APPENDIX C

Use of Piscicides and Neutralization Compounds

Historic Use of Piscicides to Manage Fisheries

Fisheries managers rely on a variety of tools to manage and assess fish populations. Historically, these have included the use of piscicides. Two piscicides, rotenone and antimycin A, are currently registered by EPA for general use in the United States under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). By law, the EPA is authorized to register a pesticide only if it will not cause unreasonable adverse effects on human health or the environment.

Originally, piscicides were mainly used to control out-of-balance or undesirable fish populations so that sport fish could be stocked for recreational purposes. Today, antimycin and rotenone are used in fisheries management for a variety of purposes, including (Finlayson et al. 2000):

- Eradication of nonnative fish,
- Restoration of threatened and endangered fish,
- Support of recreational fisheries by controlling undesirable fish,
- Eradication of fish to control disease,
- Quantification of populations of aquatic organisms,
- Eradication of competing fish in rearing facilities or ponds prior to restocking.

Although physical removal methods (e.g., nets, traps, seines, electrofishing, dewatering, and combinations of physical control techniques) are available for reducing or controlling fish communities, they are generally incapable of eradicating fish (Finlayson et al. 2000). Meronek et al. (1996) review of fish control projects found that success rates for physical removal methods ranged from 33 percent to 57 percent. In most streams, only piscicide applications or complete dewatering can eradicate entire populations of undesirable fish (Schnick 1974).

Rotenone

Rotenone has been used in the United States to manage fish populations since the 1930s and is the piscicide of choice for application in ponds and lakes. According to the American Fisheries Society (Finlayson et al. 2000), rotenone has been routinely used for management of fish populations in 34 states and several Canadian provinces. Formulations of rotenone are manufactured (under the brand names Pro-Noxfish®, Nusyn-Noxfish®, Prenfish®, and others) and shipped in either a powdered or liquid form.

In addition to applicability as a piscicide, other formulations of rotenone are registered in the United States as an insecticide for domesticated pets (dogs and cats), cattle, sheep, ornamental plants, trees, and turf; and foliar preharvest application to vegetables, berries, tree fruit, nuts, forage crops, and sugar cane.

Rotenone is a naturally occurring compound extracted from the roots of certain species of the bean family that has been used for centuries to capture fish (Finlayson et al. 2000).

As a piscicide, rotenone interrupts cellular respiration in gill-breathing animals by blocking the transfer of electrons in the mitochondria. Acute exposure to toxic levels reduces cellular uptake of blood oxygen, resulting in increased cellular anaerobic metabolism and associated production of lactic acid causes blood acidosis (Fajt and Grizzle 1998). Death results from tissue anoxia, which typically produces cardiac and respiratory failure (Ling 2003). Scientists believe that fish are more sensitive to rotenone because it is rapidly absorbed into the bloodstream from water flowing across the gill membrane. Although both fish and aquatic macroinvertebrates are highly susceptible to rotenone (Skaar 2001), most macroinvertebrate populations quickly recover to pre-treatment levels (Lennon 1971, Schnick 1974b). Gill-breathing amphibians (i.e., frog and toad tadpoles and larval salamanders) are also adversely affected (Hamilton 1941). Amphibian adults and reptiles are less sensitive than fish and should not be harmed when rotenone is applied at concentrations typically used in fisheries management (Farringer 1972). Fall applications of rotenone reduce or eliminate impacts on amphibians because most species are in the adult stage of development.

When applied at recommended doses for fish control (0.005 to 0.250 mg/L), rotenone has low toxicity to non-aquatic organisms. Extensive research has demonstrated that rotenone does not cause birth defects (Hazelton 1982), reproductive dysfunction (Spencer and Sing 1992), gene mutations (Biotech 1981, Goethem et al. 1981, NAS 1983) or cancer (EPA 1981, Tisdel 1985). The results of chronic feeding studies in which rats and dogs that were fed forms of rotenone as part of their diet for six months to two years resulted in non-lethal effects such as diarrhea, decreased food consumption, and weight loss (Skaar 2001). No adverse chronic effect was reported when rats where given 100 mg/L Pro-Noxfish® (2.5 percent rotenone) in drinking water for 70 weeks (Brooks and Price 1961). Ellis et al. (1980) found that 10 mg/kg/day rotenone administered orally to beagles for 26 weeks had no adverse chronic effect. Skaar (2001) reported that a 10-kg dog would have to consume 300,000 kg of rotenone-tainted fish to receive a lethal dose. Studies that examined avian exposure report that a 1,000 to 10,000-fold increase in levels normally used for fisheries management would be required for lethality (Skaar 2001).

Several hazard assessments for human health have also been conducted. Simulated lethal oral dosage for a human is variously estimated between 300 to 500 mg/kg (Ray 1991, Gosselin et al. 1984). Gleason et al. (1969) estimated the lowest dose for lethality would require a 60-kg person to consume 180,000 liters of water containing 0.1 mg/L rotenone, or eat 180 kg of rotenone–killed fish at one sitting. Although ingestion of rotenone-killed fish is not recommended, the rotenone level in fish considered safe for human consumption has been estimated at 10 ppm (Lehman 1950). Skaar (2001) notes that the National Academy of Sciences established in 1983 a "suggested no-adverse response level" of rotenone in drinking water of 0.014 mg/L, assuming a 70-kg person drinks 2 liters of water per day for a lifetime. In 1997, the EPA established a human ingestion risk value (reference dose for chronic exposure) of 0.004 mg/kg/day.[1]

[1] A reference dose is an estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

An Emory University study (Betarbet et al. 2000) reported finding anatomical, neurochemical, and behavioral symptoms characteristic of Parkinson's disease in laboratory rats when rotenone was administered chronically and intravenously. However, several researchers in Parkinson's disease (including J. Langston Director of the Parkinson's Institute) have stated the Emory University does not show evidence that exposure to rotenone causes Parkinson's disease. The continuous jugular vein infusion of rotenone lead to continuously high levels of rotenone in the bloodstream and included dimethyl sulfoxide to enhance tissue penetration. This mode of administration to laboratory rats was unnatural and cannot be used as a model for any environmental exposure to rotenone. The normal exposure to rotenone in humans from its use in fisheries management would be ingestion, inhalation, or through the skin. Rotenone that is ingested by mammals (and birds) is rapidly broken down by enzymatic action in the gut and excreted by the liver and kidney. Approximately 20 percent of the oral dose (and probably most of the absorbed dose) is excreted within 24 hours (Ray 1991). In the Emory University study, Betarbet et al. (2000) concluded that "rotenone seems to have little toxicity when administered orally."

Rotenone is very unstable in the environment (half-life measured in days) and completely breaks down within one to four weeks depending on pH, alkalinity, temperature, dilution, and exposure to sunlight (Schnick 1974b). It also adsorbs strongly to organic matter in sediment and is rapidly degraded (Dawson et al. 1991). Rapid neutralization (oxidation) occurs when rotenone is mixed with potassium permanganate or sodium permanganate (Engstrom-Heg 1971, 1972, 1973; Finlayson et al. 2000). Inert ingredients in the liquid formulation of rotenone consist of petroleum hydrocarbons as solvents and emulsifiers (primarily naphthaline, methylnaphthalenes, trichloroethylene, and xylenes). Studies of residual concentrations in water treated with liquid formulations indicate that solvent levels are below toxic thresholds (Ling 2003). In a study of rotenone-treated streams and lakes in Californian, concentrations of trichloroethylene never exceeded the Federal drinking water standard (Maximum Contaminant Level) of 5 ug/L and similarly the concentrations of xylene never exceeded the drinking water standard (Health Advisory) of 620 ug/L (Finlayson et al. 2001). Drinking water standards for naphthalenes and methynaphthtalenes have not been established. Finlayson et al. (2001) noted that all the volatile and semivolatile organic compounds disappeared before rotenone dissipated from the treated waters. There are no Federal or Arizona water quality standards for rotenone.

Rotenone does not easily enter groundwater because of its tendency to bind rapidly with organic material in soil and surface water (DOE 2004). In a California groundwater study, no trace of rotenone (including any of the compounds in the formulated product) was detected in 26 wells that were placed in aquifers adjacent to and downstream of 9 rotenone-treated water bodies (CDFG 1994). A similar study at Tetrault Lake, Montana, failed to detect rotenone in a nearby domestic well that was sampled two and four weeks after treatment of the lake with 90 ug/L of the piscicide (DOE 2004). The Tetrault Lake well site was studied because it was downgradient from the lake and drew water from the same aquifer that fed and drained the lake. In another study, water from a well located 65 feet from a rotenone-treated pond near Kalispell, Montana, was analyzed and showed no sign of piscicide contamination (DOE 2004).

The major risks to human health from rotenone come from exposure during application. This is the only time when humans (applicators) are exposed to high concentrations of the piscicide. Personal protective equipment is required by the product label and material safety data sheet to reduce respiratory and dermal exposure. For liquid and powder rotenone formulation application, personnel must wear approved air purifying respirators, goggles, rubber gloves, and protective clothing.

Any threat to recreational users during treatment can be readily mitigated through cautionary signing at access points, temporary closures, and posting agency personnel within the treatment area.

Antimycin A

The only other piscicide registered for general use in the United States is antimycin A (Finlayson et al. 2000). Antimycin A was first patented for piscicide use in 1964. It is also approved for use in commercial catfish farms by the Food and Drug Administration. Formulations of antimycin A are registered under the brand name Fintrol®.

Like rotenone, antimycin inhibits cellular respiration in gill-breathing animals (Schnick 1974a). When used in appropriate concentrations for fisheries management, antimycin is often less harmful to non-target aquatic organisms than rotenone. Some studies have shown effects on aquatic insects vary according to species and stream setting. Cerreto et al. (2003) found that antimycin had little to no effect on aquatic macroinvertebrates in high elevation (approximately 7,900 feet) streams in Bridger-Teton National Forest in Wyoming. Lennon et al. (1971) stated that antimycin is the ideal piscicide because of its selective effects and effectiveness at low concentrations in a wide range of water qualities, it is not repulsive to fish, and it leaves no toxic residue.

Antimycin A is an organic compound that was isolated from the bacterium *Streptomyces girseus* at University of Wisconsin in 1945 (Leben and Keitt 1948, Dunshee, et al. 1949). The chemical formula of antimycin is $C_{28}H_{40}N_2O_9$ (Rinne and Turner 1991:237), and it inhibits growth of some fungi but does not affect most bacteria. The formulations often used for fish control in streams are Fintrol-Concentrate (liquid form antimycin A) and Fintrol 15 (antimycin A coated sand). Antimycin A consists of 10 percent antimycin and inert constituent components (soy lipds, Diethyl phthalate, Nonoxyl-9 detergent [or nonylphenol polyglycol ether], and acetone).

Degradation of antimycin is by the following pathway (Hussain 1969):

antimycin A1 → blastmycic acid + antimycin lactone → fatty acids

Temperature and pH strongly influence the efficacy and rate of degradation of antimycin (Chapman et al. 2003). Antimycin degrades slower in lower temperature water, but is it also less effective, probably because at lower temperatures the metabolism and respiration rate in fish decrease, thereby affecting the rate of toxicant uptake (Berry and

Larkin 1954). Marking and Dawson (1972) reported the following half lifes of antimycin based on pH: pH 6 to 6.5 = 310 hours, pH 7.5 = 120 hours, and pH 8 = 100 hours. Degradation of antimycin occurs quickly under natural stream conditions because of dilution, adsorption to organic material and sediments, and oxidation created by sunlight and water turbulence (Lee et al. 1971). Other compounds that will readily bind with antimycin to detoxify it include leafy vegetation and water plants (Grisak 2003). Rapid neutralization occurs when antimycin is mixed with potassium permanganate or sodium permanganate (Marking and Bills 1975). The degradation compounds have very low toxicity for either fish or mammals (Herr et al. 1967). Drinking water standards have not been established for the commercial piscicide formulation of antimycin A. There are no Federal or Arizona water quality standards for antimycin A.

A review of toxicity studies relating to antimycin indicates that vertebrate animals must ingest high dosages before any adverse effect is apparent (Schnick 1974a) In laboratory tests, oral LD_{50} values for mammals ranged from 1.0 mg/kg for lambs to 55 mg/kg for mice (Herr et al. 1967). Oral LD_{50} is defined as the amount of antimycin administered orally over a specified period of time that causes the death of 50 percent of the group of test animals. For example, if a person weighing 70 kg consumed 1.5 liters from a stream treated with 200 ug/L antimycin (recommended concentrations typically range from 5 to 25 ug/L), that person would ingest 300 ug of antimycin, or 0.0042 mg/kg of body weight. A 70 kg person would have to ingest 630 liters of treated water during the period that antimycin is active in the treatment area to ingest the amount required to achieve the LD_{50} for the most sensitive mammal tested (Guinea pig, LD_{50} = 1.8 mg antimycin/kg body weight). This translates to a water consumption rate of about 105 liters per hour during an active treatment period lasting six hours. Similarly, a 363 kg horse would have to ingest about 3,265 liters of water treated with 200 ug/L antimycin to reach the oral LD_{50} value of 1.8 mg/kg for Guinea pigs.

Consumption of antimycin in water was alleged to have caused organ abnormalities and still-birth of two lambs in northern New Mexico in 1998 (Begel 2001). However, no evidence implicating antimycin in the still-birth of the two lambs was produced, and no adverse effects on animals in the surrounding area were reported (AFSFMCS et al. 2001). In addition, an independent medical microbiologist contracted by Grant and Catron counties in New Mexico to review the potential public health hazards of antimycin concluded that it was an effective and safe fish control agent for removal of fishes from streams with no potential for public health issues when applied at recommended concentrations (Brooks and Propst 2001). Vezina (1967) reported that antimycin is not hazardous to humans, livestock, and wildlife when applied at concentrations appropriate for fisheries managment.

The potential effects of consuming dead fish produced by stream renovation are poorly studied, but there have never been any reports of negative effects to humans or wildlife from ingestion of antimycin-killed fish (Berger et al. 1967, Gilderhus et al. 1969). Ritter and Strong (1966) reported that 21 humans who consumed between one and five 4-oz servings of fish killed by antimycin suffered no ill effects. Schnick (1974) reported that antimycin is not hazardous to humans whether it is consumed in food or water. In a study

on waterfowl, Vezina (1967) found that consumption of 2,900 mg antimycin/kg body weight was required to cause mortality of 50 percent of test mallard ducks in the laboratory. Similar tests on 4.5 kg domestic dogs required consumption of 5000 mg/kg antimycin to cause mortality of 50 percent of the test population. In another laboratory study, trout killed with 10 ug/L antimycin contained 76 to 388 ug/kg antimycin in their tissues (Ritter and Strong 1966).

The major risks to human health from antimycin come from exposure during application. This is the only time when humans (applicators) are potentially exposed to high concentrations of the piscicide. Personal protective equipment is required by the product label and material safety data sheet to reduce respiratory and dermal exposure. For liquid and powder antimycin formulation application, personnel must wear approved air purifying respirators, goggles, rubber gloves, and protective clothing.

Any threat to recreational users during treatment can be readily mitigated through cautionary signing at access points, temporary closures, and posting agency personnel within the treatment area.

Potassium Permanganate and Sodium Permanganate

Potassium permanganate ($KMnO_4$) is the chemical most often used to quickly neutralize (oxidize) rotenone and antimycin, and recently sodium permanganate ($NaMnO_2$) has also been used for this purpose. Since permanganate itself may be toxic to aquatic organisms at high dosages, detoxification procedures would utilize calibrated equipment to achieve minimum effective concentration of permanganate to neutralize the piscicide. Monitoring stations consisting of caged live fish would be placed at the downstream limit of the treatment area to verify detoxification of the piscicide and permanganate.

*Potassium Permanganate***.** Potassium permanganate reduces the half-life of antimycin to 7 to 11 minutes in a laboratory setting. Horton (1997) recommends a 20-minute neutralization period for rotenone in non-alpine streams. Potassium permanganate is a strong oxidizing agent that quickly breaks down to naturally occurring compounds (Archer 2001). Kemp et al. (1966) and Marking and Bills (1975) found that organic material and inorganic oxidation substances rapidly decrease the activity of potassium permanganate.

Potassium permanganate can be toxic to fish (Tucker and Boyd 1977, Archer 2001, Grisak et al. 2002). In the laboratory, exposure to 2 mg/L $KMnO_4$ was lethal to rainbow trout (Archer 2001). When applied at 1.5 mg/L in the absence of readily oxidizable substances, potassium permanganate achieved lethality in westslope cutthroat trout after 16 to 24 hours of exposure (Grisak et al. 2002). Potassium permanganate is quickly broken down when it reacts to organic material and antimycin or rotenone in stream water. Breakdown components of potassium permanganate (potassium, manganese, and water) are common in nature and have no deleterious environmental effects at concentrations used for neutralization of piscicides (Finlayson et al. 2000).

Potassium permanganate is also one of the most widely used inorganic chemicals for the treatment of municipal drinking and wastewater. According to the American Waterworks Association's Water Industry Data Base, potassium permanganate is listed as the second most widely used chemical for predisinfection and oxidation by treatment plants processing surface waters. Hundreds of drinking water treatment plants use this chemical to oxidize iron, manganese, and arsenic; to remove color; and to treat for biofilm in raw water intake pipes.

Potassium permanganate is also used in fish farming to prevent or alleviate oxygen shortages in rearing ponds. The chemical works by oxidizing decaying plant matter and other organics so that they consume less oxygen, thereby relieving oxygen depletions that otherwise could result in fish kills.

Sodium Permanganate. Sodium permanganate is another strong oxidizing agent that can be used to neutralize rotenone and antimycin. Like potassium permanganate, this permanganate compound has a low estimated lifetime in the environment and is readily degraded by organic material and inorganic oxidation substances (Sino-American 2002). In a stream, sodium permanganate will quickly degrade as it neutralizes the piscicide and reacts to any organic material. The breakdown components of sodium permanganate are sodium, manganese, and water.

Sodium permanganate is also used for in situ chemical oxidation of chlorinated organic contaminants in soil and groundwater. Liquid sodium permanganate is injected into the soil or groundwater and allowed to disperse through the contaminated media. In situ chemical oxidation using sodium permanganate is an effective and environmentally acceptable method of remediating contaminated soil and groundwater.

Piscicide Use and the Federal Insecticide, Fungicide and Rodenticide Act

FIFRA requires all persons who apply pesticides classified as restricted use (such as piscicides) be certified according to the provisions of the act, or that they work under the supervisions of a certified applicator. Commercial and public applicators must demonstrate a practical knowledge of the principles and practices of pest control and safe use of pesticides. In addition, applicators using or supervising the use of any restricted use pesticides purposefully applied to standing or running water are required to pass an exam (*Aquatic Pest Control*) to demonstrate competency as described in the Code of Federal Regulations (40 CFR 171.4) as follows:

> Aquatic applicators shall demonstrate practical knowledge of the secondary effects which can be caused by improper application rates, incorrect formulations, and faulty application of restricted pesticides used in the category. They shall demonstrate practical knowledge of various water use situations and the potential for downstream effects. Further, they must have practical knowledge concerning potential pesticide effects on plants, fish, birds, beneficial insects and other organisms which may be present in aquatic environments. Applicants in this

category must demonstrate practical knowledge of the principles of limited area application.

Piscicide Use and the Clean Water Act

Under FIFRA, EPA is charged to consider the effects of pesticides on the environment by determining whether a pesticide will perform its intended function without unreasonable adverse effects. In an agency guidance letter dated July, 11, 2003, EPA recognized the inherent value of pesticide applications to control nonnative species, and stated that when a pesticide is applied directly to waters of the United States according to its "intended purpose" as allowed under FIFRA, it is not a pollutant under the Clean Water Act. The EPA further noted that the application of a pesticide in compliance with FIFRA requirements does not require an NPDES permit under the Clean Water Act when the pesticide is applied to water to control pests. A decision by the United States Court of Appeals for the Ninth Circuit (*Fairhurst vs. Hagener*) reaffirmed EPA's decision that a pesticide applied to a river for the purpose of "eliminating pestilent fish species is not a pollutant for the purposes of the Clean Water Act…and thus not subject to the Act's permit requirements."

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