APPENDIX G: BACKGROUND INFORMATION AND EFFECTS OF ROTENONE ON ECOLOGICAL HEALTH

Rotenone and Antimycin A Regulatory History

Rotenone and antimycin A were first registered as piscicides with the US Environmental Protection Agency (EPA) in 1947 and 1960, respectively (EPA 2007A, 2007B). In 1988, the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) was amended to facilitate reregistration of products with active ingredients registered prior to November 1, 1984. The reregistration process thoroughly reviews the data on which a pesticide's registration is based, with a purpose to "reassess the potential hazards arising from the currently registered uses of a pesticide, to determine the need for additional data on health and environmental effects, and to determine whether or not the pesticide meets the "no unreasonable adverse effects" criteria of FIFRA."

In 2007, following comprehensive ecological and human health risk assessments conducted by the EPA, both rotenone and antimycin A were declared eligible for reregistration as restricted-use pesticides, but only for piscicidal use (EPA 2007A, 2007B). All other past EPA-registered uses for rotenone, including livestock, residential, home owner, domestic pet, and others, were voluntarily cancelled by the three current manufacturers of commercial rotenone products (Prentiss Incorporated, Foreign Domestic Chemicals Corporation, and Tifa International LLC).

Following these decisions, a specific formulation of rotenone, CFT Legumine™, was reregistered by the California Department of Pesticide Regulation (CDPR) for applications targeting fish in California waters (CDPR 2007). CFT Legumine™ is the newest formulation of rotenone, designed to be more benign relative to ecological and human health than older formulations. Antimycin A, however, is not currently registered by CDPR for use in California, due to the inability of the manufacturer to generate health and safety data required by the state (Finlayson B., pers. comm., 2007). The remainder of this section therefore summarizes what is known about rotenone and analyzes piscicidal use of CFT Legumine™ as a management tool.

Rotenone Origin and Use

Rotenone is a natural toxin derived from the roots of several leguminous plants, including *Derris* spp., *Lonchocarpus* spp., and *Tephrosia* spp., which are primarily found in Southeast Asia, South America, and East Africa, respectively (EPA 2007A). Because rotenone is highly toxic to fish and other aquatic life but only slightly toxic to birds and mammals, it has been used by humans for centuries to capture fish for food, and for more than 150 years as a commercial insecticide (Ling 2003). Published literature on rotenone is extensive and long-ranging, with 475 papers on insecticide use known in 1932 (Roark 1932). Since 1990, more than 1,000 papers have been published, with recent interest focusing on biochemical and possible anticancer properties (Ling 2003). Since rotenone is now considered one of the most environmentally benign piscicides available (Ling 2003), it has been used extensively to manage and research fish populations for more than 70 years, with the majority of piscicide applications in North America involving the use of rotenone (McClay 2000). To address recent public concern about piscicidal use of rotenone, a stewardship program was established by the American Fisheries Society to develop safe rotenone practices and encourage good planning and public involvement in future rotenone programs (AFS 2000).

The empirical formula and chemical name for rotenone are $C_{23}H_{22}O_6$ and (2R,6aS,12aS)-1,2,6,6a,12,12a-hexahydro-2-isopropenyl-8,9-dimethoxychromeno[3,4-b]furo[2,3-h]chromen-6-one, respectively. The chemical structure of rotenone is shown in Figure 15 (from Ling 2003), and its CAS number, a unique numerical identifier assigned by the Chemical Abstracts Service, is 83-79-4.

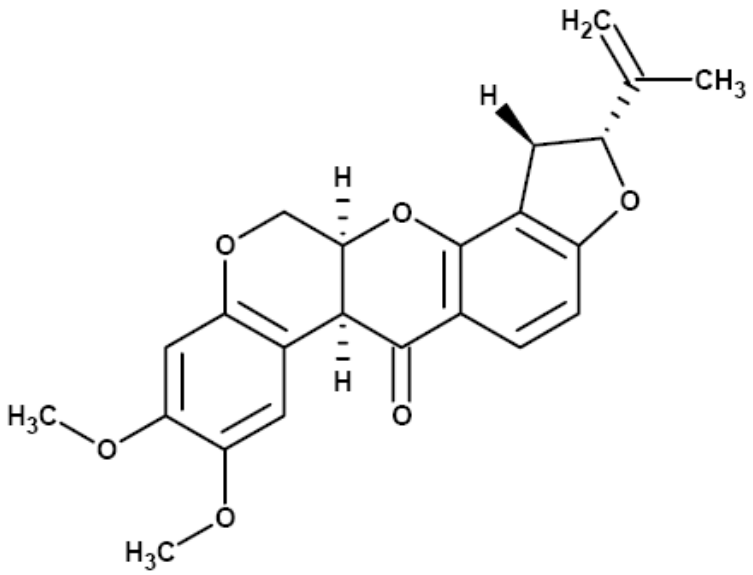

Figure 1. Chemical structure of rotenone.

The actual process by which rotenone stuns and eventually poisons fish - by disrupting cellular aerobic respiration - was only recently determined. First, rotenone blocks mitochondrial electron transport at complex I, resulting in a severance of oxidative phosphorylation (Singer and Ramsay 1994). This blocks oxygen uptake, greatly reduces cellular energy production, and increases blood pO_2. In turn, increases in cellular anaerobic metabolism and lactic acid production cause blood acidosis (Fajt & Grizzle 1998), and fatality ultimately results from tissue anoxia (Ling 2003). Because fish quickly absorb rotenone across gill surfaces, they are extremely sensitive to rotenone poisoning. Although sensitivity varies by taxa, many taxa including trout die within hours at concentrations below 1 part per million (ppm; Ling 2003).

Ingredients in CFT Legumine™

Laboratory analyses done on CFT Legumine™ batches used in a recent rotenone treatment in Lake Davis, California (CDFW 2007) show the active ingredient rotenone as 5% of the formulation. Additional main ingredients as described on the label and determined in this analysis were: methyl pyrrolidone, diethylene glycol monoethyl ether, fatty acid esters, and polyethylene glycols. These additives are necessary to make rotenone soluble in water. Several trace compounds were also detected, including naphthalene, substituted benzenes, and hexanol.

EPA (2007A) is limiting CFT Legumine™ applications to a rate of 1 ppm in flowing water and 4 ppm in standing water. At a CFT Legumine™ application rate of 1 ppm, the rotenone itself is initially present at 50 parts per billion (ppb; 1 ppm x 5% rotenone = 0.05 ppm = 50 ppb). For context, a ppb is equal to one part of a substance to a billion parts of water, or "one billionth." An example would be one ppb of Interstate 80 between New York and San Francisco (about 3,000 mi / 4,800 km) is less than ¼ inch (CDFW 2007). The trace compounds are initially present at a few ppb at the greatest, and many are not detectable in the water immediately after the rotenone is applied (CDFW 2007).

Rotenone Environmental Fate and Persistence

Rotenone mixtures, including CFT Legumine™, are chemically unstable when exposed to light, heat, and air, degrading rapidly into water-soluble, non-toxic components (Ling 2003). When applied in water, the EPA (2007A) concluded that rotenone 1) generally degrades quickly through abiotic (hydrolytic and photolytic) mechanisms, 2) is mobile in soil and sediment, and 3) has limited volatility due to its low vapor pressure and Henry's Law constant, and therefore is not persistent in the environment and has relatively low potential for bioconcentrating in aquatic organisms. Although the EPA (2007A) did not

analyze rotenone degradation through biotic mechanisms due to limited data availability, Bettoli and Maceina (1996) stated that rotenone applications can be detoxified by abundant vegetation through adsorption. Similarly, all of the compounds identified in CFT Legumine™ (rotenone and additives) are rapidly biodegraded, hydrolyzed and/or degraded by sunlight, and thus are not persistent and will not bioaccumulate in the environment (CDFW 2007).

Rotenone degradation varies depending on water temperature, however, with half-lives ranging from a few hours in summer to a few weeks in winter (Ling 2003). Summer rotenone applications should therefore strive to treat entire project areas as simultaneously as possible, or rapid breakdown may allow fish to survive and migrate back into previously treated areas. In addition, rotenone products must be stored sealed in a cool dark place or will lose much of their toxicity within weeks (Cheng et al. 1972). If rotenone products end up detoxifying in storage, the breakdown products become comparatively non-toxic, similar to degradation in the field (Marking and Bills 1976).

Rotenone Degradates and Product Additives

The EPA (2007A) also determined that rotenone degradates, including rotenoloids, occur in plants from which rotenone is derived, and thus also occur in varying amounts in manufactured rotenone formulations. The EPA concluded that rotenone degradates such as rotenoloids are structurally similar to rotenone and thus are not more toxic.

Some rotenone formulations, including CFT Legumine™, use solvents and emulsifiers to extract rotenone from derris root (EPA 2007A) and/or improve product dispersion and penetration of thermal stratifications in water (Almquist 1959). In particular, CFT Legumine™ contains the following degradates and additives (CDFW 2007):

1. Rotenolone
2. 1-Methyl-2-pyrrolidinone (or N-Methylpyrrolidone; hereafter NMP)
3. Diethylene glycol monoethyl ether (Diethylene glycol ethyl ether; hereafter DGEE)
4. 1,3,5-Trimethylbenzene (*aka* mesitylene)
5. sec-Butylbenzene
6. 1-Butylbenzene (n-Butylbenzene)
7. 4-Isopropyltoluene (p-Isopropyltoluene)
8. Methylnaphthalene
9. Naphthalene

Approximately 93% of CFT Legumine™ by weight consists of NMP and DGEE (CDFW 2007). Both are highly soluble in water, will not adsorb to sediments, and will readily volatilize or undergo hydrolysis or direct photolysis (NLM 2006). Both chemicals are therefore expected to be broken down and removed from water by aerobic biodegradation, and from air by reaction with photochemically-produced hydroxyl radicals (NLM 2006). The remaining carrier chemicals include naphthalene, methylnaphthalene and a few alkylated benzenes, which comprise less than one percent of CFT Legumine™ and are not expected to alter its overall fate and transport (CDFW 2007). CFT Legumine™ does not use the synergist piperonyl butoxide, which increases the toxicity of rotenone formulations, and therefore CFT Legumine™ has comparatively less environmental impact than rotenone formulations containing piperonyl butoxide.

NMP has low toxicity and thus is often used as a solvent, including in pharmaceuticals for oral ingestion. Toxicology data indicate that the no observable effect level (NOEL) in rats is 6,000 to 18,000 ppm and in mice is 2,500 ppm (NLM 2013). With a standard safety factor of 1,000, this translates to a safe reference dose concentration of 2.5 to 6 ppm, or approximately 25 times greater than NMP concentrations in typical field applications of CFT Legumine™. NMP is readily transformed and excreted from biological organisms, and thus does not bioaccumulate. The half-life of NMP in biological organisms is 3 to 7 hours.

In aquatic systems, NMP is not expected to bind to soils and thus biodegrades readily. For example, 210 ppm of NMP in aquatic systems biodegrades to greater than 98% within 24 hours (NLM 2013).

The other primary component used in CFT Legumine™ is DGEE. In rats and mice given DGEE in drinking water over 2 years, slight to no effects were noted at high doses, including 10,000 ppm in rats and 50,000 ppm in mice (NLM 2013). These levels are nearly 90,000 times greater than DGEE concentrations expected in a typical field application of CFT Legumine™. Additional toxicology data indicate even high concentrations of DGEE have relatively low toxicity (NLM 2013). DGEE is excreted readily through metabolic activity and thus does not bioaccumulate. Although DGEE in aquatic systems is not quickly broken down, 400 ppm DGEE was observed to degrade to greater than 90% after 28 days (NLM 2013).

NMP and DGEE would be expected to dissipate more slowly relative to the active ingredient rotenone because they would be at much higher initial concentrations. Although both are water soluble and would not readily dissipate through volatilization, both are also biodegradable, which is the primary mechanism through which they would dissipate.

The remaining components of CFT-Legumine™ include minute quantities of naphthalene, methylnaphthalene and various alkylbenzenes. In typical field applications of CFT Legumine™, the concentrations of these compounds are in the parts per trillion (ppt), and far below either drinking water standards or safe reference doses established by the EPA. From a health safety standpoint, the application concentrations of naphthalene (350 ppt) and methylnaphthalene (140 ppt) are of little concern, as they are 100 to 1,000 times lower than the safe lifetime doses determined by the EPA.

To summarize, when solvent components of CFT Legumine™ are diluted to the low concentrations expected in typical field applications, they are substantially below the safe concentrations established for drinking water contaminants by the EPA.

Although these additives may result in the presence of chemical odors and pose risks to ecological and human health, the EPA has addressed risks of concern from these additives through numerous mitigation measures, including requiring applicators to use respiratory personal protective equipment and by reducing the maximum allowable treatment concentrations to 50 ppb in flowing water and 200 ppb in standing water (EPA 2007A). The EPA concluded that these mitigations will allow periodic piscicidal use of formulations containing rotenone and additives to continue to provide benefits to society while minimizing risks to human and ecological health.

Neutralization with Potassium Permanganate ($KMnO_4$)

In reregistering rotenone for continued piscicidal use, the EPA (2007A) is requiring that all rotenone applications be deactivated or neutralized using potassium permanganate ($KMnO_4$), to ensure that rotenone toxicity does not spread downstream or linger in a treated area after project goals have been achieved. $KMnO_4$ is a strong oxidizing agent used: 1) in many industries and laboratories, 2) to disinfect water to a potable state, 3) to treat fish for parasites, and 4) to neutralize water following a rotenone application, at a ratio 2 to 4 parts $KMnO_4$ to 1 part rotenone (CDFW 2007). $KMnO_4$ eliminates the respiratory toxicity of rotenone by oxidizing it. In the process, $KMnO_4$ is reduced to potassium (K; an essential electrolyte) and manganese dioxide (MnO_2), which is generally insoluble and similar to the MnO_2 found in the earth's crust (Howe et al. 2004).

The main component of $KMnO_4$ with potential toxicity is manganese, the availability of which in water is largely controlled by pH. At pHs above approximately 5.5, colloidal manganese hydroxides typically form in water, and such colloidal forms are generally not bioavailable (CDFW 2007). Therefore, when

$KMnO_4$ is applied to water to neutralize rotenone, at least two-thirds of it will be reduced, it will not persist in the environment, and it poses little risk to human health.

The ecological toxicity of KMnO4 varies by aquatic taxa, but due to the volume of KMnO4 required to deactivate rotenone, it may be hazardous to aquatic vertebrates, eliciting toxicity at concentrations as low as 1 to 2 ppm (EPA 2006). KMnO4 is more toxic to rainbow trout at lower versus higher water temperature (Marking and Bills 1976), and more toxic in hard versus soft water due to the potential for MnO2 to precipitate on fish gills (CDFW 2007). KMnO4 is toxic to zooplankton, as represented by *Daphnia spp.*, at concentrations from 84 to 3,500 ppb (EPA 2006), however, it is less toxic to *Daphnia spp.* than rotenone, which is toxic at concentrations as low as 25 to 27 ppb (see Table 38).

Rotenone Risk Assessments for Ecological Health

General Overview

The EPA recently conducted an assessment to make a reregistration eligibility determination (RED) for rotenone based on 1) required data generated by acceptable studies following current guidelines, and 2) published scientific literature (EPA 2007A). In 1988, the EPA initiated the reregistration process for rotenone by issuing the "Registration Standard" and associated data call-ins (DCI). DCIs were also issued in 1995 to require a foliar residue dissipation study, and dermal and inhalation passive dosimetry studies; and in 2004 to require a sub-chronic (28-day) inhalation neurotoxicity study, to further investigate independent studies of intravenous rotenone injections in animals at very high doses that led to Parkinson's disease-like symptoms. Although, in 2004, there were registered uses for dust rotenone products that were of concern for inhalation exposure in areas inhabited by humans, all non-piscicidal (agricultural, residential and food) uses were voluntarily cancelled by rotenone manufacturers in 2006, and the EPA subsequently waived the requirement for this study. However, since the EPA could not quantitatively assess potential neurotoxicity at doses to which rotenone users could be exposed, an additional 10x database uncertainty factor - in addition to the inter-species (10x) uncertainty factor and intra-species (10x) uncertainty factor - was applied to the human health risk assessment to protect against potential human health effects, and thus the target margin of exposure (MOE) is 1,000.

In March 2007, the EPA concluded that "currently registered piscicidal (fish-kill) uses of rotenone are eligible for reregistration provided the requirements for reregistration identified in the RED are implemented" (EPA 2007A). The following use profile was excerpted from the RED for rotenone:

Type of Pesticide: Piscicide.

Summary of Use: Rotenone is applied directly to water to manage fish populations in lakes, ponds, reservoirs, rivers, streams, and in aquaculture. The piscicide can be applied to an entire waterbody to achieve a "complete kill" or to a portion of a waterbody to achieve a "partial kill." Complete kills are used to eliminate all fish in the treatment area; partial kills are used to reduce or sample fish populations in the treatment area.

Target Organisms: Undesired fish species.

Mode of Action: Rotenone acts through uncoupling oxidative phosphorylation within cell mitochondria by blocking electron transport at complex I.

Tolerances: No tolerance exists for the piscicidal uses of rotenone.

Use Classification: Rotenone products are classified as Restricted Use Pesticides due to acute inhalation, acute oral, and aquatic toxicity.

Formulations: Liquid.

Methods of Application: Applications are made with helicopters and boats in lakes, reservoirs, and ponds (boats would likely be used in this project); with direct metering into moving water such as streams; and with hand-held equipment such as backpack sprayers in difficult-to-reach aquatic areas.

Use Rates: Labels evaluated in this RED allow rotenone to be applied to achieve treatment concentrations up to 50 ppb in streams/rivers and up to 250 ppb in lakes/reservoirs/ponds.

Application Timing: Rotenone may be applied at any time of year. Fish management program applications typically occur during warm months because the compound degrades more rapidly in warm water than cold water. Aquaculture applications typically occur during the spring prior to stocking.

Annual Usage: Annual usage data for piscicidal applications are not available.

Ecological Risk Assessment

To estimate potential ecological risk from rotenone, the EPA calculated risk quotients (RQ) by dividing acute and chronic estimated environmental concentrations (EEC) by ecotoxicity values (LC50, EC50) taken from published studies for various taxa, and comparing RQs to levels of concern (LOC; EPA 2007A). There is presumed risk of concern when a RQ exceeds a LOC, with higher RQs suggesting greater potential risk than lower RQs. Risk characterization then provides additional information on potential adverse effects and their possible impacts, by considering the environmental fate of applied chemicals and their degradates, potentially at risk organisms, and the nature of observed effects. Toxicities and risk assessments to various biota follow.

Fish

Rotenone effects on various aquatic organisms have been reported from controlled toxicity tests that typically measure the LC50 value (median water concentration of active ingredient that kills 50 percent of test animals) over a period of time (typically 24 hrs and/or 96 hrs). Rotenone toxicity data for several fish taxa (Table 37, from Marking and Bills 1976, as presented in CDFW 2007) show the most resistant taxa as black bullhead (*Ictalurus melas*), channel catfish (*I. punctatus*), and fathead minnow (*Pimephales promelas*), with 24hr LC50 rotenone concentrations of 33.3 µg/L, 20 µg/L, and 20 µg/L, respectively. In contrast, salmonids (trout, salmon and char) were among the most sensitive taxa tested, with 24hr LC50 rotenone concentrations ranging from 1.4 to 3.6 µg/L, respectively. (All proposed fish eradication sites in SEKI only contain brook trout (*Salvelinus fontinalis*) and/or forms or rainbow trout (*Oncorhynchus mykiss spp.*), which have 24hr LC50 rotenone concentrations of 2.4 µg/L and 3.5 µg/L, respectively.)

However, these tests were conducted with laboratory water, which lacks organic materials typically present in natural water. Natural organics bind to some of the rotenone applied in a treatment, thereby increasing the total amount of rotenone needing to be applied so enough free rotenone is available to fully toxify fish (CDFW 2007). As a result, applications of commercial rotenone formulations from 1 to 3 mg/L (ppm), which result in active ingredient (rotenone) concentrations from 50 to 150 µg/L (ppb), are necessary to eliminate all fish in a treatment area (Ling 2003). In summary, trout are acutely sensitive to rotenone, quickly absorbing it through the gills and typically dying within hours at application concentrations as low as 1 ppm (Ling 2003).

The EPA (2007A) used rainbow trout to estimate toxicity, exposure, and risk to freshwater fish. Rotenone is expected to eliminate fish at labeled application rates of 200 ppb for standing water and 50 ppb for flowing water. (Since the maximum solubility concentration for rotenone is 200 ppb, it is considered that 200 ppb and 50 ppb are the maximum potential exposure, and therefore EEC, for exposed aquatic organisms in standing and flowing water, respectively.) The RQ equation (EEC/LC50 = RQ) confirms

this expectation in both lakes (200/1.94 = 103.1) and streams (50/1.94 = 25.8). Since these RQs exceed the acute risk level of concern (LOC = 0.5) when rotenone is used at labeled application rates, rotenone is likely to cause the intended effect of acute mortality for freshwater fish in the treatment area.

Table 1. Fish toxicity of Noxfish®, containing 5% rotenone, in standardized laboratory tests at 12°C water temperature.

Species	Lethal Concentration of Noxfish®		Lethal Concentration of Rotenone (x 0.05)	
	LC_{50} 24h. (µg/L)	LC_{50} 96h. (µg/L)	LC_{50} 24h. (µg/L)	LC_{50} 96h. (µg/L)
Northern Pike	44.9	33.0	2.3	1.7
Atlantic salmon	35.0	21.5	1.8	1.1
Brook trout	47.0	44.3	2.4	2.2
Chinook salmon	49.0	36.9	2.5	1.9
Coho salmon	71.6	62.0	3.6	3.1
Lake trout	26.9	26.9	1.4	1.4
Rainbow trout	68.9	46.0	3.5	2.3
Goldfish	---	497.0	---	24.9
Common carp	84.0	50.0	4.2	2.5
Fathead minnow	400.0	142.0	20	7.1
Channel catfish	400.0	164.0	20	8.2
Black bullhead	665.0	389.0	33.3	19.5
Smallmouth bass	93.2	79.0	4.7	4.0
Largemouth bass	200.0	142.0	10	7.1
Green sunfish	218.0	141.0	10.9	7.1
Bluegill sunfish	149.0	141.0	7.5	7.1
Yellow perch	92.0	70.0	4.6	3.5
Longnose sucker	67.2	57.0	3.4	2.9
White sucker	71.9	68.0	3.6	3.4
Bowfin	57.5	30.0	2.9	1.5

The RQs shown above also exceed the chronic risk level of concern (LOC = 1). Chronic effects may therefore occur if freshwater fish survive acute exposure. Based on rotenone environmental fate and labeled application rates in standing and flowing water, freshwater fish may be affected for less than two weeks in warm water and up to approximately 160 days in cold water, where rotenone is relatively more persistent.

Aquatic Invertebrates

Similar to fish, a review of many aquatic invertebrate taxa shows a range of sensitivity to rotenone (Table 38, from a variety of sources, as summarized by Ling 2003), perhaps based on differing oxygen requirements (CDFW 2007). Table 38 shows a mollusc (96hr LC50 = 7,500 µg/L), a snail (24hr LC50 = 6,350 µg/L), and a freshwater prawn (24hr LC50 = 5,150 µg/L) as the most rotenone-resistant taxa included in this review, while Branchiura (lice; 24hr LC50 = ~25 µg/L), Conchostracan (clam shrimps; 24hr LC50 = ~50 µg/L), and Hydrachnidae (water mites; 96hr LC50 = ~50 µg/L) were the most rotenone-sensitive taxa reported.

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Table 2. Rotenone toxicity reported in some aquatic invertebrates.

Species Guild	Test Species	Test Endpoint	Lethal Concentration (mg/L)
Flatworm	*Catenula* sp.	LC_{50} 24h	5.1
	Planaria sp.	LC_{50} 24h	<0.5
Annelid worms	Leech	LC_{50} 48 h	<0.100
Copepod	*Cyclops* sp.	$LC_{10}0$ 72h	<0.100
Branchiura	*Argulus* sp.	LC_{50} 24h	~0.025
Cladoceran	*Daphnia pulex*	LC_{50} 24h	0.027
	D. pulex	LC_{50} 24h	<0.025
	Diaptomus siciloides	LC_{50} 24h	<0.025
Conchostracan	*Estheria* sp.	LC_{50} 24h	~0.050
Freshwater prawn	*Palaemonetes kadiakensis*	LC_{50} 24h	5.15
Crayfish	*Cambarus immunis*	LC_{50} 72h	>0.500
Dragonfly naiad	Macromia sp.	LC_{50} 24h	4.7
Stonefly naiad	*Pteronarcys californica*	LC_{50} 24h	2.9
Backswimmer	*Notoncta* sp.	LC_{50} 24h	3.42
	Notonecta sp.	LC_{50} 24h	~0.100
Caddis fly larvae	*Hydropsychye* sp.	LC_{50} 96h	0.605
Whirligig	*Gyrinus* sp.	LC_{50} 24h	3.55
Water mite	Hydrachnidae	LC_{50} 96h	~0.050
Snail	*Physa pomilia*	LC_{50} 24h	6.35
	Oxytrema catenaria	LC_{50} 96h	1.75
	Lymnaea stagnalis	LC_{50} 96h	>1.0
Bivalve Mollusc	*Dreissena polymorpha*	LC_{50} 48h	0.219
	Obliquaria reflexa	LC_{50} 48h	>1.0
	Elliptio buckleyi	LC_{50} 96h	2.95
	Elliptio complanata	LC_{50} 96h	2
	Corbicula manilensis	LC_{50} 96 h	7.5
Ostracod	*Cypridopsis* sp.	LC_{50} 24h	0.490

However, these most sensitive invertebrate taxa are 7 to 14 times more resistant than the most resistant fish taxa in SEKI proposed eradication sites (rainbow trout: 24hr LC50 = 3.5 µg/L). Since the anatomies of many aquatic invertebrate taxa contain gill-like structures, they should theoretically be as susceptible to rotenone as fish or amphibian larvae (Bradbury 1986). In laboratory tests, however, Chandler and Marking (1982) concluded that aquatic invertebrates are generally much more tolerant of rotenone than most fishes and amphibian larval stages. A snail (*Helisoma sp.*) and the Asiatic clam (*Corbicula manilensis*) were the most resistant taxa studied, with 96hr LC50 concentrations that were 50 times greater than the most resistant fish (black bullhead) studied by Marking and Bills (1976). Another study (Sanders and Cope 1968) measured rotenone effect on subadult stages of a stonefly (*Pteronarcys californica*). They showed 24hr and 96hr LC50 concentrations of 2,900 µg/L and 380 µg/L, respectively, which are an order of magnitude greater than those reported for black bullhead. They also showed that larger, older subadults were less susceptible to given concentrations of rotenone than smaller, younger subadults of the same taxa. Although these results indicate that aquatic invertebrates are much less sensitive to rotenone than fish, acute invertebrate mortality is still expected from a typical rotenone application.

Aquatic invertebrate communities, however, tend to recover relatively quickly following rotenone treatment (Ling 2003), with studies showing rapid biomass increases following initial depletions from rotenone treatment (Neves 1975, Cook and Moore 1969). Similarly, Dudgeon (1990) found that stream rotenone treatments caused immediate invertebrate drift, particularly of mayflies, but did not cause significant mortality or a significant reduction in abundance of benthic invertebrates. Nevertheless, varied results of rotenone effect on aquatic invertebrate communities have also been reported, with some showing negligible effects (Demong 2001, Melaas et al. 2001) and others showing longer-term negative effects (Mangum and Madrigal 1999, Binns 1967).

A study of a rotenone treatment in Strawberry River, Utah (Mangum & Madrigal 1999) showed that up to 33% of benthic invertebrate taxa were never affected, 46% had recovered after one year, and 21% were still missing after five years. Most of the taxa that failed to recover were in the EPT group [Ephemeroptera (mayflies), Plecoptera (stoneflies), and Trichoptera (caddisflies)], although some taxa in each of these groups were still present and therefore resistant to rotenone. In addition, other taxa not present before the treatment were also detected and possibly filling vacated niches.

However, it is important to note that the Strawberry treatment targeted eradication of Utah chubs (*Gila atraria*), which are in the same family and functionally similar to fathead minnows – one of the most-resistant fish taxa reviewed by Marking and Bills (1976; Table 37). Therefore, assuming that Utah chubs are significantly more resistant to rotenone than trout, this treatment was applied at a concentration significantly greater than is necessary to eliminate trout taxa, and significantly greater than is currently allowed following the reregistration of rotenone for piscicidal use (EPA 2007A).

In addition, taxa in the EPT group are typically highly mobile and have short life cycles, and therefore should rapidly repopulate treated areas through dispersal and reproduction (Engstrom-Heg et al. 1978). Further, rotenone exposure to aquatic invertebrates may be reduced by behaviors such as burrowing, associating with vegetation or the ability to trap air bubbles with appendages (CDFW 2007). Moreover, rotenone toxicity to aquatic invertebrates such as freshwater shrimp may be moderated by physical and chemical attributes of the treated ecosystem (Melass et al. 2001).

Zooplankton

Table 38 shows a range of sensitivity to rotenone for two groups of zooplankton, including copepods (72hr LC100 = <100 µg/L) as the most rotenone-resistant taxa included in this review, and cladocerans (24hr LC50 = <25 to 27 µg/L) as the most rotenone-sensitive taxa reported. However, these zooplankton taxa are still 7 to 28 times more resistant than the most resistant fish taxa in SEKI proposed eradication sites (rainbow trout: 24hr LC50 = 3.5 µg/L).

Although these results indicate that zooplankton are much less sensitive to rotenone than fish, rotenone is still toxic to zooplankton (Melaas et al. 2001, Beal and Anderson 1993, Neves 1975, Anderson 1970, Kiser et al. 1963) and thus mortality is expected from a typical application in standing waters. Reductions are generally short-term, however, with populations of more-resistant taxa such as copepods recovering over periods of 1 to 8 months following treatment (Ling 2003, Beal and Anderson 1993), and populations of more-sensitive taxa such as cladocerans sometimes needing three years to recover in mountain lakes (Anderson 1970).

While many benthic invertebrates may lessen rotenone exposure by burrowing into sediment, zooplankton typically occupy open-water habitat and thus are exposed to rotenone for the entire time it is active during a treatment (CDFW 2007). As a result, zooplankton taxa such as cladocerans are generally more sensitive than larger benthic invertebrates such as mollusks, oligochaete worms and chironomid

midge larvae (Hamilton 1941, Morrison 1977). However, some zooplankton taxa have resistant life stages and/or eggs that may facilitate recovery (Kiser et al. 1963).

The EPA (2007A) used cladocerans (Daphnids) to estimate toxicity, exposure, and risk to zooplankton. Rotenone is expected to eliminate many zooplankton at labeled application rates of 200 ppb for standing water and 50 ppb for flowing water. The RQ equation (EEC/LD50 = RQ) confirms this expectation in both lakes (200/3.7 = 54.1) and streams 50/3.7 = 13.5). Since these RQs exceed the acute risk level of concern (LOC = 0.5), when rotenone is used at labeled application rates, rotenone is likely to cause acute mortality for many zooplankton in the treatment area.

The RQs shown above also exceed the chronic risk level of concern (LOC = 1). Chronic effects may therefore occur if zooplankton survive acute exposure. Based on rotenone environmental fate and labeled application rates in standing and flowing water, sensitive species may be affected for less than two weeks in warm water and up to approximately 160 days in cold water, where rotenone is relatively more persistent.

Amphibians
Table 39 (from a variety of sources, as summarized by CDFW 2007) and Chandler (1982) show a range of rotenone sensitivity values for amphibians, including northern leopard frog (*Rana pipiens*) adults (24hr LC50 = 240 to 1580 µg/L; 96hr LC50 = 240 to 920 µg/L) as the most rotenone-resistant group reported, and gilled larvae of various taxa (24hr LC50 = 5 to 580 µg/L; 96hr LC50 = 25 to 500 µg/L) as the most rotenone-sensitive group. However, even gilled larvae as the most sensitive amphibian group are still 1.4 to 165 times more resistant than the most resistant fish taxa in SEKI proposed eradication sites (rainbow trout: 24hr LC50 = 3.5 µg/L). Amphibian adults are therefore much less sensitive to rotenone than fish, and gilled larval amphibians have rotenone sensitivities similar to the most-resistant fish taxa.

Table 3. Rotenone toxicity to various amphibians in lakes.

Species	Stage	Temp °C	24 hours LC_{50} (µg/L)	96 hours LC_{50} (µg/L)	Original Reference
N. Leopard frog (*Rana pipiens*)	Juvenile/ Adult	___	10	___	Haag, 1931
	Tadpole	___	5	___	Hamiliton, 1941
	Adult	12	240	240	Farringer, 1972
	Adult	12	1200	290	Farringer, 1972
	Adult	12	1460	920	Farringer, 1972
	Adult	12	1580	640	Farringer, 1972
Tiger salamander (*Ambystoma tigrinum*)	Larvae	___	5	___	Hamilton, 1941
S. Leopard frog (*Rana sphenocephala*)	Tadpole	15-17	30	25	Chandler & Marking, 1982

The difference in rotenone sensitivity between adult and gilled larval amphibians may be due to anatomical differences in which adults primarily breathe through skin while larvae breathe through gills. Adult amphibian skin may be more of a barrier to rotenone than gills due to skin having a smaller relative surface area and a greater relative distance for rotenone to diffuse across (Fontenot et al. 1994). Amphibian adults should therefore not be harmed when rotenone is applied in accordance with labeled instructions (at required piscicidal concentrations) (Farringer 1972), and the response of gilled larval

amphibians depends on development stage (Hamilton 1941). Younger larvae that are dependent on gill respiration are far more sensitive than older larvae that are near metamorphosis and breathing air, indicating that rotenone is more readily absorbed across gills than skin. In addition, amphibian eggs are less sensitive to rotenone than fish because their rate of chemical uptake from water is much lower (Ling 2003).

At labeled rotenone application rates, some effects on amphibians are therefore expected, but significant losses would be unlikely, especially if treatments are scheduled in late summer after amphibian eggs have hatched and larvae of most amphibian taxa have metamorphosed. Indeed, these conclusions are similar to results of a rotenone application in spring of 1974 to eradicate exotic African clawed frogs (*Xenopus laevis*) in California, in which all *X. laevis* tadpoles were killed but adults were unaffected and able to reproduce later that spring (McCoid and Bettoli 1996).

Terrestrial Biota

Since terrestrial biota is largely insensitive to rotenone compared to aquatic organisms, there is a significant safety margin between maximum treatment concentrations and those needed to harm to terrestrial organisms (Ling 2003). Acute rotenone toxicities to various mammals and birds are shown in Table 40 (from a variety of sources, as summarized by CDFW 2007).

Table 4. Rotenone toxicity to various mammals and birds.

Animal Group	Toxicology Test	Median Lethal Concentration	Reference(s)
Mammals			
Human	Acute LD_{50} oral	300-500 mg/kg-body wt (Estimated)	USEPA, 1988
Rat	Acute LD_{50} oral	39.5 mg/kg (female)	USEPA, 1988
	Acute LD_{50} oral	102 mg/kg (male)	USEPA, 1988
	Chronic NOAEL TRV	0.4 mg/kg-bw/day	USFWS, 1983
	Chronic LOAEL TRV	2 mg/kg-bw/day	USFWS, 1983
Mouse	Acute LD_{50} oral	350 mg/kg	Kidd & James, 1991; USEPA, 1988
Guinea pig	Acute LD_{50} oral	12-200 mg/kg	USEPA, 1988
Rabbit	Acute LD_{50} oral	600-2000 mg/kg	USEPA, 1988
	Acute LD_{50} oral	~1500 mg/kg	Unknown reference
Dog	Chronic NOAEL TRV	0.4 mg/kg-bw/day	USFWS, 1980
	Chronic LOAEL TRV	2 mg/kg-bw/day	USFWS, 1980
Birds			
English song sparrow (nestling)	Acute LD_{50} oral	130 mg/kg	Cutcomp, 1943 (in DFG, 1994)
American robin (nestling)	Acute LD_{50} oral	200 mg/kg	Cutcomp, 1943 (in DFG, 1994)
Quail	Acute LD_{50} oral	1882 mg/kg	Unknown reference
Mallard duck	Acute LD_{50} oral	2200 mg/kg	USEPA, 1988
	Acute LD_{50} oral	> 2000 mg/kg	Extoxnet, 1996
Pheasant	Acute LD_{50} oral	1680 mg/kg	USEPA, 1988
	Acute LD_{50} oral	>1680 mg/kg	Extoxnet, 1996

Reptiles
Few studies have examined rotenone toxicity to reptiles, however, Fontenot et al. (1994) reports that acute toxicity to green anole lizards (*Anolis carolinensis*) were considered during pre-registration of piscicides including rotenone. Aquatic turtle taxa with specialized mechanisms such as buccopharyngeal respiration (*Apalone spinifera, Kinosternon minor*) or modified skin and cloaca to enhance respiration (*Trachemys scripta, K. odoratum*) may be more sensitive to rotenone than more terrestrial turtle taxa. In addition, Carr (1952) and Dundee & Rossman (1989) hypothesized that soft-shelled turtles (*Apalone* spp.) may be sensitive to rotenone but did not provide scientific data to support this. These conclusions are similar to one study of a rotenone treatment in Lake Conroe in Texas that reported aquatic turtles (*K. subrubrum*) to be sensitive to rotenone, with at least 60 dead or dying individuals observed around the lake shoreline 24 to 48 hours after treatment (McCoid & Bettoli, 1996).

Since freshwater aquatic snakes do not use aquatic respiration, it is very unlikely that absorption of rotenone will occur through the thick skin of snakes (Fontenot et al., 1994). However, Haque (1971) reported the death of one aquatic snake 48 hours after a pond rotenone treatment, while a second snake in the same pond at the same time was swimming in a healthy manner. Additional studies would therefore clarify the toxicity of rotenone to reptiles.

Birds
The EPA (2007A) concluded that: 1) birds that forage on terrestrial items have little risk of exposure to rotenone residues because rotenone is applied directly to water, and 2) although some birds that forage on fish may opportunistically feed on dead or dying fish in treatment areas, it is unlikely to result in a lethal dose. EPA based this conclusion on a study (Jarvinen and Ankley 1998) that found only 0.22 µg/g of rotenone residue in yellow perch (*Perca flavescens*; similar in size to trout) killed by rotenone. A 68 g perch would therefore contain about 15 µg of rotenone, and a 1,000 g bird would have to consume about 274,000 perch to reach the avian subacute LC50 of 4,110 mg/kg. In addition, many of the trout in a treatment area will either sink or be collected and buried and thus not be available for consumption by birds.

Mammals
The EPA (2007A) also concluded that: 1) wild mammals are not likely to have significant exposure to rotenone residues because dead fish tend to sink where they are not available for terrestrial consumption, and 2) in the unlikely event that mammals could forage on dead or dying fish, it is unlikely to result in observable acute toxicity. As stated above, a 68 g perch would contain about 15 µg of rotenone. A medium-sized (350 g) mammal with a daily food intake of 18.8 g would receive 4.1 µg of rotenone if it foraged its entire daily ration from a perch in a treatment area. This is far below the median lethal dose of rotenone (39.5 mg/kg * 0.350 kg = 13.8 mg = 13,800 µg) for similarly sized mammals. Likewise, a large-sized (1,000 g) mammal with a daily food intake of 34 g would receive 7.5 µg of rotenone if it foraged its entire daily ration from a perch in a treatment area. This is far below the median lethal dose of rotenone adjusted for body weight (30.4 mg/kg * 1 kg = 30.4 mg = 30,400 µg) for similarly sized mammals.

Insects and Plants
Although the EPA does not currently estimate RQs for terrestrial insects, a contact study on honey bees classified technical grade rotenone as practically non-toxic on an acute exposure basis to non-target terrestrial insects (EPA 2007A). Moreover, it is presumed that terrestrial insects that forage on terrestrial items have little exposure to rotenone residues because rotenone is applied directly to water.

Although no data were submitted to assess the risk of rotenone exposure to terrestrial plants, the EPA (2007A) concluded that rotenone exposure to terrestrial plants is unlikely given the protocols by which rotenone is applied.

Water

CFT Legumine™ formulations contain, in addition to the active ingredient rotenone, a variety of additional chemicals that facilitate solubility and dispersal. Table 41 (as presented in CDFW 2007) lists the chemicals present and calculated treatment concentrations for CDFW's treatment of Davis Lake in 2007. The chemicals and concentrations from the Davis Lake treatment are expected to be very similar to those expected in piscicide treatments proposed under alternatives B or D.

The rate and manner in which natural physical processes affect the breakdown or persistence of a chemical in the environment is chemical specific. All of these chemicals have characteristics that make them break down rapidly in the environment, and they are not expected to be present in environmental media for extended periods of time. Using currently available sampling and analytical tools and following EPA protocols, rotenone and many of the other compounds in the formulations proposed would not be detectable in water, sediment, or air after just a few days to weeks following the proposed treatments. Maximum conservative estimates in sediment for rotenone are assumed to persist for no longer than 45 days, and likely significantly less (CDFW 2007).

Table 5. Reported chemical composition, field concentration, persistence and toxicity of CFT Legumine™.

Ingredient	Conc. in Treatment[1] (µg/l)	Half-Life[2]	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity of Other Receptors
rotenone	42.1	Hydrolysis: 3.2 days @ pH 7, 2 days @ pH 9 Aqueous photolysis: 21 hr (1 cm), 191 days 1 m well mixed Entire Pond System (water + sediment): 20 days @ 5°C, 1.5 days @ 25-27°C Air Photooxidation: 0.05 days Soil: 3 days			LD_{50} Mice (i.p.): 2.8 mg/kg rats (oral): 132 mg/kg-bw; (i.v.): 6 mg/kg human: Ingestion or inhalation of large doses may lead to: numbness of oral muscosa, respiratory paralysis at lethal doses, tremor, trachypnea, nausea, vomiting. Chronic exposure may produce fatty changes to liver and kidney. More toxic when inhaled than ingested. Skin irritation from direct contact.
rotenolone	5.2				Oral LD_{50} mice: rotenolone I: 4.1 mg/kg rotenolone II: 25 mg/kg
1-methyl-2-pyrrolidinone	87.8	Air Photooxidation: 5 hrs Soil: 4 days in clay 8.7 in loam		NOEL=5 g/l in bacteria, algae (*Scenedes mus*), &	

Ingredient	**Conc. in Treatment[1] (µg/l)**	**Half-Life[2]**	**Water Pollution Factors**	**Aquatic Toxicity Metrics**	**Toxicity of Other Receptors**
		11.5 in sand		protozoa (*Colpoda*)	
diethylene glycol monoethyl ether	581.1	Air Photooxidation: 12 hrs	BOD: 20 NEN 3235-5.4 COD: 1.85 NEN 3235-3.3	24 hr LC_{50}: >5,000 mg/l (goldfish, static) 96 hr LC_{50}: >10,000 mg/l (*Menidia beryllina*, static)	Oral LD_{50} (single dose): rat = 8.69-9.74 g/kg guinea pig = 3.67=4.97 g/kg cat = 1 ml/kg (lethal) rat NOEL: 0.49 g/kg (repeat oral dose) rabbit, cat, guinea pig, mouse: inhalation – no injury w/ 12 day exposure to saturated vapor
1,3,5-trimethylbenzene	0.004	Aqueaceous Volitilization: est. 3 hrs for model river, 4 hrs for model lake & 5 days for model pond (includes sediment adsorption) Air Photooxidation: 7 hrs	BOD: 3% of Theoretical Oxygen Demand (ThOD) COD: 110% of ThOD	96hr median threshold limit = 13 mg/l (goldfish, flow-through)	
sec-butylbenzene	0.004	Aqueaceous Volitilization: est. 3.4 hrs for model river, 4.6 hrs for model lake & 88 days for model pond (includes sediment adsorption) Air Photooxidation: 1.9 days			Eye irritation reactivity (EIR) in humans @ 1.8
1-butlybenzene (n-butylbenzene)[3]	0.005-0.0236-0.078	Aqueaceous Volitilization: est. 3.5 hrs for model river, 4.6 hrs for model lake & 16 days for model pond (includes sediment adsorption) Air Photooxidation: 1.8 days	ThOD: 3.22		EIR: 6.4 (humans)
4-isopropyltoluene	0.005	Aqueaceous Volitilization: est. 1 hr for model river, 5 hrs for model lake & 30 days for model pond (includes sediment adsorption)			

Ingredient	Conc. in Treatment[1] (µg/l)	Half-Life[2]	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity of Other Receptors
		Air Photooxidation: 1 day			
methylnaphthalene	0.136	Aqueaceous Volitilization: est. 5.5 hr for model river, 5.3 hrs for model lake & 78 days for model pond (includes sediment adsorption) Air Photooxidation: 7.4 hrs		24, 48, 72, 96-hr LC_{50} = 39, 9, 9, 9 mg/l in FHM (static); 48 hr LC_{50} in brown trout yearlings =8.4 mg/l (static); BCF: 20 to 130 in coho salman muscle, depending on length of exposure	
naphthalene	0.255-0.341	Aqueaceous Volitilization: est. 3 hr for model river and 5 days for model lake Aqueous photolysis: 71 hrs Aqueous Biodegration: 0.8-43 days Sediment: Degradation rates in sediments are 8-20 times higher than in the above water column. Biodegradation half lives ranged from 2.4 weeks in sediments chronically exposed to petroleum hydrocarbons to 4.4 weeks in sediments from a pristine environment. Soil Biodegration: 2-18 days Air Photooxidation: 18 hrs			

Ingredient	**Conc. in Treatment[1] (µg/l)**	**Half-Life[2]**	**Water Pollution Factors**	**Aquatic Toxicity Metrics**	**Toxicity of Other Receptors**
1-hexanol	3.6		BOD: 28% of ThOD; COD: 94% of ThOD		LD_{50} orally in rats 4.59 g/kg. Toxicity threshold (cell multiplication inhibition test): bacteria (*Pseudomonas putida*): 62 mg/l; algae: *Microcystis aeruginosa*): 12 mg/l; green algae (*Scenedesmus quadricauda*): 30 mg/l; protozoa (*Entosiphon sulcatum*): 75 mg/l; protozoa: (*Uronema parduczi* Chatton-Lwoff: 93 mg/l
1,2,4,5-tetramethylbenzene	0.369	Aqueaceous Volitilization: est. 3.5 hr for model river and 4.6 days for model lake			
1,2,4-trimethlybenzene	0.0307				
1,4-diethylbenzene	0.453	Aqueaceous Volitilization: est. 3.5 hr for model river and 4.6 days for model lake			
total c_4 substitued benzenes	2.586				
total c_5 substitued benzenes	0.796				
triethylene glycol[3]	0.220-0.266		BOD5: 0.03 NEN 3235-5.4, 1.4% of ThOD; BOD10: 0.50 std.dil.sew.; 10 days: 3.7% ThOD; 15 days: 11.5% of ThOD; 20 days: 17% of ThOD; COD:	LC_{50}/96-hr values for fish are between 10 and 100 mg/l. Therefore, this material is expected to be slightly toxic to aquatic wildlife.	LD50 Oral mice, rats (g/kg): 21, 15-22; Toxicity threshold (cell multiplication inhibition test) in mg/ml: bacteria (*Pseudomonas putida*): 320; algae: *Microcystis aeruginosa*): 3,600; protozoa (*Entosiphon sulcatum*). Goldfish: 24-hr LC_{50}=>5,000 mg/l; guppy: 7 d LC_{50}: 62,600 ppm. Single oral doses LD_{50}: Guinea pig: 14.6 g/kg;

Ingredient	**Conc. in Treatment[1] (µg/l)**	**Half-Life[2]**	**Water Pollution Factors**	**Aquatic Toxicity Metrics**	**Toxicity of Other Receptors**
			1.57 NEN 3235-5.3		7.9 ml/kg. Rat (repeated oral dose): no effect@3-4 g/kg/day, 2 years, 5-8 g/kg/day, 30 days; Human: very low acute and chronic toxicity
tetraethylene glycol[3]	1.060-1.194		BOD10: 0.50 std.dil.sew.		Rats: single oral LD_{50}: 32.8 g/kg, and 28.9 ml/kg=1; Rabbit: skin LD_{50} >20,000 mg/kg
pentaethylene glycol[3]	2.00-2.471				
hexaethylene glycol[3]	3.600-4.386				Oral rat LD_{50}: 32,000 mg/kg-1; Oral guinea pig: 20,000 mg/kg-1
trichloroethylene	0.0073				
tetrachloroethylene	0.0128				
toluene	0.1667				
xylene-m/p	0.0029				
Total fatty acid esters, resin acids and neutrals[3, 4]	164.115				
Representative Rosin Acids					
abietic acid					LC_{50} values to crustaceans: 6.2 mg/l=96 hr, Nitocra spinipes; LC_{50} values in fish: 0.56 mg/l=96 hr, Oncorhynchus kisutch (i.e., coho salmon); 0.7 mg/l=96 hr, *Salmo gairdneri;* 0.41 mg/l=96 hr, Oncorhynchus kisutch.
beta-pinene					
isopimaric acid					LC50=0.4 mg/l for rainbow trout for isopimaric acid in lodgepole pine sapwood (Wang, Z. et al. Jan.1995, Applied & Env. Microbiol.).

Ingredient	**Conc. in Treatment[1] (µg/l)**	**Half-Life[2]**	**Water Pollution Factors**	**Aquatic Toxicity Metrics**	**Toxicity of Other Receptors**
Fatty Acids					
tall oil				Fish: Semistatic; 96 hour exposure; NOEC >=1000mg/L Invertebrates: (Crustacea); 48 hour exposure; NOEC >=1000mg/L Plants: (Algae); 72 hour exposure; NOEC >=1000mg/L	Oral: LD_{50}, Rat @ 74000 mg/kg bw (Oleic) LD_{50} Rat @>3200 mg/kg bw (linoleic) LD_{50}, Rat @ 7600 mg/kg bw (Rosin) Skin: Rabbit, Slight Irritant Eye: Rabbit, Slight irritant
oleic acid (112-80-1) <tall oil partition>				Fish: Fathead Minnow: LC_{50} = 205 mg/L; 96 Hr.; Static condition	LD_{50}/LC_{50}: Draize test, rabbit, eye: 100 mg Mild; Oral, mouse: LD50 = 28 gm/kg; Oral, rat: LD50 = 25 gm/kg; Human Skin Draize 15 mg/3D intermittent; REACTION: Moderate.
linoleic acid (60-33-3) <tall oil partition>			COD: 8.38% of ThOD BOD: 71% of ThOD	Invertebrate toxicity:EC_{50} (duration unspecified) purple sea urchin 0.28-1.07 mg/kg inhibited fertilisatio	Oral, mouse: LD_{50} = >50 gm/kg

Ingredient	**Conc. in Treatment[1] (µg/l)**	**Half-Life[2]**	**Water Pollution Factors**	**Aquatic Toxicity Metrics**	**Toxicity of Other Receptors**
				n (Cherr, G.N. et al. Environ.Toxicol.Chem. 1987, 6(7), 561-569).	
Linolenic (463-40-1) <tall oil partition>					
Rotenone Neutralization Compound					
potassium permanganate	4 mg/l max			96-hr LC_{50}: 3.6 mg/l (goldfish) 0.75 mg/l (channel catfish) 96-hr LD_{50}: 2.7-3.6 mg/l (bluegill)	Oral LD50 (single dose): Guinea pig: 810 mg/kg Mouse: 750 mg/kg Rat: 750 mg/kg

[1] Calculation based on application of 1 ppm

[2] River model assumes depth = 1 m, flow velocity = 1 m/sec, & wind velocity = 3 m/sec. Model lake assumes depth = 1 m,
flow velocity = 0.05 m/sec, & wind velocity = 0.5 m/sec. Do not consider sediment particulate adsorbtion.

[3] Components of Fennodefo 99™ which is 17.1% of CFT Legumine™ formulation.

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