Rotenone—a review of its toxicity and use for fisheries management

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Nicholas Ling

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Nicholas Ling

Centre for Biodiversity and Ecology Research, Dept of Biological Sciences, University of Waikato, Private Bag 3105, Hamilton, New Zealand

ABSTRACT

Rotenone is a natural plant toxin used for centuries by indigenous peoples of Southeast Asia and South America for the harvesting of fish for human consumption. It has been used as a commercial insecticide for more than 150 years and for the management of fish populations since the 1930s. Fisheries management uses include eradication of pest fishes, quantifying populations, food web manipulation, controlling fish diseases, and restoring water bodies for threatened species. Rotenone is considered one of the most environmentally benign toxicants available for fisheries management. Fish are acutely sensitive to rotenone poisoning, quickly absorbing the toxin across the gill surface and dying within hours at concentrations below 1 ppm, although individual species sensitivities vary widely. Aquatic invertebrates are generally less sensitive than fish, but it will cause significant collateral loss of invertebrate fauna, although invertebrate populations quickly recover. Humans and wildlife are comparatively insensitive to rotenone, which provides a large safety margin between concentrations required to kill fish and those that may prove harmful to non-target, non-aquatic organisms. Rotenone can be applied to standing or flowing waters as a generally dispersed toxicant, either in liquid or powdered form, or as formulated baits to target nuisance species. Rotenone is chemically unstable and breaks down rapidly in the environment, yielding water-soluble non-toxic products. It is readily metabolised to non-toxic excretable substances in the bodies of vertebrates receiving a sub-lethal dose. Rotenone is not considered to be carcinogenic. Recent experimental findings linking it to Parkinsonian effects seem unlikely to occur under normal uses. Cost and availability limit the use of rotenone to relatively small water bodies. This summary reviews the toxicity of rotenone and its use in fisheries management. A limited risk assessment for rotenone use in New Zealand is provided given that its potential uses and the sensitivities of indigenous species have not been determined.

Keywords: rotenone, piscicide, icthyocide, pesticide, fish management, fish control

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Part 1 A review of the use and toxicity of rotenone for fisheries management purposes

1. INTRODUCTION

Rotenone is a natural toxin produced by several tropical plants and has been used for centuries as a selective fish poison and more recently as a commercial insecticide. It is highly toxic to fish and other aquatic life, but has low toxicity to birds and mammals. Rotenone is non-persistent in the environment, being quickly broken down by light and heat. It does not accumulate in animals and is readily metabolised and excreted. It is registered for use in New Zealand as an insecticide and has been used experimentally for quantitative fish population surveys and to remove unwanted fishes in lake management trials. Its use in New Zealand to control pest fishes is likely to increase substantially due to increasing concern about the spread and impacts of invasive exotic fishes and the need to develop effective management tools to target these pest species.

Rotenone has been used extensively in North America since the 1930s for managing freshwater fisheries and for fisheries research. The literature on rotenone is vast. Roark (1932) published a bibliography on the use of *Derris* species as insecticides and listed 475 papers. More than 1000 papers have been published on rotenone since 1990 and the literature is currently expanding at more than 100 papers per year. Recent research interest in rotenone stems mainly from biochemical interest in its highly specific action in selectively inhibiting mitochondrial activity and its possible anticancer properties.

Rotenone is now recognised as the most environmentally benign of the commonly used fish poisons (piscicides or ichthyocides) and remains extremely useful for the chemical rehabilitation of fish habitats to remove noxious species and for research sampling. In response to recent public concerns about large-scale rotenone use in fisheries management, the American Fisheries Society has established a rotenone stewardship programme to provide advice on the safe use of rotenone, and to encourage good planning and public involvement in future rotenone programmes (AFS 2000).

This brief review summarises the toxicity of rotenone to aquatic and terrestrial animals and the use of rotenone in fisheries management and research. An ecological risk assessment for rotenone use in New Zealand is also provided.

1.1 Physical and chemical properties

Common name: Rotenone

Empirical formula: $C_{23}H_{22}O_6$

Chemical name: (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

Synonyms

(R)-1,2-dihydro-8,9-dimethoxy-2-(1-methylethenyl)[1]benzopyrano[3,4-b]furo-[2,3-h][1]benzopyran-6,12-dione

1,2,12,12aa-tetrahydro-2a-isopropenyl-8,9-dimethoxy[1]benzopyrano[3,4-b]furo[2,3-h][1]benzopyran-6(6aH)-one

1,2,12,12a-tetrahydro-8,9-dimethoxy-2-(1-methylethenyl)-[2R-(2a,6aa,12aa)]-(1)benzopyrano(3,4-b)furo(2,3-h)-(1)benzopyran-6(6aH)-one

Structure: see Fig. 1

CAS number: 83-79-4

Molecular weight: 394.43

Solubility: very limited solubility in water – 0.2 mg/L @ 20°C, 15 mg/L @ 100°C; soluble in most organic solvents including ethanol – 2g/L @ 20°C; carbon tetrachloride – 6 g/L @ 20°C; amyl acetate 16 g/L @ 20°C; xylene 34 g/L @ 20°C; acetone – 66 g/L @ 20°C; benzene 80 g/L @ 20°C; chlorobenzene 135 g/L @ 20°C; ethylene dichloride 330 g/L @ 20°C; chloroform – 472 g/L @ 20°C

Melting point: ~ 165°C

Boiling point: ~ 220°C

Vapour pressure: less than 1 mPa @ 20°C

Description: colourless to brownish crystals, or a white to brownish-white crystalline powder

Stability: sensitive to light and air

Pesticide type: botanical

Year of initial registration (USEPA): 1947

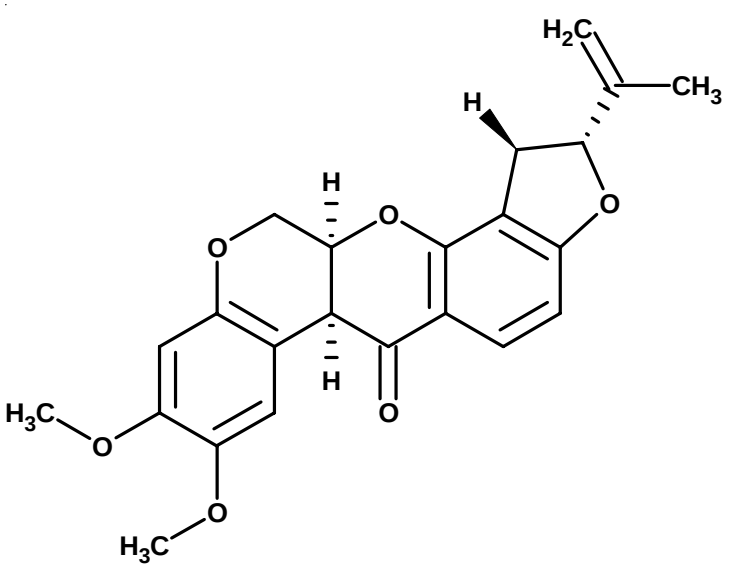

Figure 1. Structure of rotenone.

1.2 General toxicity

Rotenone is classified as either EPA toxicity class I or III (highly toxic or slightly toxic) depending on product formulation.

1.3 Trade and other names

Aker-root, Aker-tuba, Barbasco, Cenol Garden Dust, Chem-Fish, Chem-Mite, Cibe Extract, Cube, Cuberol, Cube root, Cubor, Curex Flea Duster, Dactinol, Derril, Derrin, Derris, Derris dust, Derris root, Dri-kil, ENT-133, Extrax, Fish Tox, Foliafume, Gallicide, Green Cross Warble Powder, Haiari, Mexide, Nekos, Nicouline, Noxfire, Noxfish, Nusyn-Noxfish, Paraderil, PB-Nox, Prenfish™, Prentox®, Pro-nox fish, Protex, Ro-Ko, Ronone, Rotacide, Rotefive, Rotefour, Rotessenol, Rotocide, Sinid, Synpren Fish Toxicant, Timbo, Tox-R, Tubatoxin, tuba-root, Yates Derris Dust.

Rotenone is available as crystalline preparations (approximately 95% pure), emulsified solutions (5 to 50% pure) and dusts (0.5 to 5% pure). Formulations may be synergised with piperonyl butoxide to enhance toxicity and, in the past, may have contained other pesticides including carbaryl, lindane, thiram, pyrethrins and quassia.

Rotenone is freely available from garden centres and supermarkets in New Zealand as a 0.5% (5 g/kg) dust formulation, e.g. Yates derris dust.

1.4 Sources and impurities

Rotenone is a natural poison and is the most abundant of a number of biologically active compounds derived from the roots of certain tropical species of the Leguminosae; in particular, cubé or barbasco (*Lonchocarpus utilis* and *L. urucu*), derris (*Derris elliptica*), rosewood (*Tephrosia* spp.) and Rabbit's pea (*Dalbergia paniculata*). Most commercial product comes from Central and South America. Dried derris roots contain an average of around 5% rotenone.

The ground roots or their extracts have been used for centuries to narcotise fish for human consumption by the indigenous peoples of Southeast Asia and South America, and as a commercial insecticide worldwide for at least 150 years. The use of rotenone as a selective piscicide for fisheries management is considerably more recent, dating from the 1930s (M'Gonigle & Smith 1938; Ball 1948). Cubé resin is the most commonly available extract and typically contains around 44% rotenone.

At least 28 other rotenoid compounds with similar chemical structures and varying levels of biological activity are present in cubé resin, albeit at lower concentrations. The most abundant of these are deguelin (22%), rotenolone (12ab-hydroxyrotenone, 6.7%) and tephrosin (12ab-hydroxydeguelin, 4.3%). The 25 minor rotenoids contribute less than 0.5% and may simply be decomposition products resulting from preparation and processing of the resin. Rotenone is the most toxic of the rotenoids followed by deguelin. Together, the four main compounds contribute greater than 95% of the toxicity of cubé resin (Fang & Casida 1999).

Rotenone preparations are unstable in light and air. Unlike some other pesticides, rotenone is not environmentally persistent and degrades rapidly under natural conditions. Rotenone powders lose much of their toxicity within weeks

without careful storage and must be protected from air, light and alkali, at temperatures not exceeding 25°C. Solutions of rotenone in organic solvent, when exposed to light and air, become successively yellow, orange and finally deep red due to oxidation (Cheng et al. 1972).

1.5 Common usage

The United States is the greatest consumer of rotenone and annual usage is estimated at between 25 000 and 60 000 kg (USEPA 1988). Rotenone is registered in the United States as a pesticide for the following uses:

As an insecticide for:

- Terrestrial food crops—for foliar preharvest application to vegetables, berries, tree fruit, nuts, forage crops and sugarcane; delayed dormant applications to deciduous tree fruit
- Terrestrial non-food crops—ornamentals, turf, shade trees and tobacco
- Greenhouse food crops—vegetables
- Greenhouse non-food crops—ornamentals
- Domesticated pets and their man-made premises—cats and dogs
- Livestock—cattle (beef and dairy), goats, horses, sheep and swine
- Household— flying and crawling insects
- Commercial and industrial—flying and crawling insects

As a piscicide for:

- Aquatic non-food crops—fish

2. USE IN FISHERIES MANAGEMENT AND RESEARCH

Selective piscicides such as rotenone and antimycin have been used extensively by fisheries managers in North America for more than 60 years for the management and assessment of fish populations in lakes, ponds and streams. The use of toxicants as a fisheries management tool has been practiced in at least 30 countries (Lennon et al. 1970). More than 30 chemicals have been employed as fish toxicants but rotenone and antimycin are the most widely used. Rotenone was first used in the United States in 1934 in Michigan (Ball 1948) and in Canada in 1937 (M'Gonigle & Smith 1938). Antimycin was first used experimentally in Wisconsin in 1963 (Derse & Strong 1963). Uses of piscicides in fisheries management include:

- Control of undesirable fish
- Eradication of harmful exotic fish
- Eradication of fish in rearing facilities and ponds to eliminate competing species
- Quantification of populations
- Treatment of drainages prior to impoundment
- Eradication of fish to control disease
- Restoration of threatened or endangered species

Use of rotenone as a selective piscicide for fisheries management is still common in the United States and Canada and represents a considerable proportion of total rotenone consumption although this is declining. Annual average rotenone use for fisheries management in North America decreased from 12 500 kg for the period 1988 to 1992 to 5600 kg in the following five-year period, although the data are somewhat skewed by the large-scale (20 600 kg) Strawberry Reservoir treatment in 1990. Most of this decrease was in the use of liquid formulations although there was a corresponding slight rise in the use of rotenone powders (McClay 2000).

For fisheries management purposes, liquid preparations of rotenone have been preferred because these emulsions are more easily dispersed in water than powders and they also have a greater capacity to penetrate thermally stratified water bodies (Almquist 1959). However, fish actively avoid the liquid toxicant formulations presumably because they can detect the solvents and dispersal ingredients (Dawson et al. 1998). Complete and uniform dispersal is therefore important in treated waters otherwise fish will move to toxicant-free areas and avoid exposure. Furthermore, since fish can be revived by removing them to toxicant-free water, exposed fish can recover if they move to clean water. There are instances where the efficiency of rotenone treatments was greatly reduced because of the presence of inlet springs on lake beds where fish sought refuge (Lennon et al. 1970).

Most fisheries agencies in North America now place greater emphasis on the use of powders, particularly for treating standing waters. Probable reasons for this are improvements in techniques for mechanical dispersion of the powder and its significantly lower cost. Another important factor is concerns about the environmental and public health effects of the petroleum-based solvents used in liquid products. However, liquid formulations are still preferred for treating flowing waters (McClay 2000).

The inability of some rotenone formulations to penetrate across thermoclines in thermally stratified waters has caused some researchers to suggest that this characteristic could be exploited to selectively eradicate coarse fish in some waters. Tompkins & Mullan (1958) suggested that applying rotenone to thermally stratified trout ponds would eliminate warm-water coarse fish in surface waters while the trout would be spared in the cooler water below the thermocline.

During the 1950s and 1960s the chemical 'renovation' of freshwater bodies was commonplace in North America. These treatments were designed to remove coarse fish species prior to stocking introduced game fishes such as rainbow trout. They were often highly successful, producing high quality game fisheries, although populations of coarse fish often recovered due to incomplete eradication or subsequent immigration. In recent years, rotenone has more often been used to remove pest or non-native fishes to allow recovery of indigenous stocks or for research on fish population structure and abundance (Bettoli & Maceina 1996). Rotenone has been used successfully to eliminate exotic trout in Australia to rehabilitate populations of endangered galaxiids (Sanger & Koehn 1997; Lintermans 2000) and to eradicate limited populations of European carp and mosquitofish (Sanger & Koehn 1997).

Rotenone sampling for research has been successfully applied in all freshwater environments including wetlands, ponds, lakes, streams and rivers (Bettoli & Maceina 1996). It has also been used to sample marine fish and invertebrates in estuaries (Ley et al. 1999) and coral reefs (Ackerman & Bellwood 2000). In such complex habitats as coral reefs, rotenone is particularly effective for sampling cryptic fish species. Ackerman & Bellwood (2000) found that rotenone samples increased the estimated number of species by 40% and total fish abundance by 50% compared with more traditional visual sampling methods.

Eradication of fish with rotenone has variable success depending on the type of environment and the amount of effort expended in achieving complete dispersal of the toxicant throughout the lake or drainage. In a review of large-scale treatments, Meronek et al. (1996) concluded that approximately 48% achieved their goal. In a stream or river system, it is important to achieve complete treatment of all tributaries within the drainage no matter how small. Complete dispersal of rotenone throughout a lake can be hampered by the difficulties in getting the toxicant into deeper water since applications are usually sprayed on the lake surface. A number of solutions to this problem have been tested including pumping rotenone slurry to the lake bottom with weighted pipes and sinking mud 'bombs' of rotenone. Fish toxicants have also been applied by surface-coating sand, which can then carry the chemical to the lakebed.

Dispersing toxicant in marginal zones with abundant plant growth can also present practical difficulties. The normal practice is for personnel to spray such zones with rotenone slurry using backpack sprayers. However, complete coverage is sometimes difficult to achieve, and because of the rapid loss of rotenone in such areas through chemical decay and adsorption to plants and sediment, fish may find refuge long enough to evade poisoning. Bettoli & Maceina (1996) state that wetland managers in the United States have generally abandoned toxicants for the control of carp because eradication is rarely complete and continuing management is required.

Rotenone treatments are usually most effective in small lakes or ponds with relatively clear water and without abundant aquatic macrophytes. They are therefore ideal for removing grass carp once the fish have reduced macrophyte biomass in managed systems.

A technical guidebook for the use of rotenone in fisheries management has recently been released by the American Fisheries Society (Finlayson et al. 2000).

3. NEW TECHNOLOGY—PEST FISH MANAGEMENT BAITS

A new development in the field of fish management is selective fish baits laced with rotenone, marketed as Prentox® Prenfish™ by Prentiss Incorporated. These are floating feed pellets containing around 2.5% rotenone either with or without the synergist piperonyl butoxide (Prentiss Incorporated 2000). These products have been formulated for the selective removal of grass carp and common carp (koi carp). Each pellet contains enough active ingredient to kill around 1 kg of fish. Floating bait training stations are established on a lake and

automatically release floating trainer baits onto the water surface within a retention ring to prevent wind dispersal of the baits. Following a period of training, usually up to two weeks, the trainer bait is replaced with toxic baits. The advantage of this system is that it reportedly allows selective removal of the target species without affecting other fish in the lake. Although not 100% effective at removing grass carp, it has a much higher success rate than other techniques since grass carp, especially, are notoriously difficult to catch. Estimates of catch efficiency for grass carp vary from 14 to 77% where several applications of bait are used (Fajt 1996). Kills of non-target species were either nil or very low despite the presence of up to 14 non-target fish species.

The Prentox® system has been tried in New Zealand and was moderately effective at removing grass carp from Lake Waingata (Rowe 1999). It was estimated that at least 50% of fish and probably more were eradicated by a single application of the Prentox® baits following a two-week training period.

4. MEASURING ROTENONE CONCENTRATIONS IN WATER AND OTHER MATERIALS

Fisheries management programmes should endeavour to measure rotenone concentrations in treated waters so that the efficiency of toxicant dispersal can be assessed. Post-treatment residues should also be measured to follow the rate of environmental loss. To be useful at measuring residual rotenone concentrations in water following fish treatments, analysis procedures need to be sufficiently sensitive to measure at μg/L (parts per billion) levels or below. Early methods for the chemical determination of rotenone relied on simple, qualitative, colorimetric procedures and were relatively insensitive and non-specific. With detection limits of around 20 to 50 μg/L, they are not useful for quantitative determination of rotenone residues in water, but they are still cheap, rapid and simple tests for the presence of rotenone. The original colorimetric method of Jones & Smith (1933) was improved by Gross & Smith (1934), Rogers & Calamari (1936) and Post (1955).

A much more sensitive method for quantifying rotenone in water at concentrations as low as 5 μg/L using high performance liquid chromatography (HPLC) was published in 1983 (Dawson et al. 1983) and is still recommended for verifying concentrations following fisheries management applications (Finlayson et al. 2000). More sophisticated analysis procedures, involving more complex sample preparation and concentration, have recently been published that are able to quantify rotenone residues in water and foodstuffs at lower than μg/L concentrations. These techniques are relatively complex and expensive and use HPLC, capillary gas chromatography, or liquid chromatography/particle beam mass spectrometry (Ho & Budde 1994; Draper et al. 1999; Pedersen & Shibamoto 1999; Jimenez et al. 2000).

No routine analyses for rotenone are currently offered in New Zealand, although most analytical laboratories have the capacity to develop rotenone analyses on an *ad hoc* basis with corresponding costs for method development and verification.

5. ENVIRONMENTAL PERSISTENCE

Rotenone is unstable in the presence of light, heat and oxygen. Containers of powdered formulations rapidly lose their activity when open to the air and should remain sealed whenever possible. Liquid formulations are more stable so long as they are stored sealed and kept in a cool dark place (Cheng et al. 1972).

Rotenone persistence in natural waters varies from a few days to several weeks depending on the season. The half-life of rotenone is longest in winter but may decrease to as little as a few hours in summer. Fisheries managers must carefully plan applications to take these considerations into account. For instance, unless all parts of a large water body or catchment can be treated simultaneously, the breakdown of rotenone may be so rapid in summer that fish can migrate back into previously treated areas.

Rotenone has been very widely used as a piscicide in North America and has often been used to treat large water bodies that have diverse recreational uses. Registration for such use by the U.S. Environmental Protection Agency is contingent on detailed knowledge of the environmental fate of rotenone and several studies have examined the persistence of rotenone under natural conditions in treated ponds. Bettoli & Maceina (1996) stated that both sediments and abundant vegetation can detoxify rotenone applications by adsorbing much of the rotenone applied. It is not known whether rotenone is metabolised by aquatic vegetation. Gilderhus et al. (1986, 1988) examined the fate of rotenone in shallow ponds at differing temperatures and found that rotenone loss was ten times faster at 23°C than at 1°C. The half-life of rotenone in natural waters above 20°C is generally less than one day. Gilderhus (1982) and Dawson et al. (1991) showed that the rapid loss of rotenone in treated ponds was not solely attributable to chemical decay. A significant fraction became bound to suspended particles and to bottom sediments, but the presence of aquatic plants did not significantly affect rotenone toxicity. Depending on water temperature, rates of loss were up to three times slower in a concrete-lined pond compared with an earthen-bottom pond. However, rotenone did not persist in sediments and because of chemical breakdown decreased to below detection limits within 3 days at 15–22°C, and within 14 days at 8°C.

Solutions of rotenone aged under laboratory rather than natural conditions are considerably more stable. Half-lives for laboratory-aged solutions of rotenone in soft water were 13 days at 17°C and 22 days at 12°C (Marking & Bills 1976). The toxicity of aged rotenone solutions declines in parallel with chemical decay indicating that breakdown products are comparatively non-toxic (Marking & Bills 1976).

6. THE TOXICITY OF ROTENONE

6.1 How rotenone works

Rotenone is a highly specific metabolic poison that affects cellular aerobic respiration, blocking mitochondrial electron transport by inhibiting NADH-ubiquinone reductase (Singer & Ramsay 1994). The effects of rotenone are therefore similar to those produced by other poisons that affect electron transport or oxidative phosphorylation, including antimycin, cyanide and

dinitrophenol. Cellular uptake of oxygen is blocked and production of cellular energy in the form of nucleoside triphosphates is greatly reduced. In rotenone-poisoned fish, reduced cellular uptake of blood oxygen results in increased blood pO_2, whilst compensatory increases in cellular anaerobic metabolism and associated production of lactic acid causes blood acidosis (Fajt & Grizzle 1998). Death results from tissue anoxia, especially cardiac and neurological failure.

6.2 Toxicity to aquatic invertebrates

Sensitivity to rotenone is highly variable among aquatic invertebrates although most species are more resistant than fish (Table 1). However, because field applications of rotenone invariably employ a certain degree of excess chemical to ensure a complete fish kill, most invertebrates are also eliminated. Follow-up studies of rotenone applications have shown that populations of aquatic invertebrates quickly recover to pre-treatment levels.

TABLE 1. THE TOXICITY OF ROTENONE TO SELECTED AQUATIC INVERTEBRATES.
The median lethal concentrations are those causing 50% mortality within the specified time period. LC50 – median lethal concentration; LC100 – concentration causing 100% mortality.

GROUP	SPECIES	TEST ENDPOINT	LETHAL CONCENTRATION	REFERENCE
Flatworm	*Catenula* sp.	LC50 24h	5.10 mg/L	1
	Planaria sp.	LC50 24h	< 0.500 mg/L	2
Annelid worms	Leech	LC50 48h	< 0.100 mg/L	2
Copepod	*Cyclops* sp.	LC100 72h	< 0.100 mg/L	5
Branchiura	*Argulus* sp.	LC50 24h	~ 0.025 mg/L	2
Cladoceran	*Daphnia pulex*	LC50 24h	0.027 mg/L	1
	Daphnia pulex	LC50 24h	< 0.025 mg/L	2
	Diaptomus siciloides	LC50 24h	< 0.025 mg/L	2
Ostracod	*Cypridopsis* sp.	LC50 24h	0.490 mg/L	1
Conchostracan	*Estheria* sp.	LC50 24h	~ 0.050 mg/L	2
Freshwater prawn	*Palaemonetes kadiakensis*	LC50 24h	5.15 mg/L	1
Crayfish	*Cambarus immunis*	LC50 72h	> 0.500 mg/L	2
Dragonfly naiad	*Macromia* sp.	LC50 24h	4.70 mg/L	1
Stonefly naiad	*Pteronarcys californica*	LC50 24h	2.90 mg/L	4
Backswimmer	*Notonecta* sp.	LC50 24h	3.42 mg/L	1
	Notonecta sp.	LC50 24h	~ 0.100 mg/L	2
Caddis fly larvae	*Hydropsyche* sp.	LC50 96h	0.605 mg/L	1
Whirligig beetle	*Gyrinus* sp.	LC50 24h	3.55 mg/L	1
Water mite	Hydrachnidae	LC50 96h	~ 0.050 mg/L	2
Snail	*Physa pomilia*	LC50 24h	6.35 mg/L	1
	Oxytrema catenaria	LC50 96h	1.75 mg/L	1
	Lymnaea stagnalis	LC50 96h	> 1.00 mg/L	2
Bivalve Mollusc	*Dreissena polymorpha*	LC50 48h	0.219 mg/L	3
	Obliquaria reflexa	LC50 48h	> 1.00 mg/L	3
	Elliptio buckleyi	LC50 96h	2.95 mg/L	1
	Elliptio complanata	LC50 96h	2.00 mg/L	1
	Corbicula manilensis	LC50 96h	7.50 mg/L	1

Refs: 1, Chandler 1982; 2, Hamilton 1941; 3, Waller et al. 1993; 4, Sanders & Cope 1968; 5, Meadows 1973.

Applications of rotenone in lakes and ponds to kill fish usually cause significant declines in zooplankton and may also affect some bottom fauna. These populations of more sensitive animals usually recover over periods of one to eight months following the treatment. Zooplankton species like *Daphnia* are generally more sensitive to rotenone than the larger benthic macro-invertebrates such as molluscs, crayfish, freshwater prawns, amphipods, oligochaete worms, and chironomid midge larvae (Hamilton 1941; Morrison 1977). Cladocerans and copepods seem to be the most susceptible invertebrate groups. Although copepod populations quickly recover following rotenone treatments, cladoceran abundance may take many months to recover to pre-treatment levels. Inchausty & Heckmann (1997) found that the incidence of a fish parasite (*Diplostomum*) decreased by nearly 100% for at least five years following the very large-scale treatment of Strawberry Reservoir, Utah, in 1990. This was attributed to the loss of invertebrate species such as snails that acted as intermediate hosts for the parasite.

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

Rotenone treatment of streams in Papua New Guinea caused immediate catastrophic invertebrate drift, especially mayflies, but did not cause large-scale mortality or a significant decline in benthic invertebrate abundance (Dudgeon 1990).

6.3 Toxicity to fish

Rotenone is highly toxic to fish, with 24 hour LC50 values commonly between 5 and 100 µg/L. Temperature and contact time are the two main variables that significantly affect toxicity. The time required to cause 100% mortality decreases approximately 2 to 3-fold for each five-degree rise in temperature (Gilderhus 1972). A summary of published data on the toxicity of rotenone to various species is given in Table 2 which includes a variety of endpoints, contact times and temperatures. Other variables such as water hardness, pH and rotenone formulation are not included in the table but may be obtained from the original publications in those cases where they were reported. It is generally agreed that rotenone is more effective in acidic and soft water than in alkaline or hard water when applied to natural water bodies (Foye 1964). Although Meadows (1973) found that rotenone was more toxic to roach,

Rutilus rutilus, in soft water, Marking & Bills (1976) found no significant effect of either pH or water hardness on the susceptibility of several species in standardised laboratory toxicity tests. The efficiency of rotenone is therefore difficult to predict by extrapolation of laboratory or field data, and on-site testing should be employed to determine effective concentrations. The study by Marking & Bills (1976) is the most extensive account of the toxicity of rotenone to fish including the influence of several key environmental variables. However, even under their standardised test conditions, median lethal concentrations varied by more than three-fold for repeated tests with the same species. Therefore, effective concentrations given in Table 2 should be considered as indicative only. Furthermore, these values are median (50%) lethal concentrations; concentrations required to achieve a 100% kill are likely to be much higher.

Individual species sensitivities vary widely, with salmonids most sensitive and goldfish, carp and bullhead catfish least so. Differences in the sensitivities of

TABLE 2. THE TOXICITY OF ROTENONE TO FISHES.
The median lethal or effective concentrations are those causing 50% mortality or effect respectively, within the specified time period. Data are a selection of endpoints that vary with time of exposure and test temperature. LC50 – median lethal concentration in water; EC50 – median effective concentration; LD50 – median lethal oral or injected dose; NR – not recorded.

SPECIES		TEST ENDPOINT	TEMP.	LETHAL CONCENTRATION	REFERENCE
Salmonidae					
Chinook salmon	*Oncorhynchus tshawytscha*	LC50 24h	12°C	5.6 µg/L	5
rainbow trout	*Oncorhynchus mykiss*	LC50 3h	12°C	8.8 µg/L	5
		LC50 3h	17°C	3.7 µg/L	5
		LC50 24h	12°C	3.4 µg/L	5
		LC50 24h	17°C	2.2 µg/L	5
		LC50 48h	17°C	2.0 µg/L	2
		LC50 96h	12°C	2.3 µg/L	5
		LC50 96h	17°C	2.2 µg/L	5
brown trout	*Salmo trutta*	LC50 1h	17°C	5.5 µg/L	1
Galaxiidae					
black mudfish	*Neochanna diversus*	EC50 1h	18°C	<< 100 µg/L	4
Cyprinidae – carps and minnows					
crucian carp	*Carassius carrasius*	LC50 96h	11°C	360 µg/L	1
goldfish	*Carassius auratus*	LC50 96h	12°C	24.9 µg/L	5
grass carp	*Ctenopharyngodon idella*	LC50 6h	11°C	24.5 µg/L	1
common carp (koi,	*Cyprinus carpio*	LC50 6h	12°C	13.5 µg/L	5
European carp, mirror carp)		LC50 24h	11°C	30.5 µg/L	1
		LC50 24h	NR	< 10 µg/L	3
		LC50 24h	12°C	4.2 µg/L	5
		LC50 96h	12°C	2.5 µg/L	5
		LD50 48h	25°C	8.1 mg/kg	7
		LD99 48h	25°C	11.6 mg/kg	7
rudd	*Scardinius erythropthalmus*	LC50 1h	20°C	24.5 µg/L	1
roach	*Rutilus rutilus*	LC50 1h	20°C	85 µg/L	1
		LC50 24h	15°C	24.5 µg/L	1
		LC50 6h	10°C	38 µg/L	1
gudgeon	*Gobio gobio*	LC50 1h	20°C	125 µg/L	1

SPECIES		TEST ENDPOINT	TEMP.	LETHAL CONCENTRATION	REFERENCE
Ictaluridae – bullhead catfishes					
Channel catfish	*Ictalurus punctatus*	LC50 48h	17°C	7.3 µg/L	2
		LC50 3h	12°C	86 µg/L	5
		LC50 3h	17°C	70.5 µg/L	5
		LC50 3h	22°C	37 µg/L	5
		LC50 24h	12°C	27 µg/L	5
		LC50 24h	17°C	20 µg/L	5
		LC50 24h	22C	8.2 µg/L	5
		LC50 96h	12°C	10 µg/L	5
		LC50 96h	17°C	8.2 µg/L	5
		LC50 96h	22°C	8.2 µg/L	5
Black bullhead	*Ictalurus melas*	LC50 24h	12°C	33.3 µg/L	5
		LC50 96h	12°C	19.5 µg/L	5
Centrarchidae – sunfishes					
bluegill sunfish	*Lepomis macrochirus*	LC50 24h	22°C	7.0 µg/L	5
Percidae – perches					
perch	*Perca fluviatilis*	LC50 1h	10°C	38 µg/L	1
yellow perch	*Perca flavescens*	LC50 3h	12°C	7.5 µg/L	5
		LC50 24h	12°C	4.6 µg/L	5
		LC50 96h	12°C	3.5 µg/L	5
Catostomidae – suckers					
buffalo	*Ictiobus* sp.	LC50 24h	NR	< 8.3 µg/L	3
white sucker	*Catostomus commersoni*	LC50 96h	12°C	7.2 µg/L	5
Poecilliidae – livebearers					
mosquitofish	*Gambusia affinis*	EC50 1h	18°C	84 µg/L	4
susceptible strain		LC50 24h	NR	17 µg/L	6
resistant strain		LC50 24h	NR	31 µg/L	6

Refs: 1, Meadows 1973; 2, Waller et al. 1993; 3, Hamilton 1941; 4, Willis & Ling 2000; 5, Marking & Bills 1976; 6, Fabacher & Chambers 1972; 7, Fajt & Grizzle 1993.

species have been exploited by fisheries managers to reduce or eliminate populations of unwanted species in mixed-species communities (Hooper & Crance 1960). Reasons for such variation in sensitivity may reside in differences in the levels of liver enzymes responsible for the chemical breakdown or detoxification of rotenone. Fukami et al. (1969) examined the detoxification of radiolabelled rotenone by liver enzymes in carp. Rotenone was rapidly detoxified to a variety of hydroxylated rotenoids and more water-soluble products with toxicities at least one to two orders of magnitude less than the parent rotenone. Twenty-four hours after administration, only 1.5% of the radiocarbon label was recovered from the fish as rotenone while 78% was found, mostly as water-soluble breakdown products, either in the gut or the aquarium water. The liver is the major site of xenobiotic biotransformation in fish, and hepatic conversion of low-solubility organic toxins to more readily excretable water-soluble metabolites is a common method of detoxification in all vertebrates. The formation of bile by the liver is a major route of excretion for breakdown products of endogenous compounds as well as the elimination of xenobiotics and their metabolites (Hinton et al. 2001). Rach & Gingerich (1986) examined the accumulation of rotenone and its rate of breakdown in the tissues of three warm water species: carp, perch, and bluegills. They found that rotenone was quickly eliminated in carp with rotenoid metabolites

accumulating in the bile, confirming biliary excretion from the liver in the form of excretable metabolites as the most likely route of detoxification and elimination. Bottom-feeding omnivores such as carp and catfish are known to possess greater concentrations of the mixed function oxidase (MFO) enzymes responsible for rotenone breakdown because they are exposed to a wide variety of foreign chemicals in their diets (Buhler & Rasmussen 1968). However, enhanced xenobiotic metabolism by carp cannot fully explain these species' tolerance to rotenone since carp accumulate up to six-fold higher tissue concentrations at death compared with other species (Rach & Gingerich 1986). Significantly greater dependence on anaerobic ATP production in these hypoxia-tolerant species may be another reason for rotenone tolerance, as well as the production of alternative substrates for ATP synthesis that circumvent the NADH-reliant mitochondrial pathways affected by rotenone. To date, the respective half-lives of rotenone in tolerant and susceptible species has not been examined.

Synergised formulations of rotenone containing piperonyl butoxide are around twice as toxic as non-synergised products (Marking & Bills 1976). This difference is due to the action of piperonyl butoxide in inhibiting the activity of MFO enzymes in the liver responsible for detoxifying rotenone. The use of synergised formulations is particularly useful if some degree of rotenone resistance is suspected. Fabacher & Chambers (1972) studied a population of mosquitofish that were nearly two-fold more tolerant to rotenone than unexposed individuals. This difference was solely the result of increased levels of MFO enzymes. This same population was also resistant to a wide range of other pesticides, particularly the chlorinated aryl hydrocarbons such as endrin and chlordane (Culley & Ferguson 1969). However, resistances to pesticides other than rotenone was much greater, up to 500-fold for some chemicals, indicating that the ability to develop a high degree of resistance to rotenone is probably limited.

Rotenone does not accumulate in the body with prolonged exposure. In the absence of synergists, rotenone is rapidly detoxified by the MFO system of the liver so that fish not receiving a fatal dose will recover relatively quickly. In 30 day flow-through exposures, the incipient lethal concentration was reached by 96 hours with no further increase in toxicity (Marking & Bills 1976). Repeated exposures of fish to sub-lethal doses may bring about some degree of resistance by stimulating increased levels of MFO enzymes, although the evidence of Fabacher & Chambers (1972) indicates that resistance of this kind is likely to be relatively minor. The activities of several other types of enzymes were also stimulated in livers of a tropical cyprinid species, *Labeo rohita*, by prolonged sub-lethal exposure (Medda et al. 1995).

A common misconception regarding the piscicidal action of rotenone is that it acts by blocking oxygen uptake at the gills. This fallacy appears to originate from the studies of Danneel (1933) and Hamilton (1941) who attributed death of exposed fishes to either gill necrosis or constriction of gill capillaries and subsequent asphyxia from branchial ischemia. Öberg (1959) did not find necrosis or circulatory changes in gills, and the true action of rotenone in blocking cellular respiration via the electron transport chain was established by Lindahl & Öberg (1961). Rotenone is rapidly absorbed across the gill epithelium and distributed to tissues in the blood where it blocks oxygen use by cells. Far from reducing oxygen uptake in fish, arterial oxygen actually increases significantly during the early stages of rotenone

poisoning due to increased ventilation rates. Up to ten-fold increases in venous oxygen tensions then follow because of the blockage of cellular oxygen uptake in tissues (Fajt & Grizzle 1998).

Characteristic behaviour patterns are observed in fish poisoned with rotenone. In the very early stages of poisoning, fish show reduced opercular movements and erratic bursts of swimming followed by periods of listlessness. Subsequently, ventilation rate increases with frequent coughing and fish rise to the surface to gulp air or skim the highly oxygenated water surface film. Eventually fish lose equilibrium and either float at the surface or sink to the bottom where they remain until death.

The eggs of susceptible species such as salmonids are around 100 times less sensitive than juvenile and adult fish so rotenone applications to eliminate coarse fishes should not affect redds (Marking & Bills 1976). There are no data on the sensitivities of the eggs of coarse fish species.

6.4 Toxicity to amphibians and reptiles

Amphibian adults and reptiles are less sensitive than fish and aquatic invertebrates and should not be harmed when rotenone is applied at normal piscicidal concentrations (Farringer 1972). Larval amphibians show sensitivities similar to the most resistant fish species (Table 3; Hamilton 1941; Chandler 1982). Hamilton (1941) noted that the response of larval amphibians depended on the stage of metamorphosis. Larvae that were wholly or partially dependent upon gill respiration were far more sensitive than those that were fully metamorphosed and breathing air, indicating that rotenone is far more readily absorbed across gill epithelium than skin.

6.5 Toxicity to birds

Rotenone is slightly toxic to wildfowl, and birds are extremely unlikely to be affected by normal fisheries management programmes (Table 3). Rotenone poisoning as a result of consuming poisoned fish or even fish management baits is highly unlikely. Carp management baits contain around 0.01g of rotenone and a duck would need to consume around 200 baits to receive a fatal dose. The concentration of rotenone in poisoned fish is usually 25 000 times lower than in baits.

LD50 values in mallard ducks and pheasants are greater than 2000 mg/kg and 1680 mg/kg respectively, and a dietary LC50 of between 4500 and 7000 mg/kg has been reported for Japanese quail (Hill et al. 1975). The minimum lethal dose (MLD) for robin nestlings is 195 mg/kg and for sparrow nestlings is 199 mg/kg (Cutkomp 1943). Adults of both species had significantly higher MLD values.

6.6 Toxicity to humans and other terrestrial mammals

Most mammal species are relatively resistant to rotenone (Table 3). No fatalities in humans have been reported in response to normal use of rotenone products. However, there is at least one reported human fatality in a child who ingested around 10 mL of a product called Galicide. Galicide is an insecticide for external use on animals and contains 6% rotenone as well as essential oils of clove, cinnamon, fir, rosemary and thyme. The child developed a gradual loss of consciousness over two hours and died of respiratory arrest six hours later (DeWilde et al.

TABLE 3. THE TOXICITY OF ROTENONE TO VERTEBRATES OTHER THAN FISH.

SPECIES	TEST ENDPOINT	LETHAL CONCENTRATION	REFERENCE
Amphibians			
Rana pipiens – adult	acute LC50	2 mg/L	1
Rana sphenocephala – larva	acute LC50 24h	0.58 mg/L	2
Birds			
Pigeon	acute LD50 I.V.	1 mg/kg	1
Mallard duck	acute LD50 oral	2600–3568 mg/kg	8
Ring-necked pheasant	acute LD50 oral	1608 mg/kg	8
Japanese quail	acute LD50 oral	1882 mg/kg	8
Mammals			
Mouse	acute LD50 oral	350 mg/kg	3
Rat	acute LD50 oral	132–1500 mg/kg	3
	acute LD50 oral	39.5 mg/kg female	7
	acute LD50 oral	102 mg/kg male	7
	acute LD50 I.V.	0.2 mg/kg	9
	chronic LD50 oral	~ 10 mg/kg	6
Guinea pig	acute LD50 oral	75 mg/kg	1
	acute LD50 I.P.	2 mg/kg	1
	acute LD50 I.M.	7 mg/kg	1
	acute LD50 S.C.	16 mg/kg	1
Rabbit	acute LD50 oral	~ 1.5 g/kg	1
	acute LD50 I.V.	~ 0.35 mg/kg	1
	acute LD50 I.M.	~ 5 mg/kg	1
	acute LD50 S.C.	~ 20 mg/kg	1
Cat	acute LD50 I.V.	~ 0.65 mg/kg	1
Dog	acute LD50 I.V.	~ 0.65 mg/kg	1
	chronic LD50 oral	~ 10 mg/kg (30 d)	1
	chronic LD50 oral	>> 10 mg/kg (180 d)	6
Human*	acute LD50 oral	300–500 mg/kg	4, 5

* estimated.

I.M. – intramuscular; I.P. – intraperitoneal; I.V. – intravenous; S.C. – subcutaneous

Refs: 1, Haag, H.B. 1931; 2, Chandler, J.H. 1982; 3, Kidd & James 1991; 4, Ray 1991; 5, Gosselin et al. 1984; 6, National Research Council 1983; 7, USEPA 1988; 8, Hill et al. 1975; 9, Hayes 1982.

1986). The lethal dose was estimated at 40 mg/kg, which is considerably less than the figure given in Table 3. However, the essential oils in Galicide were alleged to have enhanced toxicity, firstly, by promoting absorption of the water insoluble rotenone from the gastrointestinal tract and, secondly, by causing acute kidney damage and reducing the clearance rate of rotenone. Autopsy revealed concentrations of 2 to 4 ppm rotenone in blood, liver and kidney, and autopsy histological findings were consistent with severe rotenone poisoning.

Non-lethal symptoms have been reported in humans following prolonged occupational exposure to rotenone dusts during large-scale fisheries operations (Pintler & Johnson 1958). Headaches, sore throats and other cold-like symptoms were reported due to inhalation of dusts. Contact symptoms included sores on mucous membranes, skin rashes and severe irritation of the eyes. Poisoning is more likely as a result of inhalation rather than ingestion, since intestinal absorption of water insoluble rotenone is relatively inefficient. The simulated lethal oral dose for a 70 kg human is variously estimated at between

10 and 200 g. However, ingested carrier solvents may greatly increase intestinal absorption and enhance toxicity as described above.

7. METABOLIC FATE OF ROTENONE IN HIGHER ANIMALS (BIRDS AND MAMMALS)

Rotenone is not easily absorbed in higher animals and does not accumulate in the body. Absorption of rotenone in the stomach and intestines is relatively slow and incomplete, although fats and oils in the diet promote its uptake due to solubility effects. Large oral doses (200 mg/kg in pigeons, 10 mg/kg in dogs) usually stimulate vomiting in animals (Haag 1931). The same is reportedly true for humans following suicidal ingestion of Derris root (Ray 1991).

Once absorbed, rotenone is effectively broken down by the liver to produce less toxic excretable metabolites. Approximately 20% of the oral dose (and probably most of the absorbed dose) is excreted within 24 hours (Ray 1991); around 80% as water soluble products with the remainder as hydroxylated rotenoids (Fukami et al. 1969).

8. PATHOGENIC, TERATOGENIC, CARCINOGENIC AND ANTI-CANCER EFFECTS

Evidence for teratogenic and carcinogenic activity of rotenone is circumstantial at best. Innes et al. (1969) found no evidence of carcinogenic effects in two strains of mice given oral doses of rotenone for 18 months. Gosalvez & Merchan (1973) reported that intraperitoneal injection of 17 mg/L rotenone for 42 days resulted in the induction of mammary tumours in rats. However, higher rotenone doses did not cause increased incidence of cancer, thus these effects were not dose related. Studies by the U.S. National Institute of Health with rats fed on diets containing up to 75 mg/kg for 2 years found equivocal evidence of carcinogenic activity in male rats only, with a slightly increased incidence of rare parathyroid gland tumours. However, the incidence of some other cancers in dosed rats declined, particularly liver and subcutaneous tumours. There was no evidence of carcinogenic activity in female rats at the same doses, and no evidence of carcinogenic activity in either male or female mice fed diets containing up to 1200 mg/kg rotenone. Rotenone was therefore not mutagenic at concentrations below those that are acutely toxic to cells (National Toxicology Program 1984). A study specifically designed to repeat the work of Gosalvez & Merchan (1973) was unable to induce any carcinogenic effect, thus the reports of carcinogenic activity were not reproducible under similar experimental conditions (Greenman et al. 1993). Pregnant rats given oral doses of 5 mg/kg/day produced a significant number of young with skeletal deformities, however, these effects were not dose-dependent since higher doses had no effect. Because the effects were not dose-dependent the evidence for teratogenicity was inconclusive (Extoxnet 1996).

Rotenone has recently been reported to cause effects in rats similar to those of Parkinson's disease. Lesions were observed in dopamine producing neurons of

the substantia nigra of the brains of rats continuously exposed by I.V. infusion for 5 weeks to 2–3 mg/kg body weight per day (Betarbet et al. 2000). However, predictions for human health effects are difficult to draw from this study. Continuous intravenous injection is not a good model for occupational rotenone use where exposure would be more intermittent and via ingestion, inhalation or skin absorption. Marking (1988) observed no changes in the brains of rats given oral doses of up to 75 mg/kg per day for two years. Thiffault et al. (2000) attributed rotenone-induced damage of the substantia nigra to effects related to increased turnover of dopamine. They found that acute high doses caused increased dopamine turnover in mice although low subchronic doses did not. Other toxins known to cause Parkinsonism were, like rotenone, thought to affect mitochondrial ATP production leading to the production of reactive oxygen within cells, however, this has been discounted by Lotharius & O'Malley (2000). Therefore the mechanism by which rotenone induces lesions in the substantia nigra remains unclear.

Recent research has revealed that rotenone may be an effective agent against certain types of cancers by its action in inhibiting cellular respiration. Both rotenone and deguelin are effective at low micromolar concentrations at killing mouse liver cancer cells (Hepa 1c1c7) and human epithelial breast cancer cells (MCF-7) by inhibiting NADH:ubiquinone oxidoreductase (Fang & Casida 1998).

9. DETOXIFYING ROTENONE IN WATER AND THE USE OF FISH ANTIDOTES

Rotenone in water is rapidly detoxified by the addition of a strong oxidising agent. Potassium permanganate ($KMnO_4$) and chlorine have been used for this purpose although $KMnO_4$ is considerably more effective (Marking & Bills 1976). Potassium permanganate is commonly used during rotenone treatments to neutralise toxicity downstream from the target zone in a stream or river. In cove rotenone treatments, whereby a cove or bay in a lake is poisoned to sample fish populations, the cove is enclosed by a stop net prior to rotenone release and $KMnO_4$ may be released outside the net to safeguard the adjacent water body. However, both of these compounds are toxic to fish at concentrations only slightly greater than those required to deactivate rotenone. Given the rapid loss of rotenone when applied during warm conditions, the use of these deactivating compounds is not recommended.

Fish poisoned with rotenone usually come to the surface to gulp air or oxygenated surface water and can be caught and transferred to clean water. In the early stages of poisoning simply transferring them to uncontaminated, aerated water can revive most fish. Bouck & Ball (1965) found that methylene blue at a concentration of 5 mg/L was useful in assisting recovery of poisoned fish but its use in field situations has met with limited success and it is not to be relied on. Once fish lose equilibrium, it is usually impossible to revive them in either clean water or with methylene blue.

10. FOOD WEB MANIPULATION WITH ROTENONE

Rotenone has been shown to effectively improve water quality in small eutrophic lakes by exterminating planktivorous fishes and bottom-scavenging fishes that re-suspend bottom sediments and nutrients. A seven year biomanipulation experiment was conducted in Lake Wirbel, Poland, from 1988 to 1994 using three different biomanipulation measures to control planktivorous fishes such as roach, *Rutilus rutilus*, white bream, *Blicca bjoerkna*, and Moderlieschen, *Leucaspius delineatus*. The three biomanipulation measures used were introductions of predatory pike, extensive fishing, and eliminating all fish using rotenone. The first two measures were ineffective in achieving any improvement in water quality. However, following the application of rotenone in 1991, water transparency improved by nearly 40%, total phosphorous concentration decreased by nearly 50% and algal biomass declined 2.8 fold. These improvements were sustained for at least three years until the study concluded in 1994 and were attributed to a significant increase in abundance of a large cladoceran, *Daphnia cucullata*, that kept phytoplankton in check (Prejs et al. 1997).

Such increases in water clarity are commonly observed following rotenone treatments and are usually attributed to the elimination of bottom-scavenging fishes (Bradbury 1986). However, improvements in water clarity have also been attributed to reductions in phytoplankton and zooplankton by rotenone (Bradbury 1986). Dawson et al. (1991) observed significant reductions in turbidity, biochemical oxygen demand, total suspended solids, chlorophyll *a* and pheophytin *a* following rotenone treatment of ponds lacking fish. Treatment of a small shallow pond to eliminate grass carp and sunfish with 150 mg/L rotenone in early winter caused an immediate and almost total loss of all zooplankton. Copepods were the first group to reappear within one month, followed by rotifers. Cladocerans were the slowest zooplankton group to recover taking eight months to reach pre-treatment levels (Beal & Anderson 1993).

Part 2 Environmental risk assessment for the use of rotenone in New Zealand

11. INTRODUCTION

Risk assessment for environmental or human health involves:

- Hazard identification—an evaluation of a chemical's toxic properties
- Dose response assessment—estimates of the amount of the chemical that could potentially cause an adverse effect
- Exposure assessment—estimates of the potential exposure of the environment or people
- Risk characterisation—calculating the risk to people or the environment based on toxic hazard, dose response and occupational or environmental exposure

A review of the toxicity of rotenone and its use in fisheries management and research is provided in Part 1 of this report. Rotenone is a natural toxin occurring in several tropical plants and is used as both an insecticide and piscicide (selective fish poison).

Treating bodies of water with piscicides is the only effective way of eliminating unwanted species other than complete dewatering. Such chemical renovation is conducted for several reasons including the management of sport fisheries, quantification of fish populations, eliminating competing species in aquaculture ponds, eradicating exotic species, clearing watersheds prior to impoundment and eradication of diseases. Rotenone has been used extensively in the United States and Canada for chemical renovation of natural (ponds, lakes and streams) and artificial (aquaculture ponds) water-bodies for more than sixty years. In that time, it has been proven to be a highly effective and selective poison for fisheries management and has not produced deleterious environmental effects despite some very large-scale treatments. Its future use in New Zealand is likely to be in the application of wettable powders and pelleted baits for the eradication or control of certain pest fish species (koi carp, bullhead catfish, rudd and mosquitofish) in limited standing water bodies (wetlands, dams, ponds and small lakes), and for the targeted removal of grass carp following lake weed management.

12. ADVANTAGES OF ROTENONE

Lennon et al. (1970) conducted an extensive review of the reclamation of ponds, lakes, and streams with toxicants and presented summaries of the properties of a total of 30 candidate fish toxicants that had been used internationally. Of these compounds, rotenone was the most suitable substance based on the following criteria:

- Low cost
- Extremely high toxicity to fish
- Low toxicity to wildlife and humans
- Degrades rapidly in the environment and does not accumulate
- Rotenone is a natural product

13. DISADVANTAGES OF ROTENONE

Some of the factors that make rotenone useful for chemical treatment of waters may also been seen as disadvantages:

- Rotenone is chemically unstable and degrades rapidly on exposure to light and air. Powdered formulations lose their toxicity within weeks to months without careful storage.
- Rotenone has low solubility in water and complete dispersal throughout a water body may not be easily achieved. Rotenone strongly adsorbs to sediments and suspended particulate matter reducing effective concentrations in turbid waters.
- Liquid formulations of rotenone, although more effectively dispersed in water, generate public health concerns, produce noticeable tastes and odours in treated waters, and are easily detected and avoided by fish.
- Rotenone toxicity varies between species. Coarse fish are commonly less sensitive than valued species such as trout. There is considerable risk of poisoning non-target species during rotenone operations.
- Rotenone toxicity varies with temperature and pH.

Furthermore, given current concerns about the widespread use of pesticides in the environment, especially in situations whereby compounds could enter the human food chain, the application of a toxic, albeit chemically unstable, poison to waterways may meet with considerable public opposition.

14. ALTERNATIVES TO THE USE OF ROTENONE

Other than complete and prolonged dewatering, toxicants are the only method that is likely to completely eliminate undesirable fish in a body of water. Although the persistent application of traditional fishing methods can reduce populations to manageable levels in the short term, continually fishing any commercially undesirable species is economically unsustainable. Complete elimination of any species by fishing is unlikely given the exponential increase in effort required as catch-per-unit-effort declines.

More than 30 substances have been used internationally as fish toxicants with varying degrees of success. Some of these compounds have been used widely despite undesirable environmental effects. For instance, some large-scale applications of toxaphene in the United States were more effective at eliminating wildfowl than fish from lakes (Lennon et al. 1970). Toxaphene and some other candidate piscicides are highly persistent environmental contaminants and may pose significant long-term risks to the health of humans and wildlife.

A number of general insecticides such as dichlorvos, endrin, and malathion have been tried as fish toxicants with varying degrees of success. Many are more environmentally persistent than rotenone and often show greater toxicity to higher animals such as birds and mammals.

Rotenone and other natural products have the advantage that they usually break down rapidly in the environment and are easily metabolised by animals receiving sub-lethal doses. Natural products that have been used as piscicides are antimycin, croton seed, cunaniol, ichthyothereol, nicotine, and the plant saponins. Antimycin and saponins are the most widely used but both are probably less desirable than rotenone. Antimycin is a highly effective piscicide (effective at part per billion concentrations) and breaks down very rapidly in natural waters, but it is highly toxic to birds and mammals, is considerably more expensive than rotenone, and there are increasing concerns about the widespread environmental use of antibiotic compounds. Saponins are relatively cheap and although they show low toxicity to higher animals and high toxicity to fish, a much longer contact time (up to 24 hours) is required for effective fish kills than for rotenone. Lime is a natural product that is effective at killing fish but is likely to be non-specific and may cause long-term changes to treated water by altering pH balance and water hardness.

15. ROTENONE USE IN NEW ZEALAND

Rotenone is registered for use in New Zealand as a pesticide to control insect pests (insecticide) on ornamental and crop plants. It is sold widely in garden centres and supermarkets as Derris Dust. Rotenone is not currently registered for use as a piscicide.

Use in New Zealand for fisheries management has been very limited although both whole lake and poisoned bait treatments have been conducted. At least one large-scale experiment in bioremediation of a water body has employed rotenone in New Zealand. Lake Parkinson (a 1.9 ha dune lake south of Auckland) was treated with liquid rotenone (0.15 mg/L active ingredient—Chemfish) in 1981 to completely eradicate all fish, including grass carp introduced in 1976/77 to remove nuisance water plants (Rowe & Champion 1994).

Rotenone baits (Prentox®—see section 17)) were used to remove grass carp from Lake Waingata in 1999 following the introduction of these fish to the lake four years previously. At least 22 fish were removed by a single application of baits without collateral loss of non-target fish and with no effects on waterfowl (Rowe 1999).

Rotenone has been used in New Zealand for sampling of marine fish populations, especially for collection of museum specimens from habitats that are otherwise difficult to sample, such as reefs or burrows, and for surveying populations of cryptic reef species (Willis 2001).

Under Section 15 of the Resource Management Act 1991, it is illegal to discharge any contaminant, such as rotenone, into water unless that discharge is allowed by a regional plan, a resource consent or regulations. Future use of rotenone for experimental purposes or the control of undesirable fish will

require specific experimental use discharge consents under the Resource Management Act. Additional approvals may be required under the Hazardous Substances and New Organisms Act 1996 and the Agricultural Compounds and Veterinary Medicines Act 1997.

16. ROTENONE FORMULATIONS FOR GENERAL DISPERSAL APPLICATIONS

Rotenone is available in powders or liquid formulations. Powders are available either for insecticide (0.5% rotenone) or piscicide (5% rotenone) use. Liquids are available as 5% formulations containing up to 90% petrochemical solvents and dispersants. Products may contain synergists such as piperonyl butoxide and other pesticides. Typical fisheries applications use powders for treating small or shallow standing waters (ponds and lakes) or liquids for flowing waters (streams and rivers). Applications rates usually aim to achieve concentrations of between 1 and 3 mg/L rotenone to eliminate all fish. The use of powders is recommended to avoid environmental contamination by petrochemical solvents.

17. PRENTOX FISH MANAGEMENT BAITS

A new development in the control of certain undesirable fish species in lakes and ponds is rotenone poisoned bait pellets manufactured by Prentiss Incorporated. Prentox® Prenfish™ Grass Carp Management Baits contain approximately 2.6% rotenone (or around 0.01 g rotenone per bait). Feeding stations are established on the lake deploying floating trainer baits for up to 2 weeks prior to treatment. Once grass carp are trained to these stations and are readily taking pelleted baits, the trainer bait is switched for the Prentox® product and significant kills are achieved within a few hours. Scientific trials of this system in the United States have achieved up to 77% kills of grass carp without significant collateral loss of non-target species (Fajt 1996). A new development of this system is Prentox® Prenfish™ Common Carp Management Baits, which are essentially the same as the grass carp product, but synergised with piperonyl butoxide because of the greater tolerance of common carp to rotenone. Discrete and targeted applications of rotenone via poisoned baits are undoubtedly preferable to wholesale dispersed rotenone applications in water bodies. The most compelling argument for the use of rotenone bait products is that they are reputedly unlikely to affect other aquatic organisms, either non-target fish species or invertebrates. However, some trials of this product have met with mixed success. Targeted kills of carp were low and significant collateral loss of other species was experienced, possibly as the result of significant quantities of fine material (dust) in the bait product that could be ingested by small fish or quickly leach rotenone into the surrounding water. Bait training stations can sometimes be difficult to set up and maintain where wind or waves can disperse baits, and long training periods are sometimes needed, increasing management costs. Trials of the system usually achieve partial rather than complete kills, and total eradication seems possibly as

difficult to achieve with this method as with any other which could pose problems for highly fecund breeding species such as koi carp. However, partial eradication of non-breeding species, such as grass carp, to reduce total biomass may be adequate, depending on management requirements.

18. ROTENONE TOXICITY—GENERAL

A measure of the relative safety of rotenone for treating water bodies to sample or eliminate fish is provided by the complete absence of any known public health effects.

Rotenone has been used on a vast scale in the United States over many decades. Some lake and river treatments have employed tonnes of rotenone without any significant effects on humans or wildlife. In the mid-1950s, over 400 km of the Russian River watershed in California was treated with rotenone prior to stocking with rainbow trout (Pintler & Johnson 1958). In 1962, over 700 km of the Green River and its tributaries were treated with rotenone prior to closure of the Flaming Gorge Dam in Utah (Holden 1991). The most recent rotenone treatment on this scale was the chemical renovation of Strawberry Reservoir in Utah in 1990, which used 20.6 tonnes of active ingredient to eradicate Utah chub, *Gila atraria*, and Utah sucker, *Catostomus ardens* (McClay 2000).

Despite the lack of public health effects resulting from large-scale treatments, rotenone use remains controversial. Proposals to treat Lake Davis in California in 1997 with 3.5 tonnes of active ingredient to eradicate northern pike met with stiff public opposition. Public health concerns were not related to the use of rotenone itself but primarily due to concerns over reputed carcinogens in the chemical dispersants of the liquid formulation proposed (La Ganga 1997). The formulation consisted of 95% petrochemical solvents and contained significant quantities of trichloroethylene (TCE). While TCE is undoubtedly toxic, it is not classified as a human carcinogen as claimed by local residents. Opposition was especially vehement in this case because Lake Davis acts as the drinking water supply for the local town of Portola. The California Department of Fish and Game was required to arrange an alternate drinking water source, for the duration of the treatment, as part of its discharge consent to treat the lake.

19. OCCUPATIONAL SAFETY AND HEALTH CONCERNS FOR ROTENONE USERS

The greatest risk to human health from rotenone treatments is to operators involved in dispersing the toxicant. Pintler & Johnson (1958) report that unprotected personnel involved in the dispersal of dry rotenone powders as mechanically mixed slurries may suffer certain health effects especially if treatments last for several weeks. Effects included headaches, sore throats and other cold-like symptoms. Sores developed on mucous membranes, eyes were irritated severely and there were a number of incidences of eczema-like rashes on the skin. Very few symptomatic cases resulting from occupational exposure have been reported for non-fisheries related rotenone products such as flea

powders and may be attributable to ingredients other than rotenone (CDC 1999).

Rotenone is far more toxic to humans if inhaled rather than swallowed and every effort should be made during treatments to reduce the risk of inhaling dust or aerosols. Contact with concentrated powders and liquids may also cause skin irritation. Operators should wear full body protection including respirator mask, eye protection, protective clothing, and gloves. Dust risk from powders can be minimised by wetting where appropriate. The threshold limit value time weighted average (TLV-TWA) for aerial exposure is 5 mg/m^3, indicating that an occupational intake of 0.7mg/kg/day is considered safe (WHO 1992).

There is no specific antidote for rotenone poisoning. Medical aid should be sought immediately. In cases of ingestion, vomiting should be induced if the patient is conscious unless the rotenone preparation is in liquid form with a petroleum distillate carrier solvent. Further medical treatment should be symptomatic and supportive (WHO 1992). Effects may be aggravated by physical effort and patients should be made to rest.

The prevention of occupational poisoning depends on ensuring a safe working environment and proper work practices. The following precautions should be observed in the handling of rotenone dusts, slurries or solutions:

- Avoid contact with the skin and eyes.
- Do not smoke, drink or eat while handling rotenone. Wash hands and any exposed skin before eating, drinking or smoking, and after work.
- Avoid breathing dust from powder products. Disposable dust masks or respirator masks should be worn. Suitable eye protection and clothing should be worn were appropriate.
- When unloading and handling containers of concentrates, wear protective PVC or neoprene gloves.
- When handling leaking containers, or when dealing with leaks and spills, wear overalls and PVC or neoprene gloves and boots. If overalls become contaminated, change and wash them thoroughly before reuse.
- Store products in closed original labelled containers out of reach of children and away from food and animal feed.

Rotenone dusts and solutions in petrochemical solvents are flammable and combustion may release toxic fumes. Fight fires with foam, dry powder or CO_2 extinguishers. Avoid the use of water sprays to avoid polluted run-off from the site. Fire service personnel should be advised that self-contained breathing apparatus may be necessary because of the generation of noxious fumes.

20. PUBLIC HEALTH CONCERNS

20.1 Rotenone toxicity from treated waters

Based on established oral toxicity values for rats and other mammals, rotenone is classified as toxic class II, moderately hazardous, or III, slightly hazardous, according to the World Health Organisation classification of pesticides by hazard.

A dramatic illustration of the differences in sensitivity to rotenone between fish and humans is given by the following hypothetical example. Assuming that an average public swimming pool (25 m × 15 m × 1.5 m) was a pond containing trout and was poisoned with 50 μg/L rotenone, within 2 hours, all fish in the pond would be dead. In order for an adult human to receive a fatal dose by drinking the water, they would need to drink nearly half the contents of the swimming pool, or around 200 000 litres. Since rotenone appears to be relatively non-toxic at doses below the lethal limit it is highly unlikely that poisoning would occur from drinking treated waters.

Large-scale rotenone treatments in North America have received considerable local public opposition in recent years, particularly where the water body to be treated acts as a water supply for a local community. Public health concerns in these cases are directed at solvents and dispersants in rotenone liquid formulations rather than at rotenone itself. The use of powdered rotenone products removes this problem and is receiving increasing support in North America. Studies of residual concentrations in water treated with liquid formulations indicate that solvent levels are below toxic thresholds but that water may impart a noticeable taste or odour. It is also possible that tastes and odours may result from decay of poisoned fish and invertebrates and due to blooms of nuisance algae. Water treatment facilities that use carbon filtration will normally remove tastes and odours, and chlorine treatment would detoxify any residual rotenone (Bonn & Holbert 1961). Nevertheless, in order to allay public fears, rotenone treatments should probably be withheld from sources of public drinking water unless an alternative water source can be provided for the duration of the treatment and for a short while thereafter. Swimmers and other recreational users should probably be withheld from using rotenone-treated waters for at least twenty-four hours until the rotenone is completely dispersed.

It is highly unlikely that rotenone treatments would contaminate ground water. Rotenone adsorbs strongly to organic matter in soil and is rapidly degraded and detoxified (Dawson et al. 1991).

20.2 Rotenone residues in poisoned fish

Indigenous peoples of Southeast Asia and South America have used ground *Derris* and *Lonchocarpus* root containing rotenone for centuries to harvest fish for human consumption (Leonard 1939; Ray 1991). This was still commonly practised as recently as 1990 in Papua New Guinea (Dudgeon 1990).

Only about a quarter of the total body burden of rotenone in poisoned fish is found in the filet, with most chemical accumulating in the head, bones, skin and liver (Rach & Gingerich 1986). Concentrations of rotenone in fish filet are generally below 1 ppm, whereas the level considered safe for human consumption has been estimated at 10 ppm (Lehman 1950). Because desirable eating fish such as trout and salmon are considerably more susceptible to rotenone than less valued species such as goldfish, carp and catfish, rotenone residues in the former will be extremely low. On the basis of measured concentrations of rotenone in fatally poisoned carp filets, and assuming that all rotenone in the meal were absorbed, an adult human would need to eat approximately 10 tonnes of fish in one sitting to receive a fatal dose. Following

fisheries management treatments in North America, poisoned fish have often been given to community groups for human consumption (Bettoli & Maceina 1996). Given that rotenone is thermally labile, any residue is likely to be destroyed during cooking.

Rotenone is registered for use in New Zealand as an insecticide for use on ornamental and edible plants and sold as a 0.5% dust. The recommended withholding period for fruits and vegetables is one day following treatment. Even taking into account the rapid rate of rotenone decay in light and air it is possible that residual rotenone concentrations would be similar to those present in poisoned fish. Gosalvez & Diaz-Gil (1978) estimated that there was a residue of 14–58 μg/kg on raw green beans treated with rotenone insecticide and Jimenez et al. (2000) found rotenone residues of up to 120 μg/kg in honey from rotenone treated beehives. European food exposure limits are between 0.04 and 0.1 mg/kg (WHO 1992).

The most significant risk for humans and wildlife in eating rotenone-poisoned fish is from bacterial spoilage. Treatment programmes should provide adequate personnel and equipment to enable effective collection and safe disposal of dead fish.

21. ECOLOGICAL SAFETY

21.1 Aquatic ecosystem recovery following rotenone treatments

Almost all rotenone treatments to lakes and streams result in immediate and severe effects on the ecosystem either due to selective fish removal or because most aquatic invertebrates are also killed. Short-term effects may be either a deterioration or improvement in water quality brought about by changes in the biomass of planktonic and epiphytic algae. Deteriorating water conditions and algal blooms have often been attributed to non-removal of fish killed by the treatment. In most cases, invertebrate populations in treated waters recover within 1–8 months and there is often a long-term improvement in water quality from pre-treatment conditions (Prejs et al. 1997).

The most recent large-scale treatment in the United States involved the use of more than 20 tonnes of rotenone to treat the Strawberry Reservoir and River in Utah. A five-year post treatment monitoring program found that up to one third of the invertebrate fauna were unaffected by rotenone treatment, 46% of the invertebrate fauna in the Strawberry River had fully recovered within one year but 21% of taxa were still missing after five years (Mangum & Madrigal 1999). The long-term effects of this treatment are not attributable to the persistence of rotenone toxicity but illustrate that large scale perturbations to ecosystems may result in permanent changes in ecosystem structure. Such changes could as easily occur due to natural events.

21.2 Effects of rotenone on non-target aquatic species and wildlife

Fish are generally more sensitive to rotenone than other aquatic organisms and in theory it should be possible to apply rotenone at concentrations that would kill only fish without harming other aquatic life. However, fish species vary in their sensitivity to rotenone and toxicity is affected by numerous factors

including temperature, turbidity, pH, light, etc. It is also difficult to achieve a uniform distribution of toxicant in most water bodies, so applications normally include a substantial excess of chemical. Furthermore, individual fish vary widely in their sensitivity and a large excess of chemical may be required to kill all individuals of some resistant species. Significant effects on aquatic invertebrates are usually seen following rotenone applications but these are often short-lived. Beal & Anderson (1993) found that copepods and rotifers were the first zooplankton groups to recover in a rotenone-treated pond in Illinois, USA, reaching pre-treatment numbers within one month, while cladocerans populations took up to eight months to recover. Low winter temperatures following rotenone treatment may have limited cladoceran recovery. New Zealand zooplankton populations show less seasonal change in zooplankton abundance and species composition than those of northern temperate lakes and are dominated numerically by copepods (Burns 1991). Rotenone treatments in New Zealand lakes and ponds are therefore unlikely to cause long-term effects on zooplankton assemblages. Rotenone applications to streams cause massive invertebrate drift in the short-term, especially of mayflies, but no significant long-term reduction in total benthic invertebrate abundance (Morrison 1977; Dudgeon 1990).

Non-target fish species in New Zealand are likely to be greatly affected by rotenone treatments designed to eliminate nuisance fish species. One galaxiid species has been tested for its sensitivity to rotenone and was significantly more susceptible than mosquitofish (Willis & Ling 2000). Any eradication project should assess the potential impact on non-target species in order to compare the relative merits of dispersed applications or rotenone baits.

The eggs of fish and amphibians, and adult amphibians, are less susceptible to rotenone poisoning than are fish and aquatic invertebrates because their rate of uptake of toxicant from water is much lower. At typical application rates for fish eradication, some effects on these aquatic groups would be expected, but significant losses would be unlikely. Rotenone applications should possibly be timed to avoid periods when amphibian larvae (tadpoles) are present since these show sensitivities similar to the more resistant fish species (Hamilton 1941).

Birds and mammals are much less sensitive to rotenone than are fish and aquatic invertebrates and poisoning caused by drinking treated water or eating poisoned fish is extremely unlikely. Species at risk in New Zealand are fish-eating and scavenging birds such as shags and gulls respectively.

Although the use of rotenone baits to remove target fish species, rather than dispersed applications, would greatly reduce effects on non-target fish, aquatic invertebrates, and amphibians, there is potential for poisoning of some species by consuming uneaten floating or sunken baits. Those species most at risk in New Zealand would be scavenging macro-invertebrates such as koura, non-target fishes, especially trout, eels, smelt and bullies, and waterfowl.

22. CONSIDERATIONS FOR ROTENONE USE IN FISHERIES MANAGEMENT

A recent review of rotenone use among fisheries agencies in the United States and Canada (McClay 2000) lists the major issues that agencies considered important in fisheries management with toxicants. In order of importance they were:

- Public acceptance and understanding
- Environmental concerns
- Usability of the product
- Public health and toxicology concerns
- Availability of the product
- Animals rights and welfare concerns
- Methods and techniques

Specific issues that agencies had faced in the period 1988–97 were as follows:

- Collection and disposal of dead fish
- Impact of rotenone or formulation ingredients on public health
- Adequate public notification and education
- Animal welfare—fish and wildlife
- Impacts on non-target aquatic organisms
- Toxicant residues in fish
- Liability and property damage
- Impacts of rotenone and formulation ingredients on air quality

23. RECOMMENDED PROTOCOL

The following steps are recommended when considering the use of chemical renovation of standing freshwaters with rotenone, or the eradication of pest fish with rotenone baits.

- Conduct a biological survey to establish the need for chemical renovation or the need to eliminate target species, e.g. grass carp. Are there alternatives to the use of chemical renovation?
- Assess the risk of chemical renovation to non-target aquatic species and to wildlife, livestock and human health.
- Assess whether chemical renovation is feasible given the volume or flow of the target water body. Prepare an outline map of the target water body including all inlet and outlet streams and tributaries, sites and uses of water abstraction, toxicant delivery stations, etc.
- Conduct a public relations programme to win public support for the project.
- Consult with and gain approval of riparian landowners, Regional Councils, Department of Conservation, Fish & Game New Zealand, Iwi, and other public interest groups.

- Set a date for the project based on considerations of rotenone toxicity and breakdown, and considerations of biological importance, e.g. reproductive biology of target/non-target species.
- Measure treatment volume and establish minimum quantities of toxicant required based on target species, and hydrological features, e.g. turbidity, weed beds or deep water.
- Establish procedures for the elimination or mitigation of downstream toxicity in outlet streams, e.g. detoxification stations if necessary.
- Establish whether non-target valued species are likely to be affected by the treatment, e.g. game fish and native species.
- Calculate expected quantities of fish for disposal and establish collection and disposal procedures.
- Establish locations or methods for toxicant delivery. Determine toxicant formulation.
- Establish locations and species for live-cages for testing toxicity to target species (dispersal applications) or non-target fish or invertebrates (bait applications).
- Establish post-treatment chemical and biological survey procedures.
- Obtain required discharge permits or other permissions as required under appropriate legislation such as the Resource Management Act 1991.

Rotenone treatments in New Zealand should probably be restricted to small bodies of standing water, i.e. ponds and very small lakes with restricted outlets, to prevent downstream toxic effects within the watershed. Drains and slow flowing streams would be candidates for treatment for the same reason. Treatment of large lakes is probably impractical for a number of reasons:

- In large or deep water bodies, uniform dispersal of the toxicant is very difficult to achieve.
- Despite the low cost of rotenone, large-scale treatments become prohibitively expensive.
- Sources of rotenone are limited and it is unlikely that sufficient product would be available for large-scale treatments.

Factors to be taken into account in the application of rotenone as a fish toxicant in New Zealand:

- Rotenone is already registered and marketed in New Zealand as a pesticide but is not registered for use as a piscicide.
- Rotenone has been used successfully for fisheries management and research in many countries without significant adverse environmental or public health impacts.
- Rotenone does not accumulate in animal tissues or the environment over long periods of time, being easily metabolised in the body, and broken down within days to weeks in natural waters depending on factors such as temperature, light, pH, turbidity, etc.
- Rotenone is available as powder or liquid formulations, but the use of powders is preferred because of environmental concerns arising from the solvents and dispersants in the liquid formulations, and because fish actively avoid liquid rotenone.

- Rotenone is a natural product obtained from certain tropical plant species. Cultivation of indigenous plants for rotenone production provides employment and income in some developing nations.
- Rotenone can be detoxified in water but this should be avoided unless absolutely necessary because the substances used for detoxification can be harmful to the environment in their own right.
- Rotenone has extremely low toxicity to non-target wildlife species and humans.

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Appendix 1

PIPERONYL BUTOXIDE—SUMMARY OF PHYSICOCHEMICAL AND TOXICOLOGICAL DATA

Name: piperonyl butoxide

Empirical formula: $C_{19}H_{30}O_5$

Molecular weight: 338.4

Physical state: pale yellow oil

Solubility: not soluble in water, soluble in organic solvents

Boiling point: 180°C

Pesticide type: organic synergist

Acute toxicity: oral LD50 around 7500 mg/kg or greater

Chronic toxicity: in 2 year feeding trials, rats receiving 100 mg/kg in their diet suffered no ill effects. It is non-carcinogenic and the estimated safe level for human ingestion is 42 mg/kg in the diet.

Environmental safety: little or no hazard to fish and wildlife

Environmental stability: short lived

Reference: United States Environmental Protection Agency 1985. Piperonyl butoxide. Chemical profile 3/85. USEPA, Washington.