity through its activation of glutamate receptors.

Although rotenone-induced parkinsonism is a useful research tool, Betarbet et al. (2000) cautioned that Rotenone had little toxicity when administered orally. A continuous, intravenous administration of rotenone for 1-5 weeks is not representative of any likely exposure to rotenone. However, EPA (2006a) stated that intravenous injection may mimic the inhalation route of exposure because it is a fairly direct route of exposure that avoids any metabolic breakdown that occurs from gut uptake. A subchronic neurotoxicity study via inhalation was recommended for rotenone because inhalation is a potential route of exposure to rotenone. However, with only piscicidal uses of rotenone remaining, the requirement has been placed “in reserve” since chronic exposure to rotenone is most likely from garden, agricultural, and animal uses. For piscicidal uses, chronic inhalation is likely only for handlers and applicators of rotenone who do not wear the required Protective Personal Equipment. It is also possible that inadvertent overspray could result in inhalation exposure of rotenone, but such an event would be a one-time, acute event because treatment of an individual lake would only re-occur after at least a year, and likely several years. For applicators and other regular handlers of rotenone, the required PPE would preclude any consequential exposure to rotenone, thus removing any possibility of a Parkinson-like effect. This was apparently the same conclusion reached by EPA in waiving the inhalation neurotoxicity study.

The only logical conclusion is that, while rotenone-induced parkinsonism via chronic intravenous injection may be a useful model for studying Parkinson’s disease, there is no risk of Parkinson’s disease for humans from the piscicidal use of rotenone if the label directions, including protective equipment, are followed.

9.1.8 Epidemiology

No epidemiological studies on rotenone have been located.

9.1.9 Incident reports

EPA reported that rotenone had a similar or higher percentage of poisoning incidents reported than other pesticides. In general, the most common symptom reported was eye irritation, which was four times more prevalent than any other symptom. Other symptoms reported included dermal irritation, throat irritation, nausea, and cough/choke. This supports the finding that rotenone’s main effect is due to its irritant properties. Few neurological symptoms, other than headache and dizziness, were reported, though there were a few reports of peripheral neuropathy, numbness, or tremor. Neither fatalities nor systemic poisonings have been reported in relation to “ordinary use.” (USEPA, 2006a). There were reports of fatalities from “intentional” ingestion of rotenone. In one case an adult female drank rotenone in a successful attempt at suicide. In another case in Belgium, a young child swallowed a mouthful of a formulation containing rotenone and a variety of ethereal oils (e.g., oil of cinnamon), and died; rotenone residues found at autopsy seemed to be rather low to be the cause of death (USEPA, 2005).

9.2 Exposure assessment

9.2.1 Exposure routes

9.2.1.1 Swimming

Swimming has been allowed in rotenone-treated water after the rotenone has been thoroughly mixed into and dispersed throughout the water. Swimming can result in exposure through dermal contact as well as inadvertent ingestion of treated water. Based upon standard EPA models, there was no concern for adults swimming in treated water, assuming that 200 ppb would be the maximum concentration based upon solubility of rotenone (despite the maximum label rate being 250 ppb). However, margins of exposure were exceeded for toddlers at concentrations of 90 ppb or above; it was determined that the concentration would fall below 90 ppb after 3 days at 25°C. Therefore, EPA specified in the RED that no swimming could occur for three days after application. Alternatively, swimming may be allowed if 3 consecutive samples of treated water taken at least 4 hours apart all showed concentrations of rotenone to be below 90 ppb. It was assumed that there would be minimal swimming activity when the water temperature is below 25°C.

9.2.1.2 Drinking water

Drinking treated water is another possible route of human exposure. Although incidental ingestion of treated water could occur directly from a lake, the amount would be minimal. EPA determined that acute exposure from drinking water sources was below levels of concern.

EPA determined that chronic exposures of concern could occur for drinking water concentrations above 40 ppb. Because of the variability of rotenone degradation under differing environmental conditions, they acknowledged that they could not develop a risk estimate for chronic exposure using standard models. EPA concluded that it was necessary to be conservative, especially for potential risks to small children. They determined that the current “distance” requirements that drinking water intake locations be more than ½ mile from treated waters were insufficient. EPA did not recommend a specific solution to this situation. Rather, they called upon the registrants of rotenone products to develop a plan that would ensure that drinking water concentrations contained less than 40 ppb of rotenone. Such plans could include altering application procedures or rates, monitoring, or treatment of drinking water. Given that rotenone labels currently indicate that waters treated with rotenone may be detoxified by use of chlorine (potassium permanganate is the first mentioned detoxification chemical), it would appear that any potable water intakes that go through drinking water treatment with chlorine are likely to be below 40 ppb without additional effort.

9.2.1.3 Occupational exposure

Persons working with rotenone may be exposed to the compound at various stages of the application process beginning with those who remove rotenone from commercial packaging, through stages of handling, mixing and loading, and through the application process. EPA determined that occupational exposure should be evaluated on the expectation that dermal exposure would result in 10% uptake and inhalation exposure would result in 100% uptake. Based upon the way rotenone is used, EPA expressed concern over short-term (up to 30 days) and intermediate term (up to several months) occupational exposure, considering that custom

applicators and others may be repeatedly applying rotenone at certain seasons. Long term occupational exposure is not expected.

It is not feasible to discuss the myriad combinations of occupational exposure that may result from individuals doing one, more, or all steps in the application process along with the variety of protections that may affect exposure. EPA did find that a number of combinations exceed the margins of exposure that are considered safe. On this basis, EPA determined that Personal Protective Equipment requirements needed to be strengthened. Details on the proposed requirements are included in section 3.4 and Appendix 2.

9.2.1.4 Other

Exposure through food is not considered a concern following the deletion of all food crops from rotenone labels and the prohibition on eating dead fish from treated waters.

10. Risk Assessment and Characterization for Health Effects

10.1 Drinking water

Current labels do not provide sufficient information to determine the potential drinking water exposure. Labels require that rotenone not be used within ½ mile of potable water intakes, except where such intakes are upstream from treatment areas. This requirement appears to be based on lengthy experience, but no scientific data have been found to support the requirement as being sufficient to protect drinking water, even though it could actually be adequately protective. In the current analysis for reregistration, USEPA (2006a) determined that there was no acute risk to humans from drinking water.

It was acknowledged that no chronic exposures would be expected where rotenone was deactivated with potassium permanganate or where the drinking water was subject to an oxidative drinking water treatment regimen. But because this was only an “expectation,” a data requirement, according to established laboratory protocols, was made to confirm the assumptions.

Further, USEPA (2006a) considered that there could be a chronic risk under certain limited circumstances. First, a “Drinking Water Level of Concern” was established on the basis of chronic rat toxicity. For infants and children, this level of concern was determined to be 40 ppb. They then established that rotenone could persist in cold water “for several weeks.” The narrow circumstances under which levels of concern might be exceeded include:

- Drinking water intakes are near water intakes in cold, lentic waters; “near” was not defined.
- There would be no deactivation by potassium permanganate.
- There would be no oxidative water treatment, such as with chlorine.

To ensure that chronic or sub-chronic exposures above 40 ppb through drinking water will not occur, registrants are required to submit proposed labeling or a monitoring plan to preclude such exposures. Registrants are given several options to achieve this 40 ppb goal, including label

restrictions on where rotenone may be used with respect to water intakes (e.g., a distance between the use site and the intake), or factors of the site where rotenone can be used that might include dilution factors, temperature of water that can be treated, etc., or a monitoring system involving chemical analysis or a sentinel bioassay.

It is noted that the proposed labeling requirements include deactivation of water leaving the treated area by potassium permanganate. Assuming the confirmatory testing mentioned above indicates that deactivation would avoid chronic exposure, there would be no need for the proposed labeling or monitoring plan.

10.2 Fish consumption

Current labels prohibit the use of dead fish for food or animal feed, as do the proposed labeling requirements. If label directions are followed, there will be no consumption of treated fish. In the edible portions of fish, rotenone residues are below 1 ppm. Indigenous peoples have long used the *Derris* and *Lonchocarpus* plants containing rotenone to harvest fish for consumption, and in the past rotenone-poisoned fish have been given to community groups for consumption in the U. S. (Ling, 2003)

10.3 Rotenone exposure from swimming

Exposure to rotenone from swimming in treated water is a function both of dermal toxicity and incidental ingestion of water. The EPA's review of health effects determined that short term risks from swimming did not exceed levels of concern, but that short term risks were a concern for 3-year old toddlers at application rates of 200 ppb and above. Levels of concern for toddlers were not exceeded at rotenone concentrations of 90 ppb and below, considering both the oral and dermal exposure. Considering the dissipation rate in 25°C water, they recommended that swimming be prohibited for at least 2 days following applications at 200 ppb and at least 3 days for applications at 250 ppb (USEPA, 2006a).

The RED specified in the labeling requirements both a reduction in the maximum application rate to 200 ppb and a 72 hour prohibition for swimming or wading (USEPA, 2007). This duration exceeds the minimum 2-day prohibition recommended by the Health Effects Division. The prohibition also applies to adults, who were not considered at risk for swimming, and it applies to wading, where incidental ingestion of treated water would not be a factor.

10.4 Exposure during applications

USEPA (2006d) developed a variety of scenarios for occupational exposure that included mixing, loading, and applying rotenone for both ground and aerial applications. The scenarios included different handling activities, such as mixing or applying, different application equipment and methods, such as boat drip bars or helicopter sprays, different application rates and sites, and different levels of risk mitigation through use of Personal Protective Equipment (PPE) or engineering systems such as closed transfer systems or closed cockpits. Risks exceeded concerns for all scenarios without any risk mitigation, i.e., long-sleeve shirt, long pants, no gloves, and no respirator.

The results of the analysis indicated that certain levels of risk mitigation were sufficient to reduce risks below levels of concern for various activities in the use of liquid rotenone formulations. Occupational exposure of wettable powder formulations was found to exceed levels of concern for most activities, even with significant risk mitigation. Applicator risks were not of concern in closed cockpit or enclosed boat cabins for either kind of formulations. Conversely, the use of backpack sprayers could not be reduced below concern levels for either kind of formulation. The analysis is considered conservative, and is based on a number of assumptions because models and data used to support them are not designed to address applications to water. The HED chapter states, “There are clearly limitations and uncertainties regarding the use of the surrogate data to assess rotenone occupational handler exposure because of the distinct differences in application sites (land vs. water), however, HED can not currently define the extent of these limitations and uncertainties. Actual data for rotenone handler exposure scenarios would provide better worker risk estimates.” (USEPA, 2006a)

The proposed labeling requirements, based on this analysis, are presented in Appendix 2. In summary, these include a requirement for the use of closed systems for mixing and loading, extensive PPE including full face respirators, prohibition of certain application techniques including backpack sprayers for wettable powder formulations. See appendix 2 for details.

There was no concern for occupational exposure from postapplication activities.

10.5 Chronic exposure

With the cancellation of all uses other than piscicidal uses, USEPA (2006a) determined that long term (> 6 months) exposure is not a concern for rotenone. Short-term (1-30 days) and intermediate term (1-6 months) exposure is expected for occupational activities. Some concerns exist for intermediate-term exposure, such as from drinking water, but most exposure to piscicidal uses of rotenone is expected to be short-term.

10.6 Uncertainties

There are two major areas of uncertainty with respect to health effects from rotenone’s piscicidal use. As noted above (section 10.4), many of the parameters for the occupational exposure were extrapolated from other occupational scenarios, since models for aquatic applications are not available. Better scenarios for worker exposure would provide better risk estimates, which in turn, based on HED’s conservative approach, could reduce the need for stringent protective measures for occupational exposure.

The second major uncertainty encompasses a number of features. The HED uses uncertainty factors to quantitatively assess risk. A ten-fold uncertainty factor is used for interspecies extrapolations and another ten-fold for intraspecies extrapolations for the health effects toxicity data; these uncertainty factors apply to most chemicals. For rotenone, an additional ten-fold uncertainty factor was included because the toxicity database is incomplete. The primary data gap is a 21-day neurotoxicity study by inhalation. This study was recommended until the agricultural and home uses of rotenone were cancelled; it is now held in reserve. Additional data gaps, all held in reserve, include a metabolism study, dermal absorption study, and a rabbit developmental study. Additional studies on neurotoxicity via oral exposure and repeated-dose dermal toxicity could be required, pending the results of the other studies.

If the database were complete, then there would be a rationale for removing the extra ten-fold uncertainty factor. Assuming that the results of testing to complete the database did not provide notably unexpected results, it seems likely that most risk concerns other than for occupational exposure could be eliminated, and those for occupational exposure reduced. With reduced concerns for occupational exposure, there would likely be fewer risk mitigations specified for handlers (mixers/loaders/applicators) of rotenone. Absent the results from these studies, it is not possible to delineate what kinds of risk mitigation reductions would be appropriate which would still protect human health.

There are a number of additional uncertainties regarding the nature and applicability of toxicity and human exposure data. Many of these are incorporated into the overall uncertainty factors, but better data would still reduce some of the conservative assumptions made in analyzing these data.

10.7 Conclusions

The primary conclusion regarding health effects is that there are considerable occupational risks for rotenone. These risks have been assessed conservatively by EPA, and are most likely overstated. Even with better data to reduce assumptions and the additional data that could eliminate the database uncertainty factors, some occupational risks would exist. However, it seems that a better and more complete data set would result in fewer and/or less stringent requirements for PPE and closed system requirements, and might allow the use of backpack sprayers for wettable powders.

While there are concerns for non-occupational exposures of rotenone, e.g., drinking water and swimming, the risks are only slightly above levels of concern. Eliminating the ten-fold uncertainty factor for an incomplete database would likely reduce these risks to below levels of concern. It is also possible that some protective measures might be reduced simply by parsing the analysis to a greater degree. For example, swimming is a concern for toddlers because of the combination of dermal and oral exposure. Swimming is not a concern for adults. Yet the proposed labeling requirements would prohibit wading by adults in treated water for 3 days. While this statement is associated with recreational use, as written, the statement applies to any wading. And, as such, it contradicts the re-entry statement requiring full protective clothing for occupational exposure when going into the treated area. The full PPE in the re-entry statement includes coveralls over long-sleeved shirt and long pants, chemical-resistant gloves, chemical resistant footwear plus socks, and chemical-resistant apron (see Appendix 2). This seems quite excessive, considering that the HED analysis (US EPA, 2006a) indicates no concern for swimming by adults after the rotenone has been mixed

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Appendix 1

This appendix provides information based on current labels. Label changes are expected as a result of the requirements proposed in the rotenone RED. There are similarities and differences in various aspects of the different rotenone products. Those factors specific to a product, or several products, are included in Table App-1. Some features are common to all products or all products of a certain type. These include:

1. All uses as a fish toxicant are classified as restricted use for certified applicators trained to use fish toxicants specifically. Restricted use classification for all rotenone is based upon aquatic toxicity and acute inhalation toxicity to mammals. In addition, for powders there is also a basis from acute oral toxicity to mammals.
2. All Rotenone labels prohibit use of treated water to irrigate crops; application is prohibited within ½ mile upstream from potable water or irrigation water intakes.
3. For use in streams and rivers (other than as immediately above treated lakes, ponds and reservoirs) rotenone labels state: “Only state or federal Fish & Wildlife personnel or professional fisheries biologists under the authorization of state or federal Fish & Wildlife agencies are permitted to make applications [of this product] for control of fish in streams and rivers. Informal consultation with Fish & Wildlife personnel regarding the potential occurrence of endangered species in areas to be treated should take place.”
4. Except for bait products, all rotenone labels prohibit swimming until after application is complete and all pesticide has been thoroughly mixed into the water.
5. Rotenone labels of emulsifiable liquids have a signal word of “Danger,” or “Danger, Poison.” The Hazards to Humans and Domestic Animals statement reads (with minor variation in wording among products) “Fatal if inhaled. May be fatal if swallowed. Harmful if absorbed through the skin. Causes substantial but temporary eye injury. Causes skin irritation. Do not breathe spray mist. Do not get in eyes, or skin or on clothing. Wear goggles or safety glasses. When working with undiluted product, wear either a respirator with an organic-vapor-removing cartridge with a prefilter approved for pesticides [additional details on respirator not included here]. Wash thoroughly with soap and water after handling and before eating, drinking, or using tobacco. Remove contaminated clothing and wash before reuse.”
6. Rotenone labels of powders/dusts have signal words of “Warning,” “Danger,” or “Danger/Poison”. The Hazards to Humans and Domestic Animals statement reads (with minor variation among products), “Fatal if inhaled or swallowed. Harmful if absorbed through the skin. Causes moderate eye irritation. Prolonged or frequently repeated skin contact may cause allergic reactions in some individuals. Do not breathe dust. Use a dust filtering respirator [details of one or more described]. Avoid contact with skin, eyes, or clothing. Wash thoroughly with soap and water after handling and before eating, drinking, or using tobacco. Remove contaminated clothing and wash before reuse.”
7. Rotenone labels of baits have a signal word of “Caution.” The Hazards to Humans and Domestic Animals statement for both products reads, “Harmful if swallowed or absorbed through the skin. Causes moderate eye irritation. Avoid contact with skin, eyes or clothing. Wash thoroughly with soap and water after handling and before eating, drinking, or using tobacco.”
8. All rotenone labels have fish warning statements identical or very similar to: “This pesticide is extremely toxic to fish. Fish kills are expected at recommended rates.

Consult your State Fish and Game Agency before applying this product to public waters to determine if permit is needed for such an application. Do not contaminate untreated water when disposing of equipment wash waters.” (from TIFA 1439-157)

9. All labels, except for bait products, indicate that waters are safe for restocking when fish to be stocked survive for 24 hours after being put in live cages and placed in the coolest part of the water. Most labels say this will be in 2-4 weeks; several are silent on duration, and Chem Fish Regular indicates 3-5 days may be sufficient.
10. All labels, except for bait products and Prentox Rotenone Fish Toxicant Powder, Reg. #655-691, have a detoxification statement that reads, “In lakes, ponds, and reservoirs, natural detoxification occurs in one week to one month, depending on water chemistry. Addition of chlorine or potassium permanganate can hasten detoxification.” Some labels add that the potassium permanganate should be at a concentration of 2-4 ppm. A few labels also indicate detoxification can be determined by exposing fish in live cages.

Please note that the RED (USEPA, 2007) will result in changes to all rotenone product labels. The changes may be extensive; they should also result in more standardization on label statements among the various products. See section 3.4 and appendix 2 for details.

Table App-1. Product Label Information for Rotenone End-use Formulations

Product name, Company, EPA Reg #, Product Type	percent ai (%rotenone/ % other cube resins)	sites	application methods - lakes, ponds and reservoirs	application methods - streams immediately above lakes ponds and reservoirs	application methods - streams and rivers	notes
Cube Powder Fish Toxicant Foreign Domestic Chemicals Corp 6458-6 powder	7.4%/11.1%	Lakes, reservoirs and ponds	Mix 1 pound of product with 3-10 gal water. Uniformly apply over surface or bubble through underwater lines.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. Then the label states, "See the use directions for streams and rivers on this label for proper application instructions." However, the available label has no instructions for rivers and streams, and the label does not indicate that rivers and streams, other than immediately above treated lakes, etc., are an allowed use site.	not a registered use for this label	
Synpren Fish Toxicant Prentiss 655-421 liquid-emulsifiable	2.5%/5% + 2.5% piperonyl butoxide	Lakes, reservoirs, ponds, and streams	For still waters, mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. The label directs the user to follow the application method for streams and rivers.	For slow moving rivers use application methods as for lakes, ponds, and reservoirs: Mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines. For flowing streams and rivers, apply as a drip for 4-8 hours to the flowing portion of the stream, with application sites every 1/2 to 2 miles apart, depending upon the flow rate. Application sites are spaced at no more than 2 hours or at no less than 1 hour travel time intervals, to ensure a 2-hour minimum exposure time for fish.	directions provided for slow moving rivers and streams, but none for moderate or fast-moving rivers.
Prenfish Toxicant Prentiss 655-422 liquid-emulsifiable	5%/5%	Lakes, reservoirs, ponds, and streams	For still waters, mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a	For slow moving rivers use application methods as for lakes, ponds, and reservoirs: Mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines. For flowing	

Table App-1. Product Label Information for Rotenone End-use Formulations

Product name, Company, EPA Reg #, Product Type	percent ai (%rotenone/ % other cube resins)	sites	application methods - lakes, ponds and reservoirs	application methods - streams immediately above lakes ponds and reservoirs	application methods - streams and rivers	notes
				calculation of the amount of product to use. The label directs the user to follow the application method for streams and rivers.	streams and rivers, apply as a drip for 4-8 hours to the flowing portion of the stream, with application sites every 1/2 to 2 miles apart, depending upon the flow rate. Application sites are spaced at no more than 2 hours or at no less than 1 hour travel time intervals, to ensure a 2-hour minimum exposure time for fish.	
Prentox Rotenone Fish Toxicant Powder Prentiss 655-691 powder	8.74%/13.11 %	Lakes and ponds	For still waters, mix 1 pound product with 3-10 gal water. Uniformly apply over surface or bubble through underwater lines. Alternatively, place undiluted powder in a burlap sack and trail behind boat; put weights on bag in deeper water, 20-25 feet deep.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. Then the label states, "follow the application method."	not a registered use for this label	Methods for treatment of streams immediately above treated lakes, ponds, and reservoirs is not clear. The user is directed to "follow the application method" which is presented on this label only for lakes, etc. For labels that allow treatment of rivers and streams, in general, the labels state that the user should follow the application method for streams and rivers.
Prentox Prenfish Grass Carp Management Bait Prentiss 655-795 bait	2.64%/3.36% + 0.5% piperonyl butoxide	Lakes, reservoirs and ponds	Application is by feeding bait pellets to "trained grass carp". No specific application rate is provided, but the amount used should be the same as the amount provided in training. The label notes that one pellet contains enough rotenone to kill a typical 1 Kg fish.	not applicable	not applicable	For selective use against grass carp that have been trained to accept food pellets at designated feeding stations. It is believed that the pellets are not toxic to fish in the water that do not consume the pellets. Food pellets for training should be attractive to grass carp, but not to other fish not intended for removal (Prentiss makes such a "training bait".) This label appears to be essentially the same as for grass carp but the image is only partially available. There is a different training bait for the common carp.
Prentox Prenfish & Common Carp Management Bait Prentiss 655-803 bait	2.64%/3.33% + 0.5% piperonyl butoxide	Lakes, reservoirs and ponds	Application is by feeding bait pellets to "trained common carp". No specific application rate is provided, but the amount used should	not applicable	not applicable	

Table App-1. Product Label Information for Rotenone End-use Formulations

Product name, Company, EPA Reg #, Product Type	percent ai (%rotenone/ % other cube resins)	sites	application methods - lakes, ponds and reservoirs	application methods - streams immediately above lakes ponds and reservoirs	application methods - streams and rivers	notes
NUSYN-NOXFISH Fish Toxicant Prentiss 655-804 liquid-emulsifiable	2.5%/2.5% + 2.5% piperonyl butoxide	Lakes, reservoirs, ponds, and streams	be the same as the amount provided in training. The label notes that one pellet contains enough rotenone to kill a typical 1 Kg fish. For still waters, mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. The label directs the user to follow the application method for streams and rivers.	For slow moving rivers use application methods as for lakes, ponds, and reservoirs: Mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines. For flowing streams and rivers, apply as a drip for 4-8 hours to the flowing portion of the stream, with application sites every 1/2 to 2 miles apart, depending upon the flow rate. Application sites are spaced at no more than 2 hours or at no less than 1 hour travel time intervals, to ensure a 2-hour minimum exposure time for fish.	
NOXFISH Fish Toxicant Liquid emulsifiable Prentiss 655-805 liquid-emulsifiable	5%/5%	Lakes, reservoirs, ponds, and streams	For still waters. mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. The label directs the user to follow the application method for streams and rivers.	For slow moving rivers use application methods as for lakes, ponds, and reservoirs: Mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines. For flowing streams and rivers, apply as a drip for 4-8 hours to the flowing portion of the stream, with application sites every 1/2 to 2 miles apart, depending upon the flow rate. Application sites are spaced at no more than 2 hours or at no less than 1 hour travel time intervals, to ensure a 2-hour minimum exposure time for fish.	appears to be identical with 655-422
Cube Powder Fish Toxicant	7.4%/11.1%	Lakes, reservoirs,	Mix 1 pound of product with 3-10 gal water.	Select the treatment concentration to achieve the desired effect and	For slow moving rivers, apply as a drip for 4-8 hours to the	directions provided for slow moving rivers and streams,

Table App-1. Product Label Information for Rotenone End-use Formulations

Product name, Company, EPA Reg #, Product Type	percent ai (%rotenone/ % other cube resins)	sites	application methods - lakes, ponds and reservoirs	application methods - streams immediately above lakes ponds and reservoirs	application methods - streams and rivers	notes
Prentiss 655-806 powder		ponds, and streams	Uniformly apply over surface or bubble through underwater lines.	calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. The label directs the user to follow the application method for streams and rivers.	flowing portion of the stream, with application sites every 1/2 to 2 miles apart, depending upon the flow rate. Application sites are spaced at no more than 2 hours or at no less than 1 hour travel time intervals, to ensure a 2-hour minimum exposure time for fish.	but none for moderate or fast-moving rivers.
Sure-gard Powdered Cube Value Garden Supply 769-414 powder	5.0%/7.5%	Lakes and ponds	Distribute Powdered Cube evenly over the surface of the water and agitate to mix thoroughly. This may be accomplished by spraying on the surface and running back and forth with an outboard motor boat or a slurry can be prepared and poured directly behind the outboard motor or put material in a burlap sack and trail behind motor boat.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. Then prepare a slurry of Powdered Cube and pour directly into the center of stream with the amount of product and at the application rate as calculated in the instructions.	not a registered use for this label	
Drexel 19713-316 Drexel 7.5% Rotenone powder	7.4%/11.1%	Lakes, reservoirs and ponds	Mix 1 pound of product with 3-10 gal water. Uniformly apply over surface or bubble through underwater lines.	Select the treatment concentration to achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. Then the label states, "See the use directions for streams and rivers on this label for proper application instructions." However, the available label has no instructions for rivers and streams, and the label does not indicate that rivers and streams, other than immediately above treated lakes, etc., are an allowed use site.	not a registered use for this label	
CFT Legumine	5%/5%	Lakes,	For still waters, mix 1	Select the treatment concentration to	For slow moving rivers use	manufactured by Prentiss

Table App-1. Product Label Information for Rotenone End-use Formulations

Product name, Company, EPA Reg #, Product Type	percent ai (%rotenone/ % other cube resins)	sites	application methods - lakes, ponds and reservoirs	application methods - streams immediately above lakes ponds and reservoirs	application methods - streams and rivers	notes
CWE Properties 75338-2 not specifically stated, but appears to be liquid-emulsifiable		reservoirs, ponds, and streams	gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines.	achieve the desired effect and calculate the flow rate of the stream to calculate the application rate. Then determine exposure time to keep fish in lake from moving upstream which allows for a calculation of the amount of product to use. The label directs the user to follow the application method for streams and rivers.	application methods as for lakes, ponds, and reservoirs: Mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines. For flowing streams and rivers, apply as a drip for 4-8 hours to the flowing portion of the stream, with application sites every 1/2 to 2 miles apart, depending upon the flow rate. Application sites are spaced at no more than 2 hours or at no less than 1 hour travel time intervals, to ensure a 2-hour minimum exposure time for fish.	
Chem-Sect Brand, Chem Fish Regular TIFA Ltd. 82397-1 (was 1439-157) emulsifiable concentrate	5%/5%	Lakes and ponds	Mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines.	Allow Chem Fish Regular to drain from drum directly into center of stream at a rate of 0.85-1.7 cc per minute for each cubic foot of water flowing per second in the stream (to achieve 25-50 ppb of rotenone).	There is an introduction for use in rivers and streams, other than as immediately above treated lakes etc, but there are no further instructions. The label indicates use only for lakes and ponds.	The label statement on restocking is inconsistent with the label statement on detoxification.
Chem-Sect Brand, Chem Fish Synergized TIFA Ltd. 82397-2 (was 1439-159) emulsifiable concentrate	2.5%/2.5% + 2.5% piperonyl butoxide	Lakes and ponds	Mix 1 gal product with 10 gal water. Uniformly apply over surface or bubble through underwater lines.	Allow Chem Fish Synergized to drain from drum directly into center of stream at a rate of 0.85-1.7 cc per minute for each cubic foot of water flowing per second in the stream (to achieve 25-50 ppb of rotenone.)	not a registered use for this label	The label statement on restocking is inconsistent with the label statement on detoxification.
Chem-Sect Brand, Cube Powder Fish Toxicant TIFA Ltd. 82397-5 (was 1439-260) powder	7.4%/11.1%	Lakes, reservoirs and ponds	Mix 1 pound of product with 3-10 gal water. Uniformly apply over surface or bubble through underwater lines.	Prepare a premix by adding the calculated amount of product into a drum and add 3-10 gallons of water. Allow the premix to drain from the drum directly into the center of stream at the rate calculated.	not a registered use for this label	

Appendix 2

Table App-2. Label Changes Summary Table for Rotenone End-Use Products Intended for Occupational and Residential Use, from USEPA, 2007	
Description	Amended Labeling Language for End-Use Products
RUP	<p>“Restricted Use Pesticide”</p> <p>“Due to acute inhalation and acute oral toxicity and due to toxicity to fish and other aquatic organisms.”</p> <p>“For retail sale to and use by only Certified Applicators or persons under their direct supervision and only for those uses covered by the Certified Applicator’s certification.”</p>
SOP Manual	<p>“THIS PRODUCT MUST BE ACCOMPANIED BY AN EPA-APPROVED PRODUCT LABEL AND THE EPA-APPROVED ‘ROTENONE STANDARD OPERATING PROCEDURES MANUAL.’ THE ROTENONE STANDARD OPERATING PROCEDURES (SOP) MANUAL IS LABELING. READ AND UNDERSTAND THE ENTIRE LABELING AND SOP MANUAL PRIOR TO USE. ALL PARTS OF THE LABELING AND SOP MANUAL ARE EQUALLY IMPORTANT FOR SAFE AND EFFECTIVE USE OF THIS PRODUCT.”</p>
PPE Requirements Established by the RED for all Formulations	<p>“Personal Protective Equipment (PPE)”</p> <p>“Some materials that are chemical-resistant to this product are” [EUP registrant, insert correct chemical-resistant material]. “If you want more options, follow the instructions for category” [EUP registrant, insert A, B, C, D, E, F, G, or H] “on an EPA chemical-resistance category selection chart.”</p> <p>“All mixers, loaders, applicators (except pilots), and other handlers must wear, at a minimum, the following PPE:</p> <ul style="list-style-type: none"> * coveralls over long-sleeved shirt and long pants, * chemical-resistant gloves, * chemical resistant footwear plus socks, and * a NIOSH-approved tight-fitting full-face cartridge or canister respirator with any N, R, P, or HE filter; or a NIOSH-approved helmet or hood-style respirator with a dust/mist filter with MSHA/NIOSH approval number prefix TC-21C.” <p>“In addition, mixers, loaders, and others exposed to the concentrate, through cleaning equipment or spills must wear:</p> <ul style="list-style-type: none"> * chemical-resistant apron.” <p>[EUP registrant, drop the “N” type prefilter from the respirator statement if the pesticide product contains or is used with oil.]</p> <p>“See Engineering Controls for additional requirements and exceptions.”</p>
User Safety Requirements	<p>“Follow manufacturer's instructions for cleaning/maintaining PPE. If no such instructions for washables exist, use detergent and hot water. Keep and wash PPE separately from other laundry.”</p> <p>“Discard clothing and other absorbent materials that have been drenched or heavily contaminated with this product’s concentrate. Do not reuse them.”</p>

Table App-2. Label Changes Summary Table for Rotenone End-Use Products Intended for Occupational and Residential Use, from USEPA, 2007	
Description	Amended Labeling Language for End-Use Products
Engineering Controls for Mixing/Loading Liquid Formulations	<p>“Engineering Controls for Mixing and Loading”</p> <p>“Mixers and loaders (except mixing/loading to support backpack sprayers) must use a closed system that is designed by the manufacturer to remove the product from the shipping container and transfer the product into mixing tanks and/or application equipment. At any disconnect point, the system must be equipped with a dry disconnect or dry couple shut-off device that will limit drippage to no more than 2 ml per disconnect. The closed mixing/loading system must function properly and be used and maintained in accordance with the manufacturer’s written operating instructions. Mixers and loaders must wear the personal protective equipment required on this labeling for mixers/loaders.”</p>
Engineering Controls for Applying Liquid Formulations	<p>“Applications using a boom or other mechanized equipment must release this product below the water’s surface. Applications made with aircraft or with a backpack sprayer or other hand-held nozzle or equipment may release this product above the water’s surface.”</p>
Engineering Controls for Mixing/Loading Wettable Powder Formulations	<p>“Engineering Controls for Mixing and Loading”</p> <p>“Mixers and loaders must use a closed system. The system must be capable of removing the product from the shipping container, transferring the product into mixing tanks and/or application equipment, and applying the product below the water’s surface. At any disconnect point, the system must be equipped with a dry disconnect or dry couple shut-off device that will limit drippage to no more than 2 ml per disconnect. In addition, mixers and loaders must wear the personal protective equipment required on this labeling for mixers/loaders.”</p>
Engineering Controls for Applying Wettable Powder Formulations	<p>“Applications using a boom or other mechanized equipment must release this product below the water’s surface. Applications with a backpack sprayer are prohibited. Applications made with other hand-held nozzles or equipment or with aircraft may release this product above the water’s surface.”</p>
Engineering Controls for Aerial Applicators	<p>“Engineering Controls for Aerial Applications”</p> <p>“Open cockpits are prohibited. Pilots must use a cockpit that has a nonporous barrier that totally surrounds the cockpit occupants and prevents contact with pesticides outside the enclosed area. Pilots in enclosed cockpits may wear a long-sleeve shirt, long pants, shoes, and socks, instead of the PPE required for applicators in the PPE section of this labeling.”</p>
Engineering Controls Exception for Boat Applications	<p>“Engineering Controls for Boat Applications”</p> <p>“When boat pilots or others on the application boat are located within an enclosed area that has a nonporous barrier that totally surrounds the occupants and prevents contact with pesticides outside the enclosed area, they:</p>

Table App-2. Label Changes Summary Table for Rotenone End-Use Products Intended for Occupational and Residential Use, from USEPA, 2007	
Description	Amended Labeling Language for End-Use Products
	<p>* may wear a long-sleeve shirt, long pants, shoes, and socks, instead of the PPE required for applicators in the PPE section of this labeling.</p> <p>* must be provided and have immediately available for use in an emergency when they must exit the enclosed area while application is taking place, the PPE required for applicators in the PPE section of this labeling;</p> <p>* must take off any PPE that was worn while outside the enclosed area before reentering the enclosed area, and</p> <p>* store all such used PPE in a chemical-resistant container, such as a plastic bag, to prevent contamination of the inside of the enclosed area.”</p>
User Safety Recommendations	<p>“User Safety Recommendations”</p> <p>“Certified Applicators applying or supervising the application of this product should attend a training program for piscicide applications.”</p> <p>“Users should wash hands before eating, drinking, chewing gum, using tobacco, or using the toilet.”</p> <p>“Users should remove clothing/PPE immediately if pesticide gets inside. Then wash thoroughly and put on clean clothing.”</p> <p>“Users should remove PPE immediately after handling this product. Wash the outside of gloves before removing. As soon as possible, wash thoroughly and change into clean clothing.”</p>
Environmental Hazards	<p>“Environmental Hazards”</p> <p>“This product is extremely toxic to fish and other aquatic organisms.”</p> <p>“Do not contaminate water by cleaning of equipment or disposal of equipment wash waters.”</p> <p>“Do not contaminate water, food, or feed by storage or disposal.”</p> <p>“Do not discharge effluent containing this pesticide into sewage systems without notifying the sewage treatment plant authority (POTW).”</p>
Personal Protective Equipment When Re-entering Treated Areas	<p>“Re-entering the Treatment Area”</p> <p>“For the first 72 hours after treatment, handlers re-entering the treatment area, including shorelines, must wear, at a minimum, the following PPE:</p> <ul style="list-style-type: none"> * Coveralls over long-sleeved shirt and long pants, * Chemical-resistant gloves, * Chemical resistant footwear plus socks, and * Chemical-resistant apron.”
Complete and Partial kills	<p>“Complete and Partial Kills”</p> <p>“This product may be used to achieve a ‘complete kill’ or a ‘partial kill.’ Complete kills are intended to eliminate all fish in the treatment area whereas partial kills are intended eliminate or reduce the number of only certain (more vulnerable) species or</p>

Table App-2. Label Changes Summary Table for Rotenone End-Use Products Intended for Occupational and Residential Use, from USEPA, 2007	
Description	Amended Labeling Language for End-Use Products
	to sample fish populations. Detailed instructions for conducting complete and partial kills are presented in the Rotenone SOP Manual.”
General Application Restrictions for all Formulations	<p>“The Certified Applicator supervising the treatment must remain on-site for the duration of the application.”</p> <p>“Do not allow recreational access (e.g., wading, swimming, boating, fishing) within the treatment area while rotenone is being applied.”</p> <p>“In lakes/reservoirs/ponds, do not apply this product in a way that will result in treatment concentrations greater than 200 parts per billion.”</p> <p>“In streams/rivers, do not apply this product in a way that will result in treatment concentrations greater than 50 parts per billion.”</p> <p>“Do not apply this product in a way that will contact workers or other persons, either directly or through drift. Only protected handlers may be in the area during application.”</p> <p>"This product must not be applied to estuarine or marine environments."</p> <p>“Where practical, users should collect and bury dead fish on the surface of the treatment area.”</p>
General Application Restrictions for Liquid Formulations	“Applications using a boom or other mechanized equipment must release this product below the water’s surface. Applications made with aircraft or with a backpack sprayer or other hand-held nozzle or equipment may release this product above the water’s surface.”
General Application Restrictions for Wettable Powder Formulations	“Applications using a boom or other mechanized equipment must release this product below the water’s surface. Applications with a backpack sprayer are prohibited. Applications made with other hand-held nozzles or equipment or with aircraft may release this product above the water’s surface.”
Monitoring Requirements for Use in Aquaculture	<p>“For treated water bodies used for food production (aquaculture), the Certified Applicator or designee under his/her direct supervision must prohibit restocking of fish until monitoring samples confirm rotenone concentrations are below the level of detection for 3 consecutive samples taken no less than 4 hours apart.”</p> <p>“Detailed instructions for monitoring levels of rotenone in water are presented in the Rotenone SOP Manual.”</p>
Drinking Water Notification Requirements	<p>“Drinking Water Notification”</p> <p>If drinking water intakes are present within the treatment area, prior to application, the Certified Applicator must provide notification to the party responsible for the public water supply or to individual private water users.</p> <p>“Detailed instructions for notifications are presented in the Rotenone SOP Manual.”</p>
Notification Requirements for all	“Placarding of Treatment Areas”

Table App-2. Label Changes Summary Table for Rotenone End-Use Products Intended for Occupational and Residential Use, from USEPA, 2007	
Description	Amended Labeling Language for End-Use Products
applications	<p>“The Certified Applicator in charge of the application (or someone under his/her supervision) must placard all access areas to the treatment area. Detailed instructions for placarding are presented in the Rotenone SOP Manual. At a minimum, placards must be placed every 250 feet (including the shoreline of the treated area and up to 250 feet of shoreline past the application site to include immediate public access points) and contain the following information:”</p> <p>“NOTICE: AREA CLOSURE”</p> <ul style="list-style-type: none"> * Skull and crossbones symbol * “DANGER/PELIGRO” * “DO NOT ENTER/NO ENTRE: Pesticide Application” * The name of the product applied * The agency or entity performing the application * The purpose of the application * The start date and time of application * The end date and time of application * The duration of the area closure * “Recreational access (e.g., wading, swimming, boating, fishing) within the treatment area is prohibited while rotenone is being applied.” * “Do not swim or wade in treated water for a minimum of 72 hours after the last application.” * “Do not consume dead fish from treated water.” * The name, address, and telephone number of the Certified Applicator in charge of the application <p>“Signs must remain legible during the entire posting period and must be removed no earlier than 3 days after treatment and no later than 14 days after treatment.”</p>
Deactivation with Potassium Permanganate	<p>“Deactivation with Potassium Permanganate”</p> <p>“Effluent water must be deactivated with potassium permanganate to prevent exposure beyond the defined treatment area. Detailed instructions for deactivation with potassium permanganate are presented in the Rotenone SOP Manual.”</p>
Spray Drift Label Language for Products Applied as an Aerial Spray	<p>RELEASE HEIGHT: “Do not release spray at a height greater than 10 feet above the water.”</p> <p>BOOM LENGTH: “The boom length must not exceed 75% of the wingspan or 90% of the rotor blade diameter.”</p> <p>SWATH ADJUSTMENT: “When applications are made with a cross-wind, the swath will be displaced downwind. The applicator must compensate for this displacement at the downwind edge of the application area by adjusting the path of the aircraft upwind. Leave at least one swath unsprayed at the downwind edge of the treated area.”</p> <p>DROPLET SIZE:</p>

Table App-2. Label Changes Summary Table for Rotenone End-Use Products Intended for Occupational and Residential Use, from USEPA, 2007	
Description	Amended Labeling Language for End-Use Products
	<p>“Apply as a medium or coarser spray (ASAE standard 572).”</p> <p>WIND SPEE: “Do not apply when wind speeds are greater than 12 miles per hour.”</p>

Attachments:

Labels

MSDS sheets