U.S. DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE

A Review of the Literature

on the Use of Rotenone in Fisheries

by

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ABSTRACT

Rotenone has been used for 40 years as a piscicide in the United States and Canada. It is effective in low concentrations (0.05 to 2 mg/ℓ of the commercial formulations) against most life stages of many species of freshwater and marine fishes, but with a range of specificity among species. Thus, it can be used in selective treatments. Other desirable characteristics of rotenone include less persistence in water than organochlorine pesticides, low toxicity to birds and mammals, and rapid toxicity to fish. Rotenone has had a long history of safe use in agriculture and fish culture. The disadvantages of rotenone include resistance by undesirable carp and bullheads, repellency, failure to reach depths, and reversibility.

If needed, it can be detoxified easily by potassium permanganate or chlorine and removed by activated carbon. The activity of rotenone is limited by high pH, hardness, alkalinity, low temperatures, and certain physical situations such as weed beds and springs.

The toxicity of rotenone to birds and mammals is low. Some invertebrates are sensitive at times, but they usually recover in pretreatment numbers within a few months. Proper on-site bioassays should be conducted to ensure that an overdose of rotenone is not applied.

Several methods are available for determining the amount of rotenone in the water. Other methods are available for determining rotenone in milk, plants, and fish. The method for fish tissues may be

quantitative. Twenty degradation products have been observed after rotenone was exposed to light; this observation may pose a problem in the tolerance registration of rotenone.

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TABLES

I. History of Rotenone Use in Fish Management

The roots of rotenone-bearing plants of the family Leguminosae have been used for many centuries to stun and kill fish by primitive peoples in different parts of the world (Leonard 1939). The most common rotenone-producing plant of this family has been given the scientific name of *Derris*. Derris is a native of Australia, Oceania, and Southern Asia, whereas cubé, another form, is found in South America (U.S. Fish and Wildlife Service 1949).

The use of rotenone in the United States began in the 1930's. Rotenone was first applied as a fishery tool for pond renovation in Michigan in 1934 with only partial success (Ball 1948, Bennett 1971). Within a couple of years, complete fish kills were achieved in ponds (Eschmeyer 1938), in streams (Hoover and Morrill 1938), and in lakes (Siegler and Pillsbury 1946). The first partial treatment using rotenone was performed in 1938 when Utah chubs were killed in shallow water without harming brook trout in deeper water (Greenbank 1941). Large-scale reclamations of watersheds and pre-impoundment reclamations were begun in the late 1950's (Pintler and Johnson 1958, Thoreson 1956, Wilkins 1957, Gaffney 1959).

The results of the first extensive experimental work carried out with rotenone in aquaria for the purpose of obtaining definite data on its action was published by Leonard (1939).

Rotenone was first used in powdered derris or cubé form. Then wettable rotenone paste and emulsifiable rotenone came into being. These formulations were easier to handle, faster acting, and dispensed more readily (Solman 1950).

By 1949, 34 states and several Canadian provinces were using rotenone routinely in fishery management (Solman 1950). Lennon et al. (1970) stated from their survey that all states except Hawaii have used fish toxicants for complete reclamations since 1953, and partial treatments since 1956. Stream reclamations have been performed basically since the early 1960's and the principal toxicant used in the United States was rotenone (Walker 1969, Stroud and Martin 1968).

Rotenone apparently was introduced into Europe as a fish management tool by Swedish workers in the 1950's. In the late 1950's and early 1960's use of rotenone spread to Denmark, Finland, Ireland, and the United Kingdom (Larsen 1961, Tuunainen 1970, Almquist 1959).

South American countries also have used rotenone for fish control (Bonetto et al. 1962, Clark 1929). Fontenele (1963) recorded the prodigious effort made in Brazil to control piranhas.

Various Asian countries have used fish poisons to harvest fish. Today their efforts center on production ponds (Alikundi 1956, Bhimachar and Tripathi 1967, Haq and Tilton 1970).

S. B. Penick applied for patents for the use of rotenone as a fish toxicant starting in 1956 and obtained patents by 1967 in United States Great Britain, Sweden, Denmark, Finland, Israel, Canada, and Norway.

In addition to its use as a fish toxicant, rotenone is utilized in fisheries as a reference toxicant and a fish sampler. Rotenone has been used as a reference toxicant to determine bioassay procedures for the evaluation of fish toxicants (Saila 1954). Rotenone has also been used to sample fish populations in lakes (Baker and Cordone 1969),

reservoirs (Chance and Miller 1952), rivers (Power 1966, Power and Colman 1967), and coral reefs (Smith 1973).

Rotenone's use as an insecticide has been great since the 1800's. Between 1747 and 1931, few publications had dealt with rotenone as a fish poison; rather most literature reported on the chemical analysis and insecticidal uses (Roark 1932). The first article to urge the use of rotenone as an insecticide was published in 1848 in eastern Asia by T. Oxley (U.S. Dept. of Health, Education, and Welfare 1969). Rotenone has been used in Europe as an insecticide since 1920 (Kroller 1969). Its greatest use as an insecticide has been for control of cattle grubs and external pests of livestock and poultry (U.S. Dept. of Health, Education, and Welfare 1969, Whittaker and Whittaker 1935).

Rotenone is one of the compounds used to study and characterize mitochondrical function since it inhibits respiration catalyzed by the terminal respiratory chain (Liu et al. 1970).

II. Physical and Chemical Properties

A. Data on chemical/physical properties

Rotenone is a white crystalline material with the empirical formula of $C_{23}H_{22}O_6$. It crystallizes into orthorhombic, six-sided plates (Gaskins et al. 1972). The molecular weight of rotenone is 394.41. It consists of 70.04% carbon, 5.62% hydrogen, and 24.34% oxygen. Its melting point is 165 to 166 C. Rotenone is soluble in alcohol, acetone, carbon tetrachloride, chloroform, ether, and many other organic solvents, but it is practically insoluble in water. It combines

molecularly with acetic, dichloroacetic, and other acids. Light and air cause rotenone to decompose (Stecher 1968). Pure compound crystals melt at 163 and 180 C (Frear 1969).

Krumholz (1950) and Meyer (1963) recommended that rotenone be stored in a cool, dry location. The powdered formulation should be used within a year. Moorman and Ruhr (1951) pointed out that deterioration in strength of stored rotenone could contribute to failure of reclamations.

Powdered derris root loses toxicity upon exposure to air. Approximately 43% of its toxicity was lost in 6 months from one batch when it was exposed to air and subdued light (Leonard 1939). Rotenone changes from colourless to yellow to deep red as it decomposes. Molyneux (1972) found that, in addition to air and light, alkali accelerates decomposition.

B. Formulations

Commercially available fish toxicants include three basic types of formulations: 5% emulsifiable concentrate, 5% wettable powder, and 2.5% synergized emulsifiable concentrate. Thirty registration numbers representing 17 different chemical companies are on file in the EPA for use of rotenone. Three of the four manufacturers make rotenone for fishery use. S. B. Penick supplies other companies with rotenone products either for distribution, reformulation, or relabeling (Table 1).

Table 1.--Rotenone commercial formulations, including company and address, registration number, and ingredients

Company and Address	Reg. No.	Formulation	Comments
Chemical Insecticide Corp. (Now Blue Spruce Co.) 519 South Maple Avenue Basking Ridge, NJ 07920	1439-156	Rotenone 5% Other cubé extractives 10% Methylated naphthalene 11%	Manufacturer Chem-Fish Regular
Chemical Insecticide Corp.	1439-157	Rotenone 5% Other cubé extractives 7.5%	Chem-Fish Regular O.F.
Chemical Insecticide Corp.	1439-158	Rotenone 2.5% Other cubé extractives 5% Butoxide 2.5% Methylated naphthalene 5%	Chem-Fish Synergized
Chemical Insecticide Corp.	1439-159	Rotenone 2.5% Other cubé extractives 5% Butoxide 2.5%	Chem-Fish Synergized O.F.
Chemical Insecticide Corp.	1439-198	Rotenone 20% Other cubé extractives 30%	Powdered rotenone
Chemical Insecticide Corp.	1439-225	Rotenone 4% Other cubé extractives Sesame oil 5.3%	Chem-Fish T
Cotton Producers Assoc. P.O. Box 2210 Atlanta, GA 30301	2269-139	Rotenone 5% Other cubé resins 9%	For agricultural use

(more)

Table 1.--(cont'd.)

Crown Chemical, Inc. 4995 N. Main St. Rockford, IL 61101	7273-107	Rotenone 5% Other cubé resins 10% Xylene 75%	Supplier is S. B. Penick
J. J. Dill Co. P.O. Box 788 Kalamazoo, MI 49007	6900-113	Rotenone 2.5% Other cubé extractives 5% Sulfoxide 2.2% Related compound 0.3%	Supplier is S. B. Penick. Have own fish toxicant label
Durham's Drug Products Co. Box 443 Comanches, TX 76442	430-29	Rotenone 5% Other cubé resins 10%	For agricultural use
Fairfield Chemical Dept. P.O. Box 1616 Baltimore, MD (Manufacturing plant for FMC Corporation)	4816-166	Rotenone 5% Cubé resins (935) 14.285% Trixton X 100 10% Velsicol	Manufacturer Products for agricultural use
Niagara Chemical Division FMC Corporation Middleport, NY 14105	4816-396	Rotenone 2.5% Other cubé resins 5% piperonyl butoxide tech. 2.5% Heavy aromatic naphthalene 80%	For agricultural use
Niagara Chemical Division FMC Corporation Middleport, NY 14105	4816-397	Rotenone 5% Petroleum distillates Heavy aromatic naphthalene 75%	For agricultural use
Patterson Chemical Co., Inc. 1400 Union Ave. Kansas City, MO 64101	2169-95	Rotenone 1% Other cubé extractives 1.224% Methylated naphthalene 92.776%	Distributor of S. B. Penick products for fishery use. Rotenone vegetable garden dust
Patterson Chemical Co.	2169-101	Rotenone 5% Other cubé extractives 15% Methylated naphthalene 50%	

(more)

Table 2.--(cont'd.)

Pearson & Co. P.O. Box 431 Mobile, AL 36601	728-100	Rotenone 5%	For agricultural use
S. B. Penick & Co. 100 Church St. New York, NY 10007	432-171	Rotenone 2.5% Other cubé extractives 5% Sulfoxide 2.2% Related compounds 0.3%	Major manufacturer Pro-Noxfish
S. B. Penick & Co.	432-172	Rotenone 5% Other cubé extractives	Noxfish
S. B. Penick & Co.	432-188	Rotenone 20% Other cubé extractives 40%	Dri-Noxfish
Prentiss Drug & Chemical Co. 101 W. 31st St. New York, NY 10001	655-421	Rotenone Other cubé extractives 5% Sulfoxide 2.2% Related compounds 0.3%	Supplier is S. B. Penick. Synpren-Fish Toxicant Liquid Emulsifiable
Prentiss Drug & Chemical Co.	655-422	Rotenone 5% Other cubé extractives 10%	Prenfish. Toxicant Liquid Emulsifiable
The Puro-Gen Co. 8544 Broadway Indianapolis, IN 46240	8731-24	Rotenone 5% Other cubé extractives 15% Methylated naphthalene 50%	For agricultural use
Roberts Labs, Inc. 2995 N. Main St. Rockford, IL 61101	523-60	Rotenone 5% Other cubé resins 10% Xylene 75%	Reformulates S. B. Penick products

(more)

Table 1.--(cont'd.)

Seacoast Labs, Inc. 257 Highway 18 East Brunswick, NJ 08817	1159-39	Rotenone 0.75% Other cubé extractives 1.5% Copper as metallic 5%	Have agricultural formulation, not fishery use
Southern Mill Creek Prod. Co. P.O. Box 1096 Tampa, FL 33601	6720-119	Rotenone tech. 5% Cubé resins 10%	Manufacturer. Fish-tox. Emulsifiable
Stephenson Chemical Co., Inc. P.O. Box 188 College Park, GA 30022	4887-15	Rotenone 5% Other cubé resins 10%	Distributor of S. B. Penick products. Some for fishery use.
Stephenson Chemical Co., Inc.	4887-139	Rotenone brittle extract 5% Other cubé resins 10% Heavy aromatic naphthalene 76%	
United Co-operatives, Inc. 111 Glamorgan St. Alliance, OH 44601	1386-206	Rotenone 5% Other cubé resins 10%	Supplier is S. B. Penick. For agricultural use
Woolfork Chemical Works, Ltd. E. Main St. Fort Valley, GA 31030	769-309	Rotenone 5% Heavy aromatic naphthalene 68.2%	Supplier is S. B. Penick. Have own label of fish toxicant.
Woolfork Chemical Works, Ltd.	769-414	Rotenone 5% Other cubé resins 10%	

Instability of product and inconsistency of results were early problems with rotenone. These problems were alleviated in the 1950's when the formulations were guaranteed for content (Lennon et al. 1970).

Powdered rotenone formulations were used first by fishery managers, but they were irritating to the nose and throat and difficult to disperse. Liquid emulsifiable concentrate was easier to apply and mix, more uniform in penetration, and more effective at colder temperatures (Kirkwood 1957, Krumholz 1950, Taube et al. 1954, Price and Calsetta 1957). Then a synergist was added to the liquid to form a synergized emulsion which lowered the cost of treatment (Price and Calsetta 1957). Unfortunately the liquid formulations may be malodorous because of the solvents and carriers (Shannon 1969).

Separation occurs with several formulations if they are mixed too long before application. Engstrom-Heg (1971) recommend that Chem-Fish Regular should not be diluted with water before introduction into a stream and that stock emulsions containing more than 40% Noxfish should not be used. Smaller mixing containers (5-gallon bottle) and a diluting agent other than water should be used.

Bassett (1956) experimented with various formulations to determine if there was any difference in toxicity. He found that Pro-Noxfish, Chem-Fish, and Chem-Fish Special were basically the same in toxicity. Shannon (1969) tested nine of the formulations for toxicity. He found only small variations in amounts of formulation necessary to produce a 24-hour LC50. Noxfish and Chem-Fish Synergized OF formulations were affected significantly by water quality.

C. Interactions

Gaskins et al. (1972) reported on trials in which mixtures of rotenone with deguelin or several other rotenoids were performed using aphids and guppies. An additive effect was observed.

Because some treatment situations may require the use of more than one chemical, Howland (1969) checked the interaction of antimycin and rotenone with fish bioassay. The two chemicals used in combination appear to have an additive effect, but, more importantly, they do not nullify one another.

Brynildson (1970) reports apparent synergism where Bayluscide was applied to a pond which had been treated previously with rotenone. The pond remained toxic to fish for 6 weeks, whereas Bayluscide normally degrades within 48 hours.

Ivie and Casida (1970) surveyed 16 known chemical photosensitizers and 29 pesticides, and found rotenone to be the most effective compound for enhancing the photochemical alteration of dieldrin to photodieldrin. Rotenone also catalyzes the photochemical alteration of aldrin, isodrin, endrin, heptachlor, and heptachlor epoxide. It also increases the photodecomposition of certain methylcarbamate and phosphorothionate insecticide chemicals and of piperonyl butoxide. When certain compounds were tested on bean leaves exposed to sunlight, rotenone showed the greatest photosensitizing activity against chlorinated cyclodienes (Ivie and Casida 1971b). Ivie and Casida (1971a) studied the extent and nature of the interactions occurring when surface deposits including rotenone are exposed to sunlight.

D. Mode of Action

In the 1930's it was noted that, unlike most organic insecticides, rotenone had no specific action upon either the peripheral or control nerves of insects. Similarly, it was noted that respiration of mammals was affected by a blockage in the oxygen utilization (O'Brien 1967).

Fukami et al. (1959) showed that the primary action of rotenone was the inhibition of cell respiratory metabolism in insect tissues, where rotenone acts first on the nerve, and then the muscle. At first they thought the cockroach was deficient in its ability to oxidize succinate completely with consequent oxygen utilization, but it was shown that respiration of succinate was unaffected by rotenone and that it inhibits the respiration of NADH-linked substrate on isolated mitochondria instead and reduced cytochrome b (Ernster et al. 1963, Lindahl and Oberg 1960, 1961). Horgan et al. (1968) confirmed the site of rotenone's action in the respiratory chain as being the oxygen side of the NADH-dehydrogenase as suggested by Burgos and Redfearn (1965) and Palmer et al. (1968). Rotenone is tightly bound not only at the specific site in the dehydrogenase segment, but also at the other sites in submitochondrial particles. Teeter et al. (1969) has shown that at high concentrations, rotenone can affect electron transfer at regions other than the specific site assigned to it.

Hamilton (1941) concluded that death of fish was caused by the constriction of the gill capillaries preventing the passage of blood

through the gills. Danneel (1933) and Scheuring ahd Heuschmann (1935) reported that the action of rotenone destroys gill tissues, However, Burdick et al. (1955) found, through the examination of gills of fish exposed to various concentrations of rotenone, no apparent mechanical injury or loss of filaments. Oberg (1959, 1964) reported that in severe rotenone poisoning, the circulation in the gills was normal and the destruction of gill tissue was apparently due to secondary changes appearing in a late stage of poisoning. Lindahl and Oberg (1961), studying the effect on cellular respiration, reported that rotenone inhibited the uptake of oxygen in the presence of pyruvate and glutamate, but not in the presence of succinate as a substrate. In studying the effect of rotenone on vital organs Oberg (1964) found that glutamate-supported respiration of brain mitochondria from rotenone-treated fish, measured in a rotenone-free medium, is significantly lower than that of control fish.

Hiltibran and Johnson (1965) investigated the effect of rotenone on bluegill liver mitochondrial systems and found succinate oxidation was increased, alpha-ketoglutarate oxidation was severely inhibited, and phosphate uptake was reduced.

Oberg (1967a) suspected that the lipid solubility was a major factor involved in the movement of rotenone from the water into the gill cell membrane. This favors the entry into the cell and hence an increase in the concentration of rotenone at the rotenone-sensitive site. Once in the bloodstream rotenone associates with lipids or lipoproteins which favor its distribution to various organs. Oberg

(1967b) studied the reversibility of the respiratory inhibition in gills and found that gills are able to recover from a respiratory inhibition of up to 50% on exposure of the fish to rotenone-free water for 1 hour after the period of poisoning. A heterogenous population of mitochondria is observed in cells of recovered gills. Folding of the apical membrane is observed only occasionally.

Oberg (1967c) found that in a marine teleost (*Gadus*) exposed to rotenone there was a reduction in gill cell respiration and disruption of chloride cell mitochondria. In gills of treated fish, the apical projections are absent even after restoration of normal cellular respiration, thus the ion transporting ability of these cells may remain impaired. Fromm et al. (1971) observed that rotenone had an acute effect on the flow of perfusion fluid through isolated gills of rainbow trout. The decreased flow appears to be the result of a shift of flow into the high resistance lamellar route.

Schmidt and Weber (in review) investigated the effect of biliary excretion on the toxicity of rotenone in fish following bile duct ligation. They found no significant increase in rotenone toxicity and thus biliary excretion is probably of little consequence in influencing the toxicity of rotenone.

Fukami et al. (1970) investigated the causes of selective toxicity of rotenone between mammal, fish, and insect. In part the selective toxicity of rotenone was due to the differences in the site of action or ease of degradation of rotenone, and not from the differences in the NADH oxidation system in susceptible and resistant species (Lindahl and Oberg 1961, Ernster et al. 1963, Yamamoto 1970).

Fukami et al. (1969) found the results of *in vivo* and *in vitro* studies on rotenone detoxification indicate that the effects of components in the soluble fraction possibly are related to the selective toxicity of rotenone to mammals, fish, and insects.

Schmidt and Weber (in review) contend that the difference in toxicity between organisms may be primarily explained by a more efficient route of entry in fish, the gills.

Mammals are not highly susceptible to rotenone because they are protected by effective oxidizing enzyme systems. However, their isolated mitochondria are very susceptible (Lindahl and Oberg 1961, Ernster et al. 1963).

Rotenone is a powerful muscle relaxant both *in vitro* and *in vivo* (Santi et al. 1966). Ferrari and Maragno (1970) carried these studies further to elucidate the main features of relaxant activity of rotenone.

Most fishes tested by Bhuyan (1967) were affected within a period of 1 to 3 hours. The affected fishes show erratic and convulsive movement, dash about the surface, and lose balance gradually. Respiration is spasmodic and action of pectorals cease (Danneel 1933).

E. Degradation

The degradation of rotenone is fairly rapid, usually within 2 weeks of application (Illinois Department of Conservation 1964). The maximum is 5 months or more (Smith 1941, Leonard 1939, Meyer 1966, Cohen et al. 1960). Certain factors such as high temperatures, high alkalinity, and light intensity accelerate degradation and others, such

as turbidity, extreme depths, and overdosing with rotenone slow it down.

Post (1958) tested a variety of factors to determine the dissipation time for rotenone. The factor which seems to affect the breakdown the most is temperature. Meadows (1973) also found a rise in temperature significantly increases the rate of degradation. Degradation of 2 mg/ℓ of liquid derris occurred within 3 days at a temperature of 20 C and between 11 and 16 days at 11.5 C. However, he assumed that exposure to light, suspended matter, and adsorption by bottom deposits also accelerates degradation.

Rotenone applied under the ice and snow cover detoxifies slowly because of low temperatures and low light intensity (Meyer 1966, Cohen et al. 1960, Rounsefell and Everhard 1953).

Observers in the field have found that degradation is slower in winter months than in the summer months (Clemens and Martin 1953, Wright 1957, Siegler and Pillsbury 1946). Rotenone persists 5 to 6 days in spring, 2 to 3 days in summer, and up to 5 months or more in the winter (McKee and Wolf 1971, Meyer 1966).

Some researchers have found alkalinity and turbidity to affect degradation (Cohen et al. 1960, Rounsefell and Everhart 1953). Clemens and Martin (1953) found that clear-water ponds of low alkalinity (16 mg/ℓ) treated with emulsifiable rotenone lost their toxicity in 3 to 6 days, whereas in clear ponds of high alkalinity (60-284 mg/ℓ) degradation took place in 1 to 3 days. Turbid ponds of high alkalinity took 1 to 2 days longer. Post (1958) does not think that total

dissolved solids, pH, alkalinity, dissolved oxygen or various cations, or anions change the rate of rotenone degradation to any great extent.

Wright (1957) also found deep water affected degradation. Each increase of 1 foot in depth of a pond increased by 2 days the length of time Pro-Noxfish remained toxic to bluegill. However, even 4 to 8 times the amount normally recommended has no residual deleterious effects to bluegill production after the toxicity has dissipated.

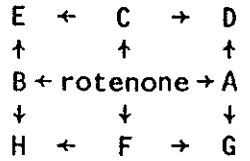
Rotenone is photochemically unstable, readily undergoing oxidative decomposition to nontoxic dihydrorotenone and water in the presence of strong light. The reaction proceeds rapidly at high temperatures (Subba-Rao and Pollard 1951).

Jones et al. (1933) exposed rotenone, dihydrorotenone, rotenone hydrochloride, rotenone-bentonite (1:1), rotenone-lampblack (1:1), ground derris root, and powdered derris extract to various light situations. All but dihydrorotenone and the rotenone-lampblack mixture lost more than half their toxicities during a 10-day exposure to sunlight. It was shown that the photochemical degradation of dry rotenone does not take place in the absence of oxygen. But, according to Ts'ai and Ke (1941), oxygen does not degrade rotenone in the dark. Gunther (1943) carried out quantitative work on the photodecomposition of rotenone and found that it is of the first order.

The type of reclamation, the amount of toxicant, and the type of formulation affect the rate of degradation of rotenone. Rotenone doesn't persist residually when partial treatments of lakes and ponds are used for corrective or selective control of fish populations (Thompkins and Mullan 1958, Bowers 1955, Huish 1958a and b, Jenkins 1956).

At times rotenone does have residual effects. Taube et al. (1954), reporting on the results of 20 years of reclamations in Michigan, found that only 6 lakes of 73 showed a reduction in the numbers of fish-food organisms. Each of these lakes had received twice as much toxicant as was normally employed and each had been treated with Fishtox : or emulsifiable rotenone, both remained toxic longer than the powdered forms. The lakes treated with Fishtox were toxic for over 18 months and those with emulsifiable rotenone, 9 months. Of the 73 Michigan lakes treated with various rotenone formulations only 11 remained toxic for over 1 month. Those 11 were treated with either emulsified rotenone or Fishtox (Taube et al. 1954). Once applied, the powdered derris and cubé formualtions lose their toxicity within 1 month. Fish-tox toxicity lasted from 8 to 36 months. Emulsifiable rotenone lasted from more than 1 month to 13.

The metabolism of rotenone was elucidated by Fukami et al. (1967). Rotenone-6a-^{14}C yielded eight metabolites *in vitro*. Each metabolite is more polar than rotenone and designated as A through H. The sequence of metabolite formation was first determined as follows:



When injected into male mice LD50 values as mg/ℓ were observed as follows: rotenone = 2.8, 8'-hydroxyrotenone (C) = 2.6, 6',7'-dihydro-6', 7'-dihydroxyrotenone (F) = 10, 6aβ, 12aβ- rotenolone (A) = 4.1, and 6aβ, 12aα- rotenolone (B) = >25 (Yamamoto 1969, 1970). The five major metabolites - A, C, D, F, and G - always occur (Yamamoto 1970).

Various hydroxylated rotenoids are naturally-occurring compounds or are formed from rotenone on metabolism in mammals, fish and insects, or by photodegradaton (Fukami et al. 1967, Fukami et al. 1969, Yamamoto et al. 1971, and Cheng et al. 1972). Two of these compounds, 8'-hydroxy rotenone and 6aβ, 12aβ-rotenolone, have toxicity comparable to that of rotenone for mammals (Fukami et al. 1967, Yamamoto et al. 1971, Burgos and Redfearn 1965).

Cheng et al. (1972) experimented with various light sources and exposures of rotenone on various surfaces. They found that rotenone photodecomposes to yield at least 20 compounds. They identified the only toxic photodecomposition product as 6aβ, 12∝β-rotenolone. Fukami et al. (1969, 1967) established the nontoxicity of some of the other products. Yamamoto et al. (1971) found the only metabolites with high potency as NADH oxidase inhibitors are those retaining the original 6aβ, 12aβ-configuration and with one hydroxyl group at the 12aβ- or 8'-position, but even these two metabolites are much less inhibitory than rotenone. The relatively high intraperitoneal toxicity to mice of 6aβ, 12aβ-rotenolone and 8'-hydroxyrotenone despite their reduced activity as NADH oxidase inhibitors suggests that these compounds are not metabolically detoxified as rapidly as rotenone.

Gersdorff (1933, 1934) performed various experiments to determine the toxicity of rotenone and its derivatives to goldfish. Gersdorff (1933) found solutions of rotenone hydrochloride and acetylrotenone are toxic to goldfish at 0.002 mg/ℓ, but with solutions of rotenolene the theoretical threshold of toxicity is 0.020 mg/ℓ. The decreasing

order of toxicity to goldfish is rotenone, acetyldihydrorotenone, dihydrorotenolone, acetyldihydrorotenolone, and acetylrotenolone.

The order of relative toxicities in mg/ℓ of rotenone derivatives to goldfish is as follows: dihydrorotenone, 1.4; rotenone, 1.0, acetyldihydrorotenone, 0.81; acetylrotenone, 0.55; dihydrorotenolone, 0.15; rotenolone, 0.097; acetyldihydrorotenolone, 0.082; and acetylrotenolone, 0.055 (Gersdorff 1935).

Unai et al. (1973a) present the details of the synthesis and sterochemical characterization of various hydroxy- and epoxy- derivatives of rotenone which are important in determining the toxicological significance of rotenone metabolites and photodecomposition products of rotenone. Unai et al. (1973b) established 3-O-Demethylation as the detoxification mechanism for 5'β-rotenone (natural rotenone) or for one of its metabolites.

The urine and feces of five rabbits and two dogs were tested for the presence of rotenone. No rotenone or conjugation product was revealed in the urine; however, rabbits continued to eliminate rotenone in the feces for about 11 days after it was administered and dogs for 8 days. No rotenone was recovered from daily unused fecal extracts (Ambrose and Haag 1937).

III. Efficacy

A. Use Pattern

Rotenone is most commonly applied at 0.5 to 1 mg/ℓ of rotenone formulations. The actual concentration is a function of fish species to be eliminated, water quality, and type of kill desired. Under usual conditions rotenone will kill fish within 24 to 36 hours (U.S. Fish and Wildlife Service 1949). Reclamations have been attempted year-around, but ideally they are conducted during the warm season. Treated waters have included ponds, lakes, reservoirs, lagoons, estuaries, and streams, either totally or partially, depending upon the type of effect desired and the location of the fish.

A fish can become a target for control when it has definite impact on man's safety, welfare, or recreational opportunity because of abundance, feeding habit, lack of sport or food value, or disease condition. Adverse effect on the environment and associated organisms may also bring control efforts.

The range of concentration varies according to target species of fish. S. B. Penick (label) claims that 0.5 mg/ℓ of rotenone formulation can produce a satisfactory fish kill; however, when bullheads are present or water is exceptionally alkaline, concentrations up to 1 mg/ℓ formulation should be used for best results. Field investigators frequently find that 1 to 5 mg/ℓ are required to control resistant species. To kill bullheads, green sunfish, and bowfin, up to 3 mg/ℓ of rotenone formulation is required (Brown 1973). In his review of reclamations in Michigan, Spitler (1970) found that concentrations

less than 1.6 mg/ℓ formulation are not very successful. Although the recommended concentration of rotenone formualtion was 0.5 mg/ℓ, many fishery managers use 1.5 to 5 mg/ℓ formulation.

Guerra (1966) decided to use aircraft to disperse 2 µℓ/ℓ of liquid rotenone formulation to remove fish from inaccessible areas. The high concentration was needed because of drift and the posssibility that rotenone might fall on dead brush and plants.

Jester (1963) successfully used several concentrations of rotenone to eliminate undesirable species before impoundment of a reservoir. Liquid formulation of rotenone at 1.5 µℓ/ℓ was used in Conchas Dam Stilling Basin, 6 to 8 mg/ℓ of wettable powder in the South Canadian River, and 4 µℓ/ℓ of liquid formulation in the Pajarito Creek.

Regan (1961) used 1.9 mg/ℓ of powdered rotenone formulation in a river and 0.7 µℓ/ℓ of liquid rotenone formulation in a lake. Of the fish collected, 97% were rough fish.

Experience from the field indicates that the following concentrations of active ingredient have generally eliminated the species mentioned: trout, native Cyprinidae, suckers - 0.025 to 0.050 mg/ℓ; sunfish - 0.050 to 0.075 mg/ℓ; bullhead - 0.1 to 0.2 mg/ℓ; carp - 0.113 to 0.2 mg/ℓ; and goldfish - 0.2 mg/ℓ. Individual fish are sometimes extremely resistant, especially in the case of goldfish, bullheads, and carp. Hence, complete kills of these species are unusually difficult to achieve (Meyer 1966).

Prévost (1960) found that the following formulation concentrations were effective on the various species: 0.5 to 0.7 mg/ℓ for yellow perch, sunfish, and minnows; 0.7 to 1 mg/ℓ for suckers, burbot (ling),

and pike; and 1 to 3 mg/ℓ for catfish and killifish.

The time rotenone takes to affect certain fish varies with certain factors (Gilderhus 1972). The effective contact times (ECT - expsoure necessary to induce 100% mortality) have been determined for 50, 100, and 250 µg/ℓ of rotenone in combination with three temperatures (12, 17, and 22 C) and certain species of fishes. The ECT's ranged from 0.5 to 25 hours with the time influenced more by water temperatures than by toxicant concentration. For example, at 12 C a doubling in concentration from 50 and 100 µg/ℓ active ingredient/reduced the ECT by 6 hours, whereas raising the temperature by 5 C reduced the ECT of 50 µg/ℓ by 14 hours. The ECT for rotenone was generally reached only after the fish had become incapacitated in the toxicant solution for 1 to 5 hours. The greater ECT's make it impractical for stream usage. Stream-side bioassays are advisable to determine the appropriate concentration and ECT.

Kinney (1968) adivsed the use of rotenone to obtain complete, partial, or selective kills of target fish. Complete treatments are recommended when the major portion of fish population is undesirable. To achieve a complete kill, rotenone is applied to the total water area at a minimum of 1 mg/ℓ rotenone formulation. Partial treatments are usually utilized in early spring before spawning to remove excessive numbers of small fish, but not to kill the entire fish population. Rotenone is applied only to the shoreline, bays, or shallow areas at a formulation concentration of 1 mg/ℓ in the areas treated.

Selective treatments where certain species of fish, such as gizzard shad, become undesirable are performed in late summer or early fall. Rotenone is applied at a weak concentration (0.1 mg/ℓ formulation) to the entire area and primarily shad will be killed, leaving the other fish unharmed. This type of treatment should be done annually for 7 years to obtain more lasting effects (Illinois Department of Conservation 1964).

The success, however, has been varied. Anderson (1970) found that the rehabilitation in Minnesota lakes lasts from 5 to 15 years. Due to reduced competition, growth rates of yellow perch, chain pickerel, and largemouth bass in warm water ponds in Massachusetts were found to be more rapid than in other natural conditions, and in some cases growth rates exceeded those recorded in history (Grice 1959). Clemens and Martin (1953) made experimental applications of emulsifiable and powdered rotenone in 30 ponds in Okalhoma. Two to 3 months later 28 of 30 ponds still contained fish.

The most successful reclamations with rotenone have been carried out in trout waters with the targets being nonnative, introduced species (Zilliox and Pfeiffer 1960). Although most lake reclamations have been accomplished in cold water, complete renovation, partial poisoning, and selective poisoning also have been used in warmwater lakes with varying degrees of success.

Rotenone has been used to improve waterfowl resting areas by eliminating carp which feed on aquatic vegetation. Emulsifiable formulation of rotenone was successfully applied at 0.5 µℓ/ℓ by a helicopter to shallow lakes and marshlands in Ohio to remove carp (Weier and Starr (1950).

Early stream reclamations were conducted by M'Gonigle and Smith (1938), Hoover and Morrill (1938), and Hagen (1940). They were trying to remove sources of disease from hatchery water supplies. Their efforts had mixed success. Smith (1950) and Miller (1950) found that reinfestation was a major problem.

The increased use of toxicants in streams prompted studies on the relative efficiency of rotenone and electrofishing. Boccardy and Cooper (1961, 1963) proved rotenone to be a more effective sampling tool than electrofishing. They concluded that stream reclamations with rotenone are effective if precautions are taken to treat the water thoroughly.

Partial reclamations have been utilized to remove carp from lakes, leaving sport fish behind. This practice has improved fishing and carp do not become abundant after treatment (Gerking 1950). This practice has also been used to control stunted panfish (Hunn 1957). Beckman (1941) found that partial treatments increased the growth of rock bass. Partial removal of bluegills resulted in improvement of bass production in a lake, but no increase in the desirable sizes of bluegills was noticed (Clark 1964). Emig (1966) noted that treating

nests and parts of lakes in Wisconsin has had uncertain success and that complete eradication is the most practical.

Swingle et al. (1953) suggest methods and techniques of correcting overcrowded pond fish populations by partial poisoning with rotenone. They suggest spring or early summer, at water temperatures above 27 C, near noon.

Byrd and Crance (1965) found that 14 years of partial poisoning to restore balance to overcrowded populations in public fishing lakes yielded successful largemouth bass fishing. However, Hooper and Crance (1960) found that bullhead or crappie crowding in bluegill-redear sunfish populations cannot be corrected by partial poisoning.

King (1953) found that partial population reduction did not result in significant growth increases, but the fishing was improved somewhat. King (1954) thought the reasons for failure of the reclamation/increased turbidity and siltation each year, low productivity were of impounded waters, and inability to remove an adequate proportion of the overpopulated species without seriously reducing bass and forage populations.

The differential in the susceptibility of various species of fish to rotenone makes selective reclamations possible. Gizzard shad have been the target of successful selective removal attempts (Bowers 1955, Huish 1959). Gizzard shad have been greatly reduced in lakes with the use of low concentrations of rotenone (Sandoz 1959, Anderson 1966, Wharton 1968, Wilcox 1965, Wood and White 1962). Wyatt and Zeller

(1965) conducted a survey and found success in removing gizzard shad from reservoirs. Dietz and Jurgens (1963), however, found success shortlived and expensive because retreatment is needed every 3 to 4 years. They suggest that only small volumes of water should be treated.

In an attempt to selectively control gizzard shad, Jergens (1957) found that treatment should be before fall turnover when water temperatures are high and shad are concentrated in shallower areas of lakes. This procedure eliminates the necessity of treating the deeper portions of the lake twice. Complete renovations were attempted to clear a lake of turbidity after other methods such as selective shad removal, drawdowns, and commercial fishing for nongame species failed (Keith 1968).

Anderson (1966) recommended that 0.15 µℓ/ℓ of Noxfish be used to remove gizzard shad from bass production ponds. Huish (1959) used 0.06 to 0.14 µℓ/ℓ of emulsifiable rotenone formulation in the fall 3 years in succession with good results. Jurgens (1957) used 0.12 mg/ℓ of rotenone formulation in shallow water and 0.15 mg/ℓ in deep water to remove both gizzard shad and freshwater drum. Mathis and Hulsey (1959) also varied the concentration used and, in addition, manipulated the water level.

Yellow perch have been a target of selective reclamation, especially in trout waters (Eschmeyer 1937 and 1938, Barrows 1939, Burdick et al. 1956). Riel (1965) found that 0.5 to 1 mg/ℓ of rotenone formulation will kill the eggs of perch. This along with netting of adults selectively reduces overpopulation of yellow perch in lakes.

B. Specificity

Various investigators have established orders of tolerance for several species of fish. Jenkins (1956) stated that the order of apparent increasing tolerance to rotenone of several species was as follows: gizzard shad, carp, largemouth bass, redear sunfish, black crappie, bluegill, white crappie, green sunfish, warmouth, and black bullhead. Burdick et al. (1955) found concentrations of 0.5 mg/ℓ of powdered derris root (5% rotenone) killed bluegills, sunfish, suckers, shiners, sticklebacks, mudminnows, and goldfish, but 0.25 mg/ℓ was nontoxic.

The species least resistant to rotenone include common shiner, golden shiner, bluegill, pumpkinseed, and brook stickleback. The mudminnow and the goldfish are the most strongly resistant (Leonard 1939).

The toxicity threshold of rotenone differs widely among species (Burdick et al. 1955). The toxicity curves indicate that species studied fall within the following order of increasing resistance to rotenone formulation (mg/ℓ): brown trout (0.2), rock bass (0.32), creek chub (0.35), smallmouth bass (0.4), white sucker (common sucker) (1.7), and brown bullhead (2.2). These curves indicate that 0.05 mg/ℓ of active ingredient would not be enough to gain a complete kill with suckers or bullheads present.

Hester (1959a) tested three formulations of rotenone (Noxfish, Pro-Noxfish, and powdered cubé) against eight species of fish. The range of 72-hour LC50's at 21 C was for carp 0.081-0.163 mg/ℓ, for largemouth bass 0.081-0.164 mg/ℓ, for fathead minnow 0.159-0.200 mg/ℓ, for green sunfish 0.165-0.246 mg/ℓ, for goldfish 0.175-0.242, for bluegill 0.179-0.268 mg/ℓ, for golden shiners 0.470-0.620 mg/ℓ, for brown bullhead 1 to 1.4 inches (speckled bullhead) 0.247-0.410 mg/ℓ, for brown bullhead 6 to 8 inches 0.794-1.033 mg/ℓ.

Meadows (1973) tested rotenone against several species of fish. Yellow perch were the most sensitive and were killed by an initial concentration of 0.1 mg/ℓ of derris formulation, but for the Crucian carp the minimum concentration lethal in 7 days was 4 mg/ℓ. Next in resistance were carp (mirror carp), roach, white amur (grass carp), gudgeon and rudd.

Marking (unpublished data) tested the toxicity of Noxfish to 11 species of fish at 12 C in reconstituted water and determined the 96-hour LC50's in µℓ/ℓ for the following species in order of their sensitivity: lake trout (0.0269), northern pike (0.0330), chinook salmon (0.0369), brook trout (0.0443), common carp (0.05), rainbow trout (0.0593), yellow perch (0.07), white sucker (0.084), channel catfish (0.196), and goldfish (0.497). The Atlantic salmon is also sensitive to rotenone with a 96-hour LC50 of 0.0215 µℓ/ℓ (Marking 1973). Green sunfish are fairly resistant with 96-hour LC50's of 0.2 to 0.253 µℓ/ℓ at 12 C in very soft to very hard water (Marking et al. 1974).

Bridges and Cope (1965) found the 24-, 48-, and 96-hour LC50's of 4.85% rotenone formulation to bluegills, rainbow trout, and channel catfish were 0.023 to 0.026, 0.027 to 0.031, and 0.028 to 0.033 mg/ℓ, respectively. Wilbur (1969) found similar values for bluegills and rainbow trout. The 24-hour LC50 was 0.024 mg/ℓ for bluegills and 0.032 mg/ℓ for rainbow trout. Bluegills died after 5 hours when exposed to 0.5 mg/ℓ of rotenone formulation (Adlung 1957).

Striped bass are extremely sensitive to rotenone. The 24- to 96-hour LC100 is 0.01 mg/ℓ (Hughes in press).

The white amur, a member of the carp family which has been widely introduced as a possible controller of aquatic vegetation, could also become a problem. Marking (1972) tested rotenone against white amur and found the 96-hour LC50 to be 63 µg/ℓ. Additional results from tests with different hardnesses and temperatures showed that rotenone would be an effective controller of white amur. Henderson (in press) tested the effects of rotenone on the white amur, and found that 0.01 mg/ℓ was the minimum concentration that would appreciably reduce a population of white amur.

The most resistant species to rotenone are bullheads and goldfish being about five to seven times, respectively, more resistant than rainbow trout held under similar conditions (Cumming in press). Although Gersdorff (1930) found the concentration just necessary to kill goldfish was 0.0125 mg/ℓ of active ingredent, the results in the field show that 1 mg/ℓ of formulation or more to eliminate goldfish in the

field (Anjaneyulu and Row 1966, Cumming in press). Krumholz (1948) found that formulation concentrations of 1.0 and 1.5 mg/ℓ failed to produce a complete kill of bullheads even though the recommended dose was 0.5 mg/ℓ.

When using pesticides, the possibility of developing rotenone-resistant strains of rough fish increases with partial treatments (Hubbs 1963). Fabacher and Chambers (1972) have found resistant strains of mosquito fish. The 24-hour LC50 was 0.017 mg/ℓ for the normal susceptible strain, but the 24-hour LC50 was 0.031 mg/ℓ for the resistant strain. Tompkins (1953) found one bluegill was resistant to treatments of 0.5 and 0.736 mg/ℓ formulation in which all other bluegills died.

The effect of rotenone on the various life stages have been investigated by several researchers. Generally, more toxicant is needed to kill adults than younger fish. This is the case with tilapia (Rowe-Rowe 1971). Toxicity to small fish ranges from 0.01 to 0.1 mg/ℓ formulation (McKee and Wolf 1971).

Eggs are usually more resistant than fry or fingerlings to rotenone. Eyed brown trout eggs were not killed in a derris concentration of 0.5 mg/ℓ, but the fry perished as soon as they broke the shell (Leonard 1939). Garrison (1968) tested the toxicity of Pro-Noxfish to salmonid eggs and fry. Over a 24-hour period more than 10 times as much Pro-Noxfish was required to kill 100% of the test batch of eggs as was required to kill an equal percent of fry.

The effect of Derris on the eggs, alevins, and fry of the mouth-breeder was determined by Rowe-Rowe (1971). Formulation concentrations (0.15 mg/ℓ) strong enough to kill 50% of the adult fish were also strong enough to kill fry within 24 hours and eggs within 48 hours, while alevin live for as long as 10 days in a 0.15 mg/ℓ Derris solution.

Hester (1959b) tested the toxicity of Noxfish and Pro-Noxfish to carp and fathead minnow eggs, finding that the LC50 using eggs was very similar to the LC50 using fingerlings of these species. The concentrations ranged from 0.091 to 0.233 mg/ℓ against the eggs and 0.081 to 0.191 mg/ℓ against the fingerlings.

Olson and Marking (in review) tested rotenone against green eggs of the chinook salmon, brook trout, and lake trout at 12 C for 24, 96, and 192 hours. The lake trout eggs were most sensitive at >0.25 to 1 mg/ℓ. The brook trout eggs were most resistant at 3.40 to 4.24 mg/ℓ.

Marking et al. (1974) found that the sensitivity of eggs to rotenone is affected by different water hardnesses. The 96-hour LC50's to rainbow trout eggs at pH 8.0 ranged from 2.5 µℓ/ℓ in very hard water to 5.6 µℓ/ℓ in very soft water when exposed to Noxfish. On the other hand, the 96-hour LC50's to fingerling green sunfish at pH 8.0 in various hardnesses ranged from 0.2 to 0.253 µℓ/ℓ.

C. Factors Affecting Efficacy

The efficacy of rotenone is affected significantly by temperature, pH and alkalinity, and to a lesser extent by physical conditions.

Rotenone is more effective at high temperatures than at low (Gersdorff 1943, Almquist 1959). Berry and Larkin (1954) state that higher temperatures increase the respiration rate of fish which increases its rate of uptake of toxicants. The survival time of roach is markedly reduced by high temperatures (Meadows 1973). However, at high temperatures, toxicity may last for only 24 hours, resulting in detoxification so rapid that toxicity is lost before the toxicant is distributed (Hooper et al. 1964).

Loeb and Engstrom-Heg (1970c) performed experiments to determine the extent to which dispersions of Noxfish change in toxicity upon standing. They found that at 8 C toxicity of Noxfish to trout remains constant for at least 10 days, but at 18 C Noxfish becomes suddenly nontoxic between 2 and 7 days after preparation. They also suggested, as had Goodhue and Haller (1939), that there may be enzymes that attack rotenone.

Turner (1959) found that summer treatments were more successful than fall treatments. The fish kills were 97% complete in summer as compared to 72% in the fall. Huntington and Jester (1958) found that temperatures as low as 1 C froze the drip system and they failed to obtain a complete kill, even with 2 µℓ/ℓ of rotenone formulation. Herrig (1964) found that some rough fish survived at a water temperature of 5.6 C. Bisbee (1968) treated the Lower Owyhee River in Oregon

with rotenone in March to take advantage of the low water flow, but the water temperatures were less than favorable at 7 to 13 C. The toxicant failed to penetrate into depths of pools and degraded rapidly.

Laboratory experiments by Leonard (1939) showed that at 16 to 24 C rotenone is the most effective. Below 9 C rotenone loses much of its effectiveness (Ball 1948). Recommendations are for applications to be made at temperature greater than 16 C (Hooper 1955). The best range of temperatures, according to Spitler (1970) was 15 to 20 C. In his survey 62.5% of the treatments within that range were successful.

Action is faster in acid than alkaline waters. Increase in water temperature from 16 to 23 C reduced the time of death almost by half. Size and species of fish, the temperature, pH, and turbidity of the water and the occurrence of weed beds affected the time needed to kill fish (Reynolds and Barry 1951, Burdick et al. 1955, Leonard 1939, Zilliox and Pfeiffer 1956, Jenkins 1956).

Leonard (1939) tested various concentrations against eight species of fish and concluded that 0.5 to 1 mg/ℓ of powdered derris root (5%) was sufficient to kill several fish species. The action was faster (3 hours vs 16 hours) at pH 6.5 than pH 8.2 for goldfish but less so for brook stickleback and golden shiner. Foye (1964) also found that rotenone is more effective in neutral pH waters.

Brooks (1961) determined the 24-hour LC50's for Pro-Noxfish to goldfish at various pH's. He found essentially no difference in the amount (0.1 to 0.12 µℓ/ℓ) at pH's 5 to 7. However at pH 8 and 9 the

requirement (0.17 to 0.19 µℓ/ℓ) increased somewhat, but at pH 10 the increase (to 0.33 µℓ/ℓ) was quite pronounced.

Spitler (1970) in reviewing reclamations with rotenone in Michigan waters found that in waters with pH's greater than 8.0 the success rate was only 28.5%. In lakes with pH of 8 or less the ratio of success was 57.1%.

Rotenone is more effective in soft than in hard water (Foye 1964). Survival times of roach were shorter in soft water than in hard. A test with 2 mg/ℓ of 5% rotenone in 20 mg/ℓ total hardness gave a median survival time some 45 minutes less than a comparable test in hard water (Meadows 1973).

Complete fish kills are common in soft, acid waters at formulation concentration between 0.25 and 0.5 mg/ℓ (Zilliox and Pfeiffer 1960). Hard, alkaline waters frequently require 1 mg/ℓ of rotenone formulation or more to effect a complete kill (Clemens and Martin 1953).

Leonard (1939) found toxic action in tests to be somewhat faster in acid than alkaline waters. Prévost (1960) and Almquist (1959) found that alkalinity does affect efficacy of rotenone. Spitler (1970) found that success was 44% when alkalinities fell within the range of 150 to 200 mg/ℓ.

However, Post (1958) found that pH (7.0-8.6), total dissolved solids, alkalinity (as mg/ℓ CO_3 ion from 14.8-227), dissolved O_2, and various cation and anion concentrations did not affect toxicity in ponds.

Additional variables may affect treatment success. Soft bottom areas, springs, floating bogs, weed beds and turbidity may affect success, especially if they are prominent environmental features (Spitler 1970, Prévost 1960). Aquatic vegetation and bottom sediments may provide surface for adsorption (Berry and Larkin 1954). Because of turbidity in a lake, 1.5 mg/ℓ of rotenone formulation was needed to remove white suckers, carp, and stunted yellow perch (Huntington 1956). Almquist (1959) recognized turbidity as a factor in efficacy of rotenone.

Orn (1962) examined various lakes using a photometric method to determine the rotenone emulsions in water. He found that rotenone emulsions are adsorbed, to a considerable extent, on bottom deposits, organisms and probably colloids. In one lake very rich in plankton, one-third of the added rotenone had been adsorbed 1 hour after treatment. In shallow lakes, the large surface area of mud may increase the adsorption of rotenone.

The efficacy of rotenone is at times affected by its repellency of undesirable species. Sayre (1969) found that rotenone delivered from a helicopter repelled fish downstream into a reservoir where they escaped rotenone poisoning.

Several investigators find that depth and thermocline affect the extent that rotenone disperses, thus causing incomplete kills (Foye 1964, Prévost 1960, Lennon 1970). Foye (1964) observed 48 pond treatments with rotenone in Maine and found that the thermocline retards

vertical dispersion, and proper mixing of chemical in deep lakes is believed to be more effective during the fall turnover.

In analyzing the results from treating ponds Turner (1959) found that complete kills occurred in 97% of the 24 ponds treated in summer and 72% of 32 ponds in the fall. He assumed that the fish were fleeing from rotenone into deep water that was homothermous and well oxygenated. He concluded that farm ponds over 2.1 meters in depth should be treated in the summer months. Richard (1962) found that the commercial formulations of Noxfish, Chem-Fish Special, Chem-Fish Regular, and cubé powder all penetrated the thermocline and caused complete kills within 30 hours at 0.1 mg/ℓ of rotenone active ingredient.

The optimum conditions considered by Spitler (1970) for a complete kill with rotenone in southern Michigan lakes include

1) Emulsifiable rotenone formulation at 1.5 µℓ/ℓ or more
2) Water surface temperatures between 16 and 21 C
3) Maximum depth between 3.4 and 6.1 meters
4) Lake size of less than 8.1 hectares
5) Shallows of from 60 to 80% of total volume
6) Alkalinity between 150 to 200 mg/ℓ
7) pH of 8 or less
8) Minimum of marshes, dense weed beds, floating bog materials, turbidity, springs, and soft bottom areas
9) Correct applications and follow-up procedures

D. Counteraction

Rotenone is detoxified largely or completely with solutions of potassium permanganate or chlorine and removed from the water with activated carbon. Potassium permanganate has been reported to be the most practical detoxifier or rotenone (Lawrence 1956, Engstrom-Heg and Loeb 1968).

Shannon (1968) conducted experiments to determine the concentration of $KMnO_4$ and the time necessary to detoxify various concentrations of Noxfish and Dri-Noxfish. $KMnO_4$ successfully detoxified Noxfish in 10 minutes at ratios of 1:40 or 1:48 rotenone to $KMnO_4$. Higher rates were needed for Dri-Noxfish. He extended his tests to seven additional formulations and found that the most readily detoxified formulations were the Chem-Fish formulations with the OF designation (Shannon 1969). Hepworth and Mitchum (1967) tested six formulations and found no apparent difference in the concentration of $KMnO_4$ needed to detoxify the various 5% formulations. A concentration of 2 to 2.5 mg/ℓ of $KMnO_4$ was required to detoxify 1 mg/ℓ rotenone formulation. Two mg/ℓ of $KMnO_4$ required 25.5 to 30 minutes to detoxify the 5% formulation and only 13 to 15 minutes to detoxify the 2.5% synergized rotenone formulation.

Engstrom-Heg (1972) determined the kinetics of rotenone-$KMnO_4$ reactions. He found that the amount of $KMnO_4$ required to counteract rotenone depends upon the concentration and formulation of rotenone, the hardness of the water, the desired detoxification time, and the background $KMnO_4$ demand. In cases of need for rapid detoxification, the

concentration of $KMnO_4$ may also be toxic; thus, reducing agents, such as sodium thiosulfate, may have to be applied to counteract $KMnO_4$. Other detoxifiers of $KMnO_4$ include tannic acid and hydrogen peroxide and possibly gallic acid, propyl gallate and manganese and ferrous salts (Loeb and Engstrom-Heg 1970a).

Loeb and Engstrom-Heg (1970b) tested the use of $KMnO_4$ as a detoxifier in a small portion of Ten Mile River. Noxfish was introduced at 1 µℓ/ℓ and 640 meters below the rotenone introduction, 4.6 mg/ℓ of $KMnO_4$ was added. The rotenone was 95% detoxified in 9 minutes and fully detoxified in 12 minutes. The neutralization was considered successful.

Hatchery managers use $KMnO_4$ to detoxify rotenone in hatchery ponds and reduce oxygen demand in highly fertile ponds (Anonymous 1971).

Recent studies by Marking et al. (1973) were executed to determine the rate of detoxification of Noxfish by potassium permanganate. Green sunfish were exposed to fresh and aged solutions of Noxfish containing 1 mg/ℓ of $KMnO_4$ to determine the half-life of rotenone at four different pH's. The deactivation indices show small and nonuniform changes in the toxicity of Noxfish for aging periods of 10 to 50 minutes. However, there is an immediate, rapid, detoxification which cannot be quantified in the 10-minute aging procedure. For instance, the 96-hour LC50 for unaged Noxfish using green sunfish was 0.378 µℓ/ℓ at pH 9.5, and the 96-hour LC50 for unaged Noxfish and 1 mg/ℓ of $KMnO_4$ was 1.55 µℓ/ℓ.

Swan (1965) used a machine which prepared a slurry of the $KMnO_4$ to be metered into the stream, thus eliminating some of the problems frequently encountered in applying permanganate crystals. Florescein dye added to rotenone helps to determine when to add the detoxifier (Meyer 1963).

Formulas have been prepared, equipment selected, and techniques developed which can be used to administer potassium permanganate in streams to detoxify rotenone (Slifer 1970, McCoy and Ratledge 1967). Fishery managers have experimented with $KMnO_4$ in Argentina (Bonetto et al. 1962). Cross (1971) reported use of $KMnO_4$ in Natal to revive fish.

Both chlorine and $KMnO_4$ may be used to detoxify rotenone-treated water. However, Jackson (1957) reports that chlorine is easier to handle as chlorinated lime than is $KMnO_4$ in crystalline form. He recommends that chlorine be used in coldwater trout ponds with a chlorine demand of 0.5 mg/ℓ or below. Both detoxifiers can be used in small streams or outlets of ponds treated with rotenone. On the other hand, Meyer (1963) finds chlorine difficult to work with, expensive, and unable to be applied continuously.

Engstrom-Heg and Loeb (1968) found that in stream situations, concentrations of chlorine which are sufficient to detoxify rotenone must in turn be detoxified with sodium thiosulfate. Chlorine is more satisfactory as a detoxifier for ponds.

When more and more treatments with rotenone were proposed in municipal water supplies, Bonn and Holbert (1961) investigated means of removing the odor and undesirable taste resulting from the application. Laboratory tests showed these conditions can be controlled with 1 mg/ℓ of activated carbon for each threshold odor number produced. Cohen et al. (1960) also recommended using activated carbon to eliminate odors from rotenone formulations which cannot be removed by chlorine. Cohen et al. (1961a) successfully treated a water supply contaminated with 2 mg/ℓ of rotenone formulation by adding 61 mg/ℓ activated carbon. Both odor and toxicity were reduced to palatable odor and safe toxicity levels. Cohen et al. (1961b) conducted further tests on odor reduction and determined that 36 to 85 mg/ℓ of activated carbon were needed for 2 mg/ℓ of various rotenone formulations. In addition, Dawson (in press) reported that 15 to 30 mg/ℓ of powdered activated carbon has been used for each µℓ/ℓ of Noxfish to detoxify it in a stream.

Dawson and Marking (in review) found the capacity of activated carbon to adsorb rotenone was 0.1 mg of Noxfish per gram of carbon.

IV. Toxicity to Nontarget Organisms

Rotenone is relatively harmless to most nontarget organisms. Its dosage can be so controlled that it creates no particular hazard to nontarget organisms except for temporary effects on zooplankton, insects, and other benthos (U.S. Dept. of Health, Education, and Welfare 1969). Hazards to humans are minimal if rotenone is handled properly.

A. Toxicity to plants

Rotenone does not affect either algae or rooted aquatic vegetation (Smith 1940). Smith (1941) found no evidence that algae or rooted aquatic vegetation is affected by rotenone. Species of algae, belong to the genera *Aphanocapsa, Anabaena, Dinobryon, Staurastrum, Tabellaria,* and other phytoplankton were plentiful before and after a treatment of 0.5 mg/ℓ of Derris.

Anderson (1950) ascertained the effect of a rotenone treatment on aquatic vegetation which eliminated a population of carp. *Chara vulgaris, Potamogeton pectinatus,* and *Vallisneria americana* increased significantly in abundance, while floating leaved and emergent species remained the same. Wollitz (1962) found little change occurred in most phytoplankton following treatment with Chem-Fish Special. *Ceratium* was absent 2 weeks after treatment but was present in 29 days. *Dinobryon* was reduced with a treatment of Pro-Noxfish.

Almquist (1959) reported that some of the phytoplankters were totally killed by 0.5 to 0.6 mg/ℓ of 5% rotenone. *Ceratium* sp. were affected by 1 mg/ℓ in 4 hours. Renewal of phytoplankton began in a few weeks after treatment.

Binns (1967) found that the aquatic flora was not adversely affected by 2.5 to 9.4 mg/ℓ of 5% rotenone. An increase in abundance was noticed due apparently to increased light, nutrients, and decreased grazing by fish. Bonn and Holbert (1961) also reported a plankton increase following rotenone treatment and believed it to be caused by a reduction in numbers of bottom feeding fish.

B. Toxicity to invertebrates

The effect of rotenone on invertebrates has been reported variously by many observers of reclamations over the past 40 years. However, if reductions in invertebrates occur, usually enough survive to continue their existence in that body of water (Lindgren 1960, Fellton 1940, Cushing and Olive 1957). Generally a sufficient food supply is available for the fish which are subsequently introduced into the treated water (Meadows 1973). In general invertebrates are much less sensitive than fish to rotenone except for the planktonic crustaceans (microcrustaceans), which are very sensitive to those rotenone concentrations which kill fish (Hamilton 1941, Smith 1940 and 1950).

Several authors have reviewed the effects of rotenone on invertebrates and have found rotenone does not adversely affect invertebrate populations. In a recent 2-year field study using ponds Houf and Hughey (1973) and Houf (1974) found no short- or long-term effects on the population abundance, relative numbers of dominant species, or species diversity of either zooplankton or benthos following treatments of 0.5 to 2 mg/ℓ formulation.

Crayfish populations were not affected by a rotenone formulation of 1.5 mg/ℓ. The trout stocked shortly after the treatment were feeding on zooplankton and *Chironomus* larvae (Huntington 1956). Anderson (1970) found that *Gammarus lacustris* survived in treated waters and stocked trout fed more frequently on this animal than had the original population.

Smith (1940, 1941), Brown and Ball (1943), Catt and Needler (1946), Ball and Hayne (1952), Hooper (1948), Pintler and Johnson (1958), Zilliox and Pfeiffer (1960), and Prévost (1960) all reported that treatment with various preparations containing 5% rotenone does not significantly affect fish-food organisms. A few of these investigators did note, however, that some organisms suffered adverse effects. Smith (1941), using 0.5 mg/ℓ derris noticed some mortality to *Chaoborus* larvae, planktonic microcrustaceans, snails (*Compeloma decisum*) and a leech. Other invertebrates, algae, and rooted vegetation are not affected. Brown and Ball (1943) conducted several experiments with caged invertebrates and found that most organisms were not seriously affected by rotenone. Only leeches, *Chaoborus (Corethra)* and aeschnine dragonflies are susceptible to rotenone, but they reappeared in 5 weeks. Only aeschnidae, Hirudinea, and *Chaoborus* died when the fish populations of a lake were removed with rotenone. However, the number or volume was only a minor fraction of the total organisms present in the lake (Ball and Hayne 1952). Hooper (1948) noted that 0.5 mg/ℓ of rotenone formulation was toxic only to Cladocera and Copepoda and not to other plankton or bottom fauna. Apparent changes in populations were believed to be due to

either sampling errors or normal flux resulting from emergence, migration, depredation, and other natural causes.

Wright (1957) used 1 µℓ/ℓ of Noxfish and Pro-Noxfish and found that Cladocera and Copepoda were absent after treatment. About 1.5 months later, a few Cladocera had returned. Copepods returned within 1 month. No effect was noticed for phantom larvae (*Chaoborus punctipennis*) or midge larvae (*Tendipes plumosus*).

Toffoli (1965) found that only the invertebrates in the immediate vicinity of the drip stations where concentrations were high were affected. None seemed to be affected downstream.

Several workers have reported rotenone as being highly toxic to aquatic invertebrates. Scheuring and Heuschman (1935) observed high mortality among *Sialis* (Megaloptera), Corixidae (Hemiptera), and *Chironomus plumosus* (Diptera) when these organisms were exposed to rotenone.

Almquist (1959) reported that most zooplankton, much of the epiphytic, and bottom animals were killed by 0.5 to 6 mg/ℓ of 5% rotenone. The most resistant groups of zooplankton were Rotatoria and Ciliata, but the mortality of the species varies greatly even within the same genus. Wollitz (1962) also noted the disappearance of zooplankton after treatment with 5.5% emulsified rotenone; dragonflies, caddisflies, and other insects also were reduced or eliminated by the treatment. Hoffman and Olive (1961) found that 1 mg/ℓ of rotenone removed protozoans, reduced rotifers, and adversely affected Entomostraca.

Kiser et al. (1963) and Donaldson et al. (1962) found that open-water zooplankton were completely removed and remained absent for 3 months from a lake treated with 0.5 mg/ℓ of 5% rotenone powder. Those species along the shore resisted the effect of rotenone, but eventually disappeared for several weeks. The populations were not reestablished until several months after the lake had become nontoxic to fish. Another shallow lake was treated with 1 mg/ℓ of rotenone formulation, and an immediate reaction by zooplankton was observed. The rotenone penetrated to the thermocline, killing Cladocera and Copepoda as it sank.

Oglesby (1964) noticed the freshwater polychoete *Nereis limnicola* was reduced greatly in Lake Merced with 0.025 mg/ℓ of rotenone.

Binns (1967) reported that invertebrate populations of Green River were drastically reduced by an overdose of rotenone ranging from 2.5 to 9.4 mg/ℓ of Chem-Fish Regular. The net-spinning caddisflies (Hydropsychidae) common before did not reappear until almost 2 years later. Two rare mayfly genera *(Pentagenia* and *Hexagenia)* did not reappear but several mayfly genera did.

The recovery of zooplankton and benthos from an application varies according to the species reduced or affected and the ecological relationships. When a balanced state is achieved in the ecosystem, the numbers of organisms return to normal levels (Lindgren 1960).

The renewal of zooplankton usually takes 1.5 to 3 months; whereas, many of the resistant benthic organisms reproduce at a very rapid rate and reach equilibrium in a few months after treatment (Almquist

1959, Smith 1941, Hooper 1948, Cushing and Olive 1957, Lindgren 1960, Brown and Ball 1943).

Even though species can be affected severely, the effect can be short-lived. Zilliox and Pfeiffer (1960) found that 0.5 mg/ℓ of rotenone formulation did not have an adverse residual effect on the food supply of the brook trout. The populations of macroscopic bottom fauna were only affected for a short period of time by a 1 mg/ℓ application (Cushing and Olive 1957). Mihajlovic and Rajevski (1960) observed both bottom fauna and zooplankton, including Cladocera in the spring after a November treatment with 0.5 mg/ℓ of Derris.

Although a rotenone treatment in a river almost destroyed the invertebrates completely, the organisms recovered to pretreatment levels both in quantity and quality within 1 month in the upper 16 km of the river. Below the 16-km mark, changes occurred both in abundance and diversity (Dexter 1965).

At times populations of invertebrates are slow to recover from rotenone reclamations. Fischthal (1947) found that plankton was reduced drastically and recovery was slow from 0.5 mg/ℓ treatment. Even after 44 days some species were still missing and those present were in reduced numbers.

Populations of bottom dwelling organisms were reduced from 34 to 100% by rotenone. All organisms except two, Plecoptera and Isopoda, recovered up to or beyond their original abundance 1 year after eradication (Little 1966).

Cook and Moore (1969) noticed a rapid and tremendous increase of organisms after sharp reductions of their numbers as a result of a Pro-Noxfish treatment of 0.05 mg/ℓ active ingredient of rotenone to the major tributaries of the Russian River. The taxa so affected included Trichoptera, Ephemeroptera, and Diptera.

Tuunainen (1970) noted that no group or species present in the lakes he studied were eliminated. In fact, *Pisidium*, *Asellus*, *Sialis*, and chironomids increased. Cushing and Olive (1957) noticed that oligochaete worms were not affected by rotenone and their numbers increased shortly after the treatment. Wollitz (1962) found that Tendipedidae and Tubificidae were adversely affected by 5.5% emulsified rotenone, but recovered to twice their original level within 1 month.

Several reasons are given for the increase in density of bottom organisms following treatment. Perhaps the elimination of direct predation by fish or the influence of fish upon food chains brings about the increase. The kills of phyto- and zooplankton can result in a new supply of food for benthic fauna (Tuunainen 1970).

Increase in invertebrates may be caused by lack of inter-specific competition. Smith (1941) observed large increases in the numbers of amphipods and molluscs which survived the treatment of a lake and Cushing and Olive (1957) found that chironomids were killed, but oligochaetes increased in population until the chironomids repopulated about 9 months later.

In some cases even though the adults or larvae of a species are reduced, the eggs are resistant. Dobie and Moyle (1945) noticed that an application of 1.4 mg/ℓ of rotenone formulation only affected protozoan plankton, but because their eggs were hard to kill, the species would recover. Prévost (1960) also noticed this to be true even with a strong concentration.

One of the results of a rotenone treatment is the possible change in the succession of invertebrate species during the recovery period. Binns (1967) observed that several groups dominated for fairly long periods of time after the treatment in contrast to short periods of dominance by various groups before the treatment. First, Tubificadae dominated in the spring following the treatment, then Tendipedidae and Baetidae recovered and dominated in the fall, and finally, Simulidae rose to dominance in the following summer. Two years after treatment, the patterns were still dissimilar to pretreatment patterns.

Table 2 lists the tests carried out in the laboratory. The invertebrates are in phylogenetic order according to Pennak (1953). All units of measurement were standardized to the metric system.

Table 2.--Toxicity of rotenone to invertebrates, including concentration, exposure, water temperatures and chemistry, and formulation

Organisms	Concentration (mg/ℓ)	Exposure	Temp. (°C)	Water Chemistry	Formulation	Comments	Citations
Turbellaria (Flatworms)							
Planaria	.5	40-50 hr			5% rotenone	Lethal to all	Hamilton 1941
Leeches	.1	50-60 hr			5% rotenone	Lethal to all	Hamilton 1941
Stylaria	.05				5% rotenone	20% reduction	Hamilton 1941
Cladocera (Water fleas)	LC50						
Daphnia pulex	0.01	48 hr	16	35 mg/ℓ TDS			U.S. Federal Water Pollution Control Administration 1968
	LC50 0.025	3 hr			5% rotenone	100% mortality	Hamilton 1941
Daphnia	.48	48 hr	27		Cubé		Wright 1957
	.24	48 hr	27		Noxfish		
	.32	48 hr	27		Pro-Noxfish		
	.57	48 hr	24		Cubé		
	.49	48 hr	24		Noxfish		
	.44	48 hr	24		Pro-Noxfish		
	.55	48 hr	20		Cubé		
	.56	48 hr	20		Noxfish		
	.57	48 hr	20		Pro-Noxfish		

(more)

Table 2.--(cont'd.)

Daphnia	0.1	48 hr	27-29	pH 7.5		Minimum lethal dose --weakest concentration of chemical which produces a kill exceeding 25%	Zischkale 1952
	EC50 .100	48 hr	16	pH 7.4-7.8			Sanders and Cope 1966
	LC50 .55	48 hr	20		Pro-Noxfish		Brooks 1961
	.44	48 hr	24				
	.31	48 hr	27				
Daphnia	LC50 0.25	1 hr					Negherbon 1959
Simocephalus serrulatus	EC50 .190	48 hr	16				Sanders and Cope 1966
Cladocera *Leptodora kindtii*	0.025	3 hr			5% rotenone	100% mortality	Hamilton 1941
Copepoda (Copepods) *Cyclops*	LC50 .22	48 hr	27		Cubé		Wright 1957
	.12	48 hr	27		Noxfish		
	.14	48 hr	27		Pro-Noxfish		
	.24	48 hr	20		Cubé		
	.14	48 hr	20		Noxfish		
	.19	48 hr	20		Pro-Noxfish		
Cyclops sp.	.1	3 days	11±1	pH 7.9 260 mg/ℓ hardness	5% rotenone liquid	None survived	Meadows 1973

(more)

Table 2.--(cont'd.)

	LC50						
Cyclops	.18	48 hr	20-27		Pro-Noxfish		Brooks 1961
	.14	48 hr					
Diaptomus siciloides	0.025	3 hr			5% rotenone	100% mortality	Hamilton 1941
Isopoda (Aquatic sow bugs)							
Asellus aquaticus	.1	6 days	11±1	pH 7.9	5% rotenone	90% survived	Meadows 1973
	.5	6 days	11±1	260 mg/ℓ	liquid	70% survived	Meadows 1973
	2.0	6 days	11±1	hardness	"	None survived	Meadows 1973
Amphipoda (Scuds, sideswimmers)							
Amphipods	.5	12 hr			5% rotenone	Lethal	Hamilton 1941
Scuds (*Hyalella knickerbockerii*)	1.0	96 hr			5% rotenone	No distress	Leonard 1939
Hyallella sp.	.2	48 hr	27-29	pH 7.2		Minimum lethal dose --concentration which produces a kill not exceeding 25%	Zischkale 1952
Amphipod (*Gammarus lacustris*)	LC50 6.0	24 hr	16	35 mg/ℓ TDS			U.S. Federal Water Pollution Control Administration 1968

(more)

Table 2.--(cont'd.)

Scuds (*Gammarus limnaeus*)	LC50						
	11.6	24 hr	12	pH 7.2-7.6	Noxfish		Farringer 1972
	6.6	24 hr	12	40-48 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
	9.2	24 hr	12	pH 7.6-8.0	Noxfish		Farringer 1972
	5.8	24 hr		160-180 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
	8.0	96 hr	12	pH 7.2-7.6	Noxfish		Farringer 1972
	1.7	96 hr	12	40-48 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
	2.5	96 hr	12	pH 7.6-8.0	Noxfish		Farringer 1972
	1.8	96 hr	12	160-180 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
Gammarus limnaeus	LC50						
	6.0	24 hr					Pimentel 1971
Gammarus lacustris	LC50						
	3.52	96 hr	15		Technical grade		Nebeker and Gaufin 1964
Gammarus pulex	0.1-0.5	6 days	11±1	pH 7.9	5% rotenone liquid	No mortalities	Meadows 1973
	2.0			260 mg/ℓ hardness		90% survived	Meadows 1973
Scuds (*Gammarus lacustris*)	6.0	24 hr	21	pH 7.1	14.39% rotenone		Sanders 1969
	3.5	48 hr	21	30 mg/ℓ hardness	"		
	2.6	96 hr	21	"	"		
Decopoda (Crayfishes, shrimps)							
Crayfish 33 mm (*Procambarus* sp.)	10.0	96 hr	20±1	pH 7.3-7.8	5% wettable powder	One died	Brown 1973
	75.0	96 hr		100 mg/ℓ hardness		Two died	

(more)

Table 2.--(cont'd.)

Crayfish *(Cambarus immunis)*	.5				5% rotenone	Not affected	Hamilton 1941
Crayfish *(Cambarus propinquus)*	1.0	96 hr	16		5% rotenone	No distress	Leonard 1939
Crayfish *(Orconectes immunis)*	LC50						
	34.5	24 hr	12	pH 7.2-7.6	Noxfish		Farringer 1972
	10.8	24 hr	12	40-48 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
	47.2	24 hr	12	pH 7.6-8.0	Noxfish		Farringer 1972
	9.6	24 hr	12	160-180 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
	1.0	96 hr	12	pH 7.2-7.6	Noxfish		Farringer 1972
	.7	96 hr	12	40-48 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
	1.2	96 hr	12	pH 7.6-8.0	Noxfish		Farringer 1972
	.4	96 hr	12	160-180 mg/ℓ hardness	Dri-Noxfish		Farringer 1972
Palaemontes	4.0	48 hr	27-29	pH 7.2		Minimum lethal dose --concentration which produces a kill not exceeding 25%	Zischkale 1952
Plecoptera (Stoneflies)	LC						
Immature Stonefly *(Pteronarcys californica)*	2.9	24 hr	16	pH 7.0	4.85% rotenone		Bridges and Cope 1965; Cope 1965
	.9	48 hr	16	35 mg/ℓ, M.O. alkalinity	"		"
	.25	96 hr	16		"		"

(more)

Table 2.--(cont'd.)

	LC50						
Pteronarcys californica	2.9	24 hr	15.5	pH 7.0	Technical	Pyrethrum is 141 to	Sanders and Cope
	1.1	48 hr	15.5	35 mg/ℓ, M.O.	grade	290 times more tox-	1968
	.38	96 hr	15.5	alkalinity		ic than rotenone	
Stonefly (*Pteronarcys californica*)	LC50 0.9	48 hr	16	35 mg/ℓ TDS			U.S. Federal Water Pollution Control Administration 1968
Stonefly nymph (*Acroneuria*)	1.0	96 hr	16		5% rotenone	No distress	Leonard 1939
Ephemeroptera (Mayflies)							
Mayfly nymph (*Siphlonurus*)	LC50 1.4	48 hr	22-23	pH 7.0-7.3 <25 mg/ℓ turbidity	95% rotenone	Should not be affected by fish-killing concentrations	Claffey and Ruck 1967
Odonata (Dragonflies, damselflies)							
Pachydiplax	3.5	48 hr	27-29	pH 7.2		Minimum lethal dose --concentration which kills less than 25%	Zischkale 1952
Dragonfly nymph (*Anax*)	LC50 2.5	48 hr	21-22	pH 7.0-7.2 <25 mg/ℓ turbidity	95% rotenone	Should not be affected by fish-killing concentrations	Claffey and Ruck 1967

(more)

Table 2.--(cont'd.)

Damselfly nymph *Agrion*	LC50 2.7	48 hr	21-23	pH 7.1-7.2 <25 mg/ℓ turbidity	95% rotenone	Should not be affected by fish-killing concentrations	Claffey and Ruck 1967
Damselfly nymphs *Argia* sp.	1.0	96 hr	16		5% rotenone	No distress	Leonard 1939
Amphiagrion	2.5	48 hr	27-29	pH 7.2		Minimum lethal dose --concentration which kills less than 25%	Zischkale 1952
Tramea	3.5	48 hr	27-29	pH 7.2			
Hemiptera (Bugs) Corixids	LC50 .5-1.0				5% rotenone	Not fatal	Hamilton 1941
Adult Hemiptera Corixids	1.0	96 hr	16		5% rotenone	No distress	Leonard 1939
Notonectids	.1	24 hr			5% rotenone		Hamilton 1941
Water bugs *Belostoma* sp.	1.0	96 hr	16		5% rotenone	No distress	Leonard 1939
Trichoptera (Caddisflies) Caddisfly larvae *Phryganea*	1.0	48 hr	21-22	pH 7.1-7.2 <25 mg/ℓ turbidity	95% rotenone	Might be reduced by high fish-kill-concentrations	Claffey and Ruck 1967
Limnephilidae	1.0	96 hr	16		5% rotenone	No distress	Leonard 1939

(more)

Table 2.--(cont'd.)

Caddisfly	LC50						
Hesperophylax sp.	10.4	24 hr	12	pH 7.2-7.6	Noxfish		Farringer 1972
	5.1	24 hr	12	40-48 mg/ℓ hardness	Dri-Noxfish		
	15.0	24 hr	12	pH 7.6-8.0	Noxfish		Farringer 1972
	5.1	24 hr	12	160-180 mg/ℓ hardness	Dri-Noxfish		
	3.4	96 hr	12	pH 7.2-7.6	Noxfish		Farringer 1972
	3.2	96 hr	12	40-48 mg/ℓ hardness	Dri-Noxfish		
	2.5	96 hr	12	pH 7.6-8.0	Noxfish		Farringer 1972
	3.6	96 hr	12	160-180 mg/ℓ hardness	Dri-Noxfish		
Diptera (Flies, mosquitos, midges)							
Cranefly larvae							
Tipula sp.	1.0	96 hr	16		5% rotenone	No distress	Leonard 1939
Culex	2.0	48 hr	27-29	pH 7.2		Minimum lethal dose --concentration which produces a kill not exceeding 25%	Zischkale 1952
Aedes	2.0	48 hr	27-29	pH 7.2			
Anopheles	2.0	48 hr	27-29	pH 7.2			
Phantom larvae	LC50						
Chaoborus punctipennis	.65	48 hr	27		Cubé		Wright 1957
	1.07	48 hr	27		Noxfish		
	1.13	48 hr	27		Pro-Noxfish		
Phantom midge larvae	LC50						
	1.13	48 hr	27		Pro-Noxfish		Brooks 1961
Chaoborus asticotopus	LC98						
	1.0				5% rotenone derris powder		Negherbon 1959
(Winter larvae)	LC97						
	0.5				5% rotenone derris powder		

(more)

Table 2.--(cont'd.)

Midge larvae *Tendipes decoris*	1.0	96 hr		pH 5.9-6.1 5-154 mg/ℓ M.O. alkalinity	Emulsifiable rotenone Cubé powder Cubé powder and Tide	Seriously affected	Taube et al. 1954
Midge larvae	LC50						
Tendipes crassicaudatus	.25	48 hr	27		Cubé		Wright 1957
	.10	48 hr	27		Noxfish		
T. plumosus	.33	48 hr	27		Pro-Noxfish		
Midge larvae	LC50						
	.31	48 hr	27		Pro-Noxfish		Brooks 1961
Aquatic midges	6.0	18 hr		pH 8.3-8.7 250-350 mg/ℓ hardness		5% mortality	Fellton 1940
	6.0	67-68 hr				100% mortality	
	3.0	46-52 hr				50% mortality	
Gastropoda (Snails, limpets)							
Snail *(Lymnaea stagnalis)*	1.0	3.5 days			5% rotenone	70% died	Hamilton 1941
	.5	3.5 days			5% rotenone	30 died	
Physa halei	0.1-.1				5% rotenone	Little effect	Hamilton 1941
Snails *Physa*	1.0	96 hr	16			No distress	Leonard 1939
Physa	4.5	48 hr	27-29	pH 7.2		Minimum lethal dose --concentration which produces a kill not exceeding 25%	Zischkale 1952
Helisoma	3.5	48 hr	27-29	pH 7.2			

(more)

Table 2.--(cont'd.)

Snail *Australorbis glabratus*	LC50 1.25	96 hr					Wasicky and Unti 1951
Oysters *C. virginica*	.1					Minimum effective concentration	Butler et al. 1960
Pelecypoda (Clams, mussels)	LC50						
Clams	2.7	96 hr	12	pH 7.2-7.6	Noxfish		Farringer 1972
Lampsilis sp.	12.9	96 hr	12	40-48 mg/ℓ hardness	Dri-Noxfish		
	2.3	96 hr	12	pH 7.6-8.0	Noxfish		Farringer 1972
	11.3	96 hr	12	160-180 mg/ℓ hardness	Dri-Noxfish		
Arachnida							
Water mites							
Hydrachnidae	0.05	4 days			5% rotenone	43% mortality	Hamilton 1941

C. Toxicity to vertebrates

All vertebrates, except fish, are extremely resistant to fish-killing concentrations of rotenone.

Amphibians and reptiles apparently are not affected by rotenone treatments. Haque (1971) reports that neither frogs or snakes were affected by 1 to 2 µℓ/ℓ of Chem-Fish Special O.F. In general, the young birds tested are about 10 times more sensitive to rotenone poisoning than older birds, the acute oral LD50 ranging from 100 to 200 mg/kg of body weight for young birds and 1,000 to 2,000 mg/kg for older birds (Cutkomp 1943b). Based on the LD50 for chicks (8 mg/kg) for Pro-Noxfish, a chick would need to consume approximately 10,900 liters per kilogram (Price and Calsetta 1957).

Chick embryos at five developmental stages ranging from full primitive streak to six pairs of somites were exposed *in vitro* to the action of 1 µg/mℓ of rotenone for 15 minutes. Definite abnormalities were noted by Rao and Chauhan (1971). The primary organizer of the embryo is not interfered with by rotenone.

Lettré (1949) observed a mitotic poison effect with rotenone on chicken heart fibroblasts and on tumor cells.

The acute oral toxicity of rotenone to mammals varies considerably between species, with the LD50 values ranging from 60 to 3,000 mg/kg. The variation was influenced by percentage of rotenone in a formulation, the particle size of rotenone, and the diluent.

The minimum acute lethal intravenous and intraperitoneal dose was 0.5 to 5 mg/kg of rotenone active ingredient, while approximately two to three times the acute intravenous dose was tolerated when administered in divided doses at various intervals (Hazleton Laboratories Inc. 1968).

Ambrose et al. (1942) found that 600 to 1,200 mg/ℓ of Derris (0.06 -9.6% rotenone) fed rats fro 200 days at dietary levels caused some growth suppression. The same effect was noted when pure rotenone was fed to rats for 107 days at dietary levels of 90 to 200 mg/ℓ. Based on the acute oral LD50 (1.7 mℓ/kg Pro-Noxfish) for rabbits, it would be necessary for a rabbit to consume at least 3,400 liters of treated water per kilogram of body weight in one drinking to obtain a dose of 1.7 mℓ of Pro-Noxfish used at the rate of 0.5 mg/ℓ (Price and Calsetta 1957, Brooks 1961). Pro-Noxfish at 100 mg/ℓ was fed to rats in their drinking water for 70 weeks and the average weight was less than that of the controls (Brooks and Price 1961).

When administered by mouth, pure rotenone produced no visible effect in dogs, cats, pigs, or sheep in doses up to 1 grain per pound of body weight (Buckingham 1930).

No gross abnormalities developed in rats fed 100 mg/ℓ of Pro-Noxfish or degraded Pro-Noxfish for 70 and 52 weeks, respectively (Brooks and Price 1961). Two-year studies by Hansen et al. (1965) also found no pathologic effects other than growth depression. In fact, the incidence of gross mammary tumors, nephritis, and pituitary lesions at the higher levels (150-400 mg/ℓ) was less than in the controls. Haag

and Taliaferro (1940) found no microscopic abnormalities in various organs or tissues of rats fed 300 mg/ℓ for 150 days. Innes et al. (1969) tested the tumorigenicity of rotenone by continuous oral administration of a maximal tolerated dose (1 mg/kg) for 18 months to mice. Rotenone did not cause a significant increase in tumors after oral administration (John I. Thompson and Co. 1969).

Rats were exposed to 5 to 15 mg of rotenone per kilogram of body weight daily for 37 days. No tumors were observed (Davis et al. 1970).

Teratology studies were conducted on three pregnant guinea pigs at a dietary level of 150 mg of rotenone per kilogram of food and on six guinea pigs at 75 mg/kg of food. All litters at the 150 mg/kg were stillborn or died with in 5 days; at the 75 mg/kg level two litters were born dead, but the other levels showed only growth suppression (Haag 1931).

Used at low concentrations rotenone suppressed the growth of New Castle disease and herpes simplex viruses, as well as decreased the necrotic spots on tobacco mosaic virus-infected leaf discs. Only derrisic acid completely inhibited the local lesion formation at subphytotoxic concentrations (Takatsuki et al. 1969),

The respiration of malignant lymphoid tissue is affected by 0.1 to 0.2 saturation in serum of rotenone, but it was nonspecific (Chambers et al. 1943).

Table 3 lists the studies performed in the laboratory on vertebrates. The vertebrates are in phylogenetic order according to Rothschild (1961). All units of measurement were standardized to the metric system.

Table 3.--Toxicity of rotenone to vertebrates including concentration or dose, exposure, and formulation

Organisms	Concentration	Exposure	Formulation	Comments	Citations
Amphibia	LC50				
Rana pipiens	4.8 µℓ/ℓ	24 hr	Noxfish	12° C, pH 7.2-7.6	Farringer 1972
	7.3 mg/ℓ	24 hr	Dri-Noxfish	40-48 mg/ℓ hardness	
	24.0 µℓ/ℓ	24 hr	Noxfish	12° C, pH 7.6-8.0	Farringer 1972
	7.9 mg/ℓ	24 hr	Dri-Noxfish	160-180 mg/ℓ hardness	
	4.8 µℓ/ℓ	96 hr	Noxfish	12° C, pH 7.2-7.6	Farringer 1972
	4.6 mg/ℓ	96 hr	Dri-Noxfish	40-48 mg/ℓ hardness	
	5.8 µℓ/ℓ	96 hr	Noxfish	12° C, pH 7.6-8.0	Farringer 1972
	3.2 mg/ℓ	96 hr	Dri-Noxfish	160-180 mg/ℓ hardness	
Frog tadpoles					
Rana pipiens	0.1 mg/ℓ	8-24 hr	5% rotenone	Lethal for all stages of tadpoles	Hamilton 1941
Metamorphosing salamanders					
Ambystoma tigrinum	0.1 mg/ℓ			Lethal for those which had completed metamorphosis	Hamilton 1941
	.017 mg/ℓ			Toxic but not necessarily fatal before resorption of gills	Hamilton 1941
Frogs	LD50				
	4.0 mg/kg			Acute oral	Haag 1931

(more)

Table 3.--(cont'd.)

Aves				
Pekin ducks	25 µℓ/ℓ	Chem-Fish Regular Pro-Noxfish	Chronic oral, nontoxic	Blue Spruce Co. 1973 Penick 1962
	50 µℓ/ℓ	Chem-Fish Synergized Pro-Noxfish	Chronic oral, nontoxic	Blue Spruce Co. 1973 Brooks 1961
White rock chickens	25 µℓ/ℓ	Chem-Fish Regular	Chronic oral, nontoxic	
	50 µℓ/ℓ	Chem-Fish Synergized Pro-Noxfish	Chronic oral, nontoxic	Blue Spruce Co. 1973 Brooks 1961
White rock chickens	LD50 6 mℓ/kg	Chem-Fish Regular Noxfish	Acute oral	Blue Spruce Co. 1973 Penick 1962
	LD50 8 mℓ/kg	Chem-Fish Synergized Pro-Noxfish	Acute oral Acute oral	Blue Spruce Co. 1973 Brooks 1961
	LD50 8 mℓ/kg	Pro-Noxfish	Acute oral	Brooks 1961
Chickens (5-day)	LD50 996 mg/kg	100% rotenone	Acute oral	Cutkomp 1943b
Chickens (28-day)	3000 mg/ℓ	100% rotenone	Acute oral	Cutkomp 1943b
Chickens (5-day)	LD50 247 mg/kg	Derris extract containing 25% rotenone	Acute oral	Cutkomp 1943b
Chicken eggs	1 mg/ℓ	Rotenone in acetone	Injected. Killed 36%	Pimentel 1971
	5 mg/ℓ	Rotenone in acetone	Injected. Killed 86%	Pimentel 1971
	10 mg/ℓ	Rotenone in acetone	Injected. Killed 100%	Pimentel 1971

(more)

Table 3.--(cont'd.)

Nestling English song sparrows	LD50 130 mg/kg		Acute oral	Cutkomp 1943b
Nestling chipping sparrows	LD50 113 mg/kg			
Nestling English sparrows	LD50 200-850 mg/kg			
Nestling American robins	LD50 200 mg/kg			
Young and old pheasants	LD50 850-1200 mg/kg		Acute oral	Cutkomp 1943b
Pheasants (female, 3-4 months)	LD50 >1414 mg/kg	Rotenone	Acute oral	Tucker and Crabtree 1970
Mallards (female, 3-4 months)	LD50 >2000 mg/kg	Rotenone	Acute oral	Tucker and Crabtree 1970
Pigeons	LD50 1.0 mg/kg	Rotenone	IV	Haag 1931
	200-500 mg/ℓ	Rotenone	Oral. Emesis noted in 15-30 minutes. No other effects	Haag 1931
Chickens (4-week)	LD50 >270 mg/kg	100% rotenone	Acute oral	Cutkomp 1943a
Mammalia				
Rabbits	1.6 g/kg		Fatal oral dose	Haag 1931
	1.25 g/kg		Survived oral dose	Haag 1931

(more)

Table 3.--(cont'd.)

Rabbits	0.35 mg/kg			Minimum lethal IV dose	Haag 1931
	5.0 mg/kg			Minimum lethal IM dose	
	20.0 mg/kg			Minimum lethal SC dose	
	LD50 100-200 mg/kg	Daily for 21 days	10% technical grade in dimethyl phthalate	Dermal toxicity	Lehman 1949, 1952
Rabbits	LD70 3000 mg/kg		Crystalline rotenone Cubé powder Derris powder	Acute oral	Haag 1937
	LD70 600 mg/kg		9.6% rotenone Derris powder	Acute oral	Haag 1937
	LD70 1000 mg/kg		4.7% rotenone Cubé powder	Acute oral	Haag 1937
Rabbits	3.0 g/kg		Rotenone	Lethal dose	Ambrose and Haag 1937
	60.0 mg/kg	Daily for 30 days	Derris	Signs of cumulative toxicity	Ambrose and Haag 1938
Rabbits	LD50 1.7 mℓ/kg		Chem-Fish Synergized Pro-Noxfish	Acute oral	Blue Spruce Co. 1973 Brooks 1961
Rabbits	LD >940 mg/kg		10% rotenone in di-methyl phthalate	Ct	Negherbon 1959

(more)

Table 3.--(cont'd.)

Rabbits	LD70 2000 mg/kg		Derris powder sans rotenone	Oral	Negherbor 1959
Rabbits	LD50 >1000- 3000 mg/kg			Acute dermal	Kenaga and Allison 1971
Mice	LD100 10 mg/kg		Rotenone in ethylene glycol	Acute ip	Shimkin and Anderson 1936
	LD80 5.0 mg/kg			Acute ip	Shimkin and Anderson 1936
White mouse	LD50 350mg/kg			Acute oral	Kenaga and Allison 1971
Rats	5.0 mg/kg	Daily	Rotenone	In diet. Caused necrosis of the liver	Cohen et al. 1960
	0.25 mg/kg	Daily	Rotenone	Chronic oral toxicity. Fed 2 years	McKee and Wolf 1971
	31.2 mg/ℓ	16 weeks	Rotenone	Minimum dose able to produce massive intoxication	Telemans and Dormal 1952
	LD 6.0 mg/kg		Rotenone in olive oil solution	IV	Negherbon 1959
	LD100 200 mg/kg		Rotenone in ethylene glycol	Acute oral	Skimkin and Anderson 1936
	LD100 5.0 mg/kg		Rotenone in ethylene glycol	Acute ip	Skimkin and Anderson 1936

(more)

Table 3.--(cont'd.)

Rats	LD80 100 mg/kg	Rotenone in ethylene glycol	Acute oral	Shimkin and Anderson 1936
	0.6 g/kg	Rotenone	Lethal dose	Ambrose and Haag 1937
	LD50 1.5±0.1 mℓ/kg	Pro-Noxfish	Acute oral	Brooks 1961
Male rats	LD50 170 mg/kg	Cubé (4.7% rotenone) in aqueous solution		Haag and Taliaferro 1940
	245 mg/kg	Cubé (4.7% rotenone) in oil suspension		Haag and Taliaferro 1940
Rats	LD50 25 mg/kg	Rotenone dissolved in in olive oil	More toxic in oil than as solid or in suspension	Lightbody and Mathews 1936
Rats	LD70 100 mg/kg	9.6% rotenone derris powder	Acute oral	Haag 1937
	LD70 200 mg/kg	4.7% rotenone cubé powder	Acute oral	Haag 1937
	LD50 132 mg/kg	Crystalline rotenone	Acute oral	Adlung 1957, Lehman 1951, Christensen 1973
	1500 mg/kg	Derris	Acute oral	Lehman 1951
	LD50 1000 mg/kg	Solid rotenone (particle size 60 mesh)	Acute oral	Lightbody and Mathews 1936

(more)

Table 3.--(cont'd.)

Rats	150 mg/kg	Solid rotenone (particle size 100 mesh)	Acute oral	Lightbody and Mathews 1936
	LD50 0.2 mg/kg	Rotenone in acetone or ethanol	Acute IV	Santi and Toth 1965
	LD50 1.60 mg/kg	Rotenone in acetone or ethanol	Acute ip	Santi and Toth 1965
	LD50 60 mg/kg	Rotenone in acetone or ethanol	Acute oral	Santi and Toth 1965
Male albino rats	130 mg/ℓ	Rotenone	Acute oral	Cohen et al. 1960
Rats	LD50 1.5 cc/kg	Chem-Fish Synergized	Acute oral	Blue Spruce Co. 1973
	50 µℓ/ℓ	Chem-Fish Regular	Chronic oral, nontoxic	Blue Spruce Co. 1973
	25 mg/ℓ		Chronic oral with no effect level	Kenaga and Allison 1971
	100 µℓ/ℓ	Chem-Fish Synergized	Nontoxic	Blue Spruce Co. 1973
Male albino rats (100-135 gm)	LD50 2.5 g/kg	Dihydrorotenone		Ambrose et al. 1953
	1.5 g/kg	Dihydrorotenone	Nontoxic	Ambrose et al. 1953
Rats	LD70 700 mg/kg	Crystalline rotenone Cubé powder Derris powder	Acute oral	Haag 1937

(more)

Table 3.--(cont'd.)

Guinea pigs	LD70 60 mg/kg	Crystalline rotenone Cube powder Derris powder	Acute oral	Haag 1937
	LD70 75 mg/kg	9.6% rotenone derris powder	Acute oral	Haag 1937
	LD70 200 mg/kg	4.7% rotenone cube powder	Acute oral	Haag 1937
	150 mg/kg	Rotenone	In food, fetuses of pregnant guinea pigs either failed to survive until birth or died shortly thereafter	Cohen et al. 1960
	15 mg/kg	Rotenone in ethylene glycol	Minimum lethal dose ip	Shimkin and Anderson 1936
	LD80 200 mg/kg	Rotenone in ethylene glycol	Acute oral	Shimkin and Anderson 1936
	0.6 g/kg	Rotenone	Lethal dose	Ambrose and Haag 1937
	LD50 2 mg/kg	Rotenone	Intraperitoneally	Cohen et al. 1960
	60 mg/kg	Rotenone	Acute oral	Cohen et al. 1960
	LD50 0.055-0.060 mg/g	100% rotenone	Oral toxicity	Cutkomp 1943a
	LD50 0.1-0.2 mg/g	100% rotenone	Oral toxicity	Cutkomp 1943a

(more)

Table 3.--(cont'd.)

Guinea pigs	LD50 12 mg/kg		Rotenone dissolved in olive oil	More toxic in oil than as solid or in suspension	Lightbody and Mathews 1936
	LD50 200 mg/kg		Rotenone in ethyl glycol	Oral	Spector 1956
	LD50 75 mg/kg		Rotenone in ethyl glycol	Oral	Spector 1956
	LD50 16 mg/kg		Rotenone in ethyl glycol	SC	Spector 1956
	LD50 7 mg/kg		Rotenone in ethyl glycol	IM	Spector 1956
	LD50 2 mg/kg		Rotenone in ethyl glycol	IP	Haag 1937
	LD50 20 mg/kg		Rotenone in ethylene glycol	IP	Haag 1937
Dogs	2 g/kg			Nonlethal oral dose	Haag 1931
	5 mg/kg	Daily for 1 month		Capsules produced slight decrease in weight gain and fatty changes in liver and kidneys	Haag 1931
	0.65 mg/kg			Lethal IV dose	Haag 1931

(more)

Table 3.--(cont'd.)

Dogs	3 g/kg	Rotenone	Acute oral	Cohen et al. 1960
	200 mg/kg		Did not kill a dog	Oliver and Roe 1957
	>400 mg/ℓ		Chronic oral with no effect level	Kenaga and Allison 1971
Dogs and cat	LD50 0.65 mg/kg	Rotenone	Intravenously	Cohen et al. 1960
Pigs	3.7 mg/kg	Rotenone	Oral administration killed within 4 hours	Oliver and Roe 1957
Lamb	50 µℓ/ℓ	Chem-Fish Synergized Pro-Noxfish	Chronic oral Nontoxic	Blue Spruce Co. 1973 Brooks 1961
Cattle	18 mg/kg	Rotenone	Slightly affected from dose in feed	Oliver and Roe 1957

D. Human safety

Rotenone is relatively free of hazards in normal use, because of the low percentage used in formulations, unstable nature of rotenone, and its low solubility in water. No human fatalities have been reported (Gleason et al. 1969, Lehman 1948). A reasonable estimate for oral toxicity to man is 0.3 to 0.5 g/kg (Gleason et al. 1969). Stecher (1968) estimated the lethal oral dose to be 0.2 g/kg and Tilemans and Dormal (1952) figured the oral LD50 for man to be 2.85 g/kg. Acute intoxication causes stimulation of respiration followed by degression and convulsions. Chronic intoxification is nonexistent (Tilemans and Dormal 1952).

Rotenone is noninjurious to humans who eat fish killed by it. Cohen et al. (1960) note that because of the peculiar injury of rotenone to the gills of fish, it is doubtful that any significant amount would enter the fleshy part of the fish. However, Meyer (1966) observed that continuing sublethal doses of rotenone in the human diet may cause fatty changes in the liver and kidneys, as well as allergies and pterygiums. The degree of skin irritation from rotenone to man is moderate. No quantities are dangerous either in single or multiple doses (Tilemans and Dormal 1952). Buccal mucous membranes become numb in man when exposed to sufficient doses (Ambrose and Haag 1936).

Pintler and Johnson (1958) observed that cubé powder caused headaches, sore throats, sores on mucous membranes, eye irritation, rashes on skin, and loss of appetite. They concluded that wettable powder and emulsifiable formulations would be safer to use.

V. Residues

Methods exist for determining the amount of rotenone in water, milk, vegetables and fruit, and fish. The method for detection in fish may be quantitative, but this has not been proven at present.

A colorimetric method was adapted by Post (1955) for determining a quantitative estimate of rotenone in water. The reagents required are chloroform, 10% thymol in chloroform, and hydrochloric-nitric acid reagent. The method is practical for field use, giving good results in natural water up to 35,000 mg/ℓ of total solids. Another colorimetric method, using methylene chloride, sodium chloride, acetone, concentrated sulfuric acid, and sodium nitrate solution was developed by Saul B. Saila at Cornell University to determine a qualitative test for rotenone in natural water (Barrows 1957).

Because the colorimetric methods are time-consuming and unreliable, Cohen et al. (1960) and Loeb and Engstrom-Heg (1971) decided to use a fish bioassay method to determine the concentration of very dilute rotenone solutions in water. The technique consisted of establishing a standard curve of the concentration of chemical to the time required to obtain a given response from an experimental animal under standard conditions. The concentration-response curves obtained through laboratory experiments have proven to be reliable indicators of residual rotenone concentrations, particularly within the 0.05 to 0.3 mg/ℓ concentration range.

There are methods for detection and measurement of rotenone residues in milk, in vegetables and fruits, and in fish.

Matthysse and Lisk (1968) found no detectable residues in cow's milk following a spraying or misting of whole dairy herds with rotenone suspensions or emulsions. Sensitivities of the method ranged from 0.05 to 0.11 mg/ℓ calculated on a whole-milk basis.

Although Gross and Smith (1934), Goodhue (1936), and others had developed and modified a very specific and sensitive colorimetric method for rotenone, the color reactions are suited only for the investigation of drugs and dressings which have a high rotenone content and contain only a few noninterfering substances. In the microgram range and especially in the presence of a large balance of different components it fails completely.

Ultraviolet spectrophotometric (Rund 1959 and others), infrared (Rund and Hambleton 1965 and others), and fluorometric methods (Yojima 1962), were developed for detection and determination of rotenone. Unfortunately these procedures are so susceptible to interference that in many cases fail even with repeated separations over silica gel and exchange columns.

Paper chromatographic methodology for rotenone was developed by Chen and Tsai (1955).

Because colorimetric, UV spectrophotometric, IR spectra, fluorometric, and paper chromatographic techniques are not sensitive in the mg/ℓ range, Kroller (1969) sought and developed a method to detect rotenone residues in fruits and vegetables. The method for analysis included extraction with methylene chloride, cleanup by thin-layer

chromatography, and colorimetric analysis of the TLC region containing rotenone. For a quantity of 100 grams of test material, the author found the limit of detection is 0.05 mg/ℓ of rotenone, with an error tolerance of ±10 to 15%.

A method of extracting and identifying rotenone in fish poisoned by rotenone was developed by Sinnappa and Thuan (1971). Acetone was used for extraction, and thin-layer chromatography was used for detection of rotenone. The identification of rotenone was supplemented with comparison studies of the alkaline hydrolysis products of the extract from fish and that of thin-layer chromatography to make it conclusive. The authors claim the TLC method can detect a minimum of 1 µg of rotenone. However, they fail to specify the concentration of derris root resin to which fish were exposed or how long they were exposed to the toxicant. Also, they did not attempt to quantitate the concentration of the rotenone residue found in the fish.

VI. Application Methodology

The historical development of methods used in reclamations can be found in reports by Siegler and Pillsbury 1946, Krumholz 1948, Solman 1950, Hayes and Livingstone 1955, Hooper 1955, Zilliox and Pfeiffer 1956, Prévost 1960, and Meyer 1966.

The development of formulations progressed from powdered derris root, to wettable powder, to emulsifiable formulations (Solman 1950). This advance was aided by better methods and equipment for distribution. The early methodology involved using motor boats to dispense

the material, dynamite to facilitate distribution and coarse meshed bags to release the material (Siegler and Pillsbury 1946). In the late 1930's pumps were employed (Leonard 1939). In the 1940's weighted hoses were used to distribute rotenone into deep lakes (Wales 1947). Then aircraft was used in 1947 on ponds and lakes (Siegler and Pillsbury 1949).

Liquid formulations can be poured into the propeller wash of an outboard motor, sprayed out or pumped into deep water using a centrifugal pump, or sprayed from shore, boat, airplane, or helicopter (Lennon et al. 1970). Weighted hoses were used to get good distribution in deep water (Hooper 1955). Later, Bone (1970) found that rotenone can be dispensed into deep water quickly and economically using a compressed CO_2 system.

Cornell University (1968) used a recording fathometer to trace the dispersion of rotenone in a pond. They found that surface applications were a more effective way to insure complete exposure of fish even in deeper waters compared to pumping because the injected rotenone remained in a narrow band with no appreciable lateral dispersal.

A practical on-site bioassay has been developed by Burress (1972) to eliminate under- or overdosing. It provides a way to determine the concentration of toxicant which is efficacious for a specific purpose in a given body of water, considering all the pertinent factors.

At times it is desirable to revive and save fish after they have come in contact with rotenone. Bouck and Ball (1965) experimented

with methylene blue to revived fish exposed to rotenone. Surfacing fish were placed in 15-quart pails containing 2 milliliters of 5% methylene blue in water and then into tanks with methylene blue. Only 11 of 270 fish died. On two other occasions, the authors found methylene blue to be effective in reviving fish. However, methylene blue should be used with care because it can kill higher aquatic plants, encourage bacterial growth on fish, and stain boats and docks.

Tate et al. (1965) found that fish can be recovered live from ponds by applying 0.5 mg/ℓ of rotenone and then picking up the fish as soon as they surface. This technique is less expensive and more efficient than seining in most nondrainable ponds.

Streams were not treated as early as lakes and ponds because the formulations and equipment were not available in the 1930's and 1940's which allowed treatment of streams. Then, electrofishing gear came into wide use in the 1950's to aid in assessing imbalances in fish populations, counteraction of a toxicant came into use in the mid-1950's, formulations became available which did not repel fish as much as the early formulations, and effective contact times were understood only in the 1950's. Only in the late 1950's were method and material for streams developed (Lennon et al. 1970, Lawrence 1956, Jackson 1957).

McCoy and Ratledge (1967) and Slifer (1970 developed formulas and suggested techniques for use of rotenone and detoxifiers in streams.

Fernholz and Slifer (1967) developed procedures for treating soft water trout streams. Gilderhus (1972) determined the effective contact time (concentration plus duration of exposure) necessary to produce kills of target fish with rotenone in flowing streams. Berry and Larkin (1954) experimented with placement of application stations in slow and fast streams. The importance of barrier dams in preventing reinfestation of treated waters by rough fish was appreciated and used in many stream treatments (Zilliox and Pfeiffer 1956, Charles 1958, Prévost 1960, Fernholz 1966, Spitler 1970).

Schoenecker and Peterson (1965) used a dye in preliminary observations to trace the flow and velocity in 115 miles of stream. Wilkens (1957) used sodium fluorescein dye to trace the path and completeness of the distribution in streams. Lennon and Parker (1959) used a salt-resistivity technique to measure the velocity, stretchout, and dilution of a toxicant as it moves downstream. Lowman (1958, 1959) developed a metering device which permitted a constant amount of liquid toxicant to flow into the stream. Price and Haus (1963) found the liquid metering device could be attached directly to drums of rotenone.

VII. Registration Status

Twenty-nine formulations of rotenone from 18 companies are registered currently by the Environmental Protection Agency for aquatic or agricultural use.

Investigations in progress to update information on rotenone to maintain the current registration include short- and long-term effects of rotenone to invertebrates in the laboratory and the field, flow-through toxicity tests on fish, counteraction, and factors influencing inactivation and degradation of rotenone.

Rotenone may qualify as a minor-use compound because it is applied infrequently, or only once, in small quantities to selected and discrete bodies of water.

When rotenone is used as an insecticide, it has been exempt from requirement of tolerance when applied in agricultural crops, but not when used on livestock or poultry (Anonymous 1967).

REFERENCES CITED

Adlung, K. G. 1957. Zur Toxizität insektizider und akarizider Wirkstoffe für Fische. Die Naturwissenschaften 44:471-472.

Alikunhi, K. H. 1956. Fish culture techniques in India, p. 63-73. In Ministry for Agriculture, Progress of Fisheries Development in India. Cuttack, Ministry for Agriculture.

Almquist, E. 1959. Observations on the effect of rotenone emulsives on fish food organisms. Institute of Fresh Water Research, Drottningholm, Report No. 40:146-160.

Ambrose, A. M., and H. B. Haag. 1936. Toxicological study of derris. Industrial and Engineering Chemistry 28(7):815-821.

Ambrose, A. M., and H. B. Haag. 1937. Toxicological studies of derris - comparative toxicity and elimination of some constituents of derris. Industrial and Engineering Chemistry 29:429-431.

Ambrose, A. M., and H. B. Haag. 1938. Toxicological studies of derris. Chronic toxicity of derris. Industrial and Engineering Chemistry 30(5):592-595.

Ambrose, A. M., H. E. Christensen, and L. J. Rather. 1953. Toxicological and pharmacological studies on dihydrorotenone. Journal of the American Pharmaceutical Association 42(6):364-366.

Ambrose, A. M., F. Deeds, and J. B. McNaught. 1942. Chronic toxicity of derris. Industrial and Engineering Chemistry 34(6):684-689.

Anderson, D. 1970. Summary of fish toxicant use in Minnesota. Minnesota Department of Conservation, Division of Game and Fish, Section of Fisheries, July 28, 1970. 7 p.

Anderson, J. K. 1966. Selective shad kill. Kentucky Progress Report for investigations projects as required by the Federal Aid to Fish and Wildlife Restoration Acts, Project No. F-26-R-1, Job No. 1-F: 44-50, plus Appendices 1-A -.5-A.

Anderson, J. M. 1950. Some aquatic vegetation changes following fish removal. Journal of Wildlife Management 14(2):206-209.

Anderson, R. S. 1970. Effects of rotenone on zooplankton communities and a study of their recovery patterns in two mountain lakes in Alberta. Journal of the Fisheries Research Board of Canada 27(8): 1335-1356.

Anjaneyulu, A. S. R., and L. R. Row. 1966. Insecticidal activity of pyrones, p. 47-57. In Symposium on synthesis of heterocyclic compounds of physiological interest, Osmania University, India, December 1964.

Anonymous. 1969. Requiem for rotenone in poultry and cattle sprays. Agricultural Chemicals 24(9):16-17.

Anonymous. 1971. A fish farmer's Rx for pondwater problems. The American Fish Farmer 2(9):6-9.

Bailey, R. M., J. E. Fitch, E. S. Herald, E. A. Lachner, C. C. Lindsey, C. R. Robins, and W. B. Scott. 1970. A list of common and scientific names of fishes from the United States and Canada. 3rd ed. American Fisheries Society, Washington, D.C., Special Publication No. 6. 150 p.

Baker, P. H., and A. J. Cordone. 1969. Distribution, size composition, and relative abundance of the Piute sculpin, *Cottus beldingii* Eigenmann and Eigenmann in Lake Tahoe. California Fish and Game 55(4):285-297.

Ball, R. C. 1948. A summary of experiments in Michigan lakes on the elimination of fish populations with rotenone, 1934-1942. Transactions of the American Fisheries Society 75(1945):139-146.

Ball, R. C., and D. W. Hayne. 1952. Effects of the removal of the fish population on the fish-food organisms of a lake. Ecology 33(1):41-48.

Barrows, M. B. 1939. Elimination of yellow perch from a lake by use of derris root. Journal of Wildlife Management 3(2):131-133.

Barrows, P. T. 1957. Notes on fish toxicants. Colorado Department of Game and Fish, Special Purpose Report No. 50:1-21.

Bassett, H. M. 1956. The economic evaluation of several fish toxicants. Quarterly Report of the Colorado Cooperative Fishery Research Unit Vol. 2(3&4):6-16.

Beckman, W. C. 1941. Increased growth rate of rock bass, *Ambloplites rupestris* (Rafinesque), following reduction in the density of the population. Transactions of the American Fisheries Society 70 (1940):143-148.

Bennett, G. W. 1971. Management of lakes and ponds. 2nd edition. Van Nostrand Reinhold Company, New York. 375 p.

Berry, G., and P. A. Larkin. 1954. Field trials with "Fish-Tox" in streams. The Canadian Fish Culturist No. 15:10-15.

Bhimachar, B. S., and S. D. Tripathi. 1967. A review of culture fisheries activities in India. FAO Fisheries Reports 2(44):1-33.

Bhuyan, B. R. 1967. Eradication of unwanted fish from ponds by using indigenous plant fish poisons. Science and Culture 32(2):82-83.

Binns, N. A. 1967. Effects of rotenone treatment on the fauna of the Green River, Wyoming. Wyoming Game and Fish Commission, Fisheries Research Bulletin, No. 1:1-114.

Bisbee, L. E. 1968. Lower Owyhee River. Oregon State Game Commission, Federal Aid in Fish Restoration, F-2-D-33, Job No. 25. 30 p.

Blue Spruce Company. 1973. Toxicological and ecological information on the effects of rotenone products. Blue Spruce Company, Basking Ridge, N.J. 11 p.

Boccardy, J. A., and E. L. Cooper. 1961. Resilience of a fish population in a stream in Pennsylvania. The Progressive Fish-Culturist 23(1):26-29.

Boccardy, J. A., and E. L. Cooper. 1963. The use of rotenone and electrofishing in surveying small streams. Transactions of the American Fisheries Society 92(3):307-310.

Bone, J. N. 1970. A method of dispensing rotenone emulsions. British Columbia Fish and Wildlife Branch, Fisheries Management Report No. 62:1-3.

Bonetto, A. A., C. Pignalberi, and E. Cordiviola. 1962. Sobre algunos ensayos preliminares relativos al empleo de la rotenona en acuicultura y control de algunas especies regionales. Direccion General de Recursos Naturales, Provincia de Santa Fe, Publicacion Tecnica No. 6:263-269.

Bonn, E. W., and L. R. Holbert. 1961. Some effects of rotenone products on municipal water supplies. Transactions of the American Fisheries Society 90(3):287-297.

Bouck, G. R., and R. C. Ball. 1965. The use of methylene blue to revive warmwater fish poisoned by rotenone. The Progressive Fish-Culturist 27(3):161-162.

Bowers, C. C. 1955. Selective poisoning of gizzard shad with rotenone. The Progressive Fish-Culturist 17(3):134-135.

Bridges, W. R., and O. B. Cope. 1965. The relative toxicities of similar formulations of pyrethrum and rotenone to fish and immature stoneflies. Pyrethrum Post 8(1):3-5.

Brooks, I. C. 1961. Research methods and findings on fish toxicants and their application. S. B. Penick & Co. Research Department, New York, N.Y. 10 p.

Brooks, I. C., and R. W. Price. 1961. Studies on the chronic toxicity of Pro-Noxfish, a proprietary synergized rotenone fish toxicant. Toxicology and Applied Pharmacology 3(1):49-56.

Brown, C. J. D., and R. C. Ball. 1943. An experiment in the use of derris root (rotenone) on the fish and fish-food organisms of Third Sister Lake. Transactions of the American Fisheries Society 72(1942):267-284.

Brown, R. T. 1973. Toxicity of antimycin and rotenone to crawfish, *Procambarus* spp., and the possible use of antimycin as a fish poison in crawfish ponds. M.S. Thesis, Louisiana State University, August 1973. 49 p.

Brynildson, C. 1970. Results of chemical fish eradication of Cox Hollow Lake, Iowa County. Wisconsin Department of Natural Resources, Division of Fish, Game and Enforcement, Bureau of Fish Management, Management Report No. 31:1-8.

Buckingham, D. E. 1930. Action on rotenone upon mammals when taken by mouth - preliminary report. Industrial and Engineering Chemistry 22(10);1133-1134.

Burdick, G. E., H. J. Dean, and E. J. Harris. 1955. Toxicity of emulsifiable rotenone to various species of fish. New York Fish and Game Journal 2(1):36-67.

Burdick, G. E., H. J. Dean, and E. J. Harris. 1956. Toxicity of emulsifiable rotenone to yellow perch. New York Fish and Game Journal 3(1):75-80.

Burgos, J., and E. R. Redfearn. 1965. The inhibition of mitochondrial reduced nicotinamide-adenine dinucleotide oxidation by rotenoids. Biochimica et biophysica Acta 110:475-483.

Burress, R. M. 1972. Resume on on-site bioassay experiments. U.S. Fish and Wildlife Service, Southeastern Fish Control Laboratory, Warm Springs, GA, typewritten, July 1972. 66 p.

Butler, P. A., A. J. Wilson, and A. J. Rick. 1960. Effect of pesticides on oysters. Proceedings of the National Shellfisheries Association 51:23-32.

Byrd, I. B., and J. H. Crance. 1965. Fourteen years of management and fishing success in Alabama's state-owned public fishing lakes. Transactions of the American Fisheries Society 94(2):129-134.

Catt, J., and A. W. H. Needler. 1946. Restoration of an abundant trout population by poisoning introduced yellow perch and restocking. The Canadian Fish Culturist 1(1):2-4.

Chambers, R., G. Cameron, and M. J. Kopac. 1943. Neoplasm studies XI. The effects in tissue culture of N,N,N',N'-tetramethyl-o-phenylenediamine and other compounds on malignant lymph nodes. Cancer Research 3:293-295.

Chance, C. J., and L. F. Miller. 1952. Fish sampling with rotenone in TVA reservoirs. Journal of the Tennessee Academy of Science 27(3):214-222.

Charles, J. R. 1958. Final report on population manipulation studies in three Kentucky streams. Proceedings of the Southeastern Association of Game and Fish Commissioners 11(1957):155-185.

Chen, Y.-L., and C.-S. Tsai. 1955. The paper chromatography of rotenone. Journal of the Taiwan Pharmaceutical Association 7(1): 31-43.

Cheng, H.-M., I. Yamamoto, and J. E. Casida. 1972. Rotenone photodecomposition. Journal of Agricultural and Food Chemistry 20(4): 850-856.

Christensen, H. E. (ed.). 1973. The toxic substances list. 1973 edition. U.S. Department of Health, Education, and Welfare, National Institute for Occupational Safety and Health, Rockville, Maryland. 1001 p.

Claffey, F. J., and J. E. Ruck. 1967. The effect of rotenone on certain fish food organisms. Proceedings of the Southeastern Association of Game and Fish Commissioners 20(1966):278-283.

Clark, C. F. 1964. Bluegill control experiment in Hamler Lake, Ohio. Ohio Department of Natural Resources, Division of Wildlife, Publication W-335:1-5.

Clark, E. P. 1929. The occurrence of rotenone in the Peruvian fish poison "cubé". Science 70:478-489.

Clemens, H. P., and M. Martin. 1953. Effectiveness of rotenone in pond reclamation. Transactions of the American Fisheries Society 82(1952):166-177.

Cohen, J. M., L. J. Kamphake, A. E. Lemke, C. Henderson, and R. L. Woodward. 1960. Effect of fish poisons on water supplies. Part 1. Removal of toxic materials. Journal American Water Works Association 52(12):1551-1566.

Cohen, J. M., Q. H. Pickering, R. L. Woodward, and W. van Heuvelen. 1961a. Effect of fish poisons on water supplies. Part 3. Field study at Dickinson. Journal American Water Works Association 53 (2):233-246.

Cohen, J. M., G. A. Rourke, and R. L. Woodward. 1961b. Effect of poisons on water supplies. Part 2. Odor problems. Journal American Water Works Association 53(1):49-62.

Cook, S. F., Jr., and R. L. Moore. 1969. The effects of rotenone treatment on the insect fauna of a California stream. Transactions of the American Fisheries Society 98(3):539-544.

Cope, O. B. 1965. Sport fishery investigations, p. 51-63. In Effects of pesticides on fish and wildlife, 1964 research findings of the Fish and Wildlife Service. U.S. Fish and Wildlife Service, Circular No. 226.

Cornell University. Department of Conservation. 1968. Rotenone detection with fathometer. Cornell University, Department of Conservation, Brandon Park, Fish Management Report 1968:44-45.

Crass, R. S. 1971. Revival of rotenone-treated fish. FAO Aquaculture Bulletin 3(3):5.

(In press).

Cumming, K. B. /History of fish toxicants in the United States. In Symposium on rehabilitation of fish populations with fish toxicants. North Central Division, American Fisheries Society.

Cushing, C. E., Jr., and J. R. Olive. 1957. Effects of toxaphene and rotenone upon the macroscopic bottom fauna of two northern Colorado reservoirs. Transactions of the American Fisheries Society 86(1956):294-301.

Cutkomp, L. K. 1943a. Toxicity of rotenone to animals. Soap and Sanitary Chemicals 19(10):107-123.

Cutkomp, L. K. 1943b. Toxicity of rotenone and derris extract administered orally to birds. Journal of Pharmacology and Experimental Therapeutics 77:238-246.

Danneel, R. 1933. Die giftwirkung des rotenons und seiner derivate auf fische. (II. Der angriffspunkt der gifte). Zeitschrift fuer Verleichende Physiologie 18:524-535.

Davis, T. R. A., A. W. Burg, J. L. Neumeyer, K. M. Butters, and B. D. Wadler. 1970. Water quality criteria data book, Vol. 1 - Organic chemical pollution of fresh water. Arthur D. Little, Inc., Cambridge, Massachusets. 379 p.

Dawson, V. K. (In press). Counteracting chemicals used in fishery operations: current technology and research. In Symposium on rehabilitation of fish populations with fish toxicants. North Central Division, American Fisheries Society.

Dawson, V. K., and L. L. Marking. (In review) Removal of toxic chemicals from water with activated carbon. Transactions of the American Fisheries Society.

Dexter, W. D. 1965. Some effects of rotenone treatment on the fauna of the Green River, Wyoming. Western Association of State Game and Fish Commissioners 45:193-197.

Dietz, E. M. C., and K. C. Jurgens. 1963. An evaluation of selective shad control at Medina Lake, Texas. Texas Parks and Wildlife Department, Inland Fisheries Report Series No. 5:1-32.

Dobie, J., and J. B. Moyle. 1945. Report on the effects of cube root (rotenone) on the fish and fish-food organisms of Dew Lake (16-414), St. Louis County. Minnesota Bureau of Fisheries Research, Investigation Report No. 64:1-22.

Donaldson, J. R., R. W. Kiser, and P. R. Olson. 1962. The effect of rotenone on zooplankton populations in freshwater lakes. Western Association of State Game and Fish Commissioners 42: 148-156.

Emig, J. W. 1966. Bluegill sunfish, p. 375-392. In A. Calhoun (ed.). Inland fisheries management. California Department of Fish and Game, Sacramento.

Engstrom-Heg, R. 1971. Stability of rotenone emulsions. New York Fish and Game Journal 18(1):73-74.

Engstrom-Heg, R. 1972. Kinetics of rotenone-potassium permanganate reactions as applied to the protection of trout streams. New York Fish and Game Journal 19(1):47-58.

Engstrom-Heg, R., and H. A. Loeb. 1968. Chemical detoxification of rotenone: a simple case of oxidation? New York State Conservation Department, Bureau of Fish, New York Fish News No. 27:3-7.

Engstrom-Heg, R., and H. A. Loeb. 1971. Recolonization of a rotenone-treated segment of the Ten Mile River. New York Fish and Game Journal 18(2):135-138.

Ernster, L., G. Dallner, and G. F. Azzone. 1963. Differential effects of rotenone and amytal on mitochondrial electron and energy transfer. Journal of Biological Chemistry 238(3):1124-1131.

Eschmeyer, R. W. 1937. Some characteristics of a population of stunted perch. Papers of the Michigan Academy of Science, Arts, and Letters 22(1936):613-628.

Eschmeyer, R. W. 1938. Further studies of perch populations. Papers of the Michigan Academy of Science, Arts, and Letters 23(1937): 611-631.

Fabacher, D. L., and H. Chambers. 1972. Rotenone tolerance in mosquitofish. Environmental Pollution 3:139-141.

Farringer, J. E. 1972. The determination of the acute toxicity of rotenone and Bayer 73 to selected aquatic organisms. M.S. Thesis, University of Wisconsin-La Crosse, La Crosse, Wisconsin. May 1972. 32 p.

Fellton, H. L. 1940. Control of aquatic midges with notes on the biology of certain species. Journal of Economic Entomology 33 (2):252-264.

Fernholz, W. B. 1966. Doctoring a sick stream. Wisconsin Conservation Bulletin 31(4):12-13.

Fernholz, W. B., and G. E. Slifer. 1967. Procedures for chemical reclamation of soft water trout streams. Wisconsin Department of Natural Resources, Bureau of Fish Management, Management Report No. 23:1-21.

Ferrari, M., and I. Maragno. 1970. Effects of rotenone on the electrical and mechanical activity of guinea pig taenia coli. Pharmacological Research Communications 2(1):71-76.

Fischthal, J. H. 1947. Fish population of Little Granite Lake, Barron County, as determined by the fin-clip method and recovery following rotenone poisoning, with observations on the effect of rotenone on the plankton and bottom organisms. Wisconsin Conservation Department, Division of Fish Management, Investigational Report No. 601:1-15.

Fontenele, O. 1963. Eradication of piranha in inland waters. Commercial Fisheries Review 25(3):46-50.

Foye, R. E. 1964. Chemical reclamation of forty-eight ponds in Maine. The Progressive Fish-Culturist 26(4):181-185.

Frear, D. E. H. 1969. Pesticide Index. 4th ed. College Science Publishers, State College, Pennsylvania. 399 p.

Fromm, P. O., B. D. Richards, and R. C. Hunter. 1971. Effects of some insecticides and MS-222 on isolated-perfused gills of trout The Progressive Fish-Culturist 33(3):138-140.

Fukami, J.-I., T. Mitsui, K. Fukunaga, and T. Shishido. 1970. The selective toxicity of rotenone between mammal, fish and insect. p. 159-178. In R. D. O'Brien, and I. Yamamoto (ed.). Biochemical toxicology of insecticides. Symposium. Academic Press Inc., New York, N.Y.

Fukami, J.-I., T. Nakatsugawa, and T. Narahashi. 1959. The relation between chemical structure and toxicity in rotenone derivatives. Japanese Journal of Applied Entomology and Zoology 3(4):259-264.

Fukami, J.-I., T. Shishido, K. Fukunaga, and J. E. Casida. 1969. Oxidative metabolism of rotenone in mammals, fish, and insects and its relation to selective toxicity. Journal of Agricultural and Food Chemistry 17(6):1217-1226.

Fukami, J.-I., Yamamoto, and J. E. Casida. 1967. Metabolism of rotenone *in vitro* by tissue homogenates from mammals and insects. Science 155:713-716.

Gaffney, J. J. 1959. Partial rehabilitation of a section of the Clark Fork River. Montana Fish and Game Department, Investigations and Development Project 29-E-1, Annual Progress Report. 13 p.

Garrison, R. L. 1968. The toxicity of Pro-Noxfish to salmonid eggs and fry. The Progressive Fish-Culturist 30(1):35-38.

Gaskins, M. H., G. A. White, F. W. Martin, N. E. Delfel, E. G. Ruppel, and D. K. Barnes. 1972. *Tephrosia vogelii*: A source of rotenoids for insecticidal and piscicidal use. U.S. Department of Agriculture, Agricultural Research Service, Technical Bulletin No. 1445:1-38.

Gerking, S. D. 1950. A carp removal experiment at Oliver Lake, Indiana. Investigations of Indiana Lakes and Streams 3(10): 373-388.

Gersdorff, W. A. 1930. The toxicity of rotenone, isorotenone, and dihydrorotenone to goldfish. Journal of the American Chemical Society 52:5051-5056.

Gersdorff, W. A. 1933. A study of the toxicity of rotenone hydrochloride, acetylrotenone, and rotenolone using the goldfish as the test animal. Journal of the American Chemical Society 55: 1147-1152.

Gersdorff, W. A. 1934. A further study of the toxicity of derivatives of rotenone with the goldfish as the test animal. Journal of the American Chemical Society 56:979-980.

Gersdorff, W. A. 1935. The quantitative relationship between the constitution and toxicity of some rotenone derivatives. Journal of Agricultural Research 50(11):893-898.

Gersdorff, W. A. 1943. Effect of change of temperature on relative toxicity of rotenone and phenol. Journal of Agricultural Research 67(2):65-80.

Gilderhus, P. A. 1972. Exposure times necessary for antimycin and rotenone to eliminate certain freshwater fish. Journal of the Fisheries Research Board of Canada 29(2):199-202.

Gleason, M. N., R. E. Gosselin, H. C. Hodge, and R. P. Smith. 1969. Clinical toxicology of commercial products. 3rd ed. The William and Wilkins Co., Baltimore, Maryland. Section III.

Goodhue, L. D. 1936. An improvement on the Gross and Smith colorimetric method for the determination of rotenone and deguelin. Journal of the Association of Official Agricultural Chemists 19 (1):118-120.

Goodhue, L. D., and H. L. Haller. 1939. Analysis of water extract of derris and cubé. Journal of Economic Entomology 32(6):877-879.

Greenbank, J. 1941. Selective poisoning of fish. Transactions of the American Fisheries Society 70(1940):80-86.

Grice, F. 1959. Elasticity of growth of yellow perch, chain pickerel, and largemouth bass in some reclaimed Massachusetts waters. Transactions of the American Fisheries Society 88:332-335.

Gross, C. R., and C. M. Smith. 1934. Colorimetric method for determination of rotenone. Journal of the Association of Official Agricultural Chemists 17(2):336-339.

Guerra, L. V. 1966. Chemical renovation of Casa Blanca Lake. Texas Parks and Wildlife Department. Federal Aid in Fish Restoration, Job Completion Report, No. F-14-D-9, Job No. 16-a-51. 13 p.

Gunther, F. A. 1943. Effects of oxygen and sunlight on decomposition of rotenone in spray mixtures. Journal of Economic Entomology 36 (2):273-281.

Haag, H. B. 1931. Toxicological studies of *Derris elliptica* and its constituents. I. Rotenone. Journal of Pharmacology and Experimental Therapeutics 43(1):193-208.

Haag, H. B. 1937. Toxicity of rotenone. A study of the toxicity to humans and animals of rotenone-bearing plants. Soap and Sanitary Chemicals 13(1):112c, 112d, 137.

Haag, H. B., and I. Taliaferro. 1940. Toxicological studies on cubé. Journal of Pharmacology and Experimental Therapeutics. 69:13-20.

Hagen, W., Jr. 1940. Sterilizing hatchery water supplies. The use of liquid chlorine and powdered derris root. The Progressive Fish-Culturist 48:19-24.

Hamilton, H. L. 1941. The biological action of rotenone on freshwater animals. Proceedings of the Iowa Academy of Sciences 48: 467-479.

Hansen, W. H., K. J. Davis, and O. G. Fitzhugh. 1965. Chronic toxicity of cubé. Toxicology and Applied Pharmacobgy 7(4):535-542.

Haq, M. F., and J. E. Tilton. 1970. Note on the use of rotenone as a piscicide in ponds on the campus of the Bangladesh Agriculture University. B.C.S.I.R. Laboratories, Bangladesh, Scientific Researches 7(2&3):111-113.

Haque, K. A. 1971. Rotenone and its use in eradication of undesirable fish from ponds. Pakistan Journal of Scientific and Industrial Research 14(4/5):385-387.

Hayes, F. R., and D. A. Livingstone. 1955. The trout population of a Nova Scotia lake as affected by habitable water, poisoning of the shallows and stocking. Journal of the Fisheries Research Board of Canada 12(4):618-635.

Hazleton, Laboratories Incorporated. 1968. Rotenone literature search. Hazleton Laboratories Incorporated, Falls Church, Virginia. 12 p.

Henderson, S. (In press). Preliminary studies on the tolerance of the white amur, *Ctenopharyngodon idella*, to rotenone and other commonly used pond treatment chemicals. Proceedings of the Southeastern Association of Game and Fish Commissioners 27.

Hepworth, W. G., and D. L. Mitchum. 1967. Study of fish toxicants. Wyoming Job Completion Report, Project No. FW-3-R-13, Work Plan 9, Job No. 2F. 19 p.

Herrig, R. G. 1964. Lower Crooked River and tributaries rehabilitation project. Oregon State Game Commission. Federal Aid in Fish Restoration, Completion Project No. F-20-D-22, Job No. 13. 17 p.

Hester, F. E. 1959a. The tolerance of eight species of warm-water fishes to certain rotenone formulations. Proceedings of the Southeastern Association of Game and Fish Commissioners 13(1959): 121-133.

Hester, F. E. 1959b. The toxicity of Noxfish and Pro-Noxfish to eggs of common carp and fathead minnows. Proceedings of the Southeastern Association of Game and Fish Commissioners 13(1959):325-331.

Hiltibran, R. C., and M. G. Johnson. 1965. The effect of rotenone on oxygen uptake by liver mitochondria of the bluegill *Lepomis macrochirus*. Transactions of the Illinois State Academy of Science 58(2):140-143.

Hoffman, D. A., and J. R. Olive. 1961. The effects of rotenone and toxaphene upon plankton of two Colorado reservoirs. Limnology and Oceanography 6(2):219-222.

Hooper, A. D., and J. H. Crance. 1960. Use of rotenone in restoring balance to overcrowded fish populations in Alabama lakes. Transactions of the American Fisheries Society 89(4):351-357.

Hooper, F. F. 1948. The effect of derris root (rotenone) upon plankton and bottom fauna organisms of a small Minnesota lake. Proceedings of the Minnesota Academy of Science 16:29-33.

Hooper, F. F. 1955. Eradication of fish by chemical treatment. Michigan Department of Conservation, Fish Division Pamphlet No. 19:1-6.

Hooper, F. F., J. E. Williams, M. H. Patriarche, F. Kent, and J. C. Schneider. 1964. Status of lake and stream rehabilitation in the United States and Canada with recommendations for Michigan waters. Michigan Department of Conservation, Institute for Fisheries Research, Report No. 1688:1-56.

Hoover, E. E., and G. W. Morrill, Jr. 1938. Autecology of brook trout *(Salvelinus fontinalis)* in two primitive streams of northern New Hampshire. New Hampshire Fish and Game Department, Technical Circular - Brook trout No. 2:1-34.

Horgan, D. J., T. P. Singer, and J. E. Casida. 1968. Studies on the respiratory chain-linked reduced nicotinamide adenine dinucleotide dehydrogenase. XIII. Binding sites of rotenone, piericidin A and amytal in the respiratory chain. Journal of Biological Chemistry 243(4):834-843.

Houf, L. J. 1974. Effects of antimycin A and rotenone on shallow benthic pond communities in mid-Missouri. M.S. Thesis, University of Missouri, Columbia. May 1974.

Houf, L. J., and R. E. Hughey. 1973. The effect of fish toxicants antimycin A and rotenone on pond communities. Progress report. Missouri Cooperative Fishery Unit, Columbia, Missouri. April 1973. 38 p.

Howland, R. M. 1969. Interaction of antimycin A and rotenone in fish bioassays. The Progressive Fish-Culturist 31(1):33-34.

Hubbs, C. 1963. An evaluation of the use of rotenone as a means of "improving" sports fishing in the Concho River, Texas. Copeia No. 1:199-203.

Hughes, J. S. (In press). Acute toxicity of thirty chemicals to striped bass *(Morone saxatilis)*. Presented at the Western Association of State Game and Fish Commissioners in Salt Lake City, Utah, July 1973. 15 p.

Huish, M. T. 1958a. Gizzard shad removal in Deer Island Lake, Florida. Proceedings of the Southeastern Association of Game and Fish Commissioners 11(1957):312-318.

Huish, M. T. 1958b. Studies of gizzard shad reduction at Lake Beulah, Florida. Proceedings of the Southeastern Association of Game and Fish Commissioners 11(1957):66-70.

Huish, M. T. 1959. A summary of methods used during Florida's gizzard shad control experiments. Proceedings of the Southeastern Association of Game and Fish Commissioners 12(1958):178-183.

Hunn, J. B. 1957. Methods for the control of stunted panfish populations. M.S. Thesis, Michigan State University. 57 p.

Huntington, E. H. 1956. Bluewater Lake rehabilitation. New Mexico Department of Game and Fish, Job Completion Report, Federal Aid Project F-13-D. 10 p.

Huntington, E. H., and D. B. Jester. 1958. Rehabilitation of Negrito Creek in Catron County, New Mexico. New Mexico Department of Game and Fish, Federal Aid in Fish Restoration. 2 p.

Illinois Department of Conservation. 1964. Rotenone for eradication of undesirable fish in management of lakes and ponds. Illinois Department of Conservation, Division of Fisheries, Fisheries Leaflet No. 5:1-3.

Innes, J. R. M., B. M. Ulland, M. G. Valerio, L. Petrucelli, L. Fishbein, E. R. Hart, A. J. Pallotta, R. R. Bates, H. L. Falk, J. J. Gart, M. Klein, I. Mitchell, and J. Peters. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: a preliminary note. Journal of the National Cancer Institute 42 (6):1101-1114.

Ivie, G. W., and J. E. Casida. 1970. Enhancement of photoalteration of cyclodiene insecticide chemical residues by rotenone. Science 167:1620-1622.

Ivie, G. W., and J. E. Casida. 1971a. Sensitized photodecomposition and photosensitizer activity of pesticide chemicals exposed to sunlight on silica gel chromatoplates. Journal of Agricultural and Food Chemistry 19(3):405-409.

Ivie, G. W., and J. E. Casida. 1971b. Photosensitizers for the accelerated degradation of chlorinated cyclodienes and other insecticide chemicals exposed to sunlight on bean leaves. Journal of Agricultural and Food Chemistry 19(3):410-416.

Jackson, C. F. 1957. Detoxification of rotenone-treated water. New Hampshire Fish and Game Department, Technical Circular No. 14: 1-28.

Jenkins, R. M. 1956. Some results of the partial fish population removal technique in lake management. Proceedings of the Oklahoma Academy of Science 37:164-173.

Jeppson, P. W. 1963. Control of peamouth and suckers by eradication of spawning populations. The Progressive Fish-Culturist 25(3):154.

Jester, D. B. 1963. Rehabilitation of Ute Lake Watershed, New Mexico. New Mexico Department of Game and Fish, Federal Aid in Fish Restoration Act, Job Completion Report, Project F-19-D-5, Amendment No. 2. 6 p.

Jones, H. A., W. A. Gersdorff, E. L. Gooden, F. L. Campbell, and W. N. Sullivan. 1933. Loss in toxicity of deposits of rotenone and related materials exposed to light. Journal of Economic Entomology 26:451-470.

Jurgens, K. C. 1957. Selective control of gizzard shad in Lake Inks. Texas Fish and Game Commission, Job Completion Report, Project No. F-14-D-1, Job No. 16a-1. 10 p.

Keith, W. E. 1968. Turbidity control and fish population renovation on Blue Mountain Lake, Arkansas. Proceedings of the Southeastern Association of Game and Fish Commissioners 21(1967):495-505.

Kenaga, E., and W. E. Allison. 1971. Commercial and experimental organic insecticides. (1971 Revision). Reprinted from the Bulletin of the Entomological Society of American 15(2):85-148, 1969 and revised according to changes in 16(1):68, 1970.

King, J. E. 1953. Growth rate of fishes of Lake Hiwassee, Oklahoma, after two years of attempted population control. Proceedings of the Oklahoma Academy of Science 34:53-56.

King, J. E. 1954. Three years of partial fish population removal at Lake Hiwassee, Oklahoma. Proceedings of the Oklahoma Academy of Science 35:21-24.

Kinney, E. C. 1968. Rotenone in fish pond management. U.S. Bureau of Sport Fisheries and Wildlife, Leaflet No. 576:1-7.

Kirkwood, J. B. 1957. Small lake and stream investigation and management. Kentucky Department of Fish and Wildlife Resources, Division of Fisheries, Federal Aid in Fish Restoration, Project No. F-10-R-2, Job I-A. 25 p.

Kiser, R. W., J. R. Donaldson, and P. R. Olson. 1963. The effect of rotenone on zooplankton populations in freshwater lakes. Transactions of the American Fisheries Society 92(1):17-24.

Kroller, E. 1969. Untersuchungen zur Restmengen-Bestimmung von Rotenon. Deutsche Lebensmittel-Rundschau 65(2):41-45.

Krumholz, L. A. 1948. The use of rotenone in fisheries research. Journal of Wildlife Management 12:304-317.

Krumholz, L. A. 1950. Some practical considerations in the use of rotenone in fisheries research. Journal of Wildlife Management 14(4):413-424.

Larsen, K. The fish population of a peat pit as determined by rotenone poisoning. Meddelelser fra Danmarks Fiskeri-og Havundersøgelser 3(5):117-132.

Lawrence, J. M. 1956. Preliminary results on the use of potassium permanganate to counteract the effects of rotenone on fish. The Progressive Fish-Culturist 18(1):15-21.

Lehman, A. J. 1948. The toxicology of the newer agricultural chemicals. Association of Food and Drug Officials. Quarterly Bulletin 12:82-89.

Lehman, A. J. 1949. Pharmacological considerations of insecticides. Association of Food and Drug Officials. Quarterly Bulletin 13: 65-70.

Lehman, A. J. 1951. Chemicals in foods: a report to the Association of Food and Drug Officials on current developments. Part II. Pesticides. Association of Food and Drug Officials. Quarterly Bulletin 15:122-133.

Lehman, A. J. 1952. Chemicals in foods: a report to the Association of Food and Drug Officials on current developments. Part II. Pesticides. Association of Food and Drug Officials. Quarterly Bulletin 16:3-9.

Lennon, R. E. 1970. Control of freshwater fish with chemicals, p. 129-137. In Proceedings: Fourth Vertebrate Pest Conference, March 3-5, 1970, West Sacramento, California.

Lennon, R. E., and P. S. Parker. 1959. The reclamation of Indian and Abrams Creeks in Great Smoky Mountains National Park. U.S. Fish and Wildlife Service, Special Scientific Report-Fisheries, No. 306:1-22.

Lennon, R. E., J. B. Hunn, R. A. Schnick, and R. M. Burress. 1970. Reclamation of ponds, lakes, and streams with fish toxicants: a review. FAO Fisheries Technical Paper No. 100:1-99.

Leonard, J. W. 1939. Notes on the use of derris as a fish poison. Transactions of the American Fisheries Society 68(1938):269-279.

Lettré, H. 1949. Mitosegiftforschung und ihre Beziehung zu Problemen der Enzymforshcung. Ergebnisse der Enzymforschung 10:269-301.

Lightbody, H. D., and J. A. Mathews. 1936. Toxicology of rotenone. Industrial and Engineering Chemistry 28:809-811.

Lindahl, P. E., and K. E. Oberg. 1960. Mechanism of the physiological action of rotenone. Nature 187:784.

Lindahl, P. E., K. E. Oberg. 1961. The effect of rotenone on respiration and its point of attack. Experimental Cell Research 23: 228-237.

Lindgren, P. E. 1960. About the effect of rotenone upon benthonic animals in lakes. Institute of Freshwater Research. Drottningholm. Report No. 41:172-184.

Little, J. D. 1966. Reclamation of Pine Creek, Tennessee. Proceedings of the Southeastern Association of Game and Fish Commissioners 19(1965):302-315.

Liu, M., M. Siess, and P. C. Hoffman. 1970. Inhibition of the mitochondrial respiratory chain in isolated atria - a comparison of rotenone and amytal. Biochemical Pharmacology 19:197-207.

Loeb, H. A., and R. Engstrom-Heg. 1970a. Potential chemical systems for stream reclamation. New York Fish News No. 32:1-3.

Loeb, H. A., and R. Engstrom-Heg. 1970b. Poisoning of the Ten Mile River. New York Fish News No. 32:4-7.

Loeb, H. A., and R. Engstrom-Heg. 1970c. Time-dependent changes in toxicity of rotenone dispersions to trout. Toxicology and Applied Pharmacology 17(3):605-614.

Loeb, H. A., and R. Engstrom-Heg. 1971. Estimation of rotenone concentration by bio-assay. New York Fish and Game Journal 18(2): 129-134.

Lowman, F. G., Jr. 1958. Experimental selective rotenone killing of undesirable fish species in flowing streams. Texas Game and Fish Commission, Federal Aid to Fish Restoration, Job Completion Report, Project F-9-R-4, Job E-3. 2 p.

Lowman, F. G., Jr. 1959. Experimental selective rotenone killing of undesirable fish species in flowing streams (Continuation of Job E-3, Project F-9-R-5). Federal Aid to Fish Restoration, Segment Completion Report, Project F-9-R-6, Job No. E-3. 5 p.

Marking, L. L. 1972. Sensitivity of the white amur to fish toxicants. The Progressive Fish-Culturist 34(1):26.

Marking, L. L. 1973. Atlantic salmon: their responses to fishery chemicals. U.S. Department of the Interior, Fish and Wildlife Service, Fish Control Laboratory, La Crosse, Wisconsin, mimeo, October 1973. 5 p.

Marking, L. L., J. H. Chandler, and T. D. Bills. 1973. 1973 End-of-Year report. U.S. Fish and Wildlife Service, Fish Control Laboratory, La Crosse, Wisconsin, typewritten. 21 p.

Marking, L. L., T. D. Bills, and J. H. Chandler. 1974. Quarterly report - January-March 1974. U.S. Fish and Wildlife Service, Fish Control Laboratory, La Crosse, Wisconsin, typewritten. 19 p.

Mathis W., and A. Hulsey. 1959. Rough fish removal from Lake Catherine, Arkansas. Proceedings of the Southeastern Association of Game and Fish Commissioners 13(1959):197-203.

Matthysse, J. G., and D. Lisk. 1968. Residues of diazinon, coumaphos, Ciodrin, methoxychlor, and rotenone in cow's milk from treatments similar to those used for ectoparasite and fly control on dairy cattle, with notes on safety of diazinon and Ciodrin to calves. Journal of Economic Entomology 61(5):1394-1398.

McCoy, E. G., and H. M. Ratledge. 1967. Manual of stream renovation techniques. North Carolina Wildlife Resources Commission, Division of Inland Fisheries, mimeo, June 1967. 29 p.

M'Gonigle, R. H., and M. W. Smith. 1938. Cobequid hatchery - fish production in Second River, and a new method of disease control. The Progressive Fish-Culturist No. 38:5-11.

McKee, J. E., and H. W. Wolf. 1971. Water quality criteria. 2nd edition. California State Water Resources Control Board, Publication 3-A. 548 p.

Meadows, B. S. 1973. Toxicity of rotenone to some species of coarse fish and invertebrates. Journal of Fish Biology 5:155-163.

Meyer. F. A. 1963. Rough fish control. California Department of Fish and Game, Inland Fisheries Administrative Report No. 63-10: 1-22.

Meyer, F. A. 1966. Chemical control of undesirable fishes, p. 498-510. In A. Calhoun (ed.). Inland fisheries management, California Department of Fish and Game, Sacramento.

Mihajlovic, I., and V. Rajevski. 1960. Primena "Rotenona" za iskorenjivanje riblje faune u vodama gde se uvodi intenzivno gazdovanje, Ribarstvo, Jugoslavije 6:1-2.

Miller, R. B. 1950. A critique of the need and use of poisons in fisheries research and management. The Canadian Fish Culturist No. 8:30-33.

Molyneux, F. 1972. Derris--a natural pesticide. Australian Chemical Processing and Engineering 25(11):9-12.

Moorman, R. B., and C. E. Ruhr. 1951. Suggestions for improving the collection of fish with rotenone. The Progressive Fish-Culturist 13(3):149-152.

Nebeker, A. V., and A. R. Gaufin. 1964. Bioassays to determine pesticide toxicity to the amphipod crustacean, *Gammarus lacustris*. Utah Academy of Sciences. Proceedings 41(1):64-67.

Negherbon, W. O. 1959. Handbook of toxicology. Vol. III: Insecticides. W. B. Saunders Co., Philadelphia and London. 854 p.

Nikol'skii, G. V. 1954. Special ichthyology. 2nd ed. Sovetskaya nauka, Moskva, U.S.S.R. (English translation Israel Program for Scientific Translations No. 233, 1961. 538 p.)

Oberg, K.-E. 1959. The structure of gill epithelium and the circulation in gills of fishes poisoned with rotenone. Arkiv för Zoologi 12(25):383-386.

Oberg, K.-E. 1964. The inhibition of the respiration of brain mitochondria of rotenone-poisoned fish. Experimental Cell Research 36(2):407-410.

Oberg, K.-E. 1967a. On the principal way of attack of rotenone in fish. Arkiv för Zoologi 18(11):217-220.

Oberg, K.-E. 1967b. The reversibility of the respiratory inhibition in gills and the ultrastructural changes in chloride cells from the rotenone-poisoned marine teleost, *Gadus callarias* L. Experimental Cell Research 45:590-602.

Oberg, K.-E. 1967c. On the mode of action of rotenone upon the cell respiration and the structural pattern of the chloride cells of the gills of the rotenone-poisoned marine teleost *Gadus callarias* L. Acta Universitatis Upsaliensis 85:1-17.

O'Brien, R. D. 1967. Insecticides: action and metabolism. Academic Press, N.Y. 332 p.

Oglesby, L. C. 1964. Mortality of a freshwater polychaete, *Nereis limnicola* Johnson, attributed to rotenone. California Fish and Game 50(4):268-270.

Oliver, W. T., and C. K. Roe. 1957. Rotenone poisoning of swine. Journal of the American Veterinary Medical Association 130:410-411.

Olson, L. E., and L. L. Marking. (In review). Toxicity for four toxicants to green eggs of salmonids. Transactions of the American Fisheries Society.

Orn, B. 1962. Quantitative analysis of technical rotenone preparations. Sodra Sver. Fisk For, 1961-1962:26-38. (Water Pollution Abstracts 39(11):374, 1966).

Palmer, G., D. J. Horgan, H. Tisdale, T. P. Singer, and H. Beinert. 1968. Studies on the respiratory chain-linked reduced nicotinamide adenine dinucleotide dehydrogenase. XIV. Location of the sites of inhibition of rotenone, barbiturates, and piericidin by means of electron paramagnetic resonance spectroscopy. Journal of Biological Chemistry 243(4):844-847.

Penick and Co., S. B. 1962. I. Noxfish. II. Pro-Noxfish. Application for registration of economic poisons, August 7, 1962. S.B. Penick & Co., N.Y. Exhibits A-T.

Pennak, R. W. 1953. Fresh-water invertebrates of the United States. The Ronald Press Company, New York. 769 p.

Pimentel, D. 1971. Ecological effects of pesticides on non-target species. Executive Office of the President, Office of Science and Technology, Washington, D.C. 220 p.

Pintler, H. E., and W. C. Johnson. 1958. Chemical control of rough fish in the Russian River drainage, California. California Fish and Game 44(2):91-124.

Post, G. 1955. A simple chemical test for rotenone in water. The Progressive Fish-Culturist 17(4):190-191.

Post, G. 1958. Time versus water temperature in rotenone dissipation. Proceedings of the Western Association of Game and Fish Commissioners, 38:279-284.

Power, G. 1966. Analysis of the movement and growth of juvenile brook trout *(Salvelinus fontinalis)* from rotenone collections taken in the Nabisipi River and vicinity, Quebec. The Canadian Field-Naturalist 80:213-223.

Power, G., and J. R. Colman. 1967. Winter rotenoning as a means of collecting and studying trout in an ice and snow covered Canadian stream. Transactions of the American Fisheries Society 96(2): 222-223.

Prévost, G. 1960. Use of fish toxicants in the Province of Quebec. The Canadian Fish Culturist No. 28:13-35.

Price, R. W., and D. R. Calsetta. 1957. Pro-Noxfish. A new synergized rotenone formulation for fish control. Proceedings of the Southeastern Association of Game and Fish Commissioners 10(1956): 68-75.

Price, R. W., and J. B. Haus. 1963. Aids for stream reclamation. The Progressive Fish-Culturist 25(1):37-39.

Rao, K. V., and S. P. S. Chauhan. 1971. Teratogenic effects of rotenone on the early development of chick embryos *in vitro*. Teratology 4(2)191-197.

Regan, D. M. 1961. Rehabilitation of Caballo Lake and the Rio Grande River between Caballo Lake and the Elephant Butte Dam. New Mexico Department of Game and Fish, Federal Aid in Fish Restoration, Job Completion Report, Project No. F-19-D-3, Job No. 16-D. 9 p.

Reynolds, H. J., and J. V. Barry. 1951. Scrap fish control with toxicants. Standard Supply Distributors, Wenatchee, Washington. 34 p.

Richard, J. B. 1962. Tests on thermocline penetration by several rotenone products. The Progressive Fish-Culturist 24(4):177-181.

Riel, A. D. 1965. The control of an overpopulation of yellow perch in Bow Lake, Strafford, New Hampshire. The Progressive Fish-Culturist 27(1):37-41.

Roark, R. C. 1932. A digest of the literature of Derris (Deguelia) species used as insecticides, 1747-1931. United States Department of Agriculture, Miscellaneous Publication No. 120:1-78.

Rothschild, L. 1961. A classification of living animals. John Wiley and Sons Inc., New York. 106 p.

Rounsefell, G. A., and W. H. Everhart. 1953. Fishery science, its methods and applications. John Wiley and Sons, Inc., New York. 444 p.

Rowe-Rowe, D. T. 1971. Rotenone tolerances of some freshwater fishes of Natal. The Progressive Fish-Culturist 33(4):206-209.

Rund, R. C. 1959. Report on rotenone. Journal of the Association of Official Agricultural Chemists 42(1):96-100.

Rund, R. C., and L. G. Hambleton. 1965. Collaborative study of rotenone methods. Journal of the Association of Official Agricultural Chemists 48(3):566-569.

Saila, S. B. 1954. Bio-assay procedures for the evaluation of fish toxicants with particular reference to rotenone. Transactions of the American Fisheries Society 83(1953):104-114.

Sanders, H. O. 1969. Toxicity of pesticides to the crustacean *Gammarus lacustris*. U.S. Bureau of Sport Fisheries and Wildlife, Technical Paper No. 25:1-18.

Sanders, H. O., and O. B. Cope. 1966. Toxicities of several pesticides to two species of cladocerans. Transactions of the American Fisheries Society 95(2):165-169.

Sanders, H. O., and O. B. Cope. 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnology and Oceanography 13(1):112-117.

Sandoz, O. 1959. Changes in the fish population of Lake Murray following the reduction of gizzard shad numbers. Proceedings of the Oklahoma Academy of Science 37(1956):174-181.

Santi, R., and C. E. Toth. 1965. Toxicology of rotenone. Farmaco - Ed. Sc. 20(4):270-279.

Santi, R., M. Ferrari, and E. Toth. 1966. Pharmacological properties of rotenone. Farmaco - Ed. Sci. 21(10);689-703.

Sayre, R. C. 1969. Powder River rehabilitation. Use of Fintrol-5 for fish eradication. Oregon State Game Commission, Federal Aid in Fish Restoration, Project F-80-R-1, Job No. 26. 47 p.

Scheuring, L., and O. Heuschmann. 1935. Ueber die giftwirkung von derrispräparaten auf fische. Allgemeine Fischerei Zeitung 60: 370-378.

Schmidt, D. C., and L. J. Weber. (In press). The effect of surgical impairment of biliary excretion on the toxicity of rotenone to rainbow trout and rats. General and Comparative Pharmacology.

Schoenecker, W., and J. Peterson. 1965. Rough fish control projects: Niobrara River and Box Butte Reservoir. Nebraska Game, Reforestation and Parks Commission, Federal Aid in Fish Restoration, Project F-7-D-1 & 2. 16 p.

Shannon, E. H. 1968. The toxicity and detoxification of the rotenone formulations used in fish management. North Carolina, Wildlife Resources Commission, Federal Aid in Fish Restoration, Project F-16-R, Job VIII-B. 5 p.

Shannon, E. H. 1969. The toxicity and detoxification of the rotenone formulations used in fish management. North Carolina Wildlife Resources Commission, Federal Aid in Fish Restoration, Project F-19-1, Job IV-B. 8 p.

Shimkin, M. B., and H. H. Anderson. 1936. Acute toxicities of rotenone and mixed pyrethrins in mammals. Proceedings of the Society for Experimental Biology and Medicine 34:135-138.

Siegler, H. R., and H. W. Pillsbury. 1946. Use of derris to reclaim ponds for game fish. Journal of Wildlife Management 10(4):308-316.

Siegler, H. R., and H. Pillsbury. 1949. Progress in reclamation techniques. The Progressive Fish-Culturist 11(2):125-129.

Sinnappa, S., and C. E. Thuan. 1971. Identification of rotenone in fish poisoned by derris root resin. Malaysian Agricultural Journal 48(1):20-24.

Slifer, G. E. 1970. Stream reclamation techniques. Wisconsin Department of Natural Resources, Bureau of Fish Management, Management Report No. 33:1-35.

Smith, C. L. 1973. Small rotenone stations: a tool for studying coral reef fish communities. American Museum Novitates No. 2512: 1-21.

Smith, M. W. 1940. Copper sulphate and rotenone as fish poisons. Transactions of the American Fisheries Society 69(1939):141-157.

Smith, M. W. 1941. Treatment of Potter's Lake, New Brunswick, with rotenone. Transactions of the American Fisheries Society 70(1940): 347-355.

Smith, M. W. 1950. The use of poisons to control undesirable fish in Canadian fresh waters. The Canadian Fish Culturist No. 8:17-29.

Solman, V. E. F. 1950. History and use of fish poisons in the United States. The Canadian Fish Culturist No. 8:3-16.

Spector, W. S. 1956. Handbook of toxicology. Vol. I. Acute toxicities of solids, liquids and gases to laboratory animals. W. B. Saunders Company, Philadelphia and London. 408 p.

Spitler, R. J. 1970. An analysis of rotenone treatments for elimination of fish populations in southern Michigan lakes, 1957-1967. Michigan Academician 3(1):77-82.

Stecher, Paul G. (ed.). 1968. The Merck Index. 8th edition. Merck and Co. Inc., Rahway, N.J. 1713 p.

Sterba, G. 1963. Freshwater fishes of the world. The Viking Press, New York. 878 p.

Stroud, R. H., and R. G. Martin. 1968. Fish conservation highlights 1963-1967. Sport Fishing Institute, Washington, D.C. 147 p.

Subba-Rao, N. V., and A. G. Pollard. 1951. Photodecomposition of rotenone in spray deposits. III. Kinetics of the photodecomposition. Journal of the Science of Food and Agriculture 2:462-472.

Swan, R. L. 1965. Fall Creek Reservoir - pre-impoundment chemical treatment to remove undesirable fish. Oregon State Game Commission, Completion report for U.S. Army Engineer Contract No. DA-35-026-CIVENG-66-19, Number 17. 25 p.

Swingle, H. S., E. E. Prather, and J. M. Lawrence. 1953. Partial poisoning of overcrowded fish populations. Alabama Polytechnic Institute, Agricultural Experiment Station, Circular No. 113:1-15.

Takatsuki, A., N. Nakatani, M. Morimoto, G. Tamura, M. Matsui, K. Arima, I. Yamaguchi, and T. Misato. 1969. Antiviral and antitumor antibiotics. XX. Effects of rotenone, deguelin, and related compounds on animal and plant viruses. Applied Microbiology 18:660-667.

Tate, B., T. Moen, and B. I. Severson. 1965. The use of rotenone for recovery of live fish. The Progressive Fish-Culturist 27(3):158-160.

Taube, C. M., K. G. Fukano, and F. F. Hooper. 1954. Further studies on the use of fish poisons in Michigan lakes. Michigan Academy of Science, Arts, and Letters, Report, No. 1414:1-29.

Teeter, M. E., M. L. Baginsky, and Y. Hatefi. 1969. Ectopic inhibition of the complexes of the electron transport system by rotenone, piericidin A, demerol and antimycin A. Biochimica et Biophysica Acta 172:331-333.

Thompson and Company, John I. 1969. Survey of compounds which have been tested for carcinogenic activity. 1968-1969 Volume. DHEW Publication No. (NIH) 72-35, Public Health Service Publication No. 149.

Thoreson, N. A. 1956. Marias River fishery restoration. Montana Department of Fish and Game, Federal Aid in Fish Restoration, Job Completion Report, Project F-15-D-2. 11 p.

Tilemans, Ém., and S. Dormal. 1952. Toxicité des produits phytopharmaceutiques envers l'homme et les animaux à sang chaud. Parasitica 8(2):64-91.

Toffoli, E. V. 1965. Chemical treatment of the Merced River, Mariposa County. California Department of Fish and Game, Inland Fisheries Administrative Report No. 65-14:1-12.

Tompkins, W. A. 1953. An apparent case of rotenone tolerance in a bluegill, *Lepomis macrochirus*. Transactions of the Kentucky Academy of Science 14:43.

Tompkins, W. A., and J. W. Mullan. 1958. Selective poisoning as a management tool in stratified trout ponds in Massachusetts. The Progressive Fish-Culturist 20(3):117-123.

Ts'ai, L.-S., and T.-S. Ke. 1941. The photochemical decomposition of rotenone. Journal of the American Chemical Society 63:1717-1718.

Tucker, R. K., and D. G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. U.S. Bureau of Sport Fisheries and Wildlife, Resource Publication No. 84:1-131.

Turner, W. R. 1959. Effectiveness of various rotenone-containing preparations in eradicating farm pond fish populations. Kentucky Department of Fish and Wildlife Resources, Fisheries Bulletin No. 25:1-22.

Tuunainen, P. 1970. Relations between the benthic fauna and two species of trout in some small Finnish lakes treated with rotenone. Annales Zoologici Fennici 7:67-120.

Unai, T., I. Yamamoto, H.-M. Cheng, and J. E. Casida. 1973a. Synthesis and stereochemical characterization of hydroxy- and epoxy-derivatives of rotenone. Agricultural and Biological Chemistry 37(2):387-401.

Unai, T., H.-M. Cheng, I. Yamamoto, and J. E. Casida. 1973b. Chemical and biological O-demethylation of rotenone derivatives. Agricultural and Biological Chemistry 37(8):1937-1944.

U.S. Department of Health, Education, and Welfare. 1969. Report of the Secretary's Commission on Pesticides and their Relationship to Environmental Health. U.S. Department of Health, Education, and Welfare, Washington, D.C. 677 p.

U.S. Federal Water Pollution Control Administration. 1968. Water quality criteria; report of the National Technical Advisory Committee to the Secretary of the Interior. U.S. Department of the Interior, Federal Water Pollution Control Administration, Washington, D.C. 234 p.

U.S. Fish and Wildlife Service. 1949. The use of rotenone as a fish poison. U.S. Fish and Wildlife Service, Fishery Leaflet 350:1-8.

Wales, J. H. 1947. Castle Lake trout investigation 1946 catch, and chemical removal of all fish. California Fish and Game 33(4): 267-286.

Walker, C. R. 1969. Problems in clearance and registration of chemical tools used by fish culturists and fishery biologists, p. 1-139. In Fish and Chemicals: a symposium on registration and clearance of chemicals for fish culture and fishery management. U.S. Bureau of Sport Fisheries and Wildlife. Washington, D.C.

Wasicky, R., and O. Unti. 1951. Experiencias com algumas substancias sobre *Australorbis glabratus*, transmissor de esquistossomose. Anais da Faculdade de Farmacia e Odontologia da Universidade de Sao Paulo 9:41-50.

Weier, J. L., and D. F. Starr. 1950. The use of rotenone to remove rough fish for the purpose of improving migratory waterfowl refuge areas. Journal of Wildlife Management 14(2):203-205.

Wharton, D. A. 1968. Selective removal of gizzard shad from Wye Mills Lake (1968). Maryland Department of Game and Inland Fisheries. 5 p.

Whittaker, R. M., and A. L. Whittaker. 1935. Plant fish poisons as insecticides. Poultry Science 14:351-354.

Wilber, C. G. 1969. The biological aspects of water pollution. Charles C. Thomas Publisher, Springfield, Illinois. 296 p.

Wilcox, J. F. 1965. Rough fish control in San Angelo Reservoir by selective and contour rotenone treatment. Texas Parks and Wildlife Department, Federal Aid in Fish Restoration, Job Completion Report, Project No. F-14-D-8, Job No. 16-a-49. 13 p.

Wilkins, P. 1957. Reclamation of Chilhowee Lake Site. Tennessee Game and Fish Commission, Federal Aid in Fish Restoration, Job Completion Report, Project F-18-D-1. 13 p.

Wollitz, R. E. 1962. Effects of certain commercial fish toxicants on the limnology of three cold-water ponds, Montana. Proceedings of the Montana Academy of Sciences 22:54-81.

Wood, J. G., and R. L. White. 1962. Selective control of gizzard shad in Lake Inks, Texas. Texas Game and Fish Commission, Federal Aid in Fish Restoration, Project No. F-14-D-6, Job No. 16-a-35. 8 p.

Wright, T. W. 1957. The rates of dissipation of certain rotenone preparations, their residual effects on bluegill production, and their effects on populations of fish-food organisms. M.S. Thesis, Alabama Polytechnic Institute, Auburn, Alabama, June 5, 1957. 47 p.

Wyatt, H. N., and H. D. Zeller. 1965. Fish population dynamics following a selective shad kill. Proceedings of the Southeastern Association of Game and Fish Commissioners 16(1962):411-418.

Yamamoto, I. 1969. Mode of action of natural insecticides. Residue Reviews No. 25:161-174.

Yamamoto, I. 1970. Mode of action of pyrethroids, nicotinoids, and rotenoids. Annual Review Entomology 15:257-272.

Yamamoto, I., T. Unai, H. Ohkawa, and J. E. Casida. 1971. Stereochemical considerations in the formation and biological activity of the rotenone metabolites. Pesticide Biochemistry and Physiology 1:143-150.

Yojima, H. 1962. Fluorometric analysis of derris. Kagaku Keisatsu Kenkyusko Hokuku 15(1):73-76.

Zilliox, R. G., and M. Pfeiffer. 1956. Restoration of brook trout fishing in a chain of connected waters. New York Fish and Game Journal 3(2):167-190.

Zilliox, R. G., and M. Pfeiffer. 1960. The use of rotenone for management of New York trout waters. The Canadian Fish Culturist No. 28:3-12.

Zischkale, M. 1952. Effects of rotenone and some common herbicides on fish-food organisms. Field and Laboratory 20:18-24.

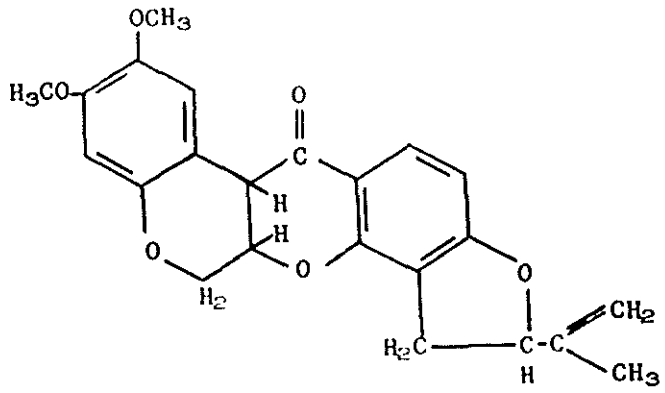
Appendix A--Technical Data on Rotenone

Alternative names : Ronone, Rotenon, Barbasco, Haiari, Noxfish, Pro-Noxfish, Chem-Fish Regular, Chem-fish Special, Fish-Tox, Derris, Cubé, Derrin, Nicouline, Tubatoxin, Timbo powder, Drymac, Cube root, Ro-Ko, Exelolo, Extrax, Cubor, Chem-mite, Rotefine, Rotessenol, rotocide, Warbicide, Akertube, Nekoe

Chemical name : Extracts from *Derris, Lonchocarpus, Tephrosia, Nillettia,* and *Mundulea* plants 1,2,12,12a,tetrahydro-2-isopropenyl-8,9-dimethoxy-(1)benzopyrano-(3,4-b)furo(2,3-n)(1)benzopyran-6(6aH) one.

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

Structural formula :



Formulations : Liquid, synergized liquid, powdered plant roots

Primary uses : Insecticide, parasiticide for external use on domestic animals

Secondary use : Fish toxicant. Some formulations are registered for fishery use.

Mode of action : Inhibitor of cellular respiration

Toxicity to plants : Nonphytotoxic

Toxicity to invertebrates: Stonefly (*Pteronarcys* sp.) - 24-hour LC50 = 2.9 mg/ℓ
Amphipod - 24-hour LC50 = 0.35-6 mg/ℓ
Water flea - 48-hour LC50 = 0.010 mg/ℓ
Honeybee - oral LD50 = 3 mg/kg
American cockroach - oral LD50 = 1,000 mg/kg
Phantom midge larvae - oral LD50 = 1.13 mg/ℓ

Toxicity to fish : Extremely toxic
Bluegill - 96-hour LC50 = 0.150 mg/ℓ
Channel catfish - 96-hour LC50 = 0.118 mg/ℓ
Yellow perch - 96-hour LC50 = 0.056 mg/ℓ
Striped bass - 96-hour LC100 = 0.01 mg/ℓ

Toxicity to amphibians and reptiles: *Rana* sp. - LC = 0.1 mg/ℓ
Ambystoma - LC = 0.1 mg/ℓ

Toxicity to birds : Slightly toxic
Mallard - oral LD50 = >2,000 mg/kg
Pheasant - oral LD50 = >1,414 mg/kg
Chicken - oral LD50 = 996 mg/kg
American robin - oral LD50 = 200 mg/kg
(Young birds are about 10 times more sensitive to rotenone than older birds)

Toxicity to mammals: Moderately toxic
Rat - oral LD50 = 132 mg/kg
Guinea pig - oral LD50 = 200 mg/kg
Rabbit - oral LD50 = 1.7 mℓ/kg

Safety hazards : Inhalation of powder causes headache, sore throat and other cold symptoms, and sores on mucous membranes; contact causes irritation of eyes and rash on skin. Protective clothing is advised when using powdered root. Use of wettable powder or liquid formulations reduces risks to safety and health.

Persistence in the environment: Seldom over 2 weeks; longer in very soft water

Appendix B--Common and Technical Names of Fishes

The following fish classification was obtained by utilizing Bailey et al. (1970), Sterba (1963), and Nikol'skii (1954).

Common Name	Technical Name
Bowfins	Amiidae
Bowfin	*Amia calva*
Herrings	Clupeidae
Gizzard shad	*Dorosoma cepedianum*
Trouts	Salmonidae
Chinook salmon	*Oncorhynchus tshawytscha*
Rainbow trout	*Salmo gairdneri*
Atlantic salmon	*Salmo salar*
Brown trout	*Salmo trutta*
Brook trout	*Salvelinus fontinalis*
Lake trout	*Salvelinus namaycush*
Midminnows	Umbridae
Mudminnow	*Umbria* sp.
Pikes	Esocidae
Northern pike	*Exox lucius*
Chain pickerel	*Esox niger*
Characins	Characidae
Piranha	*Serrasalmus* sp.
Minnows and carps	Cyprinidae
Crucian carp	*Carassius carassius*
Carp	*Cyprinus carpio*
Gudgeon	*Gobio gobio*
Roach	*Rutilus rutilus*
White amur	*Ctenopharyngodon idellus*
Rudd	*Scardinius erythrophthalmus*
Goldfish	*Carassius auratus*
Utah chub	*Gila atraria*
Golden shiner	*Notemigonus crysoleucas*
Common shiner	*Notropis cornutus*
Fathead minnow	*Pimephales promelas*
Creek chub	*Semotilus atromaculatus*

Suckers	Catostomidae
White sucker	*Catostomus commersoni*
Freshwater catfishes	Ictaluridae
Black bullhead	*Ictalurus melas*
Brown bullhead	*Ictalurus nebulosus*
Channel catfish	*Ictalurus punctatus*
Codfishes	Gadidae
Burbot	*Lota lota*
Killifishes	Cyprinodontidae
Killifish	*Fundulus* sp.
Livebearers	Poeciliidae
Mosquitofish	*Gambusia affinis*
Sticklebacks	Gasterosteidae
Brook stickleback	*Culaea inconstans*
Temperate basses	Percichthyidae
Striped bass	*Morone saxatilis*
Sunfishes	Centrarchidae
Rock bass	*Ambloplites rupestris*
Green sunfish	*Lepomis cyanellus*
Pumpkinseed	*Lepomis gibbosus*
Warmouth	*Lepomis gulosus*
Bluegill	*Lepomis macrochirus*
Redear sunfish	*Lepomis microlophus*
Smallmouth bass	*Micropterus dolomieui*
Largemouth bass	*Micropterus salmoides*
White crappie	*Pomoxis annularis*
Black crappie	*Pomoxis nigromaculatus*
Perches	Percidae
Yellow perch	*Perca flavescens*
Drums	Sciaenidae
Freshwater drum	*Aplodinotus grunniens*
Cichlids	Cichlidae
Mouthbreeder	*Pseudocrenilabrus philander*
Tilapia	*Tilapia* sp.