

A Review of Rotenone as Piscicide With Notes on Its' Application in a
Census of Fishes in Mangrove Habitat in Southern Everglades
National Park

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I. Introduction

Rotenone is a naturally occurring, nonsystemic, selective insecticide and piscicide that occurs in the roots of several species of plants of the Leguminsae family. Primitive peoples throughout the tropics have been using rotenone bearing plants to harvest fish for centuries (State of California, 1985). In the 1940's, commercial use of rotenone became popular and was sold as wettable powders and emulsifiable concentrates for ease of handling and increased effectiveness (State of California, 1985). Rotenone is one of only two (antimycin is the other) piscicides registered for fisheries use by the United States Environmental Protection Agency (USEPA) according to the Federal Environmental Pesticide Control Act (Davies and Shelton, 1983).

Rotenone kills fish, not by suffocation, as was previously believed (Davies and Shelton, 1983) but by blocking a biochemical process at the cellular level (Sousa et al., 1985). The poison acts as a powerful inhibitor of the respiratory enzyme chain system. Specifically, rotenone binds at two sites on reduced NADH molecules, blocking oxidation (State of California, 1985). Rotenone has also been used as a garden pesticide and as a dust to control insect parasites on cattle, sheep, and dogs (Sousa et al., 1985).

The reason rotenone is lethal to many aquatic organisms and not terrestrials is because of the method of respiration; the specialized structure of the gills favors entrance of rotenone to the blood where it is transported to vital organs and blocks cellular respiration (State of California, 1985). Airborne dust is simply not as pervasive to terrestrials as dissolved organics in water are to aquatics. Rotenone is a powerful poison to all animal life if it enters the blood stream unaltered; an injected lethal dose of rotenone was found to be 0.65 ppm in dogs (State of California, 1985).

Inadvertent ingestion of rotenone due to treatment as a piscicide or insecticide is of no consequence. Rotenone is quickly oxidized, being broken down into harmless products before absorption can occur (State of California, 1985). Oral LD₅₀ for adult birds was found to be 1.0-2.0 g/kg of food (1000-2000 ppm) (State of California, 1985). The recommended maximum treatment for killing fish is 0.25 ppm (Sousa et al., 1985; State of California, 1985). It would simply be impossible for a bird to ingest enough water for acute toxicity to be a factor. Gilderhus et al. (1988) reported that the accumulation of rotenone from a treatment of 0.25 ppm can be as high as four times that of the dose itself in various aquatic animals (fish, crustaceans, and mollusks). This bioconcentration still presents no threat of acute toxicity to birds that might feed at rotenone treatment sites. Young birds are more susceptible to oral rotenone poisoning; LD₅₀'s were found to be between 100 and 200 ppm (State of California, 1985). Still, there is no threat; young birds would have to be fed a single meal of 100 kg of fish killed exclusively by rotenone with the highest level of bioconcentration for a legal

treatment of rotenone to be lethal. Similar results were found in mammals. Even with a safety factor of 1000 times, Sousa et al. (1985) reported that a 132 lb human would have to eat a single meal of 535 lbs of raw fish killed by rotenone to receive a lethal dose of rotenone. No human fatalities by rotenone poisoning have ever been reported (State of California, 1985)

Chronic toxicity is also very unlikely. Hansen et al. (1965) fed rats food with 115.2 ppm pure rotenone every day for 2 years with no gastrointestinal or neural damage. The rats did exhibit a decreased growth rate but this was attributed to a lower rate of food consumption. Rats presumably did not eat as much food because rotenone made it less palatable. Similar results were found in dogs and mice (State of California, 1985). Davies and Shelton (1983) reported that rotenone was, for the most part, not a hazard to organisms that feed on, or drink from rotenone treated areas. However, they did report that swine were "particularly susceptible to rotenone" but no indication was made as to what a harmful dose was. In direct contradiction, a report by the State of California (1983) stated that swine were regularly treated with rotenone for insect infestations.

Chronic doses, such as those found to be harmful in laboratory experiments, could not be encountered in the field simply because of the small doses applied and the quickness with which it biodegrades and dissipates. Daily over-applications of rotenone in a confined area would be necessary to expose wildlife to quantities of rotenone similar to those of lab experiments.

Current literature holds that there are no teratogenic, mutagenic, or carcinogenic effects of rotenone (State of California, 1985). Khera et al. (1982) fed pregnant Kester rats rotenone in corn oil on days six through 15 of gestation. The dose used was large enough to kill half of the females, but, those that survived had viable offspring in similar numbers to controls. Goethem et al. (1981) conducted E. coli DNA repair tests for rotenone induced mutagenic activity. At 10 ppm no DNA modifying activity occurred. Biotech research (1981) analyzed the bone marrow cells of rotenone treated rats and concluded that rotenone was not clastogenic and did not cause chromosomal breaks. Hansen et al. (1965) fed rats rotenone at a dose of 1000 ppm and dogs at 400 ppm for more than 2 years. There was no increase of mammary tumors, nephritis or pituitary lesions over controls. Surprisingly, at lower doses (<50 ppm) rats fed rotenone had significantly fewer cancerous tumors. Similar studies showed no increase between treatment and control groups for mice (Innes et al., 1969), golden hamsters (Leber and Pershing, 1978), and Sprague-Dawley rats (Leber and Hake, 1978). The USEPA concluded in 1980 that rotenone is not a carcinogen and has been shown to be a powerful inhibitor of cancer cells in culture (State of California, 1985).

II. Forms of Rotenone

Rotenone is sold commercially, under various brand names, in basically four forms; 5% emulsified solution, 2.5% emulsified solution with 2.5% synergist, rotenone powder (reported in the literature as

being anywhere from 5% to 45% pure) and crystalline rotenone (97% active ingredient). Gilmore et al. (1981) compared the effectiveness of a 5% emulsified solution to both crystalline rotenone and powdered rotenone dissolved in various chemical solutions. Crystalline rotenone was dissolved in acetone and a DMSO-ethanol solution. Powered rotenone was dissolved in the same solutions as the crystalline as well as in a solution of a commercial emulsifier, Atlox 3335. All solutions were standardized to 5% active ingredient. The solutions used to dissolve the powdered rotenone were also tested for their effects on fish. Several species of anemone and teleost fish, as well as one species of palomonetes shrimp, were treated with a dosage of 0.25 ppm active rotenone for 30 minutes. The results show that none of the solutions killed or critically effected any of the anemones. The crystalline solutions and the powder in DMSO-ethanol had no effect on the shrimp, however the powder in acetone and the 5% emulsified solution highly disoriented the shrimp. Powder in Atlox 3335 killed the shrimp. All solutions killed or disoriented the fish except for the powder in acetone which had no effect on one of the species tested (Gobiosoma robustum). Atlox 3335, when tested without rotenone, had strong effects on the anemones and killed the teleosts. DMSO-ethanol and acetone solutions without rotenone had no effect on any of the organisms tested. Of particular importance to our current proposal of sampling fishes of mangrove habitat in the southern Everglades is that Cyprinodon variegatus, one of our target species, was killed by 0.25 ppm rotenone regardless of how it was applied.

Marking and Bills (1976) determined the 1 hr LC₅₀s of rainbow trout (Salmo gairdneri) for rotenone as a 5% emulsified solution, as a 2.5% emulsified solution with a synergist, and powdered rotenone (no solvent identified so assume dissolved in water). They concluded that the emulsified solution with a synergist was the most effective and the 5% emulsified solution to be the least effective of the three tested. Powdered rotenone required a concentration of only 0.0165 ppm to kill 50% of the fish in one hour ✓ or less.

There are advantages and disadvantages to each form of rotenone. The 5% emulsified and 2.5% emulsified with synergist are very effective and are simple to store and use, but, as pointed out by Gilmore et al. (1981) the emulsifiers themselves do damage to non-target organisms. Also the emulsifiers cloud the water, making it difficult to collect dying fish. Pre-mixed rotenone solutions are also very expensive. Powdered rotenone is much cheaper but it is also harder to keep for long periods; light, moisture, and temperature ✓✓✓ facilitate the breakdown of rotenone (Gilmore et al., 1981) and as a result the powder must be stored in a dark, cool, dry place. Since powder is usually sold in 50 lb increments it is hard to store it properly so that it will not go bad before all 50 lbs is used. To reduce the incidence of powder being oxidized into a useless form, it should be placed in small, resealable plastic bags. Another potential problem with the powder is that it is difficult to dissolve in water. Crystalline rotenone is easy to store and keep fresh, being a concentrate, but it is also harder to dissolve in solution. The crystalline form is also not as effective as the emulsified forms or the

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powder (Marking and Bills, 1976; Gilmore et al., 1981). Crystalline rotenone is in very short supply, and if a retailer can be found, the price is exorbitant (McIvor, pers. comm.)

For our study, powdered rotenone, dissolved in acetone and water to 5% just before a field operation, is probably the best option. Since we are working in a national park and wish to leave the environment as pristine as possible, emulsifiers should be avoided because of their toxicity to non-target organisms. Crystalline rotenone is not as effective, more expensive, and a guaranteed supply is not likely. Powdered rotenone is cheap and plentiful at this time. Our needs are small enough that we can stockpile enough to complete our study without a storage problem. Acetone is also cheap and represents no hazards to researchers or the environment. Though rotenone in acetone did not kill G. robustum at 0.25 ppm, it did kill C. variegatus and did disorient palominetes shrimp (two of our target species). Also, the experiment was only conducted for 30 minutes and then all of the fish were terminated. It is quite possible that it takes longer than that for the physiological effects of rotenone to manifest themselves in some of the less active species. Since our enclosures will be sealed and collection will occur for 48 hrs after application, this type of delayed reaction will not be a problem.

III. Recommended Dosages

A review of the literature revealed that there is no standard in reporting concentration of rotenone. Most commonly, researchers reported the concentration of rotenone product they were using

(Marking and Bills, 1976; State of California, 1983), others reported the concentration of pure rotenone (Gilmore et al., 1981), and some made no mention of the concentration they used (Theyer et al., 1987). Many times a concentration is reported but whether it is of pure rotenone or of the rotenone product is not defined (Bayley and Austen, 1988) and some reports vary in their presentation of concentrations (State of California, 1983). While reading these papers, I recalculated, whenever necessary, to ppm of active ingredient. This is the standard that most toxicologists use and seems to make the most sense. All concentrations reported in this paper are in ppm of active ingredient (pure rotenone) regardless of how they were reported in the papers cited.

Recommended doses of rotenone range from 0.025 ppm (Davies and Shelton, 1983) to 3.0 ppm (Bayley and Austen, 1988). Thankfully, most works report the recommended dose be 0.1 ppm or, when dealing with some what resistant fish or less than ideal conditions, a maximum concentration of 0.25 ppm (Gilderhus, 1972; State of California, 1985; Sousa et al., 1986, Gilderhus et al., 1988). Only Bayley and Austen (1988) used more than 1.0 ppm (they used 3.0 ppm) but they were not very clear about whether they were reporting the concentration of active ingredient or the amount of rotenone product they were using.

The label for Noxfish brand 5% rotenone in emulsified solution states "It is in violation of federal law to use this product in a manner inconsistent with its labeling" and recommends for normal pond use, that Noxfish should only be used at 0.5-1.0 ppm (0.025-0.05 ppm active ingredient). To remove fish that are resistant to

rotenone or in less than ideal conditions (for example; an organic pond) no more than 4.0 ppm of Noxfish (0.20 ppm active ingredient) should be used. The label also states that the no more than 5.0 ppm Noxfish (0.25 ppm active ingredient) should be necessary and that higher concentrations should not be used. The State of California (1985) reported that it is illegal to use rotenone in concentrations grater than 0.25 ppm within its boarders.

All of the 3 hr LC50s reported by Marking and Bills (1976) were significantly lower than the recommended dose (0.0025-0.086 ppm). However, slightly higher doses would be needed in the field because the spread of rotenone outside the sampling area would not be contained as it was in their lab experiments and a 100% kill is usually desired, not just 50%.

The permit to use rotenone in Everglades National Park states that rotenone is not allowed to be used in concentrations greater than 5.0 ppm. Because the permit is ambiguous as to whether this specifies the limit of rotenone product or pure rotenone, I believe that the park was negligent in the drafting of the permit and that it should state the limit as 0.25 ppm of the active ingredient in rotenone products. It would not constitute a criminal act for us to use greater amounts than 0.25 ppm because of the wording of the permit, but it would be irresponsible and contemptible for us to do so. As for myself, I will not use more than 0.25 ppm active ingredient inside the boundaries of Everglades National Park.

Sousa et al. (1986) reported that fish are most susceptible to the poison between 50° and 75° F (10°-24° C) and Marking and Bills (1976) reported that an increase in temperature results in quicker

death, probably due to the faster metabolism of fishes in warmer water. Marking and Bills (1976) also stated that softer and more acidic water is conducive to extermination. They concluded that, though acidity, total hardness, and, especially, temperature, have an effect on the action of rotenone in the lab, these results should not be extrapolated to determine dosages in the field. They recommended that field concentrations should be determined on site. However, Marking was dealing with concentrations of less than 100 ppb. By using a relatively excessive amount (250 ppb) in our study, we eliminate the need to test for the effects of hardness and acidity. Also, since the temperatures at our site will probably not drop below 10° C, adjustments of concentration due to temperature will not be necessary because we will be working at the optimum temperature (Sousa et al., 1986).

IV. Application and Collection

Ideally, rotenone should be applied when water is between 15.5° and 20.0° C, alkalinities between 150 and 200 ppm, and pH <8.0 (Marking and Bills, 1976). Most reports concerning the application of rotenone and the collection of dead fishes use much larger sampling areas (>0.5 ha) and are much deeper than our sampling sites. In these cases, boat engines (Burress, 1982) and the forcing of pressurized solutions through perforated, submerged hoses (Davies and Shelton, 1983) are methods generally used to distribute the toxicant. Shallow areas were treated with a hand-pumped sprayer or by using buckets (Gilderhus et al., 1988). Because many of the

fish we wish to sample take refuge in the mangrove prop roots, it would be beneficial for us to use a hand-pumped sprayer with a directional nozzle. This would allow us to release rotenone directly within the prop root structure. A sprayer will also insure that the entire water column is treated, not just the surface, as may be the case when buckets are used to spread the poison (personal observation by the author). Hand-pumped sprayers would also help to minimize movement through sampling sites because the need for mixing by physical activity (stirring the water by walking through it) would be reduced. Walking at our study sites has the potential to radically alter the topography of the substrate. One of our objectives is to leave the sites in a condition as close to the way we found them as possible. Use of sprayers will help accomplish this goal.

Fish tend to swim near the surface for a brief period just after the effects of rotenone are felt. If the fish are not collected with dip nets at this time, they will sink to the bottom. Fish are collected in many studies through the use of SCUBA or seines (Davies and Shelton, 1983; State of California, 1985). Our study sites and target species make these collection methods inadequate. In the eastern sites, collection is not a problem because of the clarity of the water; fish anywhere in the water column can be seen. In the western sites, however, water clarity is very poor and dead fish on the bottom would not be seen. Davies and Shelton (1983) proposed that return visits to the site should be made for up to seven days after rotenone application. As dead fish decompose, gases are created inside the fish and they become buoyant. Floating fish can then easily be collected. Parker (1970) demonstrated that an overwhelming

number of fish do eventually surface (1105 collected/1119 in sample). Parker also showed that colder and deeper water inhibits surfacing while warmer, shallow water facilitates it. He concluded that in water with a temperature higher than 60° F (15.5° C), a depth of less than 10 ft deep (3 m), and no rooted aquatic plants, fish will surface within a week. He also indicated that in less than 3 ft (1 m) of water, under the same conditions, fish will surface within 3 days. These guidelines fit our study very well. Though mangroves can be considered an aquatic rooted plant, Parker was probably referring to submerged vegetation that would "catch" rising fish. Mangroves would not prevent fish from surfacing. Parker's study indicates that our proposed method of collection, i.e., making two returns to the site at 24 and 48 hrs after application, would be effective in collecting most of the fish within an enclosure.

V. Efficiency

Efficiency is usually calculated using mark and recapture (Weinstein and Davis, 1980; Shireman et. al., 1981; Thayer et al., 1987; Bayley and Austin, 1988). Fish should be collected at each site by an alternate method (throw trap, minnow trap, etc.), marked (usually by clipping of a certain fin), and released inside the enclosure for every rotenone sample (Davies and Shelton, 1983). Though this does not create a problem for our western sites (where throw trapping is quite effective) it does create a problem at our eastern sites. During the wet season throw traps are ineffective in the east because density are so low. An alternative is to use minnow traps, but these

must be set a day in advance, creating two more field days a month. During the dry season, when densities are high in the East, throw trapping may be possible.

Efficiencies of reported field studies are highly variable. The most influential factor in recapture is the size of the marked fish. Bayley and Austen (1988) graphed recapture success of fishes from lakes in midwestern United States as a function of length (Figure 1). Larger fish had much higher recapture rates than smaller fish. Weinstein and Davis (1980) reported significantly lower recoveries of fish less than 40 mm (11.5%-46.7%) than fish over 40 mm (52.7%-66.7%) from tidal creeks in North Carolina. Ley (unpublished) reported efficiencies of 35.8% and 35.7% for fishes between 2.5 cm and 5.0 cm and 5.0 cm to 7.5 cm respectively. A 68.0% recovery was reported for fish between 7.5 cm and 15.0 cm in the same study. Preliminary data indicates that the vast majority of fish at our study sites will be under 7.5 cm, so we can expect similar efficiencies. However, in an unreplicated sample at our Highway Creek site, the net efficiency was 56% with all marked fish being smaller than 7.5 cm.

A second factor effecting recapture is species differences. Some species become very conspicuous after death, while other may seem even more cryptic than when they were alive (personal observation by author, supported by Shireman et al, 1981). Table 1 lists the efficiencies found by Ley (unpublished) for fishes common with our study and that were collected in a similar manner to our proposed sampling method. Note that Floridychthys carpio and

Cyprinodon variegatus, two very similar species in both appearance and behavior, have vary different recapture percentages.

Efficiency should be unaffected by those factors that effect collection. For example; cold, deep water should keep fish from surfacing, but, in equal proportions of marked and unmarked fish. If 10% of the marked fish remain missing because they failed to rise, then 10% of the unmarked fish should fail to rise as well. There is no mention in any of the literature of any environmental factor that would effect efficiency. Efficiency, from one sample to the next, would be affected if fish had some way to emigrate from one of the enclosure, or if two different marking processes were used, with one of the methods biased towards recapture or lack thereof. For example, if a certain marking process made the fish more susceptible to immediate predation after reintroduction, while the second method did not, than the first method will have a lower efficiency. As long as the marking methods are the same, and the nets are carefully checked before each sample, the nets of a given sample from a particular site should have similar efficiencies.

Figure 1. Fish retrieval efficiency as a function of fish size. Collection made by using rotenone (reconstructed from Bayley and Austin, 1988).

Table 1. Fish retrieval efficiency (percent abundance recovered) for fishes captured in south Everglades National Park during November, 1989 through the use of block nets and rotenone (from Ley, unpublished)

VI. Prevention of Damage to Untargeted Species and Sampling Areas

Rotenone is a chemically and biologically unstable substance.

Breakdown of rotenone is influenced by light and temperature (State of California, 1985), among other things. Davies and Shelton (1983) reported that temperature is the most influential factor in the degradation of rotenone. The time needed for rotenone to breakdown naturally is calculated using the equations

$$d=93-1.33T$$

for temperatures between 45° and 60° F, and

$$d=38-0.43T$$

for temperatures between 60° and 80° F, where d is the number of days, and T is the temperature in degrees Fahrenheit (Post, 1958). Since we can expect temperatures between 60° and 85° F, the range of time required for breakdown to occur is from 12.2 to 1.45 days (assuming that 85° F can be used in the above equation). It is paramount that damage to the environment at our sites be kept to a minimum. Therefore, having active rotenone moving about in the system for even a day is unsatisfactory. Light also plays a role in the oxidation of rotenone, but since water absorbs sunlight very quickly,

photodegradation of rotenone in solution will be unlikely (State of California, 1985) even in South Florida's famous sunshine.

To prevent unnecessary destruction to sampled areas, potassium permanganate (KMnO_4) can be applied (Davies and Shelton 1983). KMnO_4 is a strong oxidizing agent that breaks down rotenone almost immediately (Marking and Bills, 1976). Chlorine has a similar effect but to a much lower degree (State of California, 1985). Davies and Shelton (1983) recommend that KMnO_4 be applied outside enclosed areas where rotenone is being used. In the case of net enclosures, plastic bags of KMnO_4 solution can be placed in the water and secured to the outside of the net. Just after rotenone application, the bags should be perforated so that the KMnO_4 can diffuse from the bags at a rate similar to that of rotenone from the net. Rotenone diffusing from the net will be oxidized by the halo of KMnO_4 surrounding the net.

KMnO_4 should be used in a solution equal to that of rotenone. For example, if a 5% emulsified solution of rotenone is used than a 5% solution of KMnO_4 should be used to neutralize it (State of California, 1985). After rotenone has had time to complete its function, it is necessary to neutralize the enclosed area to prevent spread. Five percent KMnO_4 at a 4:1 ratio to the 5% rotenone should be applied inside the enclosure. The greater ratio of KMnO_4 to rotenone is to account for the reduction of KMnO_4 by decaying organic material in the water (State of California, 1985). Unfortunately, KMnO_4 in doses over 1.5 ppm can be lethal to fish and other aquatic organisms (Marking and Bills, 1976) and can result in blooms of phytoplankton

(State of California, 1985). Special care should be taken not to exceed the 4:1 ratio and to prevent KMnO_4 spills.

The effects of rotenone are reversible. Davies and Shelton (1983) reported that fish, disoriented by rotenone, can recover if placed in a solution of methylene blue. Gilderhus (1972) reported that some fish removed from rotenone after equilibrium loss, did recover.

VII. Deleterious Effects on Other Aquatic Organisms

The effects of rotenone on terrestrial organisms is reviewed in the introduction. Since wading birds are integral part of the aquatic ecosystem in question, they are of great importance to the study and will briefly be discussed here. The major goal of this study is to determine why breeding success in wading birds has declined drastically in recent years. To use any sampling method that may further reduce breeding success in these birds, or pose a threat to their overall well being in any way, would be counter productive. The only threat to wading birds would be through the feeding of nestlings with rotenone killed fish; rotenone will be used at such a level that acute or chronic toxicity to adults would be an impossibility (see section I) Also, as stated previously, a nestling would have to consume 100 kg of fish killed with rotenone for it to be acutely toxic. Unfortunately there is no mention of the chronic effects of rotenone on nestlings. Though damage to any organism through consumption of rotenone in these quantities is highly dubious, precautions should be made. We will attempt to prevent

the consumption of any poisoned fish by birds by covering the enclosures with 3/4 inch mesh nets. This will also prevent sampling loss due to the feeding activity of birds.

The effects of rotenone on aquatic organisms other than fish is highly variable. A laboratory study of the effects of rotenone on microflora of soil, sediment, and water revealed that nitrogen fixation was neither enhanced nor inhibited at normal treatment levels (State of California, 1985). Microbial activity was, however, inhibited when treated at levels between 5 and 25 ppm pure rotenone (State of California, 1985). Davies and Shelton (1983) reported that 1 to 2 ppm of rotenone is lethal to zooplankton and many aquatic invertebrates. Burress (1982) reported that phytoplankton increase in number after treatment because of the reduction of plankton eating organisms. Regardless of the conflicting reports about what should be considered a harmful dose to microorganisms, our study will not be a threat. The combination of small dosages, small treatment areas, and the immediate neutralization will result in almost immediate recolonization of the sampled area even if rotenone application is at all lethal to the microflora or microfauna.

The State of California (1985) reported that, in general, aquatic invertebrates are less sensitive than vertebrate fishes except for the planktonic crustaceans (eg; daphnia). Houf and Campbell (1977) reported that microcrustaceans are not immediately effected by 0.10 ppm rotenone, however, Burress (1982) reported that there is an immediate 96% decline of microinvertebrates just after a 0.25 ppm treatment. Conflicting results concerning the deleterious effects of

rotenone are reported for crustaceans (State of California, 1985 vs. Gilderhus et al., 1988), mollusks (State of California, 1985 vs. Burress, 1982) and larval insects (Houf and Campbell, 1977 vs. Burress 1982). The reason for such conflicts is because techniques are not standardized and individual field tests with no replication are reported as fact (eg; Burress, 1982). To make such statements about the toxicity of rotenone to different organisms, standard LC₅₀ and LD₅₀ guidelines should be followed, and a wide variety of species from a given taxon tested. Until procedures are standardized, no general statements can be made about the toxicity of rotenone on micro- or macroinvertebrates. Current literature does, however, predominantly report that micro- and macroinvertebrates are more resistant to rotenone than fish and that a temporary reduction in non-fish fauna will occur in a treatment area (Houf and Campbell, 1977; Burress, 1982; Davies and Shelton, 1983, State of California, 1985).

VII. Rotenone Requirements for Proposed ENP Survey

Our current proposal calls for the use of block nets 40 ft long (12.2 m) at our western sites and 80 ft (24.4 m) at our eastern sites. If placed in a perfect circle the smaller nets would enclose an area of 11.8 m² and the larger nets would enclose 47.4 m². We will be using five nets at each site and two site will be sampled in both the east and the west. Our sampling area will 118 m² per month in the west and 474 m² per month in the east for a total monthly sampling area of 592 m². Using this figure, the necessary amounts of rotenone,

for various depths and concentrations are provided in Table 2. As already stated, the maximum concentration of rotenone to be used will be 0.25 ppm of active ingredient and probably no more than a depth of 1.5 ft will be sampled.

Table 2. Amount of rotenone needed (in lbs and kg) for a single months' sample. Calculations are for various concentrations and are for sampling methods proposed as of December 8, 1989 (see section VIII). Depths are given in ft and (m), Volume is in l, and Rotenone/Month is in lbs and (kg).

Depth	Volume	<u>Rotenone/Month</u>		
		1.00ppm	0.25ppm	0.10ppm
0.5 (0.15)	80,000	4.0 (1.8)	1.0 (0.5)	0.4 (0.2)
1.0 (0.31)	181,000	7.9 (3.6)	2.0 (0.9)	0.8 (0.4)
1.5 (0.46)	271,000	11.9 (5.4)	3.0 (1.4)	1.2 (0.5)
2.0 (0.61)	361,000	15.8 (7.2)	4.0 (1.8)	1.6 (0.7)

IX. Conclusion

In 1981 the USEPA stated that there was no reason to restrict the use of rotenone in waters intended for irrigation, livestock consumption, or recreational swimming use because 1) the combined effects of relatively low volume use, rapid degradation in the environment, and rapid dilution that occurs in treated bodies of water make significant human exposures unlikely and 2) current available information on the chemistry and toxicology of rotenone does not suggest that acute or chronic effects are likely to occur.

For these reasons, I feel that use of rotenone in Everglades National Park is of no threat to the preservation of its' current state. By using a dosage of 0.25 ppm in relatively small areas, the knowledge that we gain will be substantial, while the system as a whole will remain unaffected.

X. Literature Cited

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