

W DEPARTMENT OF WILDLIFE Washington

August 1992



**FINAL — SUPPLEMENTAL
ENVIRONMENTAL IMPACT STATEMENT
LAKE & STREAM REHABILITATIONS —1992-1993
HABITAT & FISHERIES MANAGEMENT DIVISIONS**

Report #92-14

Washington Department of Wildlife
Habitat and Fisheries Management Divisions
Report #92-14

FINAL
SUPPLEMENTAL ENVIRONMENTAL IMPACT STATEMENT
LAKE & STREAM REHABILITATIONS
1992-1993

August 1992

FINAL
Supplemental Environmental Impact Statement
LAKE AND STREAM REHABILITATIONS

Description:

The Washington Department of Wildlife (WDW) proposes to continue **Lake and Stream Rehabilitations** to improve fishing for game fish in selected waters via the elimination of other non-game or competitor species of fish.

The following alternative methods area considered in this EIS:

Use of the pesticide Rotenone
Use of other fish toxicants
Use of predator or competitor species
Stocking with legal size fish
Mechanical methods
No Action

Proponent:

Washington Department of Wildlife

Proposed Date of Implementation:

Fall 1992

Lead Agency:

Washington Department of Wildlife
600 North Capitol Way
Olympia, Washington 98504

Responsible Official:

Curt Smitch, Director, Washington Department of Wildlife

Contact Person:

Greg Hueckel
Fish Management Division
Washington Department of Wildlife
600 North Capitol Way
Olympia, Washington 98504
Phone: (206) 753-2895

Licenses Required:

Water Quality Modification - Washington Department of Ecology (DOE)
National Pollution Discharge Elimination System Permit - (DOE)
Approval by Washington Wildlife Commission

Authors and Principal Contributors:

This document was prepared by the regulatory services program, Habitat Management Division, and various staff of Fisheries Management Division, Washington Department of Wildlife.

Date of Issue:

July 1, 1992

Public Hearings:

August 15, 1992

Wildlife Commission
Red Lion Inn
1225 N. Wenatchee Ave
Wenatchee, Washington 98801

Date Final Action is Planned:

September 30, 1992

Type and Timing of Subsequent Environmental Review:

None

Location of EIS Background Data:

SEPA Public Information Center
Washington Department of Wildlife
Habitat Management Division
600 North Capitol Way
Olympia, Washington 98504

Cost to the Public:

None

TABLE OF CONTENTS
Lake and Stream Rehabilitation
Draft Programmatic Environmental Impact Statement

FACT SHEET	i
TABLE OF CONTENTS	iii
LIST OF FIGURES	v
LIST OF TABLES	vi
SUMMARY/ALTERNATIVES	1
DESCRIPTION OF PROPOSED ACTION	6
DETAILED ASSESSMENT OF IMPACTS	
Earth	12
Air	12
Water	12
Plants	18
Zooplankton	57
Benthic Fauna	77
Fish	99
Amphibians and Reptiles	121
Birds	123
Mammals	125
Human Health	127

CONTENTS (continued)

APPENDIX A:	FORMS
APPENDIX B:	HISTORY OF ROTENONE
APPENDIX C:	GLOSSARY
APPENDIX D:	REFERENCES
APPENDIX E:	LAKES PROPOSED FOR REHABILITATION IN 1992

LIST OF FIGURES

<u>Figure</u>	<u>Title</u>	<u>Page</u>
1	Flow chart showing the most important ways in which rotenone poisoning and subsequent trout stocking may affect algae levels in a lake.	19
2	Relationship between carp density and phosphorus release.	22
3	Release of various forms of phosphorus by different sized carp in 22° C water.	23
4	Volume weighted mean chlorophyll <u>a</u> content in the epilimnion of Pine Lake, Washington, before and after rotenone.	31
5	Phytoplankton levels in Bonham State Park Lake, Texas, before and after rotenone.	32
6	Abundance of algae in Fern Lake, Washington over a twelve year period.	33
7	Secchi disc transparency in two ponds treated with rotenone, and one untreated control pond.	34
8	Diatom and green algae levels in Hodges Reservoir, California, before and after rotenone.	35
9	Phytoplankton levels in Lake Lavon (Price Creek Cove), Texas, before and after rotenone.	36
10	Phytoplankton levels in Carls Lake, Minnesota, before and after rotenone.	37
11	Algae, phosphorus, transparency and <i>Daphnia</i> in Wirth Lake, Minnesota.	46
12	The effects of adding various numbers of zooplanktivorous fish to enclosures of lake water containing <i>Daphnia</i> .	51
13	The effects of adding zooplanktivorous and benthivorous fish to enclosures in two Swedish lakes.	52

<u>Figure</u>	<u>Title</u>	<u>Page</u>
14	Effects of rainbow trout stocking on <i>Daphnia</i> and phytoplankton in previously unstocked Medical Lake Washington.	53
15	Effect of toxaphene poisoning and subsequent restocking with planktivores and bottom feeders on water clarity in Clear Lake, Minnesota.	55
16	Hypothetical scenario following rotenone poisoning and trout restocking in a lake.	56
17	Recovery times for the zooplankton in several lakes and ponds following rotenone.	70
18	Mean LC50's of rotenone formulation for various groups of lake and pond benthos.	84
19	Effect of bottom muds on survival of midge larvae <i>Chironomus plumosus</i> in aquariums subjected to various dosages of rotenone.	85
20	Effect of fish removal on benthos in two lakes where fish were not restocked following rotenone treatment.	96
21	Effect of fish removal and subsequent restocking on benthos in two lakes.	97
22	Skipped	
23	96-hour median lethal concentration (LC50) of Noxfish for several fish held under standardized laboratory conditions.	105
24	96-hour median lethal concentration (LC50) of Noxfish for several fish held standardized laboratory conditions.	106
25	Relationship between water temperature and the percentage of dead fish that surface following rotenone treatment.	116
26	Relationship between fish size and surfacing rate for various species in Nebish Lake, Wisconsin.	118
27	Paths of possible human exposure to rotenone.	128

LIST OF TABLES

<u>Table</u>	<u>Title</u>	<u>Page</u>
A	Comparative Impacts Matrix	3
B	Phosphorus content of fish.	26
C	Response fo phytoplankton shortly after rotenone treatment in selected lakes.	29
D	Magnitude of algal blooms following rotenone treatment in some test waters.	39
E	Estimated annual amounts of phosphorus added and removed by fingerling trout stocking in selected Washington lakes.	42
F	Algal types unaffected and suppressed by grazing.	48
G	Toxicity of rotenone to zooplankton in laboratory bioassays.	58
H	Data summary of zooplankton studies in lakes and ponds. I. immediate effects rotenone on mid-water zooplankton.	60
I	Data summary of zooplankton studies in lakes and ponds. II. long term effects of rotenone.	64
J	The effects of rotenone treatment and susequent fish stocking on the kinds and size of zooplankton in six lakes.	74
K	The effects of rotenone treatment and subsequent fish stocking on the kinds and size of zooplankton in eight Swedish lakes.	75
L	Toxicity of rotenone to benthic animals in laboratory bioassays.	78
M	Short and long-term effects of rotenone treatment on the benthos of various lakes and ponds.	86
N	Toxicity of rotenone to fish in laboratory bioassays.	100

<u>Table</u>	<u>Title</u>	<u>Page</u>
O	Toxicity of rotenone formulations to fish eggs.	108
P	Percentage of dead fish surfacing following rotenone treatment in mark-recapture experiments.	114
Q	Toxicity of rotenone to amphibians in laboratory bioassays.	122
R	Median lethal dosages (LD50) of pure rotenone formulations administered orally to birds.	124
S	Median lethal dosages (LD50) of pure rotenone formulations administered orally to animals.	126
T	Estimated lethal oral doses of rotenone for humans.	130
U	Results of long-term oral dosages of rotenone on dogs, rats and hamsters.	133
V	Studies on the cancer causing potential of long-term exposure to rotenone.	136

SUMMARY

The Washington Department of Wildlife manages approximately 5.9% of the state's lowland lakes throughout the state according to public desires, recreational demands habitat considerations and previous management efforts. Although surveys have shown that trout are the most popular of the state's game fish, some lakes are managed to improve populations of bass, bluegill or crappie. In response to these needs WDW proposes the elimination of non-game or competitor species in a portion of these lakes to allow stocking and optimal populations of trout fingerlings and selected warmwater species. The overall objective of the program is to improve public fishing opportunities.

Alternative Methods:

In Table A the alternative methods are broken into groups; Fish Toxicants, Predator/Competitor, Mechanical and No Action. The methods included in these groups are explained below.

Fish Toxicants:

Rotenone . Rotenone is widely regarded as the safest and least persistent of the poisons.

Other poisons . The list of poisons used to kill undesired fish in lakes and ponds is huge (Lennon et al., 1970; Eschmeyer, 1975). Currently only the Streptomyces derived antibiotic antimycin (marketed as Fintrol) is registered for use as a general fish toxicant with the EPA (Cumming, 1975).

Baits . Baits have been used with limited success, either as an attractant to draw fish to a secluded area of the lake to be eliminated by other means or as a coating for calcium carbide pellets that would produce acetylene gas and float the fish after the bait had been digested.

Predator/Competitor

Predator stocking . Actual experiments with predator stocking as a fish-control technique are scarce, and success has been limited (Dunst, et al., 1974). Both northern pike and largemouth bass failed to control bluegills in Michigan (Shapiro, et al., 1975) and in California (State of California, 1983). California's efforts to control carp, suckers, and squawfish with predators have failed although striped bass reduced shad and bluegills in some reservoirs (State of California, 1983). Since large apex predators would also eat trout fry, this is not an option in most Washington state waters.

Mechanical

Water level drawdown . Completely draining a pond or reservoir is the most foolproof way to destroy all the fish in it (Prevost, 1960); where pockets of water remain, they can be easily and thoroughly poisoned, netted or electroshocked (Barry, 1967). Partial drawdowns that expose carp spawning beds have also been reasonably successful (Sprague, 1961). In Washington State, however, few of the program lakes have water level control facilities.

Lakewide netting and trapping . There are no published accounts of lakewide netting programs that have been successful. Most lakewide attempts using commercial fishermen have failed because they are not cost-effective and are extremely labor intensive.

Dams and barriers . Barriers are used to block migrating fish from their spawning streams. This method has little practical value in Washington where the important target species (carp, perch, and sunfish) are lake spawners.

Electrofishing . Electrofishing on a lakewide basis has never been successful as a control measure and, like netting, is very labor intensive.

Removing congregations of spawning fish . There are several accounts of success with this method, whereby adult fish are allowed to congregate in spawning areas which are subsequently blocked off in most cases. The fish are then poisoned, electroshocked or netted. To actually eliminate a nuisance fish population, this technique would have to be repeated yearly, at least until all year-classes had reached spawning size.

Table A - Comparative Impacts Matrix, by element of the environment.

Method	Significant Impacts	Mitigation Measures
Earth		
Fish Toxicants Predator/Competitor	Rate and distribution of lake soil sediment may be altered with changes in species abundance and diversity	
Mechanical	Change in sediment transport through/around dams or barriers Changes in plant/benthic from drawdown	
Air		
Fish Toxicants Mechanical	Adverse odors may be present while fish killed decompose	Extended fishing season to increase opportunity to harvest fish prior to rehabilitation
Surface Water		
Fish Toxicants Mechanical Predator/Competitor	Changes in bacteria levels and turbidity, change or elimination of phyto/zoo-plankton, benthic fauna, fish species and diversity, algae blooms, change to water taste and odor. Algae blooms.	Lakes can recover from algae blooms, loss of phyto/zoo-plankton, benthic fauna and changes to taste and odor in two to twelve months.

Table A. continued.

Method	Significant Impacts	Mitigation Measures
		Mitigation measures include actions to restrict the use of rotenone to targeted waters only, and to include potassium permanganate dip stations and temporary sand bag dams
		Lakes would be restocked with desired species.
Terrestrial Resources		
Fish Toxicants	Larval amphibians and some adults may be killed. Adult amphibians or reptiles may be temporarily affected by loss of aquatic fish food source.	Treatments are timed to produce the desired rehabilitation with the least impact to other species
Predator/Competitor		
Mechanical	Birds or mammals which depend on fish/benthic organisms for food may be temporarily impacted	
	Humans in direct contact with the powder rotenone may experience temporary skin, eye or mucous membrane irritation.	Protective clothing
Environmental health		
Fish toxicants	The rotenone label precludes the consumption of rotenoned fish as food or feed.	Disposal of fish or prevention of use.
Predator/Competitor		
Mechanical		

Table A. continued.

Method	Significant Impacts	Mitigation Measures
Aesthetics	Increases in human activities as a result of increased fishing pressure may cause erosion, air, water and noise pollution, trampling of vegetation, and other impacts to recreational, religious, or scientific use of the area.	Monitoring, education and enforcement.
Fish toxicants	Water will be brown in color following treatment with rotenone or disturbance by mechanical means.	Water will recover in a few hours to a few days.
Mechanical	Floating or beached fish	Disposal or education.

DESCRIPTION OF THE PROPOSED ACTION:

Type of Action

The proposed action is to continue the Washington Department of Wildlife's rehabilitation of selected lakes and streams by eliminating undesired fish species using rotenone followed by restocking with a preferred fish species, to improve public fishing. Lake and stream rehabilitations occur throughout the state. Almost all treatments have occurred in lakes and ponds, with only occasional stream or slough treatments.

In the last 20 years approximately 5.9% of the state's lowland lakes have been treated with rotenone. This equals about 3.4% of the state's total standing water acreage below 2500 feet in elevation. The average per year has been 0.3% of total surface acreage of the state's lowland lakes.

Justification

Fisheries Management

To satisfy the annual demand for productive gamefish fishing by over 600,000 anglers, Washington Department of Wildlife stocks selected waters with trout from hatcheries and transplanted bass, crappie, walleye, and additional warmwater fish species from other waters. Many waters are managed for specific fisheries, such as trout only or warmwater species. The management emphasis for state waters is decided according to habitat parameters, public desires, recreational demands, and previous management efforts. Occasionally, these waters become overpopulated with fish species outside this management emphasis. This often results in increased predation and/or competition, hence poor growth and survival, of targeted game fish. If carp overpopulate, fish survival decreases and nesting bird habitat is degraded due to siltation and uprooting of emergent vegetation. Infestations of these fish species occur through migration from other waters or through illegal transport and introductions. Three management options are available if this happens:

- 1) Take no action;
- 2) Change the management emphasis for the water;
- 3) Eliminate competing species and stock with desired gamefish species.

Option 1 will result in an increase in numbers of fish outside the management emphasis to a point where the water no longer supports a viable gamefish fishery.

Option 2 allows for a viable fishery, but is relatively costly. For example, to establish a trout fishery, the cost of producing a fingerling trout in a state hatchery is about 4% of the cost of a legal-sized trout (Washington Department of Wildlife, 1983). Even though fry survival is lower when compared to legal-sized trout, they can still be more economical in some cases (see below). Furthermore, legal-sized trout are considered a lower quality fish than naturally-reared fry-origin trout, and are usually smaller as well.

Option 3 is the only alternative that allows the lake to continue to provide a viable fishery. Rotenone is the method currently used by WDW to eliminate fish in lakes and is far more economical than either options 1 or 2 above. Washington Department of Wildlife (1984) compared the costs of three different management strategies for a typical lowland trout lake in western Washington (Lake Erie, Skagit County).

These options were:

- 1) trout-only lake maintained by fry stocking and periodic rotenone treatment;
- 2) mixed-species lake maintained by trout fry stocking (no rotenone); and
- 3) mixed species lake maintained by legal-sized trout stocking (no rotenone).

The cost of Option 1 was about one-third the cost of either Option 2 or 3. Also note that Option 2 is not likely to be a viable alternative in many lakes for the reasons already discussed.

Wildlife Management

Lakes are also rehabilitated by the Department of Wildlife to improve the quality of waterfowl habitat. The primary objective is to remove carp from potentially productive nesting and rearing duck habitat to increase the amount of food (aquatic invertebrates) and vegetative cover. Candidate waters are primarily one to three feet deep.

Pre-Treatment Procedures

A lake or stream is selected for rotenone treatment when a viable fishery can only be provided with introductions of legal-sized fish. These determinations are made by the WDW Area Fisheries Biologist directly charged with managing the lake's gamefish. Standard indicators of fishery performance are the average catch per hour on opening day, and fish size and abundance from annual pre-season gillnet sets. When poor performance is coupled with gillnet and/or electroshocking data showing and increase in species

outside the management emphasis, the Area Biologist may recommend treatment to his Regional Biologist.

A Pre-Rehabilitation Plan (See Appendix A) containing vital information on the proposed treatment must be completed by the biologist.

In calculating the dosage of rotenone needed, the biologist considers a variety of physical and biological factors, the most important being target species, water chemistry, past successes or failures in the lake and presence of weedy shorelines.

Dosage is initially calculated based on powder or liquid containing 5% rotenone, and is expressed as parts of powder or liquid - not pure rotenone itself - per million parts of lake water (ppm) on a weighted basis. One ppm is equivalent to one milligram per liter (1 mg/l).

The powders used by WDW rarely contain only 5% rotenone. WDW receives most of its rotenone dust from Peruvian suppliers, and shipments are chemically assayed by batch for rotenone content. Powders used from 1977 through 1984 ranged from 6.6% rotenone to 8.1% rotenone. Liquid preparations consistently contain 5% rotenone. When these formulations are received and the exact assay known, biologists adjust the amount of powder used to conform to the initial calculation based on 5% powders.

The actual amount of rotenone needed is based on the estimated weight of water in the lake. This is determined by volumetric calculations using WDW surveys on the particular lake.

The Regional Fisheries Program Manager presents his list of proposed treatments along with justifications for each water to the Fisheries Management Division of WDW. Approval at this stage may depend not only on the validity of the biological justifications, but on other considerations such as the lake's public use and its importance as a recreational fishery, and the availability of rotenone itself. Statewide priorities are established, and a list of candidate lakes drawn up.

After developing a list of candidate lakes, the public is notified through a general news release, usually in late spring. Area Biologists also solicit public opinion from lakeshore residents and other groups in the area. Public meetings are held in the vicinity of the waters proposed for treatment prior to a final decision.

At its annual August public hearing, the Washington State Wildlife Commission - a group of private citizens chosen by the Governor to oversee WDW - is presented with the list of candidate lakes. The Commission approve or denies treatment on individual lakes at its annual August meeting. Even after a lake has been approved by the Commission, WDW may opt not to treat that lake.

Treatment Procedures

Shortly before treatment, the lake is divided into sections of similar volumes, and these sections are marked using buoys and shoreline markers.

On the day slated for treatment, each section of the lake is assigned to a WDW employee. Rotenone is applied by towing burlap sacks of commercial dust behind a boat, the outboard prop wash helping to diffuse the poison. Shoreline and marshy areas are often sprayed with liquid rotenone by motorized pump or are dusted by hand. Aerial applications are sometimes made. Common dosages of rotenone (5%) in lakes treated in Washington range between 1-4 ppm.

Fishing regulations are liberalized when possible, and upon approval by the Wildlife Commission, to utilize fish in waters scheduled for rehabilitation. Warmwater game fish, usually mature bass, are collected (depending on need) prior to rehabilitation, to be utilized as broodstock for waters nearby which are managed for warmwater fisheries. On some lakes, bass that have floated to the surface have been netted by WDW employees and bass club volunteers, revived by dipping the fish in potassium permanganate, and moved to mixed-species or spiny ray lakes to augment or start a population (Fletcher, 1976). WDW has typically transplanted 200-300 fish from a single lake during this type of procedure. The use of potassium permanganate also requires a short-term water modification (permit) to the water quality standards issued by the Washington Department of Ecology.

Post-Treatment Procedures

In lakes with a stream outlet, runoff from the lake must be controlled or detoxified. In some cases, the runoff is small enough that it can be dammed off (using sandbags, for example) until the rotenone is naturally degraded. When this is not possible, and oxidizing agent - usually potassium permanganate - is dripped into the outlet stream to detoxify the rotenone before it can harm fish and invertebrates downstream. Between 1977 and 1984 such detoxification was necessary in only 16% of the lakes treated. Pfeifer (1985) provides a detailed account of outlet detoxification procedures, including dosage/detoxification curves and case histories in Martha and Silver Lakes (Snohomish County).

In the lake itself, rotenone degrades naturally over time. At intervals following treatment, WDW Area Biologists usually perform a simple bioassay to determine how long the lake remains toxic to fish: hatchery rainbow trout are commonly suspended in the water column in wire cages and when these fish survive 1-6 days in the lake, it is considered nontoxic.

The biologist submits a Post-Rehabilitation Form (see Appendix A) for each treated water; it describes, among other things, the possibility of a complete kill, water conditions at the time of treatment, and any detoxification measures taken.

Fish are restocked the following spring. During the post-treatment years, the Area Biologist continues to monitor fish survival and growth, as well as catch rates for the water.

Number and size of Waters Treated

The first rotenone treatment in Washington State took place in September, 1940 on King Lake (Pend Oreille County). Since that time 473 state waters have been treated at least once. The chlorinated hydrocarbon insecticide toxaphene was occasionally used instead of rotenone; its use was discontinued in the late 1960's, and since then, rotenone has been the only fish poison applied in Washington State.

Almost all treatments have occurred in lakes and ponds, with only occasional stream or slough treatments. Waters treated since 1940 represent [5.72%] of the total surface acreage of all lakes below 2,500 feet elevation in the state.

Frequency of Rotenone Treatments

Rotenone rarely if ever kills all the fish in a lake. Problem species often repopulate the lake naturally over the course of time. In addition, problem species are often reintroduced illegally by anglers or lakeside residents. These may be the same species that originally degraded the targeted fishery, or new ones. The net result of any of these cases is the same: fish production will eventually decline, and the lake may have to be rehabilitated again.

Of 473 Washington State lakes that have been treated, 240 (55%) have been treated more than once. The average length of time between treatments has been 7.74 years ($n = 522$ intervals, $s = 4.49$ years).

Target Species

In the eastern half of the state pumpkinseed sunfish was most frequently targeted for elimination, in the western half of the state yellow perch was most frequently targeted. Other important target species statewide include carp, crappie, brown bullhead (catfish), and largemouth bass. All are introduced, non-native species.

A particular lake may experience recurring problems with the same target species over the course of many years. Often, however, the target species on frequently-rotenoned lakes changes over the years. This is often the case in "urban" lakes which are frequent targets for illegal fish introductions.

Timing of Rotenone Treatments

Seventy-eight percent of rotenone treatments in the state have taken place in the fall, mostly in September and October. Only 22% have been spring treatments, and these occurred mostly in March. All spring treatments were on eastern Washington lakes.

Rotenone is usually applied in the fall because water levels are low, aquatic vegetation is sparse, recreational use of the lake is reduced and since most lake's summer thermal stratification has ended (allowing rotenone to circulate throughout the water column). Spring rotenone treatment are occasionally performed on certain lakes with extensive shallow or weedy areas; higher water levels in the spring make these areas more accessible by boat.

Legal Standing

RCW 77.12.420 empowers the Wildlife Commission to eradicate "undesirable types of fish. The Commission's right to rehabilitate lakes and streams was affirmed by Thurston-Mason County Superior Court in the case of Patrick vs. Biggs (#27476), January, 1954.

Funding

Lake and stream rehabilitation operations are funded through fishing license fees and through taxes collected by the federal government on fishing tackle at the manufacturing level and apportioned to states under the Dingell-Johnson Act. Dingell-Johnson funds are limited to 75% of total project costs. A 25% contribution on Department of Wildlife monies is required by federal law. Lake and stream rehabilitation with rotenone is an approved fishery management activity under Dingell-Johnson funding.

DETAILED ASSESSMENT OF IMPACTS

Earth

Lake and stream rehabilitation may have some effect on lake soils since changing diversity of fish can influence rate and distribution of organic sedimentation. No specific data are available on this subject.

By enhancing fishing in a lake, more fishermen may visit the area. Increased human activity may also increase erosion if vegetation becomes trampled and undeveloped trails are used more frequently.

Air

Rotenone droplets or mist may be carried in the air from the liquid applications. Powder rotenone is applied by towing an open sack underwater, so escape of particles in the air should be minimal. Decomposing fish emit an adverse odor to the surrounding atmosphere. Since the rate of decomposition is influenced by temperature and moisture, rehabilitation projects are usually scheduled during periods that minimize the undesirable aspects of decomposition. In residential areas, dead fish are sometimes used in gardens and flower beds as fertilizer by local residents.

Also better fishing in an area usually attracts more people during fishing season. This may increase noise and air pollution from cars and boats.

Water

From a human use standpoint, important water quality parameters in lakes include dissolved oxygen, fecal coliform levels, total dissolved gas, temperature, Ph, turbidity, and aesthetic values (Title 173 WAC, Water Quality Standards, pages 187-1988, 1983). Where lakes supply drinking water for people or livestock, safety and palatability of the water are obvious concerns. A variety of other chemical and biological parameters are also considered here as water quality factors.

Some important aspects of water quality that are affected indirectly by rotenone treatment include phytoplankton, which affects water transparency and thus aesthetic values and dissolved oxygen levels at the sediment/water interface, and the effect of fish stocking on lake phosphorous loads.

There has been only one comprehensive study of how rotenone treatment indirectly affects all routinely-measured water quality parameters : Bonn and Holbert (1961) conducted tests on 18 water quality indicators in Lake Lavon and Bonham State Park Lake, Texas. Their goal was to determine the indirect effects of rotenone treatment on municipal drinking water supplies. Only coves in Lake Lavon were treated, with non-treated coves serving as controls. In Bonham State Park Lake, all 49 acres were treated for a complete fish kill and results were compared with pretreatment data. Standard rotenone formulations and dosages were used, and after dead fish were weighed, their carcasses were punctured and scattered back into the water to create a natural post-treatment environment. Samples of water were taken from various depths at two-week intervals during the year, and at shorter intervals immediately prior to and after the treatment. Bonn and Holbert tested the following parameters.

- | | |
|---------------------|-------------------------------------------------|
| 1) temperature | 10) total nitrogen |
| 2) Ph | 11) phosphorous |
| 3) turbidity | 12) potassium |
| 4) dissolved oxygen | 13) total phytoplankton |
| 5) carbon dioxide | 14) generic makeup of phytoplankton |
| 6) total alkalinity | 15) total hardness |
| 7) calcium | 16) odor number |
| 8) NH ₄ | 17) most probable number (of coliform bacteria) |
| 9) organic nitrogen | 18) bacterial colonies per milliliter |

Of these 18 parameters, only four showed significant change due to the treatment: turbidity decreased, phytoplankton increased, noncoliform bacteria increased, and the water took on a disagreeable taste and odor. The change in taste and odor of the water was by far the greatest of the water quality changes noted.

Scattered water quality data from other studies (which gathered them from ancillary information) are also available:

Brown and Ball (1943a) measured water temperature, dissolved oxygen, carbon dioxide, methyl orange alkalinity, and pH throughout the water column in Third Sister Lake, Michigan. None of the factors changed significantly within four days of rotenone treatment when compared to pre-treatment data.

Houf and Campbell (1977) compared three small, fishless Missouri ponds treated with rotenone and two untreated control ponds, concluding that rotenone treatment "had no noticeable effect on water chemistry." The monitored pH, water temperature (pond surface and bottom), dissolved oxygen (pond surface and bottom), hardness and alkalinity. These parameters were measured throughout the experiment, which began three months before treatment and ended a year after treatment.

Wollitz (1962) measured several chemical and physical properties of two Montana ponds before and after rotenone treatment. He found that oxygen saturation, alkalinity, pH, nitrate, and inorganic phosphate levels did not change significantly after treatment. In one of the ponds turbidity decreased and transparency increased after poisoning.

Bandow (1980) found no significant changes in the surface temperature, dissolved oxygen (surface and subsurface), or nitrate nitrogen levels in Carls Lake, Minnesota, after it was poisoned with rotenone. Transparency increased dramatically, however, due to lower algae levels.

Based on these studies and those of Bonn and Holbert (1961), it can probably be concluded that water quality parameters unaffected by rotenone treatment, either directly or indirectly are: water temperature, dissolved oxygen, pH, alkalinity and carbon dioxide.

Those water quality parameters that have been shown to be affected indirectly by rotenone treatment are:

- 1) Phytoplankton levels - Both increases and decreases in the level of phytoplankton have been documented following rotenone.
- 2) Bacteria levels - Bonn and Holbert (1961) saw an increase in the number of bacteria per milliliter in both Texas lakes they rotenoned. They felt the increase could be due to the decay of dead fish and/or the agitation of the water and bottom sludge during the treatment. Since there was no corresponding increase in the Most Probable Number of coliforms, bacteria other than coliform constituted the increase. The bacterial increase was temporary, and the authors noted that most modern water treatment plants could cope with it without difficulty.
- 3) Turbidity/Transparency - Turbidity in water is caused by suspended matter, either organic or inorganic (American Public Health Association, 1971). Strictly speaking, it is not the same thing as transparency or visibility (usually measured by Secchi disc), though it is obviously related. In lakes that are turbid because bottom-scavenging fish constantly stir up sediments, poisoning with rotenone or other toxicants almost always results in reduced turbidity. However, in a deep lake with a coarse or gravelly substrate, turbidity from bottom-scavenging fish is not likely a problem. It is possible that nutrient re-suspension resulting in bloom conditions following a rehabilitation can reduce water transparency, although no studies were found to substantiate this speculation.

Increased water transparencies following carp poisoning have been reported in lakes in Illinois (Bennett, 1943), North Dakota (Needham, 1966), Colorado, (Tanner and Hayes, 1955),

Ohio (Weier and Starr, 1950), Wisconsin (Klingbeil, 1975), and Oklahoma (Eschmeyer, 1953). In Bass Lake, Indiana, removing the carp by seining produced the same results (Ricker and Gottschalk, 1940). None of these results were quantified and only refer to increased "visibility" making it difficult to determine which if two important factors - suspended silt, or algae - was responsible for the improvement. Other work has shown that carp and other bottom-feeders cloud the water not only by stirring up mud but also by increasing algae levels, and that the latter may be far more important in some lakes (Lamarra, 1975; Smeltzer and Shapiro, 1982). Some researchers specifically mentioned reductions in suspended silt or mud as the reason for improved water clarity (Cushing and Olive, 1957; Hoffman and Olive, 1961; Hoffman and Payette, 1956).

Only two studies have actually quantified turbidity (as distinct from transparency) following rotenone treatment: Bonn and Holbert (1961) recorded an 85% reduction in turbidity five days after poisoning Bonham State Park Lake, Texas. Wollitz (1962) cited a 54% drop in turbidity in Middle Pond, Montana. In both cases, the authors attributed the improvements to the elimination of bottom feeding fish. Wollitz (1962), however, reported no turbidity changes in a nearby pond containing few bottom-feeders that was also poisoned.

While decreased turbidity is generally considered a good thing, Bonn and Holbert (1961) suggested that clear water might allow a surge in algae growth. They cautioned that this would be undesirable in drinking water supplies if the algae consisted either of unpalatable blue-greens, or filter-clogging forms.

- 4) Water Taste and Odor - Researchers at several municipal water supplies have reported changes in the taste and odor of rotenone-treated water.

Of the 18 water quality tests performed by Bonn and Holbert (1961) on two Texas lakes, the greatest changes occurred in water taste and odor. They rated these changes using the Odor Number Test established by the American Water Works Association (American Public Health Association, 1971). Drinking water normally rated a "5-musty" before treatment changed to a "30-kerosene" odor number the day following treatment with rotenone. This was attributed to the hydrocarbon solvents in the rotenone formulations (Noxfish and Chemfish Special). Five percent rotenone powder produced no such kerosene odor in treated water. The kerosene odor disappeared five days after treatment.

A fishy odor was detected 17 days after treatment in one of the Texas lakes. The odor number in a treated lake cove became as high as "30-fishy" three days after treatment, then

disappeared six days later. These changes obviously occurred as a result of decaying fish.

Since it contains no petroleum-based carriers, Bonn and Holbert (1961) recommended 5% rotenone powder as a first choice when treating drinking water supplies. Their laboratory tests confirmed that rotenone powder by itself produced no change in odor number.

While both the kerosene and fishy odors were temporary, Bonn and Holbert's (1961) lab tests showed that both odors could be eliminated by a 1.0 ppm of activated carbon for each threshold odor number produced.

Cohen et al. (1960; 1961a; 1961b) made detailed laboratory and field tests of rotenone in drinking water supplies. They also concluded that the solvents, rather than rotenone itself, caused the kerosene odor. Like Bonn and Holbert, they concluded that activated carbon was the most effective way to reduce obnoxious odors resulting from emulsified rotenone formulations. Depending on the commercial rotenone formulation used, between 36 and 85 ppm activated carbon would be needed to make water with 2 ppm formulation immediately palatable.

Residual Toxicity in Drinking Water - Municipal drinking water supplies have been treated with fish-killing concentrations of rotenone in at least six states, with no harmful effects: Texas (Bonn and Holbert, 1961); Massachusetts (Stroud, 1956); California (Hoffman and Payette, 1956; State of California, 1983); Oklahoma (Eschmeyer, 1953); Indiana (Barry, 1967); and North Dakota (Cohen et al. 1961b). In some cases, rotenone treatment has been used specifically to improve or protect the drinking water quality (Hoffman and Payette, 1956; Barry, 1967). Cohen et al. (1960; 1961a; 1961b) performed the most extensive research on the effects of rotenone in public drinking water, and they concluded that rotenone treatment was "consistent with the objective of a water treatment: namely, to produce a safe and potable water".

Despite rotenone's relative safety, the U.S. Environmental Protection Agency (EPA), as a matter of policy, does not set tolerances for pesticides in drinking water. States such as California therefore require that whenever drinking water reservoirs are treated, that the rotenone be detoxified to undetectable levels (less than 0.005 ppm pure rotenone; Dawson et al., 1983) before it reaches the public. Detoxification can occur through natural breakdown, chemical treatment or both (State of California, 1985).

Rotenone breaks down quickly in the environment (Schnick, 1974), and retention time is long enough in most public reservoirs to allow complete natural detoxification (Bonn and Holbert, 1961;

Cohen et al., 1960). There are occasions when water may reach the treatment plant with some residual toxicity. Although there is little likelihood that it could have any effects on humans or livestock (Cohen et al., 1960; U.S. EPA, 1981), this residue must be removed, or chemically altered, to produce a finished drinking water of good quality. Cohen et al. (1960) made detailed recommendations for eliminating any residual toxicity using activated carbon. They also tested their laboratory finding in a drinking water supply in North Dakota (Cohen et al., 1961b), using 61 ppm activated carbon to detoxify a water supply treated with 2 ppm rotenone.

Both the State of California (1985) and the National Academy of Science (1983) have computed "safe" levels of rotenone in drinking water. California's figure was in the form of an Action Level (AL = the concentration of material in water above which human health may be adversely affected), while the Academy computed a Suggested No-Adverse-Response Level (SNARL). Both the AL and SNARL were based on long-term dosing study of the Midwest Research Institute (1980). Both California and the Academy applied a safety factor of 1,000 to the study's no-effect levels (10 for variability within species, 10 because the study was less than a lifetime, and 10 because the study is to be applied to humans). The SNARL for a 150-pound person who drinks half a gallon of treated water per day was 0.014 ppm pure rotenone; California's more conservative AL for a 22-pound child who drinks a quart of treated water per day was 0.004 ppm.

The detection of pure rotenone in water is approximately 0.005 ppm, slightly below the SNARL and slightly above the AL. The State of California (1985) therefore concluded that a conservative and justifiable requirement for human safety would be that no measurable levels of rotenone be allowed in public drinking water.

Effects of Trout Stocking - Bottom-feeding fish directly influence turbidity levels, and indirectly influence algae levels. Planktivorous fish - among them both stocked trout and numerous "target species" for rotenone - can also exert an indirect influence on algae. Algae levels, in turn, can affect the levels of ammonia, hydrogen sulfide, and hypolimnetic oxygen in a lake.

A lake stocked with trout or any other planktivorous fish will generally support higher algae levels than the same lake if it were fishless. This may partially offset by the periodic removal of other planktivores (e.g., perch or bluegills) with rotenone, and possibly the removal of nutrients from certain lakes through trout angling.

In annual stocking of trout-only lakes in Washington state, no change beyond those which have historically occurred as part of previous rehabilitation and stocking of trout only lakes in any

water quality parameter would be expected due to a post-rotenone introduction of the same (i.e., historical) magnitude.

Plants

According to most studies, phytoplankton is not directly affected by rotenone at concentrations of up to 3 ppm of the 5% dust (Bandow, 1980; Anderson, 1970; Wrenn, 1965; Kiser et al., 1963; Bonn and Holbert, 1961; Hooper, 1948; Smith, 1940; Smith, 1941; Brown and Ball, 1943a; Stenson, 1972).

Only two authors have reported toxic effects on phytoplankton: Wollitz (1962) stated that *Dinobryson* was absent for two weeks in a Montana pond treated with 0.7 ppm Pro-Noxfish. It returned to its former abundance two weeks later, and no other phytoplankters were affected. Almquist (1959) reported that concentrations of 5% rotenone above 2 ppm killed all *Volvox*, while 1 ppm was capable of destroying *Ceratium*. Anderson (1970), however, noted no decrease in either genus when subjected to 0.75 ppm.

Indirect Effects of Rotenone and Trout Stocking - It is difficult to summarize the indirect effects of rotenone and subsequent trout stocking as there are a greater number of trophic links involved.

Figure 1 is a flow chart showing the most important ways in which rotenone poisoning and subsequent trout introductions may influence lake algae levels. It is assumed for simplicity's sake that the two main factors that influence algae growth are the amount of phosphorous (P) available and the level of grazing by zooplankton.

While productivity in some lakes is limited by other nutrients (e.g. nitrogen, silicon, CO₂) algal growth in most culturally eutrophic lakes is controlled by the amount of phosphorus available (Schindler, 1974; Vollenweider, 1968). Within the limits normally found in lakes, Figure 1 illustrates the valid generalization that when phosphorus increases, so do algae levels; when phosphorus decreases, algae is reduced.

There is also ample evidence in the literature supporting the second assumption made in Figure 1: increased grazing by zooplankton generally crops down algae, while decreased grazing boosts algal biomass (Gliwicz, 1975; Shapiro et al., 1975). There are important exceptions, the first to be discussed is the pathways in which rotenone and trout stocking affect phosphorus levels. Rotenone treatment of a lake potentially affects phosphorus levels in two ways:

- 1) the numbers of bottom-feeding fish (such as carp and bullhead) decrease, which in turn may reduce phosphorus levels; and
- 2) dead fish decay on the lake bottom releasing the phosphorus

bound in their carcasses and possibly creating anoxic bottom conditions which could release phosphorus from the lake sediments.

Trout stocking also affects phosphorus levels in two ways:

- 1) trout not caught by anglers die and decay on the bottom, increasing phosphorus as in (2) above; and
- 2) trout caught by anglers represent a loss of phosphorus from the system.

Effect of Bottom-Feeding Fish on Phosphorus Levels - Bottom-feeding fish such as carp, goldfish, and bullheads have for years been associated with murky water (Moyle, 1968). Some of the reduced transparency is due to suspended silt stirred up by the fish as they scavenge the bottom especially in shallow lakes. But algal blooms associated with these fish can play an important, if not overriding role in clouding the water.

It is possible to separate the effects of silt and algae by plotting the reciprocal of secchi disk transparency against chlorophyll concentrations (Brezonik, 1978); the intercept of the regression represents the amount of murkiness due to substances other than algae (e.g. silt) in the water. Smeltzer and Shapiro (1982) did this in a carp and bullhead infested Minnesota lake, and found that most of the light attenuation (71%) was caused by algae; stirred-up silt was only a minor contributor.

Empirical evidence that bottom-feeding fish can cause algae blooms comes from lakes where these fish have been poisoned: Hoffman and Payette (1956) killed 107 tons of carp with rotenone in a San Diego reservoir and within a month noted marked decreases in most algal counts and increased transparency (though a diatom bloom took place). Needham (1966) found that chlorophyta decreased steadily and remained at low concentrations after poisoning bottom fish in North Dakota lakes. Bandow (1980) reported that reduced algal levels followed bullhead removal in a number of Minnesota lakes. Hrba'cek et al. (1961), Stenson et al. (1978), and Schindler and Comita (1972) have all documented similar improvements following the demise of bottom-feeders.

It was once widely accepted that bottom fish release nutrients (such as phosphorus) into the lake by stirring up the bottom sediments; in turn, these nutrients fostered algae blooms. While agitation does release phosphorus (Zicker et al., 1965), there is usually more phosphorus absorbed by aerobic sediments than lost (Fitzgerald, 1970); if bottom fish were releasing phosphorus and causing algal blooms, some other mechanism must be involved. Using carp, Lamarra (1975) proved that it was mostly the digestive activity of benthivorous fish that released phosphorus from the sediments and, more importantly, raised chlorophyll levels. Simple

mechanical stirring of the bottom, on the other hand, did not release appreciable amounts of phosphorus nor did it increase algae levels. Lamarra also showed that release of phosphorus in all its forms was negatively correlated with fish size (i.e., bigger carp release less phosphorus) and that 50% of the total phosphorus excreted by all sizes of carp was in the form of orthophosphate, which is immediately available for algal growth, Figures 2 and 3 display the relationships between carp size, carp density, and sediment phosphorus release from Lamarra's experiments. The actual excretion rate of dissolved phosphorus for a specific weight class of carp or bullhead may be computed from Lamarra's regression equations:

$$1) \quad \text{for carp:} \quad \log_{10} E(\text{DP}) = -.49 \log_{10} W + .027T + .77$$

$$2) \quad \text{for bullhead:} \quad \log_{10} E(\text{DP}) = -.379 \log_{10} W + .027T + .344$$

where:

$E(\text{DP})$ = specific excretion rate of dissolved phosphorus
(micrograms/gram wet weight per hour)

W = wet weight of fish (g)

T = temperature ($^{\circ}\text{C}$)

With an estimate of fish biomass for various size classes in a lake, it is possible to compute the annual phosphorus loading due to carp and bullhead. Lamarra performed these calculations for the typical "rough-fish" lake in Minnesota. Such a lake contains about 200 kg of carp/ha, and Lamarra estimated that they recycled between 1.07 mg and 2.18 mg total P/m²/day, or 0.52 mg orthophosphate/m²/day. Even the smaller, more conservative estimate is surprisingly high, and Lamarra concluded that carp were probably liberating amounts of sediment phosphorus that were significant in terms of the lakes' total phosphorus budgets.

In view of its ability to liberate large amounts of phosphorus from lake sediments, Lamarra termed the carp a "phosphorus pump". This ability is not confined to carp alone; the bullhead is also an important "phosphorus pump" (Lamarra, 1976; Shapiro et al., 1975; Bandow, 1980). Although no quantitative data exist, we can probably add the goldfish to this list in view of its genetic similarity to the carp (the two interbreed in the wild).

Smeltzer and Shapiro (1982) further investigated the significance of these experimental findings in a lake dominated by black bullheads and carp. They found that bullheads at a density of 59 kg/ha and carp at 43 kg/ha were contributing 88 mg of P/m²/year to Lake Marion, Minnesota. This same eutrophic lake was receiving 84 mg of P/m²/year from external sources. The conclusion that benthivorous fish were supplying the lake with as much phosphorus as all external sources combined (drainage, rain, and septic tank seepage) is astounding, and implicates them as major contributors to algae blooms.

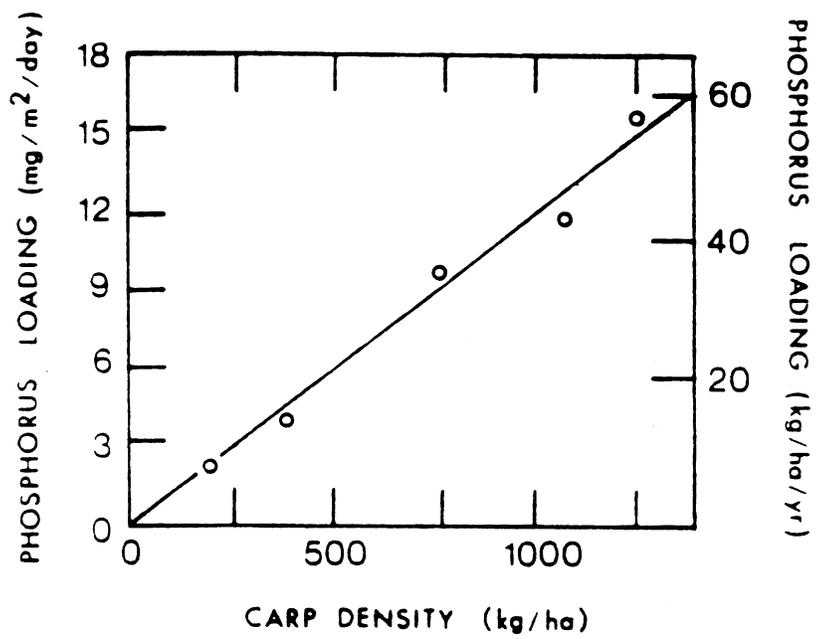


Figure 2. Relationship between carp density and phosphorus release. Carp were between 140 and 180 grams wet weight. Source: Lamarra 1975.

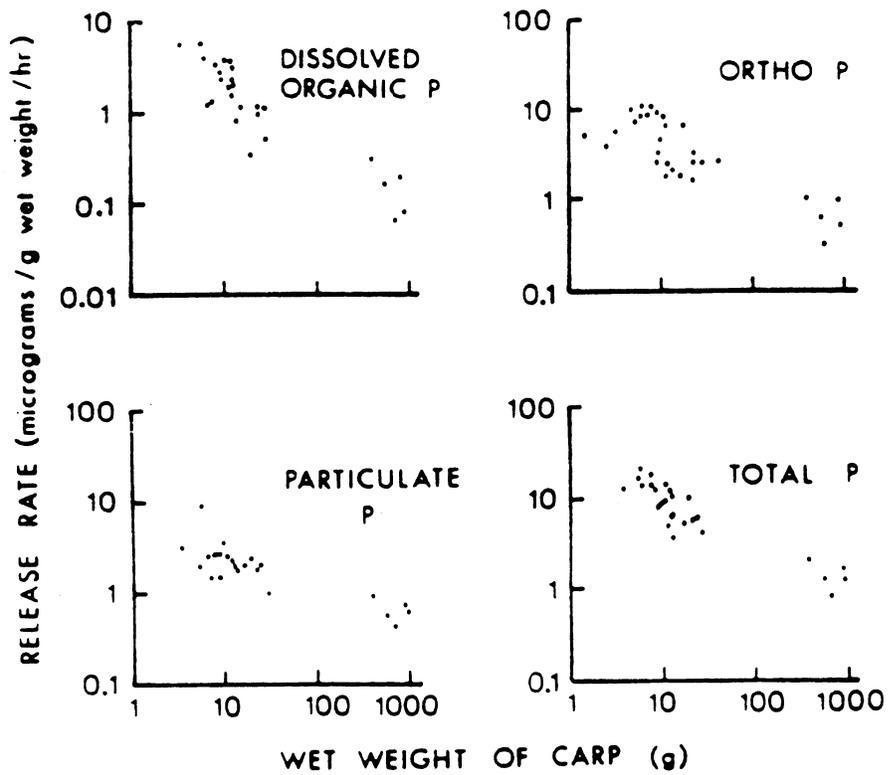


Figure 3. Release of various forms of phosphorus (P) by different-sized carp in 22° C water. Source: Lamarra 1975.

It may be concluded that in lakes infested with carp or bullheads, their removal by rotenone or other methods will substantially decrease phosphorus levels and thus algae levels. There are three complications which may amend this conclusion:

- 1) This same phosphorus can also be released from lake sediments in the absence of fish, particularly when the bottom becomes anoxic (Hutchinson, 1957). In lakes where this is a major source of annual phosphorus loading, the effect of bottom-feeding fish may be negligible. At least in Marion Lake, Smeltzer and Shapiro (1982) believed that excretion by carp and bullheads was by far the most important internal source of phosphorus.
- 2) Aquatic macrophytes such as *Elodea* also act as "phosphorus pumps" (Bandow, 1980; Welch, 1980) and these may proliferate in the absence of bottom fish which uproot and destroy them. This was the case in 14-foot deep Carls Lake, Minnesota (Bandow, 1980); after black bullheads were poisoned with rotenone, aquatic macrophytes (including *Elodea*) were no longer held in check, and expanded to occupy the entire lake bottom. These plants released large amounts of phosphorus and ammonia from the sediments, essentially negating the water quality benefits gained by killing the bullheads. Bandow did note, however, that in deeper lakes the growth of rooted aquatic plants should be less extensive.
- 3) When benthivorous fish are eliminated by any method which leaves their carcasses in the lake (e.g., rotenone), the phosphorus released by decay will at least temporarily mollify the beneficial effects of destroying them. For example, Smeltzer and Shapiro (1982) calculated that the phosphorus released by decaying carp and bullhead carcasses following rotenone treatment of Marion Lake would be equal to a third of the sediment phosphorus liberated by the fish if they were alive. As a consequence, phosphorus levels and algae would not decline as rapidly as expected following treatment. However, the authors pointed out that phosphorus release from fish decay constituted only a single "pulse" of loading, whereas excretion was a chronic source.

In summary, elimination of bottom-feeding fish with rotenone can be expected to lower phosphorous levels and thus algal abundance in lakes where:

- 1) algal abundance is limited by phosphorus; 2) lake sediments contain substantial amounts of phosphorus; and 3) potential sediment phosphorus released by fish is high compared to other mechanisms (e.g., sediment phosphorus release during periods of oxygen-depletion in the bottom muds).

The degree of phosphorus reduction may be roughly predicted from Lamarra's (1976) equations when estimates of fish biomass, size distribution and phosphorus loading from other source are available.

Effect of Decaying Fish on Phosphorus Levels - The phosphorus content of fish has been studied by several authors (Table B). Phosphorus made up between 0.3 and 1.6% of the whole-fish wet weight in these studies. While phosphorus content of fish varies somewhat with species, age, sex season, sexual maturity, and trophic state of the lake (Vinogradov, 1953; Dunst et al., 1974), Bull and Mackay (1976) suggested that an average value of 0.4% is adequate for a wide range of fish populations.

When fish die, the phosphorus bound in their carcasses must be broken down into the dissolved inorganic or organic form, mostly by bacterial action and autolysis, before it is usable by phytoplankton. This breakdown is extremely rapid; Wetzel (1983) states that "small fish" lose 7% of their total substance immediately upon death, and that 28% has been released within 24 hours under aerobic conditions in 20-25° C water. Once phosphorus is in an available form it is taken up so rapidly by algae and other plants that it is often not measurable. The release of this phosphorus from fish carcasses following rotenone treatment has been suggested as a cause of algae blooms (Funk and Moore, 1984).

Most fish killed by rotenone sink to the bottom of lakes undetected. It has been estimated that at 57-58° F (the average fall surface water temperature of lakes treated in Washington), only about 20-30% of the dead fish would surface within 24 hours. Thus, even when a concerted effort is made to recover all carcasses, at least 70% of the phosphorus content of the fish stock will be released into the lake through decay.

One final figure is necessary to estimate the amount of phosphorus (in g/ha) released by decaying carcasses: the total weight of fish per unit of surface area, or standing crop. This can vary considerably depending on the lake and the fish present. Bennett (1962) presented mean standing crop values for nineteen fish (usually in combination with other species) in U.S. lakes and reservoirs. Mean values for fish found in Washington state ranged from 4 lbs/acre (4.5 kg/ha) where trout dominated, to 100 lbs/acre (112 kg/ha) where carp dominated. The maximum standing crop recorded for U.S. waters was 1,235 lbs/acre (1,384 kg/ha) in Iowa ponds (Bennett, 1962; Dunst et al., 1974). Two Indiana reservoirs that contained a mixed population of warmwater species (mostly bullheads, bluegill, and carp) were rotenoned and then completely drained; this procedure provided standing crop figures of 153 lbs/acre and 300 lbs/acre (171 and 336 kg/ha) for the two lakes (Barry, 1967).

Table B. Phosphorus (P) content of fish.

Species	% of Wet Weight	Reference
Fish in general	0.3	
Atlantic salmon	0.168 a/	Vinogradov (1953)
Brown trout	0.246 a/	
Black crappie	0.7 b/	Burgess (1966)
Bluegill	0.8 b/	
Redear	0.6 b/	
Warmouth	0.5 b/	
Gizzard shad	0.6 b/	
Golden shiner	0.5 b/	
Brown bullhead	0.5 b/	
Longnose gar	1.6 b/	
Sockeye salmon (prespawning)	0.384	Donaldson (1967)
Sockeye salmon (spent)	0.345	
Rainbow trout	0.4	Bull and Mackay (1976)
Carp	0.5	
Northern squawfish	0.4	
Largescale sucker	0.3	

a/ Listed as phosphorus content of "soft part" of fish; may not reflect percentage of the whole fish.

b/ Burgess' figures originally reflected percentage of phosphates in the fish. Here his figures have been modified in accordance with Dunst et al. (1974), who reported percentage of phosphorus.

In Washington lakes proposed for rotenone treatment - often characterized by an out-of-balance fish population - total standing crop is often on the order of 75 lbs/acre (84 kg/ha); but where carp or goldfish dominate, this figure can be much higher (Fletcher, WDW, pers. comm.). For example, Picnic Point Pond in western Washington contained about 362 kg of goldfish (and almost no other fish) when rotenoned in 1980, yielding 228 kg/ha or 203 lbs/acre (calculated from data collected by University of Washington students and WDW biologist; Washington Department of Wildlife, 1981; and length-weight regressions for goldfish in Carlander, 1969).

Roughly, the decay of fish killed by rotenone could release as much as 0.3 kg P/ha into an out-of-balance mixed species water with 84 kg fish/ha for a carp or goldfish infested water with 300 kg fish/ha, the estimate jumps to 1.2 kg P/ha. These estimates are based on P = 0.4% of the wet weight of a fish.

In one respect, whatever the biomass of decaying fish and consequent phosphorus release, the phosphorus released by carcasses is phosphorus that would be released in any event when the fish die a natural death. On the other hand, in addition to the large biomass of "target" fish that are killed, there are always some residual trout left in the lake at the time of rotenone treatment. These are stocked fish, and thus the phosphorus in their carcasses represents an addition of phosphorus to the lake that occurs as a result of the trout program. Generally, however, the biomass of trout in a lake designated for rotenone treatment is small. These lakes are usually taken out of production as fry growing lakes the year of the treatment and given a nominal stocking of legal sized trout. Most of these fish are readily caught before the fall treatment. For example, 38% of the stocked legals in Pine Lake, Washington were taken by fishermen on Opening Day, 1980, and the majority removed within a week (Zisette, 1981). From a nutrient-loading standpoint, the essential difference between natural mortality and rotenone poisoning is that in the case of rotenone, all the phosphorus contained in the lake's standing crop of fish is released at the same time, rather than gradually.

There is no way to carry the estimate of how much of this phosphorus could become available for algae growth without some knowledge of a particular lake's limnology; too many factors influence the fate of phosphorus, of which the most important are:

- 1) Flushing rate of the lake . In rapidly flushing lakes, even high phosphorus loads can be insignificant (Welch, 1980). This may occur in some Washington lakes that are rotenoned in the fall, just prior to relatively massive rainfall and flushing. Naturally, the effect of sudden nutrient release in a lake with little outflow would be greater.

- 2) Conditions at the water-sediment interface . If the lake's hypolimnion is aerobic, much of the phosphorus released from the carcasses will be quickly tied up by metal complexes and resettle, unavailable for algae growth. Anaerobic conditions, on the other hand, would allow much of this phosphorus to reach the overlying water where it could be used by algae.

In many instances algae blooms occur shortly after rotenone treatment and some authors suggest that the release of phosphorus from decaying carcasses is a contributing factor. Table C shows the results of several studies where algal abundance was measured or noted shortly after rotenone treatment. In nine of eleven bodies of water, an algae bloom developed following rotenone treatment (although on Fern Lake, an application two years later produced no bloom). A "bloom" in this case is any increase in total algae (measured in chlorophyll *a*, cells/l, etc.) thought to be significant by the investigator(s).

As Table C shows, it is impossible to determine exactly why blooms occurred following rotenone. The two most likely hypotheses are phosphorus released from carcasses and/or a decrease in grazing following the annihilation of zooplankton, but it is impossible to separate the effects of the two. While no definitive answer exists, it is interesting to note that there were no fish in Burress' (1982) ponds, yet a bloom still developed following rotenone. Clearly, carcasses played no role in that case. Also, no bloom developed on Third Sister Lake (Brown and Ball, 1943a), zooplankton was only mildly affected by rotenone, cladocerans were never absent from the open water. These two examples seem to suggest that phosphorus released from fish carcasses is not nearly as important as reduced phosphorus grazing in causing algal blooms. Contradictory evidence from Carls Lake (Bandow, 1980) and Fern Lake 1962 (Fowler, 1973) - where no blooms developed despite the near annihilation of grazers - make firm conclusions impossible. Where they occur, it is likely that both phosphorus release from carcasses and reduced grazing are responsible for post-rotenone algae blooms, with the relative importance of each determined by the particulars of each lake.

Quantitative data are available from seven of the studies listed in Table C. These are graphed in Figures 4 through 10, showing the timing and magnitude of post-rotenone algae blooms where they developed.

Comparing "bloom" levels in a rotenone year with the algae levels during that same period in a nonrotenone year is perhaps the best way to gauge the magnitude of these blooms. These type of data are available for Pine Lake, Washington and Hodges Reservoir, California. On Fern Lake, Washington, a continuous 12-year record of phytoplankton levels provides us with five seasons of data in nonrotenone years for comparison with the bloom that followed

Table C Response of phytoplankton shortly after rotenone treatment in selected lakes.

Name, location	Month of treatment	Length of study before treatment	Length of study after treatment	Did Algal bloom occur shortly after rotenone?	REASON FOR BLOOM			Comments	References
					Increase in nutrients due to decay of zooplankton	Decrease in grazing by zooplankton	Other		
Pine Lake, western Washington	October	1 year, 3 months	2 months	yes	x	x		<p>"...chlorophyll <u>a</u> increase... likely influenced by absence of grazing pressure." "Phosphorus remineralization of the decomposing organisms may have been at least partially responsible for an observed elevation in phosphorus." -Zisette 1981 -Walch et al. 1981 -Municipality of Metropolitan Seattle 1981</p>	
Fern Lake western Washington	June	2 years	10 years	yes (1960) no (1962)		x		<p>"slight increase in phytoplankton... occurred several weeks after the application of rotenone" in 1960. -Kiser et al. 1963 -Foeller 1973</p>	
Bonham State Park Lake Texas	April	1 day	17 days	yes	x	a/ x	b/ x	<p>"There was an increase in the number of (phyto-)plankton after addition of rotenone products." Born & Holbert 1961</p>	
Lake Lavon (Price Creek Cove) Texas	June	unclear ^{c/}	unclear ^{c/}	yes	x	a/ x	b/ x	<p>Born & Holbert 1961</p>	
Patricia & Celestine Lakes Alberta, Canada	September	1 month	3 years, 5 months	yes				<p>"Compared with subsequent years and pre-rotenone abundance, there was a small phytoplankton bloom shortly after treatment." Anderson 1970</p>	

Table C Continued

Name, location	Month of treatment	Length of study before treatment	Length of study after treatment	Old Algal bloom occur shortly after rotenone?	REASON FOR BLOOM			Comments	References
					Increase in nutrients due to grazing by zooplankton	Decrease in grazing by zooplankton	Other		
Ponds I & II Georgia	---	1 day	1 week	yes			"Substantial phytoplankton blooms developed within 3 days" [compared to control pond].	Burress 1982	
Hodges Reservoir California	February	1 year, 1 month	3 weeks	yes			"Subsequent to treatment with Pro-Horfish it was observed that a diatom pulse was developing."	Hoffman & Payette 1956	
Carls Lake Minnesota	September	1 year, 5 months	2 years, 1 month	no			Although chl <u>a</u> and phytoplankton in general declined, author noted a "strong increase" of <u>Melosira</u> and <u>Cryptomonas</u> following rotenone, which he credited to decrease in grazing following the kill.	Bandow 1980	
Third Sister Lake Michigan	May, August	1 year	1 year	no			"Most of the phytoplankton groups showed little or no change following the introduction of rotenone. A gradual decrease throughout the summer was noticeable for <u>Chroococcales</u> with an accompanying increase in diatoms... and <u>Dinobryon</u> ."	Brown & Ball 1983	

a/ authors actually gave reason as "reduction in plankton-feeding fishes," but none existed in the lakes; we assume they were referring to herbivorous zooplankton.

b/ "additional depth of light penetration" given as other reason for bloom.

c/ samples taken "at approximate 2-week intervals during the year resulted in a total of 180 analyses for 18 trips."

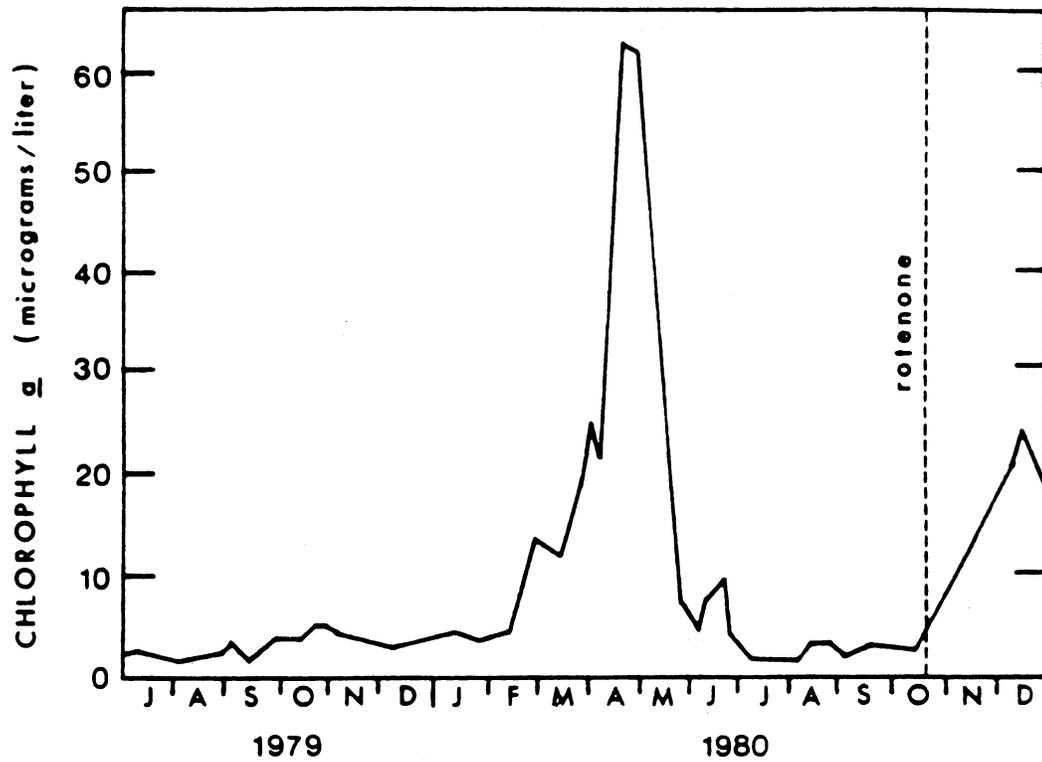


Figure 4 Volume weighted mean chlorophyll a content in the epilimnion of Pine Lake, Washington, before and after rotenone. Source: Welch et al. 1981.

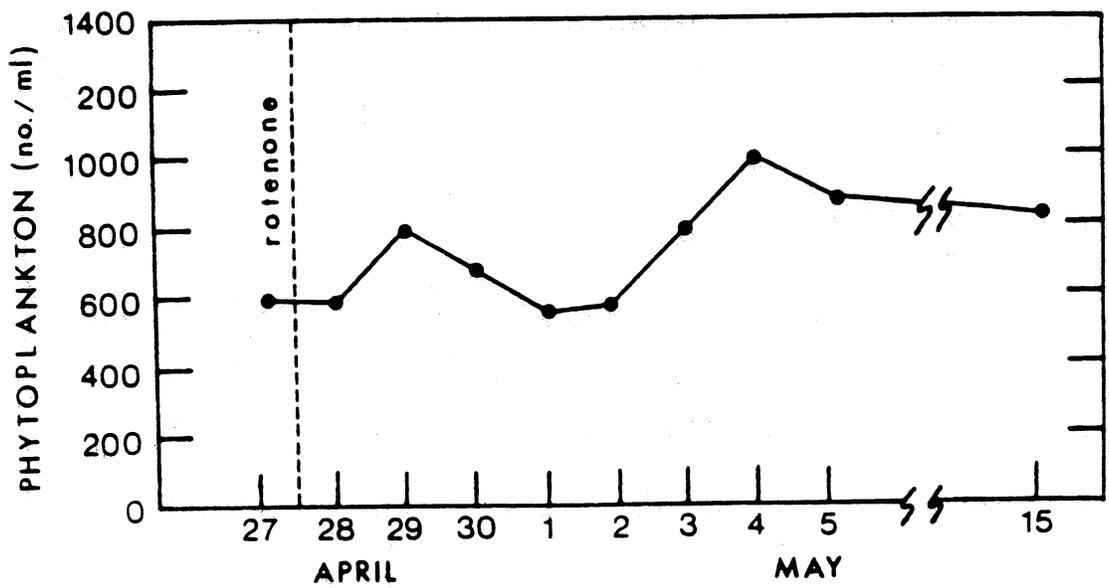


Figure 5 Phytoplankton levels in Bonham State Park Lake, Texas, before and after rotenone. Source: Bonn and Holbert 1961.

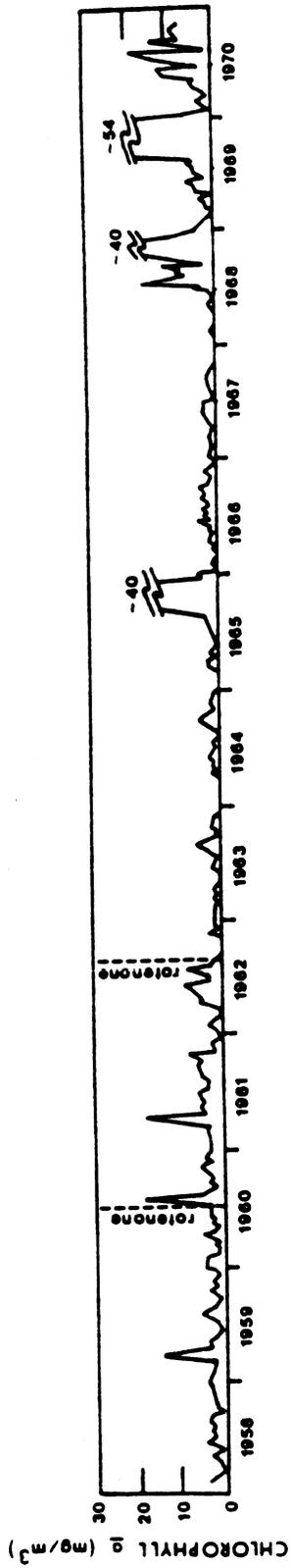


Figure 6 Abundance of algae (as chlorophyll a) in Fern Lake, Washington over a twelve-year period. Rotenone was applied in 1960 and 1962. Artificial fertilizers were applied in 1965, 1968, and 1969. Source: Fowler 1973.

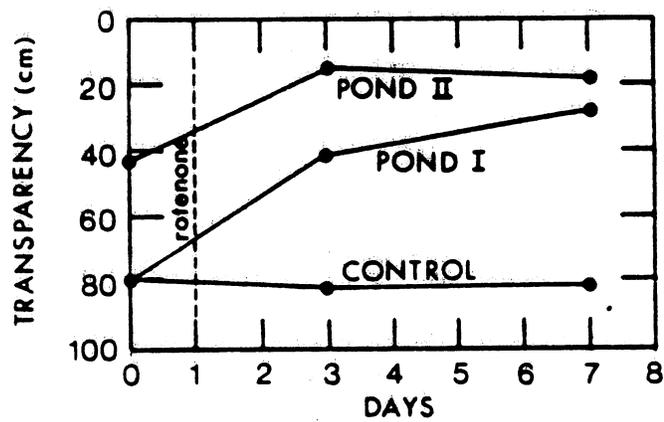


Figure 7. Secchi disc transparency in two ponds treated with rotenone, and one untreated control pond. Source: Burress 1982.

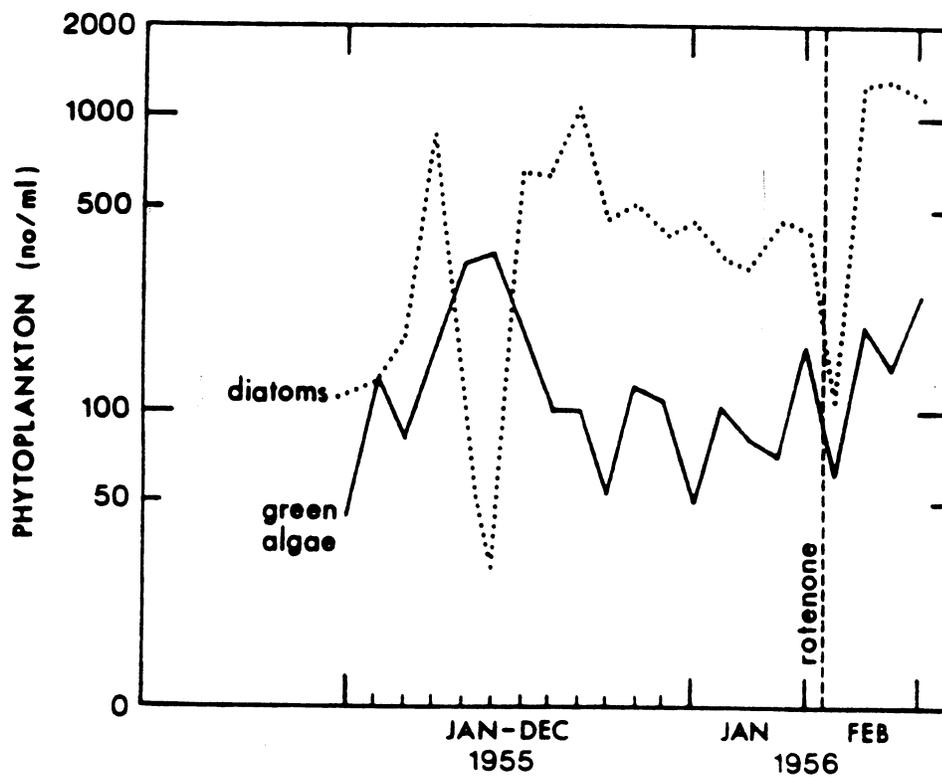


Figure 8. Diatom and green algae levels in Hodges Reservoir, California, before and after rotenone. Source: Hoffman and Payette 1956.

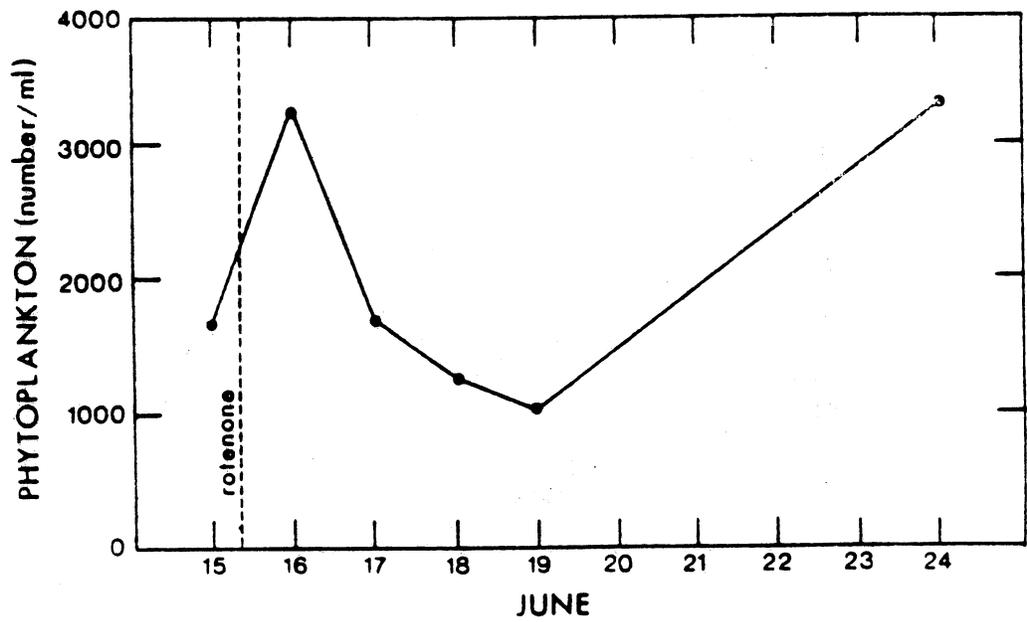


Figure 9. Phytoplankton levels in Lake Lavon (Price Creek Cove), Texas, before and after rotenone. Source: Bonn and Holbert 1961.

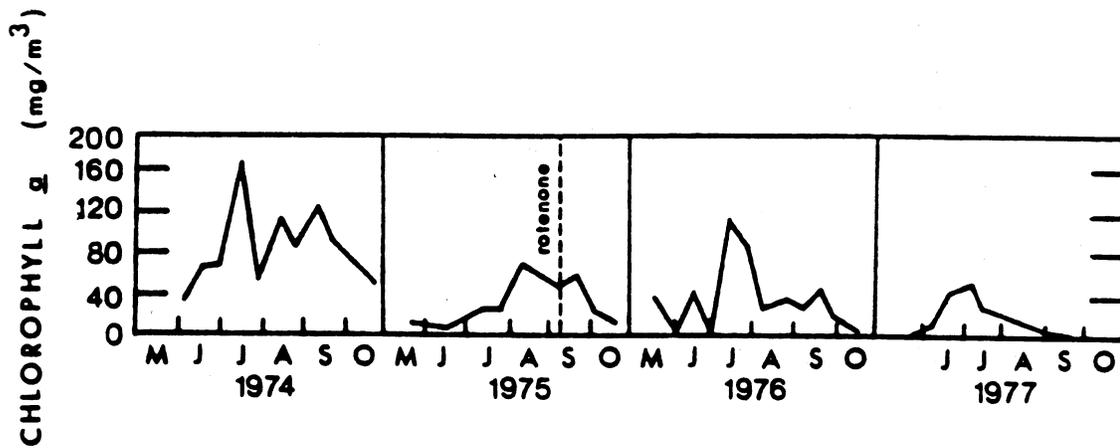


Figure 10. Phytoplankton levels (as measured by chlorophyll *a*) in Carls Lake, Minnesota, before and after rotenone. Source: Bandow 1980.

the rotenone treatment in 1960 (artificial fertilization and a second rotenone treatment obscure results in the other years).

The algae levels in rotenone and nonrotenone years are shown for these three lakes in Table D. Algae levels increased 4 to 6 fold shortly after rotenone treatment, when compared with "normal" levels in nontreated years. It is important to remember that blooms do not always occur following rotenone, even in a particular lake (i.e., Fern Lake). Table D demonstrates the magnitude of post-rotenone blooms where they occurred. These blooms generally lasted 1-2 months, judging by the studies on Pine, Fern and Hodges lakes. Most rotenone applications in Washington take place in the fall; a bloom, if one occurs, would be expected to subside sometime in December with decreasing sunlight and flushing, as was the case on Pine Lake in 1980.

There is little information whether the increased phosphorus from fish carcasses decaying on the bottom will cause algae blooms (or intensify regularly-occurring blooms) the following season. Algae levels in Fern Lake in 1961 and 1963 (following the 1960 and 1962 treatments) indicate nothing out of the ordinary when compared to other years. These data agree with the wealth of information available from lakes and ponds that have been artificially fertilized; Shapiro (1970) cited the early work of Einsele and Edmondson, noting that "single-shot" fertilization with superphosphates was ineffectual - the results lasted only for the year of application and did not carry over to any extent to the next year unless the fertilization was repeated. More recent experiments in Canada have confirmed this: when Schindler and Fee (1974) fertilized a lake with phosphorus, algae increased dramatically during the two treatment years, but fell to pretreatment levels as soon as phosphorus input was curtailed. Figure 15 shows the same pattern in Fern Lake, which was fertilized in 1965, 1968 and 1969. This is generally true of lakes in the low or medium productivity range (Wetzel, 1983). In eutrophic lakes this fertilizer might be recycled from the sediments, causing further blooms.

There is an essential difference between artificial fertilization and phosphorus released from rotenone killed carcasses: artificially fertilized lakes (including the great many eutrophic lakes that receive phosphorus from septic tanks, runoff, etc.) are enriched by phosphorus from external sources. In a rotenoned lake, the sudden enrichment comes from fish that obtained all their phosphorus from the lake. Thus, there is no net increase in phosphorus, only a sudden and unusual availability that takes place following the poisoning. This essentially confines the bloom potential to the year of treatment, even in eutrophic lakes. For example, researchers concluded that on culturally-eutrophic Pine Lake, "phosphorus remineralization of decomposing organisms (following the 1980 rotenone treatment) may have been at least partially responsible for an observed elevation in whole-lake phosphorus

Table D. Magnitude of algal blooms following rotenone treatment in some test waters. Values are approximations only, in some cases based on interpretation of graphic data. Blooms on these lakes lasted 1-2 months.

Lake, year	Method of comparison	Unit	ALGAE LEVELS			Reference
			Nonrotenone year(s)	Rotenone Year	% Increase	
Pine Lake western Washington 1980	means of November and December chl <u>a</u> levels in 1979 (no rotenone applied) and 1980 (rotenone applied on October 23).	ug chl <u>a</u> / l	3.5	18.5	429%	-Zisette 1981 -Welch et al. 1981
Fern Lake western Washington 1960	means of peak chl <u>a</u> levels nearest the end of June in 1958, 1959, 1961, 1963, and 1964 (no rotenone applied) with peak in 1960 (rotenone applied on June 4).	mg chl <u>a</u> / m ³	13.4	59.7	346%	Fowler 1973
Hodges Reservoir California 1956	mean diatom level in February 1955 (no rotenone applied) with February 1956 (rotenone applied January 31).	#/ml	125	730	484%	Hoffman & Payette 1956

(about 5 micrograms/l while inflow phosphorus was negligible). Available data, though inconclusive, suggest that existing autumn rotenone applications do not have a significant impact on annual phosphorus dynamics and algal blooms in Pine Lake" (Municipality of Metropolitan Seattle, 1981).

As shown in Figure 1, there is a second way in which decaying fish carcasses have the potential for increasing phosphorus levels: decomposition requires oxygen in most cases, and large numbers of fish carcasses may turn a lake bottom anaerobic. Sediment phosphorus is normally trapped (or release is insignificant) in aerobic bottoms where the overlying water contains more than 1 mg oxygen per liter (Wetzel, 1983). But when oxygen levels fall below this point, redox potentials also decrease, and a sudden release of phosphate phosphorus occurs from the sediments. When the water is reoxygenated, phosphate phosphorus is again resettled and trapped in the sediments. In this case, the source of phosphorus is not the fish carcasses themselves, but the lake sediment that has become depleted of oxygen by their decay. Phosphorus released from the sediments by any means can be a major cause of algae blooms (Cook et al., 1977).

Almost no data are available from rotenoned lakes on this subject. While a number of studies routinely recorded oxygen levels at various depths before and after treatment, there is often no mention of whether or not the fish carcasses were removed from the study lake or allowed to decay. In others, such as Pine Lake, (Welch et al., 1981), the bottom was anoxic even in nonrotenone years.

In research designed to determine the indirect effects of rotenone on municipal water supplies, Bonn and Holbert (1961) poisoned two Texas lakes, killing 79 pounds of fish per acre on one and 145 lbs/acre on the other. After a weigh-in, the body cavities of the fish were punctured and they were scattered back into the lakes to create a natural post-kill condition. Oxygen levels as well as other chemical and biological parameters were measured at various depths, including the bottom. Water temperatures were high (70-91° F) and the carcasses decayed rapidly, with significant increases in total organic nitrogen and bacterial levels. Although some oxygen must have been consumed by the bacterial decomposition, Bonn and Holbert reported no significant change in oxygen levels. A "bloom" did develop in both lakes, but not because of anaerobic phosphorus release from the sediments. However, these Texas lakes were fairly shallow (maximum depths of 15.5 and 9 feet), and oxygen depletion in deeper lakes, where oxygen diffusion from the surface takes more time, might still occur.

The hypolimnia of many lakes - especially eutrophic ones - typically become anoxic during the summer and winter (Welch, 1980). Where this yearly pattern occurs, rotenone-killed fish carcasses cannot be expected to aggravate sediment phosphorus release, since

oxygen levels are already below 1 mg oxygen/liter. In culturally eutrophic Pine Lake, for example, the October 1980 rotenone treatment occurred well after the bottom became completely anoxic in early July; as in other years, the lake turned over in early December and the bottom was reoxygenated (Welch et al., 1981).

Effect of Trout Stocking on Phosphorus Levels - When a fingerling trout is stocked in a lake, the 0.4% of its wet weight that is phosphorus is added to the system. When and if the trout is caught by an angler, this amount of phosphorus plus 0.4% of the added weight that the trout has gained during its growth period in the lake is removed.

Phosphorus tied up in the tissue of living fish is not immediately available for use by algae; but it is quickly released back into the water if the fish dies in the lake. In the same way, a fish removed from the lake represents a loss from the total phosphorus pool and ultimately from the available phosphorus pool.

Fish stocking records and catch estimates can be used to determine if the process of stocking and harvesting trout fertilize lakes or reverse fertilization. While fish stocking records are readily available for all of Washington's stocked lakes, creel survey estimates of season-long catch are more difficult to come by. Where season-long catch estimates are not available, catch estimates from opening day of lowland fishing season (usually occurring on the third or fourth Sunday in April) can be used as a minimum value for phosphorus removal by angling. Creel surveys are performed on all of Washington's lowland trout lakes on opening day, and the statistical methodology for estimating catches of fingerling-origin trout on these lakes is well developed (Brown, 1978).

Table E displays the estimates of phosphorus added and removed by trout stocking and harvest on selected Washington lakes. All are lowland "trout-only" waters, most have been treated with rotenone, and they are fairly representative in terms of stocking and catch rates. These particular lakes were selected because reliable season long (or opening day) catch estimates were available.

Only the Kitsap County lakes surveyed by Johnston (1973) showed a net gain in phosphorus; Table E indicates that in most cases, more phosphorus is removed from trout-only lakes than is added. The amount varies considerably, however, mostly depending on the percentage of the fingerling introduction that is caught. In eastern Washington lakes, this percentage is typically high, while in western Washington it is usually much lower. This difference is due mostly to fingerling survival: mark-recapture studies have shown that survival from stocking to opening day ranges from 2% to 61% in western Washington lakes and from 70% to 87% in eastern Washington lakes (Washington Department of Wildlife, 1968).

Table E Estimated annual amounts of phosphorus (P) added and removed by fingerling trout stocking in selected Washington lakes.

Name Year(s) Reference	Various Lakes									
	Quincy Lake 1981 Jackson 1983	Susan Lake 1981 Jackson 1983	Lower Hampton 1981 Jackson 1983	Upper Hampton 1981 Jackson 1983	Liberty Lake 1978 WDG files	Lake Morton 1974 Cummins 1975	Martha Lake 1978, 1980/ 1972, 1973	In Kitsap Cty. 1972, 1973	Pine Lake 1979	WDG files
fingerling stocking rate (fish/acre/yr)	610	607	668	225	387	377	463	400	454	
fingerling size (fish/lb)	130 c/	130 c/	130 c/	130 c/	80	110	170	128	150	
fingerling stocking rate (kg/ha/yr)	5.26	5.23	5.76	1.94	5.42	3.84	3.05	3.50	3.39	
P added by fingerling stocking (g/ha/yr)d/	21	21	23	8	22	15	12	14	14	
Survey period	season long	season long	season long	season long	season long	season long	Opening Day	Within 3 weeks of Opening Day	Opening Day	
% of fingerling plant caught	45.3	37.9	65.3	45.8	60.0	21.0	5.4	1.8	3.0	
catch (fish/ha/yr) ^{e/}	541	568	1078	256	574	196	62	18	34	
catch (kg/ha/yr)	162	170	323	77	86	27	9	3	5	
P removed by fishery (g/ha/yr)	648	680	1292	308	344	108	36	12	20	
Δ P (g/ha/yr)	-627	-659	-1269	-300	-322	-93	-24	+2	-6	
P load (g/ha/yr)					3700 f/				4146 g/	
P removal as a % of P load					8.70%				0.14%	

a/ refers to harvest year - fingerlings were planted the previous year.

b/ mean of both years' data.

c/ average size for these lakes (Joe Foster, WDG biologist, personal communication).

d/ assumes that P = 0.4% of a trout's wet weight (Bull and Mackay 1976).

e/ weight/fish = 0.14 kg for lakes in Kitsap County, and lakes Morton, Martha, and Pine, based on average Opening Day fork length of 8.75 inches (Johnston 1973) and length/weight regressions in Carlander (1969). Weight/fish \approx 0.15 kg for Liberty Lake, based on average Opening Day size (John Hisata, WDG biologist, personal communication). Weight/fish \approx 0.3 kg for lakes Susan, Quincy, and Hampton, based on Opening Day size of 11.5-12.0 inches (Jackson 1983) and regressions in Carlander (1969).

f/ 1974-78 pre-restoration figures (Ron Pine, D.O.E., personal communication).

g/ from WDG files 1981

Table E shows that net removal of phosphorus in the eastern Washington lakes varied between 300 and 1,269 g/ha/yr. In the western Washington lakes, net removal ranged from 6 to 93 g phosphorus/ha/yr., with a net addition of 2 g of phosphorus/ha/yr in the Kitsap County lakes. While most of the lakes in western Washington are represented by only Opening Day or partial season catch estimates, it is well documented that a major portion of the season long catch occurs on Opening Day (Johnston, 1973; Cummins, 1975).

Phosphorus-loading data are available for only two lakes, Liberty Lake and Pine Lake. Both of which are culturally eutrophic lakes, suffering from nuisance algae blooms, and both are phosphorus limited, making analysis of the significance in terms of the lakes' total phosphorus budgets difficult.

In the case of Pine Lake, Table E makes it clear that the net phosphorus removed by trout harvest (6 g/ha/yr) is insignificant when compared to the total phosphorus loading of 4,146 g/ha/yr. Since no phosphorus budgets exist for the other western Washington lakes shown in Table E, it can only be assumed that they lie within the broad range of values suggested by Vollenweider (1968): between 700 and 2500 g/ha/yr on low nutrient lakes, and between 1300 and 5000 g/ha/yr on "problem lakes". It is easy to see that either addition or removal of phosphorus in the range given for western Washington lakes in Table E (+2 to -93 g/ha/yr) is negligible compared to the phosphorus loads.

The situation is different in eastern Washington lakes, where phosphorus was removed by anglers in much larger amounts - 300 to 1,269 g phosphorus/ha/yr. These withdrawals of phosphorus from lakes could play an important role in counteracting the eutrophication process. On Liberty Lake, for example, anglers removed 322 g/ha in 1978. Total phosphorus loading in 1974-1978 was 3,700 g/ha/yr so that 8.7% of the phosphorus added to the lake in 1978 from all sources could have been removed by anglers that year.

Bull and Mackay (1976) drew a similar table for two Canadian lakes: one eutrophic, the other oligotrophic - and concluded that even at the maximum sustainable yield (MSY), less than 1% of the phosphorus entering the lakes could be removed by anglers. Thus, fish harvest could neither slow nor reverse the process of eutrophication. While this conclusion agrees closely with our analysis of the western Washington lakes, it contradicts the eastern Washington data. The reason for the difference is that Bull and Mackay estimated the top catch from their lakes was less than 3 kg/ha/yr, an extremely low figure that compares only with the Kitsap County lakes in Table E. A likely explanation for this low estimate is that the Canadian lakes were not sustained by

hatchery stocks; a much lower catch-per-unit-surface-area would be expected from a natural population of salmonids than from a stocked lake.

Burgess (1966) noted that sport harvest of warmwater fish removed over 5,000 lbs. of phosphates from Lake Harris, Florida, over a 15-month period. The author likened this to removing all the phosphates from the annual untreated waste of over 1,000 persons. He further noted that while phosphorus removed by anglers would not cause an immediate reduction of nutrients in the lake, it would serve as a deterrent to eutrophication. Dunst et al. (1974) concurred that harvesting fish might significantly reduce nutrients in some problem lakes.

While fishing is not a consequence of a "trout-only" program, the large catches associated with eastern Washington "trout-only" lakes do not ordinarily occur in waters managed for "mixed-species". On the "trout-only" waters in eastern Washington cited in Table E, for example, the average harvest was 1,491 trout/acre/year; the average on four similar-sized "mixed-species" lakes (two in eastern and two in western Washington) was only 120 "warmwater" fish/acre/year. Trout catches were not included in the latter calculation because trout planted as "legals" just before Opening Day do not constitute a net loss of nutrients when caught. These figures indicate that harvest (and thus nutrient removal by anglers) is roughly ten times greater on high-yield "trout-only" lakes than on "mixed-species" lakes.

Effects of Rotenone on Trout Stocking and Grazing - Figure 1 shows that both rotenone treatment and subsequent trout stocking affect grazing by zooplankton.

Poisoning a lake with rotenone temporarily destroys the zooplankton and thus decreases grazing (Figure 1). 95 to 100% of the open-water zooplankton are destroyed within a few days, and crustacean plankters are generally absent from open-water tows for two to twelve weeks. Since these are the most important grazers, a decrease in grazing followed by a surge in phytoplankton could logically be expected.

Algae blooms commonly follow rotenone treatments and reduced grazing is often cited as a cause (Table C). Phosphorus released by carcasses probably also contributes to these blooms, and it is impossible to separate the effects of the two. Long-term (i.e., beyond the year of application) effects of rotenone on grazing are unlikely; in most cases, zooplankton have recovered in abundance and diversity to prerotenone levels within two to twelve months after treatment.

Zooplankton populations eventually recover from rotenone poisoning, and they usually do so before fish re-enter the lake or are restocked. Figure 1 shows that in the absence of fish, large

zooplankters such as *Daphnia pulex* usually dominate the recovered community, and that they will continue to dominate if fish are not restocked or otherwise re-enter the lake. Also, already common plankters often increase in body size when they recover in the absence of fish.

These large zooplankters are more efficient grazers than small ones (Kerfoot, 1980). Burns (1969) found that the filtration rates of several *Daphnia* species were roughly proportional to the square or even cube of their body lengths. This difference in grazer body size may be even more important than grazer population size in controlling algae. Hrbacek et al. (1961) found that algae levels in a rotenoned Czechoslovakian backwater decreased despite a smaller standing crop of zooplankton, the reason being that a large daphnid suddenly became dominant following the fish kill.

This sequence - large grazers becoming dominant in the absence of planktivorous fish and reducing algae levels - has been repeatedly demonstrated in both small-scale enclosure experiments (Lynch and Shapiro, 1981; Andersson et al., 1978) and whole-lake situations (Shapiro, 1979). Reducing algae levels by increasing large grazer populations (either by killing planktivorous fish with rotenone or by introducing large piscivorous fish which accomplish the same thing) is one of the cornerstones of the "biomanipulation" movement, and the literature is abundant and detailed (i.e., Shapiro et al, 1982; Goad, 1982).

Shapiro and Smeltzer (1982) reviewed data from 13 Minnesota lakes poisoned with rotenone or toxaphene; seven showed transparency increases following poisoning, two probably became clearer, and four showed no change. Unfortunately, the species of "undesirable" fish eliminated - presumably planktivorous in at least some of the improved lakes - were not mentioned; these lakes were managed for bass, pike and walleye. The authors made the reasonable assumption that the lakes that cleared did so because of reduced algae levels, and that either increased grazing in the absence of fish (or both) was the cause.

Eutrophic Wirth Lake, Minnesota was intensively studied by Shapiro (1982) before and after rotenone treatment. The fish population, made up of crappies (*Pomoxis spp.*) (50%), bluegill sunfish (*Lepomis macrochirus*) (25%), carp (*Cyprinus carpio*) (15%) and perch, bullheads (*Ictalurus spp.*), suckers (Catostomidae), northern pike (*Esox lucius*) and largemouth bass (*Micropterus salmoides*) (10%), was poisoned in September 1977. The zooplankton population was virtually wiped out by the rotenone, but partial recovery was apparent 25 days later. Due to complications, it is impossible to tell whether or not a post-rotenone bloom occurred during this period. The following year a huge population of large daphnids (*D. pulex*) appeared, and algae levels decreased dramatically with a concurrent increase in transparency (Figure 11). The *Daphnia* population averaged 256,000 per m², or 32 individuals per liter, a

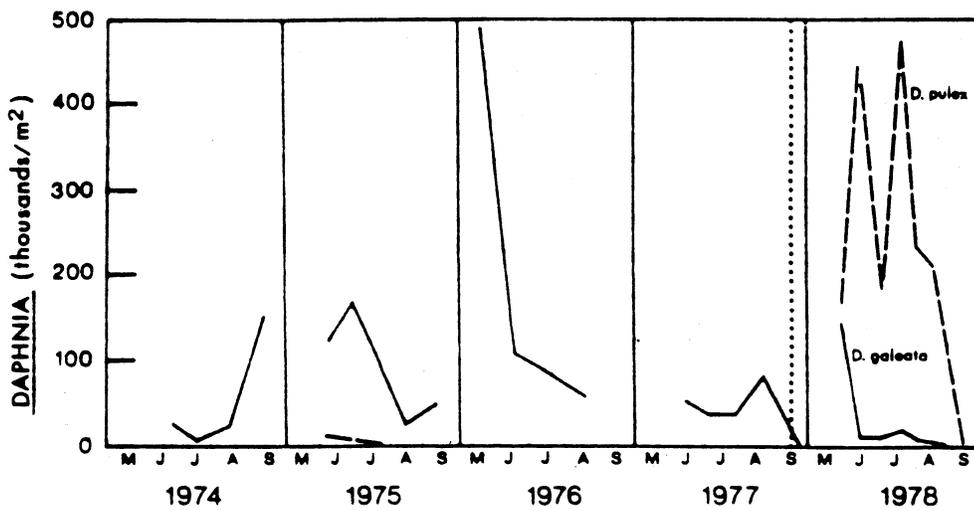
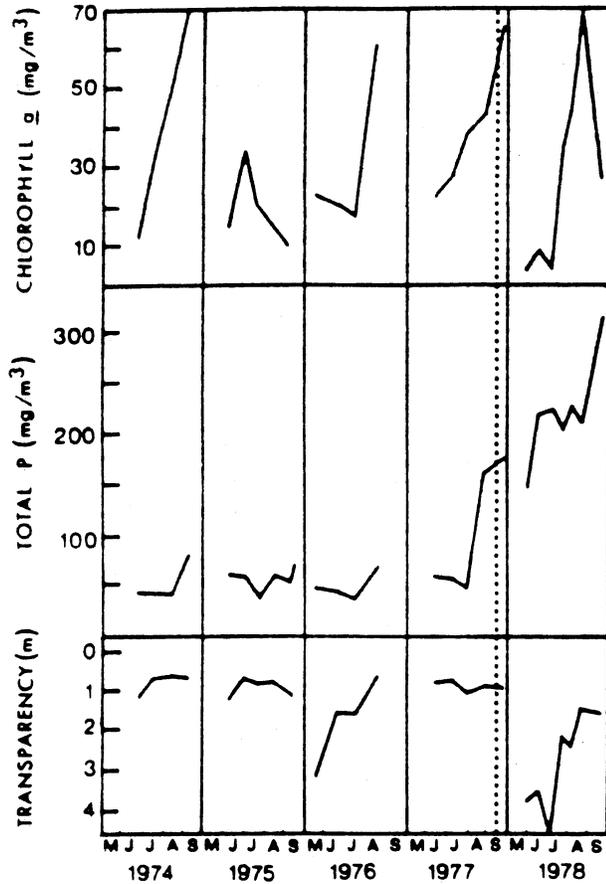


Figure 11. Algae (as chlorophyll a), phosphorus (P), transparency, and *Daphnia* in Wirth Lake, Minnesota. Dotted vertical line indicates date of rotenone treatment. Phosphorus levels in 1978 were increased by mechanical circulation, not through any biological process. Source: Shapiro et al. 1982.

concentration theoretically capable of removing virtually all filamentous blue-green algae from the lake (Lynch and Shapiro, 1981).

Within a short time, the blue-greens *Aphanizomenon*, *Oscillatoria* and *Anabaena* were reduced to low levels and replaced by green algae. Overall, algae levels in early 1978 were much lower than in the four years before rotenone, and Shapiro credited the large zooplankters. What makes Wirth Lake a particularly interesting case is that phosphorus levels were dramatically increasing (as a result of mechanical water circulation) during the same time that rotenone treatment and large daphnid increases were taking place; thus, algae levels decreased due to grazing despite "fertilization". However, later in 1978, chlorophyll levels increased and transparency decreased due to certain complications.

Clear Lake, Minnesota provides another example: Smeltzer (1982) noted that algae levels declined sharply the year following rotenone, and he credited increased grazing by large zooplankton in the absence of fish. Based on these and other experiments, Shapiro and Smeltzer (1982) concluded that "in balance, then, it would appear that use of fish toxicants does reduce algal abundance and that in some cases the effect has been longlasting".

Grazing Effects on Different Algae - Generally, when large grazers become dominant algae levels decrease, although there are some important exceptions to this pattern:

- 1) not all algae can be eaten by zooplankton;
- 2) some algae, though eaten, can pass through a zooplankter's gut unaffected; and
- 3) some algal species increase in number or change to inedible forms when grazing on them increases.

In the first two cases the net result is that algae levels are unaffected by increases in the number of grazers. In the third case, increased grazing may actually reverse the usual pattern and cause algal levels to increase. The dotted line in Figure 1 leading to an "*Aphanizomenon* flake bloom" depicts this pathway; it is a dotted rather than a solid line to indicate that it is an exception that only seems to occur under special circumstances.

Table F lists some algae that are not significantly affected by zooplankton grazing. In most cases, these forms are too large for plankters to eat. In some cases, gelatinous green algae like *Sphaerocystis* are eaten but pass intact through the grazer's gut. In still other cases, algal species secrete something that causes grazers to reject them, or that is actually toxic to zooplankton. The net result is the same: if these forms are dominant in a lake, grazing by itself can rarely be expected to reduce their abundance.

Table F. Algal types unaffected and suppressed by grazing. Numbers in parentheses refer to references in the literature (see below).

	USUALLY UNAFFECTED BY GRAZING	USUALLY SUPPRESSED BY GRAZING	
BLUE GREEN ALGAE	<u>Anacystis nidulans</u> (1)	<u>Chroococcus limneticus</u> (5)	BLUE GREEN ALGAE
	<u>Merismopedia</u> sp. (1)	<u>Aphanizomenon flos-aquae</u> (2) b/	
	<u>Synechocystis</u> sp. (1)	<u>Cryptomonas</u> (5)	
	<u>Gloeocapsa alpicola</u> (1)	<u>Rhodomonas</u> (5)	
	<u>Microcystis aeruginosa</u> (2, 3)	<u>Cyclotella comta</u> (5)	
	<u>Oscillatoria rubescens</u> (4) a/	<u>Asterionella formosa</u> (5)	
	<u>Oscillatoria agardhii</u> (4) a/	<u>Oocystis lacustris</u> (5)	
	<u>Anabaena flos-aquae</u> (5, 1)	<u>Chlamydomonas</u> (1, 5)	
	<u>Anabaena affinis</u> (5)	<u>Coelosphaerium dubium</u> (2)	
	<u>Anabaena</u> sp. (6)	<u>Ankistrodesmus falcatus</u> (1)	
	<u>Anabaena</u> ("some species") (2)	<u>Chlorella vulgaris</u> (1)	
	<u>Gloeotrichia</u> sp. (6)	large diatoms (5)	
	<u>Lyngbya</u> sp. (6)	flagellates (5)	
	<u>Synechococcus elongata</u> (1)	euglenids (5)	
	<u>Aphanizomenon</u> sp. (7) b/	ciliates (5)	
	<u>Synechococcus cedrorum</u> (1)		
	<u>Mallomonas caudata</u> (5)		
	<u>Peridinium willei</u> (5)		
	<u>Cosmarium depressum</u> (5)		
	<u>Sphaerocystis schroeteri</u> (5)		
<u>Elakatrothrix gelatinosa</u> (5)			

- (1) Arnold (1971)
 (2) Sorokin (1968)
 (3) Lampert (1981)
 (4) Edmondson and Litt (1982)
 (5) Porter (1973)
 (6) Edmondson (1957)
 (7) Lefevre (1950)

- a/ Oscillatoria apparently inhibits only Daphnia sp.
 b/ note that this species is listed in both columns; although Lefevre does not actually mention it, he probably was referring to the "flake" colony form of Aphanizomenon as inedible. Sorokin and others have found A. flos-aquae in the filamentous or non-flake form to be readily eaten. See discussion on this species in text.

Many of these inedible forms of algae are the very ones responsible for obnoxious blooms in eutrophic "problem" lakes. Blue-green algae are usually the most objectionable, and as shown in Table F many of these are unaffected by grazing.

This has far-reaching implications for "biomanipulators" interested in cleaning up problem lakes. For example, Goad's (1982) proposal to increase grazing in Green Lake, Washington by eliminating yellow perch (*Perca flavescens*) and other planktivores was dismissed by Perkins (1982) because the problem-causing algae there were primarily *Gloeotrichia* and other inedible blue-greens. Unless the phytoplankton community could be changed to more edible forms - and there are ways to do this - no great improvements would likely occur.

Not all blue-greens are inedible, however. *Aphanizomenon*, for example, especially *A. flos-aquae*, is commonly found in many eutrophic and mesotrophic lakes in Washington; Liberty Lake and Lake Washington being two examples. *Aphanizomenon* has the ability to exist in two forms: when filamentous, it is readily eaten, especially by large grazers, and is a good food source (Sorokin, 1968). But it also can clump together in the "flake" or "grass-blade" form, which is inedible. Interestingly, this "flake" form often develops in lakes with abundant large grazers such as *Daphnia pulex* or *D. pulicaria*, apparently in response to heavy grazing pressure (Hrba'cek, 1964; Lynch, 1980; Shapiro, 1979; Bandow, 1980). This phenomenon occurs on a wide variety of lakes (Shapiro, 1979), and Lynch (1980) substantiated the fact that the large matted colonies of *Aphanizomenon* appears when *D. pulex* became abundant, and disappeared when the large grazers were eliminated by fish. Straskraba and Straskraba' (1969) likewise reported that "extraordinarily high numbers of fish (reducing the grazing population) decreased the *Aphanizomenon* blooms" in a Czech reservoir.

But "flake" blooms of *A. flos-aquae* are not inevitable with abundant large *Daphnia*. Lynch (1980) found that "flake" blooms occurred only when the lake bottom was oxygenated. Shapiro (1979) showed that this was the case in a wide range of lakes: both large daphnids and oxygenated hypolimnia were necessary for such a bloom.

This, then, is a special case in which increased grazing by large zooplankters actually promotes nuisance algae blooms. There is yet another complication with *Aphanizomenon* blooms: since the "flake" or "grass-blade" form is clumped, water transparency in a lake usually increases rather than decreases. This leads to the paradox of high algae and chlorophyll a levels, but clear water at the same time. Edmondson (1980) noted that when large colonies of *Aphanizomenon* and *Anabaena* formed in Lake Washington in the presence of large *Daphnia*, the water "looked crumby but clear". While Lake Washington was enjoying the clearest water ever recorded, residents on the downwind side of the lake were

complaining about the huge matted colonies of blue-green algae (Litt, UW, pers. comm.; Shapiro, 1979). Because of this problem, Shapiro cautioned against using any technique to increase large *Daphnia* in a lake unless it could be ascertained that *Aphanizomenon* would not become dominant.

Grazing After Fish Are Restocked - Figure 1 shows that when trout are restocked in a rotenoned lake, the large grazers that developed in the absence of fish are again cropped back. Additionally, Figure 10 shows that when the large plankters are cropped back, a reduction in grazing can be expected, since the small grazers are less efficient at filtering algae. This should lead to an increase in algae.

There is a great deal of literature suggesting that when trout or other planktivorous fish are introduced into a formerly fish-free environment dominated by large grazers, the following things happen:

- 1) large grazers are reduced, or even eliminated entirely;
- 2) small grazers take their place;
- 3) grazing on algae is reduced as a result; and
- 4) algae levels increase, often dramatically.

Evidence comes in part from enclosure experiments where planktivorous fish have been added to plastic bags suspended in lakes. Lynch and Shapiro (1981) demonstrated that algal biomass increased and transparency decreased as they added more planktivorous fish to enclosures containing *Daphnia* (Figure 12). Andersson et al. (1978) also noted dramatic increase in algae when they added fish to enclosures in two Swedish lakes (Figure 13). Since they used fish that were both planktivorous and benthivorous, some of the increased algae was due to higher phosphorus levels. In both of the experiments above, the algae produced in the presence of fish were mostly edible blue-greens.

Similar studies in natural, whole-lake situations are rare. While a number of authors have investigated the effect of trout introductions on grazer populations in fish-free lakes (Kitchell and Kitchell, 1980; Anderson, 1972; Galbraith, 1967; 1974), almost none have extended their studies to include the phytoplankton.

Medical Lake in eastern Washington is an exception, with the fish, zooplankton, and algae extensively studied since 1974 (Scholz et al., 1985; Mires et al., 1981; Knapp and Soltero, 1983; Soltero et al., 1981). Medical Lake was treated with alum in 1977 to reduce phosphorus levels and clear the lake of nuisance algae. The lake responded with a change from blue-green to green algae, reduced algae levels, greater water clarity, and a predominance of large *Daphnia pulex*. The enhanced water quality prompted WDW to stock the lake - which previously could not support trout - with rainbow fingerlings beginning in 1978. These trout began feeding almost

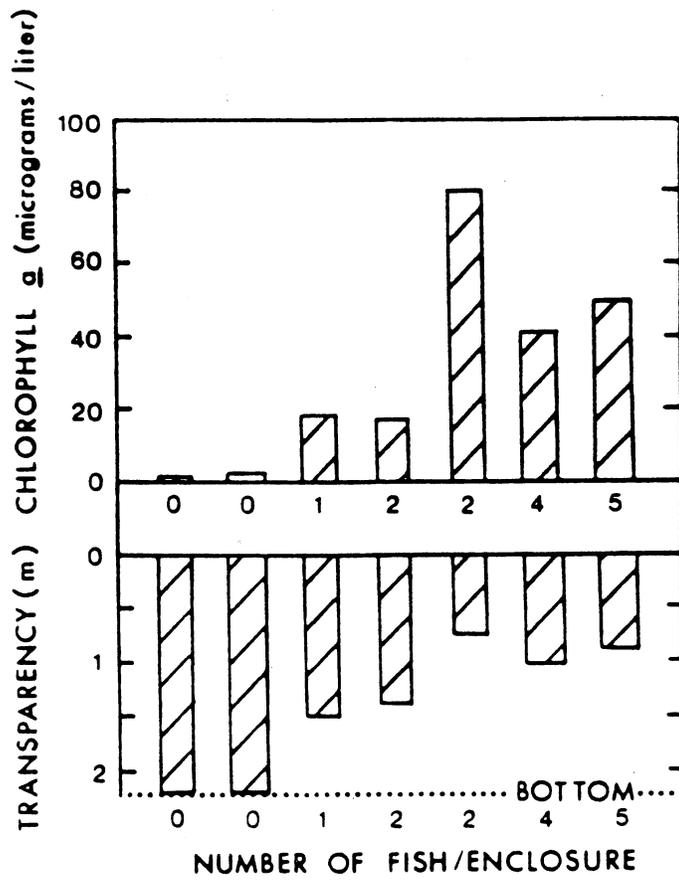


Figure 12 The effects of adding various numbers of zooplanktivorous fish to enclosures of lake water containing *Daphnia*. Source: Lynch and Shapiro 1981; Shapiro 1979.

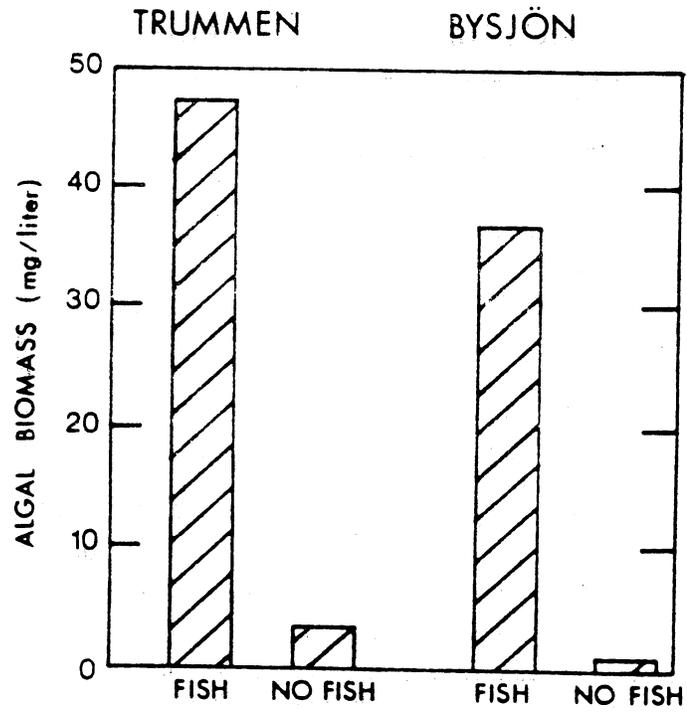


Figure 13 The effects of adding zooplanktivorous and benthivorous fish to enclosures in two Swedish lakes. Source: Andersson et al. 1978.

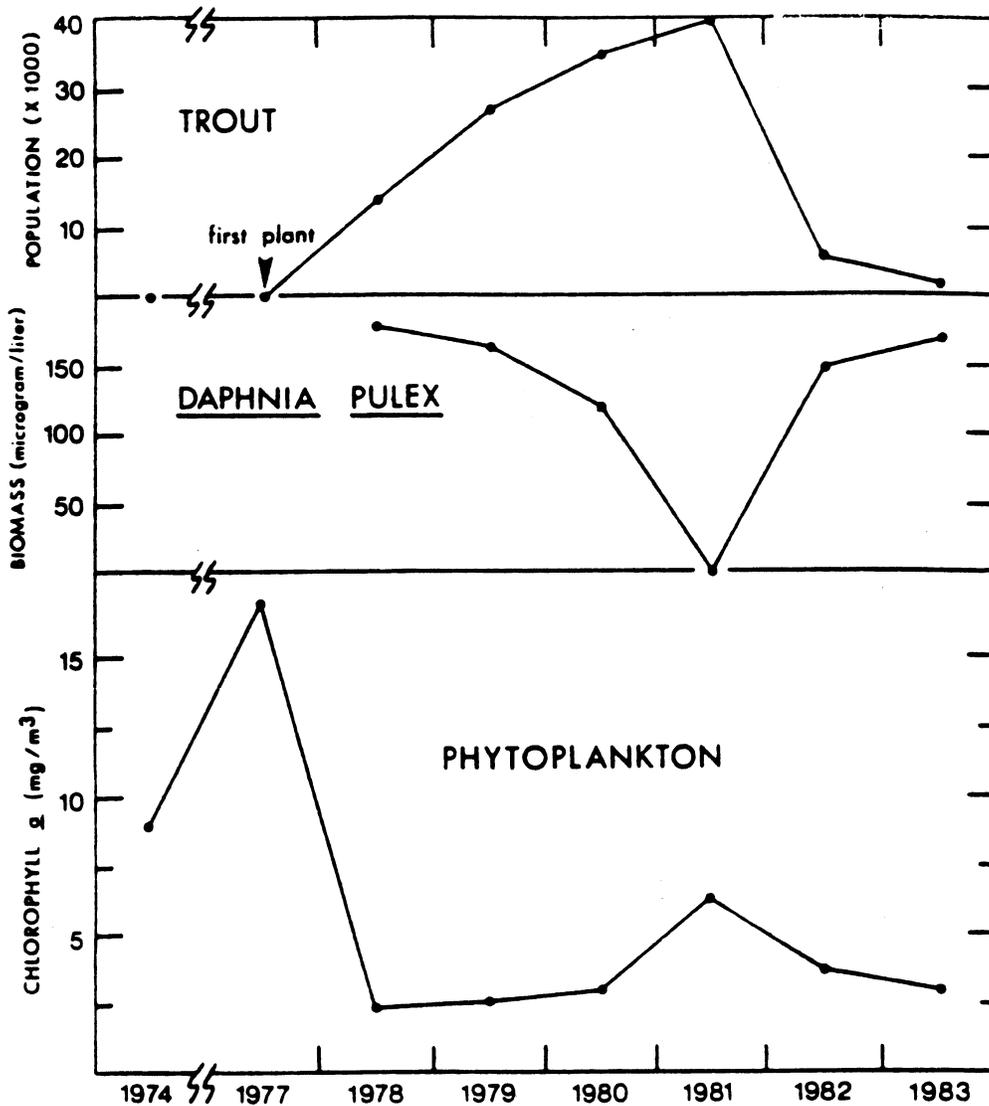


Figure 14 Effects of rainbow trout stocking on Daphnia and phytoplankton (as chlorophyll a) in previously unstocked Medical Lake, Washington. Source: Scholz et. al. 1985.

exclusively on the large *Daphnia* population. From 1978 to 1981, the average size of *D. pulex* decreased from 2.3 to 1.0 mm due to size selective predation by all age-classes of trout. Figure 14 displays the changes in standing crop of *D. pulex* and phytoplankton in relation to the rainbow trout population. *Daphnia* abundance was clearly related to the numbers of trout in the lake, steadily decreasing through 1981. Consequently, algae levels rose. These changes were almost certainly due to the reduction in grazing since phosphorus levels were relatively constant during this period.

Both the enclosure experiments and the studies on Medical Lake compared algae levels in a fishless environment with algae levels after fish stocking. Collectively, these studies suggest that a lake stocked with trout or any other plantivorous fish will contain more algae than the same lake without fish.

The situation is somewhat different in most rotenoned Washington lakes, which have been routinely stocked for many years with a fairly uniform number of trout. The lakes are fishless for only about 5-8 months following rotenone, at which time fingerling trout are restocked, generally at the prerotenone levels. Although it is logical to assume that algal abundance would return to the prerotenone level in such a case, no studies have actually addressed this question. Shapiro and Smeltzer (1982) did examine a somewhat analogous situation on Clear Lake, Minnesota: the lake was poisoned with toxaphene and responded with slightly increased transparency. The year after treatment, Clear Lake was restocked with both planktivorous and bottom feeders and clarity was reduced to about pretreatment levels (Figure 15).

Figure 16 shows that virtually all zooplankton and residual trout are killed by the rotenone treatment. Shortly thereafter, an algae bloom occurs owing to some combination of decreased grazing and increased phosphorus levels. This bloom subsides in the winter. Zooplankters then recover to at least their former levels of abundance; grazing itself increases due to the large, more efficient grazers that dominate while fish are absent. As a consequence, algal abundance decreases to some level that is lower than before (i.e., when fish are restocked in the lake at prerotenone levels). They crop back or even eliminate the large grazers that developed in their absence, and the zooplankton community returns in number and kind to the prerotenone state. As a consequence, algae levels increase to their prerotenone state, negating any short-term water quality benefits that might have occurred while fish were absent. (Since the fish-free period generally runs from late fall through early spring - a time of normally low algae and grazer levels - actual decreases in algal abundance may be unnoticeable, if they occur at all).

In Figure 16 it was assumed that the only fish in the lake were stocked trout, although this is not the case; both target and non-target species share the lake with trout and are killed along with

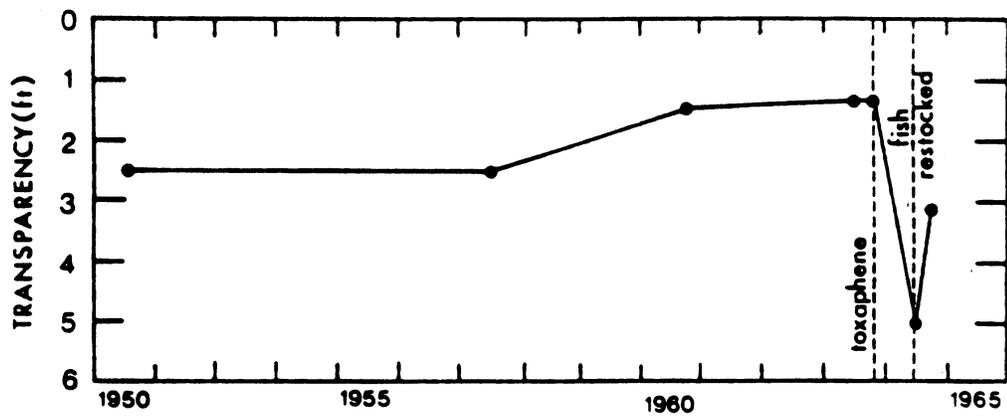


Figure 15 Effect of toxaphene poisoning and subsequent restocking with planktivores and bottom feeders on water clarity in Clear Lake, Minnesota. Source: Shapiro and Smeltzer 1982.

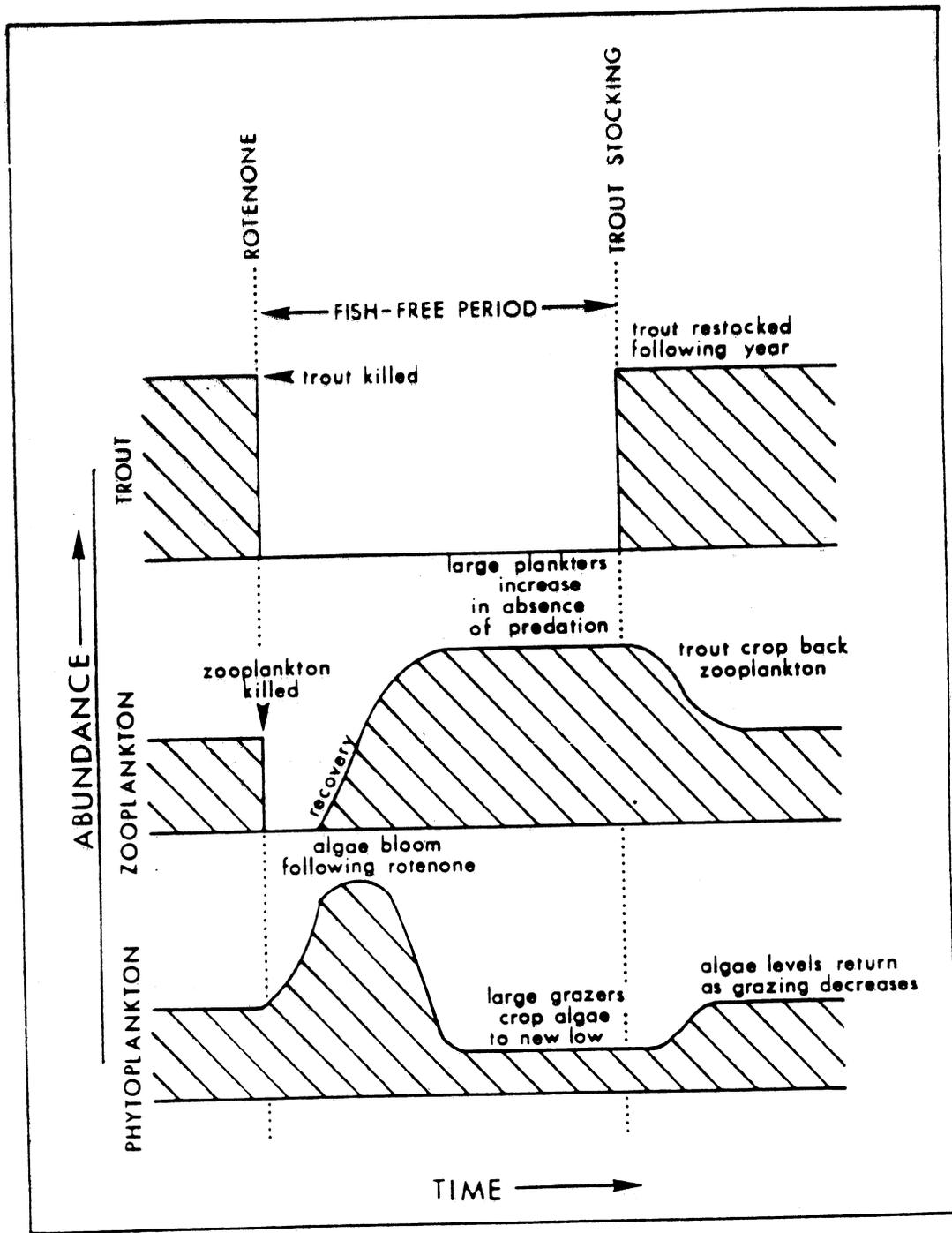


Figure 16. Hypothetical scenario following rotenone poisoning and trout restocking in a lake. Time and abundance not necessarily to scale.

them by rotenone. Many of these other fish are planktivorous, including yellow perch and sunfish which selectively feed on large grazers in much the same way as trout. Unlike the trout, which are stocked again the following spring, these other species must either repopulate the lake from survivors or be illegally reintroduced. In either case they do not usually reach their prerotenone levels of abundance for several years. If these target fish are highly planktivorous, it can be hypothesized that predation on the grazers will be somewhat reduced during this period, with trout the only important planktivores. If this hypothesis is correct, the net effect would be reduced algae levels when compared to the prerotenone years, during which time the total planktivore population was higher. There is no evidence in the literature to either support or refute this hypothesis. It is equally possible that trout alone could decimate the large grazers, even in the absence of other planktivorous fish.

Zooplankton

Short Term Effects - Table G displays the results of bioassays performed on various zooplankters. The cladocerans ("water fleas"), especially *Daphnia*, are well represented in the tests. While the 48-hour LC50's for cladocerans range widely from 0.01 ppm to 0.57 ppm, most are within the range 0.1-0.5 ppm of formulation. LC50's for the copepod *Cyclops* are somewhat lower, between 0.10 and 0.22 ppm. Based on laboratory findings, at least 50% of the cladocerans and copepods could be expected to die from exposure to the rotenone concentrations commonly used in fisheries work (0.5 ppm and up).

The effects of rotenone on the two other major components of the zooplankton community, rotifers and protozoans, have not been studied in the laboratory.

There is almost unanimous agreement among researchers that rotenone's immediate effect on the zooplankton is catastrophic. Table H shows the results of nineteen studies in lakes and ponds where zooplankton abundance was recorded before and shortly after (within four days in 12 of the studies) treatment. In 17 of the 19 cases, the immediate reduction in total numbers of mid-water zooplankton was between 75 and 100%. In 16 cases, the reduction was between 95 and 100%.

In the other two cases there was no reduction at all due to rotenone; in one of these (Libey and Holland, 1980), the very light dosage (0.1 ppm Noxfish) probably accounts for the absence of a zooplankton kill. This concurs with the bulk of laboratory bioassays cited above, which show that cladocerans and copepods (the important plankters in Libey and Holland's ponds) generally require more than 0.1 ppm to kill 50% of the population in 48

Table 9 Toxicity of rotenone to zooplankton in laboratory bioassays.

Species	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	References
CLADOCERA (water fleas)							
<u>Daphnia pulex</u>	0.01	48 hr.	16°	35 mg/l IDS		LC50	U.S. Federal Water Pollution Administration 1968
<u>Daphnia pulex</u>	0.025	3 hr.			5% rotenone	100% mortality	Hamilton 1941
<u>Daphnia pulex</u>	0.0275	24 hr.	16±1°	20 mg/l total hardness pH 6.6	Noxflish	LC50	Chandler & Marking 1962
<u>Daphnia</u>							
	0.48	48 hr.	27°		cubd	LC50	Wright 1957
	0.24	48 hr.	27°		Noxflish	LC50	
	0.32	48 hr.	27°		Pro-Noxflish	LC50	
	0.57	48 hr.	24°		cubd	LC50	
	0.49	48 hr.	24°		Noxflish	LC50	
	0.44	48 hr.	24°		Pro-Noxflish	LC50	
	0.55	48 hr.	20°		cubd	LC50	
	0.56	48 hr.	20°		Noxflish	LC50	
	0.57	48 hr.	20°		Pro-Noxflish	LC50	
<u>Daphnia</u>	0.1	48 hr.	27-29°	pH 7.2	4.9% rotenone	Minimum lethal dose-weakest concentration of chemical which produced a kill exceeding 25%	Zischke 1952
<u>Daphnia</u>	0.100	48 hr.	16°	pH 7.4-7.8		LC50	Sanders & Cape 1966
<u>Daphnia</u>	0.55	48 hr.	20°		Pro-Noxflish	LC50	Brooks 1961
	0.44	48 hr.	24°		Pro-Noxflish	LC50	
	0.31	48 hr.	27°		Pro-Noxflish	LC50	
<u>Daphnia</u>	0.25	1 hr.				LC50	Neighan 1959

Table 9 Continued

Species	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
<u>Simoccephalus</u> <u>serrulatus</u>	0.190	48 hr.	16°			LC50	Sanders & Cape 1966
<u>Leptodora kindtii</u>	0.025	3 hr.			5% rotenone	100% mortality	Hamilton 1961
<u>COPEPODA (copepods)</u>							
<u>Cyclops</u>							
	0.22	48 hr.	27°		cubé	LC50	Wright 1957
	0.12	48 hr.	27°		Noxfish	LC50	
	0.14	48 hr.	27°		Pro-Noxfish	LC50	
	0.24	48 hr.	20°		cubé	LC50	
	0.14	48 hr.	20°		Noxfish	LC50	
	0.19	48 hr.	20°		Pro-Noxfish	LC50	
<u>Cyclops</u>							
	0.18	48 hr.	20-27°		Pro-Noxfish	LC50	Brooks 1961
	0.14	48 hr.	20-27°		Pro-Noxfish	LC50	
<u>Cyclops</u>	0.1	3 days	11±1°	pH 7.9 260 mg/l hardness	5% rotenone liquid	none survived	Meadows 1973
<u>Diaptomus</u> <u>siciloides</u>	0.025	3 hr.			5% rotenone	100% mortality	Hamilton 1961

Table H Data summary of zooplankton studies in lakes and ponds. I: Immediate effects of rotenone on mid-water zooplankton.

Name, location	Surface area (acres)	Formulation	Dosage (ppm)	Post-rotenone sampling	Immediate reduction in zooplankton abundance	Reference
Fern Lake western Washington	21	powdered rotenone (5% rotenone)	0.5	within 3 days	nearly 100%	Kiser et al. 1963
Celestine Lake Alberta, Canada	96	derris root powder (5% rotenone)	0.75	within 24 hours	more than 95%	Anderson 1970
Patricia Lake Alberta, Canada	170	derris root powder (5% rotenone)	0.75	within 24 hours	95-100%	Anderson 1970
McCormick Lake Nova Scotia, Canada	6	derris (5% rotenone)	1.33	within 24 hours	nearly 100%	Smith 1940
Ljogdttjern Norway	6	Pro-Noxfish	0.5	28 days after	99%	Haugve 1977
Smith Lake Colorado	24	derris powder (5% rotenone)	1.0	within 4 days	nearly 100%	Hoffman & Ollive 1961
Velká Arazilova Czechoslovakia	0.1	"Lonchocarpus extract"	---	7 days after	nearly 100%	Hrabčok & Novotná-Ovošková 1965
West Pond Montana	13	Pro-Noxfish	0.7	within 1 month	0%	Mollitz 1962
Third Sister Lake Michigan	10	derris root powder (5% rotenone)	0.5	within 24 hours	100%	Brown & Ball 1943a
South Branch Lake (cove only) Maine	11	Noxfish	0.6	within 24 hours	97%	Neves 1975
Middle Pond Montana	20	Chem-Fish Special	0.7	within 1 month	98%	Mollitz 1962

Table H Continued

Name, location	Surface area (acres)	Formulation	Dosage (ppm)	Post-rotenone sampling	Immediate reduction in zooplankton abundance	Reference
Silver Lake western Washington	106	powdered rotenone (5% rotenone)	1.0	2 days later	100%	Kiser et al. 1963
Halmjón Sweden	94	Pro-Noxfish	0.5	3 days after 10 days after	90% 97%	Almqvist 1959
Potter's Lake New Brunswick, Canada	113	derris powder (5% rotenone)	0.5	"after"	nearly 100%	Smith 1941
Ponds A & B Indiana	1	Noxfish	0.1	within 3 days	0%	Libbey & Holland 1980
Bug Lake Wisconsin	11	Pro-Noxfish	2.5	within 12 days	100%	Serns 1979
Caris Lake Minnesota	110	Chem-Fish Pro-Noxfish	3.0 3.0	within 12 days	100%	Bandow 1980
Wirth Lake Minnesota	40	"rotenone"	---	within 10 days	99%	Shapiro 1982
Dening Lake Minnesota	13	derris powder (5% rotenone)	0.5	4 days later	75%	Hooper 1948

hours. Libey and Holland's research is interesting in that it is one of the few reported cases where rotenone was used to purposely poison zooplankton; the authors wished to starve a stunted bluegill population by reducing the plankters.

The results of the other study in which there was no zooplankton kill (Wollitz, 1962 on West Pond, Montana) are difficult to explain, especially since the same author noted a 98% reduction in the similarly treated nearby Middle Pond (Wollitz, 1962).

Zooplankters feel the effects of rotenone shortly after application: Kiser et al. (1963) made tows every 15 minutes on Silver Lake, Washington, during the day it was treated, noting a 34% decrease in plankton counts within 30 minutes after treatment began. The greatest reduction in total zooplankton counts came between 15 minutes and one hour after treatment began, a reduction of 70%.

Susceptibility of Different Species - While virtually all plankters are affected by rotenone, some are more tolerant than others.

There is general agreement that the planktonic crustaceans, especially the cladocerans, are the group most quickly or thoroughly eliminated (Anderson, 1970; Bandow, 1980; Hrba'cek and Novotna'-Dvora'kova', 1965; Hongve, 1977; Neves, 1975; Smith, 1940; 1941; Wollitz, 1962; Brown and Ball, 1943a; Almquist, 1959; and Hooper, 1948). Almquist (1959) ranked various plankters according to their sensitivity to rotenone in Lake Erken, Sweden, and found that among the ten most sensitive, eight were cladocerans and two were rotifers. *Diaphanosoma* and *Daphnia* required the lowest exposure (all died between 30 minutes and two hours in 0.5 ppm formulation) of all test animals. Almquist found wide differences in tolerance even among the cladocerans, however; *Alonella* and *Pleuroxus* withstood 1.5 ppm and 2 ppm Pro-Noxfish for up to 8 hours, and *Alona* was one of the most tolerant of all 44 organisms test, requiring seven hours in 4.5 ppm Pro-Noxfish for a 100% kill. Kiser et al. (1963) reported these same three genera resisting rotenone in Fern Lake, Washington, though habitat within the lake may have contributed. And *Bosmina* remained present in the open water of Silver Lake, Washington after treatment far longer than *Daphnia* or *Holopedium* (Kiser et al., 1963).

Rotifers are generally considered to be more tolerant of rotenone than the cladocerans or copepods. *Keratella* has been singled out as highly resistant by several authors (Bandow, 1980; Almquist, 1959; Anderson, 1970; Smith, 1940; 1941; Walters and Vincent, 1973; Neves, 1975), along with *Conochilus* (Neves, 1975; Smith, 1941; Almquist, 1959).

Susceptibility According to Habitat - Sensitivity to rotenone apparently varies not only to the species of plankter, but with the habitat type within the lake as well, though only one study has

adequately addressed this question. Kiser et al. (1963) separately sampled three different habitats in Fern Lake, Washington: the open water, the margin between the brush and open water, and the shallow weedy shoreline. Immediately after treatment they noted that the reduction in total zooplankton counts was most severe in open water and least severe in the weedy shoreline. The margin was intermediately affected.

Naturally, as each habitat supports a different assortment of plankters, it could be concluded that these results were mostly due to the varying sensitivities of the species involved rather than the habitat. But a few plankters in Fern Lake, such as the cladoceran *Alonella* and the rotifer *Chydorus sphaericus*, live in all three habitats, and they suffered greater losses in the open water than in the weedier areas.

It is well documented that vegetation and heavy organic debris detoxify rotenone, and the Fern Lake researchers suggested this as a reason why the weedy area plankters were somewhat less affected. They also admitted the possibility that the inaccessible brushy regions weren't as well dusted with rotenone. All other field studies have confined themselves to open-water sampling, or failed to break down their results by habitat type.

Long Term Effects - Recovery - Although they are drastically reduced immediately following rotenone treatment, zooplankton communities do recover in almost all cases. Even in those lakes where not a single living plankter appeared in the post-rotenone samples, enough escaped or survived treatment to eventually repopulate the lake.

As previously noted, some plankters escape treatment in densely weeded areas where rotenone is quickly detoxified (Almquist, 1959; Kiser et al., 1963). Others may survive simply by virtue of their tolerance to rotenone. Certain plankters may survive by means of parthenogenetic summer eggs and tough ephippial eggs which are unaffected by rotenone (Bandow, 1980; Anderson, 1970; Kiser et al., 1963). Both cladocerans and cyclopoid copepods produce ephippial eggs, which lie dormant in the lake sediments throughout the winter. They are normally produced in the late fall, but unfavorable environmental factors may stimulate early production. Kiser et al. (1963) observed female cladocerans with early ephippial eggs, and suggested that it was the rotenone that acted as this unfavorable factor. Finally, it has been suggested that zooplankton for repopulation may also come from other nearby bodies of water (Hrba'cek et al., 1961; Kiser et al, 1963), though this has never been documented in rotenoned lakes.

In most lakes there is a period following the rotenone treatment during which plankters (at least crustaceans) are scarce or absent from tow samples. Table I shows the results of 15 studies where the long-term effects of rotenone on zooplankton were recorded. In

Table I Data summary of zooplankton studies in lakes and ponds. II: long-term effects of rotenone.

Lake	Rotenone formulation	Dosage (ppm)	Time that Lake was toxic to fish	Crustacean-free period	Rotifer-free period	Fish restocked or re-entered	Period of study before rotenone	Period of study after rotenone	Time to complete recovery	Any species fell to reappear?	Any new species appear?	Reference
Fern Lake western Washington	powdered rotenone (5% rotenone)	0.5	33 days	2 weeks	---	steelhead	2 years	6 months	17 weeks	no	yes	Kiser et al. 1963 Fowler 1973
Celestine Lake Alberta, Canada	derris root powder (5% rotenone)	0.75	---	9 months	never absent	rainbow trout	1 month	2 years, 1 month	3 years	yes	yes	Anderson 1970
Patricia Lake Alberta, Canada	derris root powder (5% rotenone)	0.75	---	6 months	never absent	rainbow trout	1 month	3 years, 5 months	3 years	no	yes	Anderson 1970
McComack Lake Nova Scotia, Canada	derris (5% rotenone)	1.33	~1 month	9 months	never absent	---	3 days	1 year, 3 months	1 year	no	yes	Smith 1940
Ljøgdittjern Norway	Pro-Noxfish	0.5	---	---	---	rainbow trout	1 year, 3 months	1 year, 2 months	1 year	no	yes	Haugve 1977
Salth Lake Colorado	derris powder (5% rotenone)	1.0	---	3 months	never absent	---	14 days	11 months	6 months			Hoffman & Olive 1961
Velka Arazimova Czechoslovakia	"Lonchocarpus extract"	---	---	1 month	never absent	carp, bass, roach, bream	2 years	4 years, 1 month	4 months	yes	yes	Hrbáček & Novotná-Dvořáková 1965

Table I Continued

Lake	Rotenone formulation	Dosage (ppm)	Time that lake was toxic to fish	Crustacean-free period	Rotifer-free period	Fish restocked or re-entered	Period of study before rotenone	Period of study after rotenone	Time to complete recovery	Any species fall to reappear?	Any new species appear?	Reference
West Pond Montana	Pro-Noxfish	0.7	less than 17 days	---	---	---	1 year, 3 months	9 months	Immediate	---	---	Mollitz 1962
Third Sister Lake Michigan	derris root powder (5% rotenone)	0.5	7 days	never absent	never absent	none	1 year	1 year	2 months	yes	---	Brown & Ball 1943a
South Branch Lake (cove only) Maine	Noxfish	0.6	---	never absent	never absent	---	2 weeks	2 weeks	1 week	no	---	Neves 1975
Emaline Lake Colorado	Pro-Noxfish	1.0	---	---	never absent	cutthroat trout	2 months, 2 weeks	4 years	2 years	---	---	Wren 1965 Walters & Vincent 1973
Potter's Lake New Brunswick Canada	derris powder (5% rotenone)	0.5	between 18-45 days	2 months	never absent	brook trout	52 days	11 months, 9 days	10 months	---	---	Smith 1941
Ponds A & B Indiana	Noxfish	0.1	---	---	---	---	1 year, 2 months	1 month, 2 weeks	Immediate	---	---	Libby & Holland 1980
Bug Lake Wisconsin	Pro-Noxfish	2.5	5 months, 20 days	5 months, 19 days	---	fathead minnows, brook trout	3 months, 24 days	2 years	7-8 months	yes	yes	Sems 1979
Car's Lake Minnesota	Chem-Fish Pro-Noxfish	3.0 3.0	---	26 days	never absent	bass, walleye, channel catfish, brown trout	1 year, 3 months	2 years	did not fully recover	no	---	Bendow 1980

11 of these investigations, there were sufficient data to establish the length of time following treatment that plankters were absent from open-water tows.

In two examples, some crustacean plankton was always present in tow samples, but these appear to be special cases: Neves (1975) poisoned only an isolated cove within a lake, and immigration from the non-treated areas took place immediately following treatment; and Brown and Ball (1943a) observed an unusually short toxic period of seven days, possibly accounting for the continued presence of plankters in small numbers.

In the other nine cases, cladocerans and copepods were entirely absent from open-water tow samples from two weeks to as long as nine weeks.

Considering first the four lakes in which crustacean plankters remained absent for the longest time (Serns, 1979; Anderson, 1970; Smith, 1940):

Serns found no crustacean plankters for five months and nineteen days following treatment in Bug Lake, Wisconsin. The lake was toxic to fish, however, for at least five months and twenty days, possibly as a result of the heavy dosage (2.5 ppm Pro-Noxfish).

It is somewhat more difficult to understand the other three lakes where the crustacean-free period lasted from six to nine months (Smith, 1940; Anderson, 1970). While dosages were somewhat higher (0.75 ppm-1.33 ppm) than the bulk of the lakes studied, Bandow (1980) and Hoffman and Olive (1961) saw crustaceans much sooner after using 3 ppm and 1 ppm. Sampling bias may be partly responsible in the example of McCormick Lake (Smith, 1940); tows were made for only two months following treatment with rotenone, after which there was a seven-month period when no samples were taken. In the case Anderson's (1970) two lakes, the extremely long absence of crustacean plankton (6-9 months) may well be due to the oligotrophic nature of the high mountain lakes involved. Anderson (1972) and Wrenn (1965) pointed out that plankton recovery was slower in the relatively sterile alpine lakes than in nutrient-rich lowland lakes.

For Washington's rehabilitated lakes, the best estimate of this crustacean-free period probably lies between two and twelve weeks, the range of the other five studies (Kiser et al., 1963; Hrba'cek and Novotna'-Dvora'kova', 1965; Bandow, 1980; Smith, 1941; and Hoffman and Olive, 1961).

Since cladocerans and copepods are the plankters that juvenile trout eat most frequently, this period when they are virtually absent from open water may have important management implications in cases where restocking is planned shortly after treatment.

Generally, this is not the case in Washington; 78% of the lakes in the program have been treated in the fall and restocked no sooner than five months later. On several cases crustacean plankton reappeared before the lakes were nontoxic to fish.

Factors Affecting Recovery of Different Plankters - Once plankters reappear, the community begins to rebuild itself, eventually returning in most all cases to prerotenone levels of abundance and diversity. But just as the various plankters respond differently to rotenone when it is applied, they also recover at different rates.

Anderson (1970) stated that the speed of recovery for different plankters was likely related to four factors:

- 1) Susceptibility to rotenone . Most researchers found that the plankton groups most tolerant of rotenone recovered the quickest. Rotifers usually reached prerotenone levels of abundance before the cladocerans and copepods. In Smith Lake, Colorado, rotifers recovered after five months, while the crustacean plankters required six months (Hoffman and Olive, 1961). Hrba'cek and Novotna'-Dvora'kova' (1965) found cladocerans and copepods recovered between 3 and 4 months after poisoning, while protozoans and rotifers reappeared in just 1 - 2 months. In the alpine lakes studied by Anderson (1970), the rotifers had completely recovered to their former levels of diversity and abundance in 11 - 12 months, a full two years before the crustaceans did so.
- 2) Time of reproduction . Anderson (1970) states that rotenone is more devastating to those species which have not reached reproductive maturity by the time rotenone is applied. This was the case with the copepod *Diaptomus sicilis* in a Canadian alpine lake, and it was the last species to recover. In general, the major reproductive peak occurs in the spring, with a lesser one in the fall; but the precise timing depends on the species and water conditions (Arni Litt, UW, pers. comm.).
- 3) Ability to form resistant stages . All cladocerans, copepods, and rotifers have the ability to form ephippial or other overwintering eggs; in Washington this occurs mostly in the eastern half of the state where ice cover forms (Arni Litt, UW, pers. comm.). Anderson (1970) suggests that such eggs - depending on when in the fall a particular species produces them - could resist the poisoning and aid in recovery. Bandow (1980) suspected that this ability allowed *Daphnia* to become the dominant crustacean in a rotenoned lake. Brynildson and Kempinger (1973) stated that the "comeback" of *Daphnia* in a Wisconsin lake after rotenone may have been partly due to ephippia which hatched the following spring.

Partial Recovery - Long before zooplankton communities recover to the point where all species have reappeared in their approximate prerotenone levels, there is usually a point when most species are present in large numbers.

Anderson (1970) noted that even in the extreme case of alpine lakes where complete recovery took as long as three years, most species of crustaceans had reappeared within 10 months of poisoning. More relevant to Washington's lowland lakes is the case of Fern Lake, Washington, where although complete recovery in all habitat types took 17 weeks, the authors suggested that zooplankton populations had recovered to the point where trout could be restocked in just 9 - 10 weeks (Kiser et al., 1963). Two weeks later, all open water crustaceans had returned to prerotenone levels. WDW restocked Fern Lake 37 days after poisoning, and the authors suggested that this may have been about five weeks too soon in view of the reduced plankton levels.

During this period to complete recovery, there are often shifts in the zooplankton community structure. One of the more gross changes is the temporary disappearance of the cladocerans and copepods, while rotifers dominate. Researchers have reported other unusual changes in the community during recovery as well: Neves (1975) noted minor rotifer "blooms" during recolonization of a poisoned lake cove, probably due to lack of competition and/or low predation by grazing plankters. Walters and Vincent (1973) also observed a temporary rotifer "bloom" during recovery. Patricia and Celestine lakes saw an increase in small sized cladocerans (Anderson, 1970). In Fern Lake, a number of weedy shoreline plankters unaffected by rotenone invaded the open-water areas of the lake and became dominant for about nine weeks. By the twelfth week they had been gradually excluded by the original open-water species which had returned. (Kiser et al., 1963). Anderson (1970), though he did not sample the shoreline areas, noted "new" species in the open-water tows on Patricia and Celestine lakes, and suggested that the same invasion by resistant shoreline plankters seen in Fern Lake was occurring. Neves (1975) did not observe this phenomenon in a treated cove, claiming that quick recolonization from outside by open-water plankters was the reason.

These changes in community structure were all relatively minor and temporary. Probably the most commonly observed change in zooplankton community structure during the recovery period is the dominance of large sized cladocerans after treatment (Bandow, 1980; Hrba'cek and Novotna'-Dvora'kova', 1965; Anderson 1970; Gustafson et al., 1981; Serns, 1979; Walters and Vincent, 1973; Stenson, 1972). All of these authors trace this change to the absence of predatory fish in the lake following poisoning. And, depending on whether or not fish are restocked, the change can be temporary or permanent.

Complete Recovery - Zooplankton recovery times in 15 test waters are shown in Table I. Figure 17 graphically displays the recovery time on most of these waters. For interpretation of these results "complete recovery time" means the time it took for all, or nearly all, of the important or sampled elements of the zooplankton community to reappear and reach approximate prerotenone levels of abundance. In a number of instances, the authors have actually stated a recovery time. In others, recovery time must be inferred from the data. In both cases, there are three main reasons why the assignment of recovery times must be regarded as an approximation. 1) the zooplankton community cannot be expected to reappear exactly as before rotenone, there is often a characteristic shift to larger sized plankters in the absence of predatory fish; 2) some studies identified samples down to the generic or specific level, while others used only broad taxonomic descriptions such as "rotifers" and "protozoans". This makes it difficult to establish when diversity has been restored; and 3) zooplankton counts vary widely from year to year, making it difficult to establish prerotenone levels of abundance to use as a "yardstick" in measuring recovery.

The most reliable studies are those in which plankters were identified to the generic or specific level, and samples were collected regularly for several years prior to treatment. Table I shows that recovery times ranged from "immediate" to three years, and in one case (Bandow, 1980) recovery was not complete when the study ended two years after rotenone treatment.

Carls Lake, Minnesota (Bandow, 1980) appears to be a special case: while calanoid copepods never returned to full abundance even after two years, there was a sharp increase in total standing crop of zooplankton, mostly *Daphnia*. Several other factors confuse the picture on Carls Lake: the severe winterkill that disrupted the ecosystem not long before treatment, the double application of rotenone at high dosage (3 ppm), and the post-rotenone introduction of at least six species of fish. The combined effect of these unusual variables makes it hard to draw conclusions on recovery time from this study. Other special cases include Libey and Holland (1980), where the small dosage never affected zooplankton; Wollitz's (1962) treatment of West Pond, an anomaly; and Neves (1975), where the poisoned cove recovered completely in one week due to rapid immigration from the untreated areas.

The remaining examples all show recovery times ranging from two months to three years. Where complete recovery required two and three years (Emmaline, Celestine and Patricia Lakes), it is perhaps relevant to note that all three are oligotrophic alpine lakes. Zooplankton in sterile alpine lakes require an unusually long time to recover (Anderson, 1972; Wrenn, 1965). Moreover, Anderson and Wrenn were only able to sample the lakes one and two months prior to poisoning, making it difficult to say with certainty what the prerotenone abundance levels were.

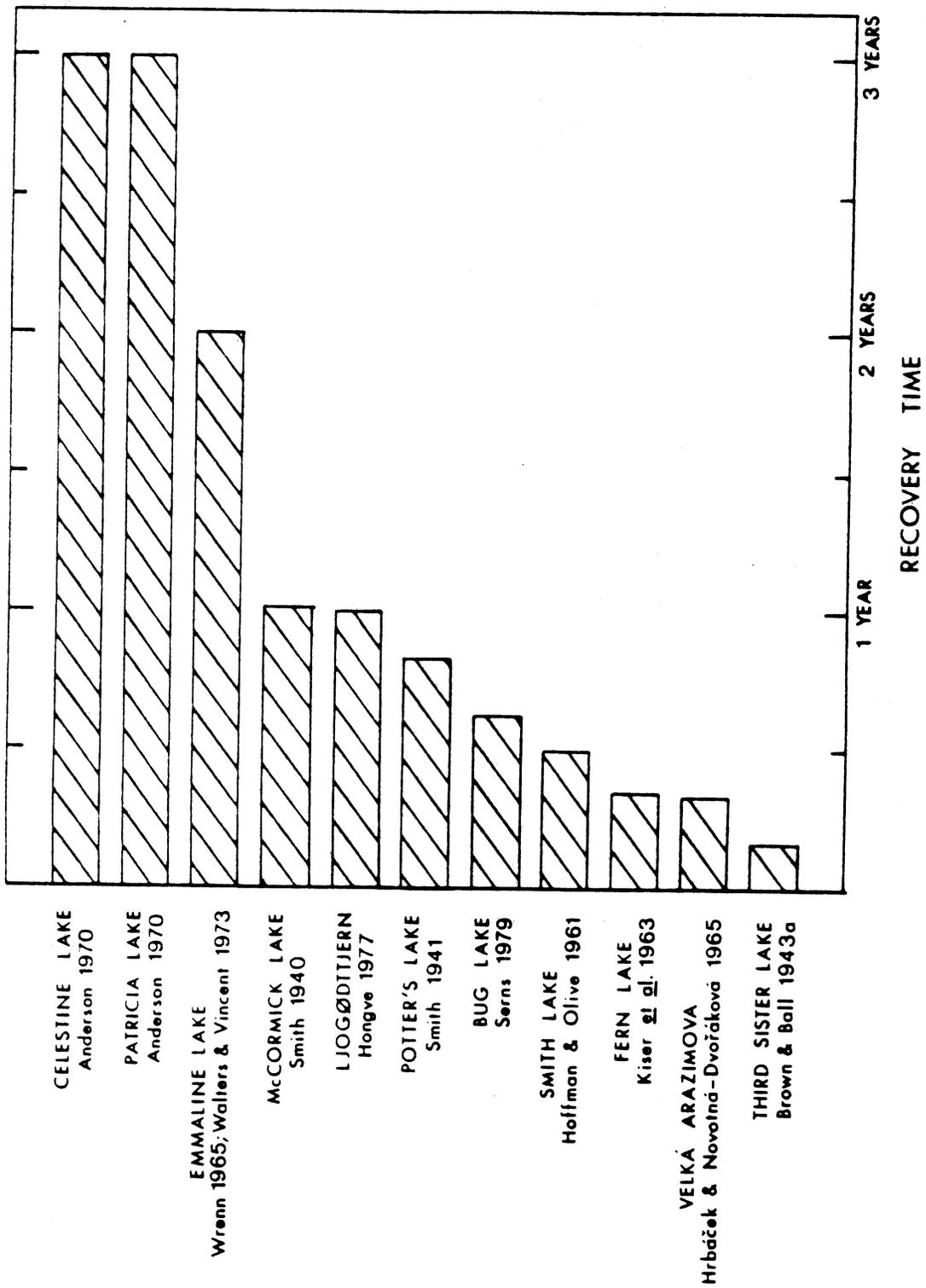


Figure 17. Recovery times for the zooplankton in several lakes and ponds following rotenone. Data are from Table 17.

Complete recovery based on the remaining studies required anywhere from two to twelve months. Eliminating the two month example (Brown and Ball, 1943a) due to their unusually short toxic period for fish, we are left with a range of four months to a year. The most thorough of these studies (based on the three criteria discussed above) is that of Kiser et al. (1963) on Fern Lake, Washington, where recovery took 17 weeks.

In several lakes cited in Table I, zooplankton populations not only recovered to previous levels after poisoning, but exceeded them (Hongve, 1977; Anderson, 1970; Woolitz, 1962; Serns, 1979).

Since none of these four studies involved extensive sampling before treatment, the "increased" populations may not be significant.

The dosages used in most of the studies cited are somewhat less than the statewide average applied in Washington (1.23 ppm). Considering the effect of different lake chemistries on rotenone as well as other variables, it is impossible to say whether or not this is significant. There is no apparent correlation between dosages shown in Table I and the corresponding times to complete recovery. There is also no clear correlation between recovery and either pH or water temperature, despite the fact that a wide range of pH's (5.9-8.9) and water temperatures (38° - 82°F) are represented in the test waters. Two other authors have made statements on recovery time that should be mentioned here, although their data cannot be included in Table I: Schnick (1974), after a review of the literature, concluded that "recovery takes from 1.5 - 3 months"; possibly referring to what has been described as "partial" recovery, where most but not all the important elements of the plankton community have reappeared. Galbraith (1974), reviewing unspecified data on Michigan trout lakes, stated that after rotenone "it takes at least on full year before the *Daphnia spp.* regain their original densities."

Brynildson and Kempinger (1973) recommended speeding up the recovery of *Daphnia*, *Leptodora*, and *Holopedium* in rotenoned Wisconsin lakes by stocking these crustacean plankters shortly after treatment.

Disappearance of Species/Appearance of New Species - Table I shows that in four of ten cases, a species observed before treatment failed to reappear in samples taken after recovery was "complete". In three of these cases (Anderson, 1970; Brown and Ball, 1943a, Serns 1979), the authors suggested that incomplete sampling or the sporadic prerotenone appearance of a rare specimen was responsible for the "disappearance". In the other example, the disappearance of *Daphnia cucullata* from a pond was traced to exclusion by a larger daphnid in the absence of fish (Hrba'cek and Novotna'-Dvora'kova', 1965).

None of the 42 crustacean species in Fern Lake, Washington, disappeared permanently after rotenone (Kiser et al., 1963), and the authors believed that complete elimination of a species was "quite unlikely".

The post-rotenone appearance of species never collected before treatment was common, although not explained or assigned any significance. Kiser et al. (1963) observed that after treatment, many weedy-shoreline plankters invaded open-water areas where they were normally never found. Had the authors not towed the shoreline, these species may have been classed as "new" to the lake. Undoubtedly this happened in some of the other lakes, where shoreline habitat was not sampled. And, since only two of the other studies where "new" species were reported included more than a few months sampling before treatment (Hongve, 1977; Hrba'cek-Novotna'-Dvora'kova', 1965), the chances of missing a seasonal or sporadic species were very high in the others.

These "new" species appearing after rotenone never attained dominance in any of the lakes cited in Table I. Even in the case of Fern Lake, Washington, the large population of cladocerans which dominated nearby untreated lakes never gained a foothold in Fern Lake during its recovery (Kiser et al., 1963).

Fish/Zooplankton Interactions - Because fish are consumers near the top of a lake's trophic "pyramid", and because they make up only a small percentage of the lake ecosystem's total matter and energy, they were once considered unimportant in controlling the plankters. There is now a great deal of evidence to the contrary; fish can and do have a dramatic influence on the zooplankton in a lake (Shapiro et al., 1975; Brooks and Dodson, 1965; Galbraith, 1967).

While zooplankton is not always the main food source for fish (Walters and Vincent, 1973), almost all fish in a lake eat zooplankton to some degree, at some life stage. Naturally, a great deal depends on the lake itself and what other foods it supplies. In general, though, rainbow trout of all ages and sizes often feed heavily on zooplankton in Washington lakes, mainly cladocerans and copepods (Wydoski and Whitney, 1979; Carlander, 1969). They do not feed indiscriminately; instead, they individually select and eat only the largest cladocerans (Galbraith, 1967).

It is not only trout that feed on zooplankton; many of the nonindigenous fish that are targeted for eradication from Washington's trout-only lakes also eat zooplankton. Yellow perch (*Perca falvescens*) of all ages and sizes prey on cladocerans and copepods (Wydoski and Whitney, 1979; Serns and Hoff, 1984) and have been called one of the most important zooplanktivores in the U.S. (Shapiro et al., 1975). Like trout, they select and eat only the largest plankters (Galbraith, 1967; Serns and Hoff, 1984). Zooplankton is important in the diet of fathead minnows (*Pimephales promelas*) (Serns, 1979; Galbraith, 1974), pumpkinseed sunfish

(*Lepomis gibbosus*) (Beard, 1971) and bluegills (*Lepomis macrochirus*) (Krska and Applegate, 1984; Shapiro et al., 1975). Even the brown bullhead catfish (*Ictalurus nebulosus*), commonly regarded as a bottom feeder, is often planktivorous (Bandow, 1980), at times exclusively so (Olson and Koopman, 1976).

When all, or almost all the fish in a lake are eliminated, zooplankton return in large numbers within 2 - 10 weeks of treatment, usually closer to three weeks and major shifts in dominance during the period of time before fish are reintroduced to the lake may reasonably be expected.

In some lakes, when the period of time before reintroduction is very short (e.g., 37 days), nothing of importance happens (Kiser et al., 1963). But most lakes in Washington treated with rotenone in the fall or winter are restocked the following spring, allowing at least five months during which the zooplankton suffer no predation.

Many investigators have reported a shift in community structure, with some plankters exceeding prerotenone levels while others decline. By far the most common shifts observed were:

- 1) an increase in the number of large sized plankters (usually *Daphnia*), with a corresponding decline in the smaller-sized plankters that cannot graze as effectively; and
- 2) an increase in the body size of the already existing plankters.

Table J show the results on six lakes where body-size relationships were examined following rotenone and fish stocking. In all six lakes, large-sized plankters became dominant when fish were absent or scarce; and in the two studies where carapace lengths were measured, the small-sized plankters increased in body size (this may have occurred in the other four lakes as well). In all but one lake, the large-sized species that became dominant already existed in the lakes but in smaller numbers; in Lake Sarvsjon, however, the new dominant plankters had never before been recorded in the lake (Gustafson et al., 1981). In all six cases, the authors stated that these shifts were due to the absence of predation by fish following rotenone. And in every case, the zooplankton community reverted to the "normal" prerotenone conditions once fish were restocked and firmly established again.

Stenson (1972) confirmed these results with an experiment in eight Swedish lakes, which all contained the same types of fish and zooplankton. He poisoned four with rotenone and left the other four as untreated "controls". Zooplankton began to repopulate all four rotenone lakes, and Stenson stocked new fish species (trout) in three of them, while allowing the original species (perch, pike, eels) to re-enter the fourth. During the experiment, predation was low in the newly stocked "trout" lakes, but quickly returned to

Table 3 The effects of rotenone treatment and subsequent fish stocking on the kinds and size of zooplankton in six lakes.

Test water, location	BEFORE ROTENONE		FISH-FREE PERIOD AFTER ROTENONE		FISH & ZOOPLANKTON RE-ESTABLISHED		Reference
	Fish present before rotenone	lake chub mountain whitefish longnose sucker lake trout brook trout rainbow trout	Increase in body size of zooplankton	Large-sized species more abundant	Fish introduced after rotenone	Did community revert to pre-rotenone state?	
Patricia Lake Alberta, Canada	lake chub mountain whitefish longnose sucker lake trout brook trout rainbow trout	not studied	yes	yes	brook trout rainbow trout	yes	Anderson 1970
Celestine Lake Alberta, Canada	lake chub rainbow trout	not studied	yes	yes	rainbow trout	yes	Anderson 1970
Velké Aravimova Czechoslovakia	perch roach bitternling carp	not studied	yes	yes	carp largemouth bass roach bream	yes	Hrbáček & Novotná-Dvořáková 1965
Lake Särvsjön Sweden	whitefish perch northern pike	yes	yes	yes	brown trout arctic char	not studied	Gustafson et al. 1981
Carls Lake Minnesota	brown bullhead bluegill brown trout channel catfish fathead minnow golden shiner green sunfish largemouth bass northern pike walleye	yes	yes	yes	brown bullhead largemouth bass walleye channel catfish brown trout	unclear	Bendow 1980
Emmeline Lakes Colorado	brook trout	not studied	yes	yes	cutthroat trout	yes	Walters & Vincent 1973

normal in the lake repopulated by the original fish. Stenson's results are shown in Table K.

Table K. The effects of rotenone treatment and subsequent fish stocking on the kinds and size of zooplankton in eight Swedish Lakes. Source: Stenson, 1972.

Treatment	Increase in Body Size of Plankters	Shift in Dominance to Large Cladocerans

NO ROTENONE (Control)		
-Original fish species	NO	NO
-High predation (4 lakes)		

ROTENONE		
-Original fish species re-entered	NO	NO
-High predation (1 lake)		

ROTENONE		
-New fish species stocked	YES	YES
-Low predation (3 lakes)		

Clearly, the scarcity of fish in the newly stocked trout lakes allowed the larger cladocerans to become dominant, and also allowed the mean body size of the cladoceran *Bosmia* to increase. Most interesting are the results in the single rotenoned lake where the original fish repopulated after rotenone; the zooplankton community recovered and was identical to the nonrotenoned lakes. These results concur with those in Table J where communities reverted to their prerotenone state once fish were restocked. Stenson showed conclusively that it was the lack of predation, not the rotenone that changed the community.

In general, large daphnids are not found in lakes with many planktivorous fish, although there are some notable exceptions, i.e., Lake Washington (Edmondson and Litt, 1982).

There is concern that when trout are stocked following poisoning that not only will they return the zooplankton community to former levels of abundance, but that they will eventually eliminate it.

When trout are stocked in formerly fish-free lakes, for example, dominant plankters are often dramatically reduced or eliminated. Anderson (1972) stocked trout in an alpine lake that had never supported a fish population before, and with no other food source available (e.g., benthic invertebrates), they eliminated the dominant plankters within two to six years. When rainbow trout were introduced into Medical Lake, Washington, they largely eliminated the dominant plankter, *Daphnia pulex*. Knapp and Soltero (1983) felt that this loss of a preferred food item would jeopardize the newly established trout fishery.

Most of the Washington state lakes treated with rotenone have been routinely stocked with fingerling trout for many years and poisoned at more or less regular intervals. To be considered for lake rehabilitation, the lake must provide good fingerling survival and growth as indicated by yearly gill-net sets and creel checks. This empirical evidence suggests that trout stocking at historical levels does not reduce zooplankton to the point where trout growth is affected.

There is also no evidence in the literature to suggest that continued stocking in traditionally successful trout waters eliminates zooplankton as a food source. Galbraith (1967) reported that trout reduced the *Daphnia pulex* population in Sporley Lake, Michigan, to the point where the fishery deteriorated. Further research, however, showed that perch, fathead minnows, and smelt were important contributors to the *Daphnia* decline; even when trout stocking was discontinued for four years, the daphnid population stayed at very low levels. Only after the perch, fathead minnows, and smelt were poisoned with rotenone did *Daphnia* return (Galbraith, 1974).

A similar situation developed on Nebish Lake, Wisconsin, after rotenone; while both hatchery trout and yellow perch preyed heavily on large *Daphnia*, it was the exploding perch population that eventually overgrazed the lake (Brynildson and Kempinger, 1973).

The data from lakes with established fish populations at the time of rotenone treatment (Kiser et al., 1963; Anderson, 1970; Hrba'cek and Novotna'-Dvora'kova', 1965; Walters and Vincent, 1973; Stenson, 1972) show that when fish are restocked, the zooplankton community returns in kind and number to the prerotenone state.

In those lakes which contain planktivores other than trout (such as yellow perch, fathead minnows, bluegills, etc.), it is reasonable to assume that even after restocking with trout, there could be a net decrease in predation on zooplankton due to the absence of the other planktivorous fish. If this occurred, it would be a temporary situation, since the target fish populations usually re-establish themselves after a few years.

Benthic Fauna

Short Term Effects - Table L displays the results of bioassays performed on various benthic animals found in lakes and ponds. The widely cited results of Leonard (1939) have been omitted; in his tests, Leonard found that rotenone dosages as high as 2 ppm had no effect on a variety of benthic animals, but since that time, several authors have cast doubt on the quality of his rotenone formulations (Almquist, 1959; Kiser et al., 1963). Many of these studies were performed before the standardization of laboratory toxicity test (96-hour LC50's being the current standard), so it is impossible to perform any meaningful quantitative comparison which includes all the data. In addition, all of the research except for that of Zischkale (1952) involved testing of benthic animals in bare aquariums devoid of any natural substrate. Since Lindgren (1960) has shown this to be an important, if not overriding factor in benthic mortality with rotenone, the results cannot be reliably extrapolated to a real lake environment.

Laboratory tests are not without value since they can be used to understand the relative susceptibilities of different benthic animals. Figure 18 broadly groups several types of benthic animals, giving a rough idea of the varying susceptibility of each to rotenone. Data are drawn from Table L, utilizing only LC50's for exposures ranging from 24 to 96 hours. Some other data from Table L are included as well, where tests indicated that a particular concentration killed 50% of the animals; in a strict sense, these are not LC50's, though their inclusion here is justified since they provide extra data.

Figure 18 shows the decapod crustaceans (mostly crayfish) to be the most tolerant group, followed in descending order by caddisfly larvae, aquatic snails, and clams, the larval stages of dragonflies and damselflies, phantom midges, true midges and mayflies. This figure includes all the important components of lake benthos except for the oligochaete worms (aquatic earthworms, or Tubificidae), which have not been tested in the laboratory. True midges (chironomids) generally make up the bulk of the benthic biomass in most lakes and ponds (Merritt and Cummins, 1978).

Lindgren's (1960) laboratory tests showed what an important influence access to the bottom sediments has on the survival of benthic fauna exposed to rotenone. Figure 19 shows clearly that when midge larvae had access to the bottom muds, they sustained only a 50% mortality when subjected to a dosage ten times that which killed all midges in a bare aquarium (3.0 ppm as opposed to 0.3 ppm).

Rotenone's immediate effect on benthic animals in lakes and ponds varies, but it does not affect them as drastically as it does plankton. Table M displays the results of thirteen studies on 23 lakes and ponds; in nine of these, the investigators recorded

Table L Toxicity of rotenone to benthic animals in laboratory bioassays.

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
TURBELLARIA (flatworms)							
<u>Planaria</u>	0.500	48 hrs.			5% rotenone	100% mortality	Hamilton 1941
<u>Catenula</u> sp.	1.72	96 hrs.	16±1°	20 mg/l total hardness pH 6.6, limed water	Noxfish	LC50	Chandler & Marking 1982
HIRUDINEA (leeches)							
und. leeches	0.100	48 hours			5% rotenone	90% mortality	Hamilton 1941
	0.100	55 hrs.			5% rotenone	100% mortality	
CONCHOSTRACA							
<u>Estheria mexicana</u>	0.050	48 hrs.			5% rotenone	90% mortality	Hamilton 1941
	0.050	58 hrs.			5% rotenone	100% mortality	
OSTRACODA							
<u>Cypridopsis</u> sp.	0.340	96 hrs.	16±1°	20 mg/l total hardness pH 6.6 limed water	Noxfish	LC50	Chandler & Marking 1982
<u>Eucypris</u>	0.1	48 hrs.	27-29°	pH 7.2	4.9% rotenone	Minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
MALACOSTRACA							
ISOPODA							
<u>Asellus aquaticus</u>	0.5	6 days	11±1°	pH 7.0 260 mg/l hardness	5% rotenone	30% mortality	Meadows 1973
	1.0	47 hrs.	18±1°		Chem-Fish Special P10-Noxfish	60% mortality	Lindgren 1960
AMPHIPODA							
<u>Gammarus pulex</u>	2.0	6 days	11±1°	pH 7.9 260 mg/l hardness	5% rotenone	10% mortality	Meadows 1973
<u>Hyallela</u>	0.2	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimal lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952

Table C Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water Chemistry	Formulation	Comments	Reference
<u>MAJUSIRACA</u> (cont'd.) und. amphipods	0.500	12 hrs.			5% rotenone	100% mortality	Hamilton 1941
DECAPODA							
<u>Palaeomonetes</u> <u>kadiakensis</u> (fresh-water prawn)	1.12	96 hrs.	16 ± 1°	20 mg mg/l total hardness pH 6.6 lined water	Noxfish	LC50	Chandler & Marking 1982
<u>Palaeomonetes</u> (freshwater prawn)	4.0	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischke 1952
<u>Cambarus immunis</u> (crayfish)	0.500				5% rotenone	unaffected	Hamilton 1941
<u>Procambarus</u> sp. (crayfish)	10.0	96 hours	20±1°	pH 7.2-7.8 100 mg/l hardness	5% wettable powder	2.5% died	Brown 1973
	75.0	96 hrs.	20±1°	pH 7.3-7.8 100 mg/l hardness	5% wettable powder	5% died	
<u>Orconectes immunis</u> (crayfish)	34.5	24 hrs.	12°	pH 7.2-7.6	Noxfish	LC50	Fairinger 1972
	10.8	24 hrs.	12°	40-48 mg/l hardness	Dri-Noxfish	LC50	
	47.2	24 hrs.	12°	pH 7.6-8.0	Noxfish	LC50	
	9.6	24 hrs.	12°	160-180 mg/l hardness	Dri-Noxfish	LC50	
	1.0	96 hrs.	12°	pH 7.2-7.6	Noxfish	LC50	
	0.7	96 hrs.	12°	40-48 mg/l hardness	Dri-Noxfish	LC50	
	1.2	96 hrs.	12°	pH 7.6-8.0	Noxfish	LC50	
	0.4	96 hrs.	12°	160-180 mg/l hardness	Dri-Noxfish	LC50	
EPIHEMEROPTERA (mayflies)							
<u>Siphonurus</u>	1.25	48 hrs.	21-23°	pH 7.0-7.3	5% rotenone	50% mortality	Claffey & Ruck 1967
<u>Caenis</u> sp. (mayfly)	0.1	30 hrs.	18±1°		Chem-Fish Special Pro-Noxfish	50% mortality 50% mortality	Indgren 1960
<u>ODONATA</u> (dragonflies & damselflies)							
<u>Macromia</u> sp.	1.00	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness lined water	Noxfish	LC50	Chandler & Marking 1982

Table 1 Continued

Organism	Dosage (ppm)	Exposure	Water Temp. °C	Water Chemistry	Formulation	Comments	Reference
COGNATA (cont'd.)							
<u>Amphigrion</u> sp.	2.5	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischewale 1952
<u>Pachydiplax</u> , <u>Iraema</u>	3.5						
<u>Basiaeschna janata</u>	0.22	96 hrs.	22°	pH 7.2 140 mg/l total hardness	"rotenone"	LC50	Watkins & Tarter 1974
<u>Anax</u>	2.25						
<u>Aurion</u>	2.6	48 hrs.	21-23°	pH 7.0-7.3	5% rotenone	50% mortality	Claffey & Ruck 1967
HEMIPTERA (bugs)							
<u>unid. corixids</u> (water boatmen)	1.000	48 hrs.			5% rotenone	40% mortality	Hamilton 1941
<u>unid. notonectids</u> (backswimmers)	0.01 0.025 0.100	24 hrs. 24 hrs. 24 hrs.			5% rotenone	unaffected unaffected 50% mortality	Hamilton 1941
<u>Notonecta</u> sp. (backswimmers)	0.1 0.5		18±1°		Chem-Fish Special Pro-Noxfish	29% mortality 72% mortality	Indgren 1960
<u>Notonecta</u> sp. (backswimmers)	1.58	96 hrs.	16 ± 1°	pH 6.6 limed water 20 mg/l total hardness	Noxfish	LC50	Chandler & Marking 1982
COLEOPTERA (beetles)							
<u>Gyrinus</u> sp. (whirligig beetle)	0.700	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
TRICHOPTERA (caddisflies)							
<u>Hydropsyche</u> sp.	0.605	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
<u>Hesperophylax</u> sp.	10.4 5.1	24 hrs.	12°	pH 7.2-7.6 40-48 mg/l hardness	Noxfish Orl-Noxfish	LC50 LC50	Farringer 1972

Table L Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
TRICHOPTERA (cont'd.)							
<u>Hesperophylax</u> sp.	15.0 5.1	24 hrs.	12°	pH 7.60-8.0 160-180 mg/l hardness	Noxfish Dri-Noxfish	LC50 LC50	
	3.4 3.2	96 hrs.	12°	pH 7.2-7.6 40-48 mg/l hardness	Noxfish Dri-Noxfish	LC50 LC50	Farringer 1972
	2.5 3.6	96 hrs.	12°	pH 7.6-8.0 160-180 mg/l hardness	Noxfish Dri-Noxfish	LC50 LC50	
DIPTERA (true flies)							
<u>Culex</u> , <u>Aedes</u> , <u>Anopheles</u> (mosquitoes)	2.0	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
midges							
	3.0 6.0	18 hrs. 18 hrs.				10% mortality 5% mortality	
	12.0	18 hrs.			derris	25% mortality	
	2.0			pH 8.3-8.7		5% mortality	Fellton 1940
	3.0	46-52 hrs.		250-350 mg/l hardness	(5% rotenone)	50% mortality	
	4.0					90% mortality	
	5.0					95% mortality	
	6.0					100% mortality	
unid. midges	0.31	48 hrs.	27°		Pro-Noxfish	LC50	Brooks 1961
<u>Tendipes</u> <u>crassicaudatus</u> ,	0.25 0.10	48 hrs.	27°		Cubé Noxfish	LC50 LC50	Wright 1957
<u>I. plumosus</u> (midges)	0.33				Pro-Noxfish	LC50	
<u>Tendipes decorus</u> (midges)	1.0	96 hrs.		pH 5.9-6.1 5-154 mg/l m.o. alkalinity		seriously affected	Taube et al. 1954
<u>Chironomus</u> (midges)	0.1	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952

Table L Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
DIPTERA (cont'd.)							
unld. phantom midges	1.13	48 hrs.	27°		Pro-Noxfish	LC50	Brooks 1961
<i>Chaoborus punctipennis</i> (phantom midges)	0.65 1.07 1.13	48 hrs.	27°		Cubé Noxfish Pro-Noxfish	LC50 LC50 LC50	Wright 1957
<i>Chaoborus astictopus</i> (winter larvae)	1.0 0.5				derris (5% rotenone)	98% mortality 97% mortality	Neighbon 1959
<i>Palpomyia</i> sp. (biting midges)	3.0	36 hrs.	18±1°		Chem-Fish Special Pro-Noxfish	80% mortality 50% mortality	Indyren 1960
unld. chironomid	0.5	32 hrs.	18±1°				
ACARI (water mites)							
unld. water mites	0.0166 0.05	4 days			5% rotenone	unaffected 43% mortality	Hamilton 1941
MOLLUSCA (snails & clams)							
<i>Physa halei</i> (snail)	0.1	48 hrs.			5% rotenone	20% mortality	Hamilton 1941
<i>Lymnaea stagnalis</i> (snail)	1.0 0.5	3.5 days			5% rotenone	70% mortality 30% mortality	Hamilton 1941
<i>Physa pomilla</i> (snail)	4.00					LC50	
<i>Dytrema catenaria</i> (snail)	1.75					LC50	
<i>Heliosoma</i> sp. (snail)	7.95	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
<i>Elliptio buckleyi</i> (Buckley's filter clam)	2.95					LC50	
<i>Elliptio complanata</i> (flattened filter clam)	2.00					LC50	

Table L Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
<u>MOLLUSCA (cont'd.)</u> <u>Corbicula manilensis</u> (Asiatic clam)	7.50	96 hrs.	16±1°	ph 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
<u>Physa</u> (snail)	4.5	48 hrs.	27-29°	ph 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
<u>Helisoma</u> (snail)	3.5						
<u>Viviparus viviparus</u> (snail)	1.0 5.0	110 hrs. 57 hrs.	18±1° 18±1°		Chem-Fish Special Pro-Noxfish	unaffected 50% mortality	I Indgren 1960
<u>Planorbis planorbis</u> (snail)	10.0	47 hrs.	18±1°		Chem-Fish Special Pro-Noxfish	unaffected	I Indgren 1960

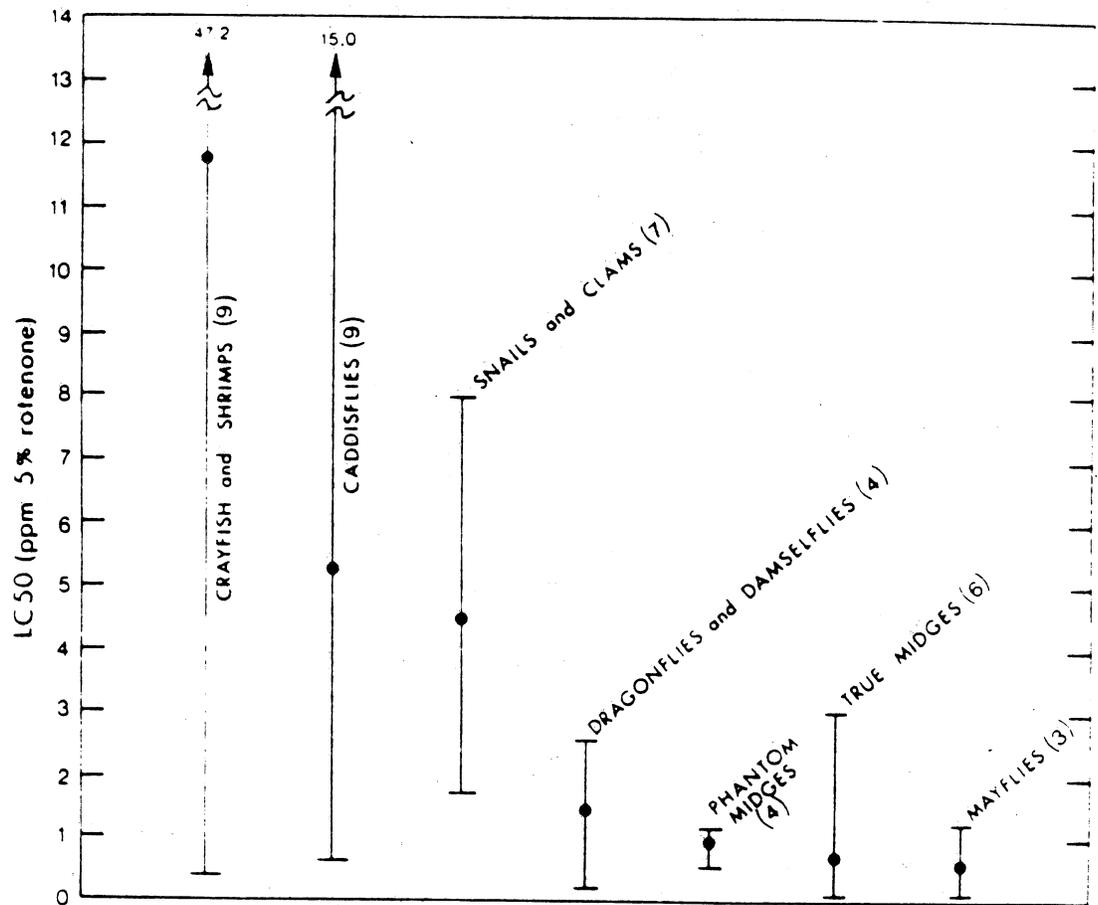


Figure 18 Mean LC50's of rotenone formulation for various groups of lake and pond benthos. Data are drawn from Table 13. Vertical bars represent the range of LC50's found in the literature. Numbers in parentheses represent the number of data points (tests) used in computing the means.

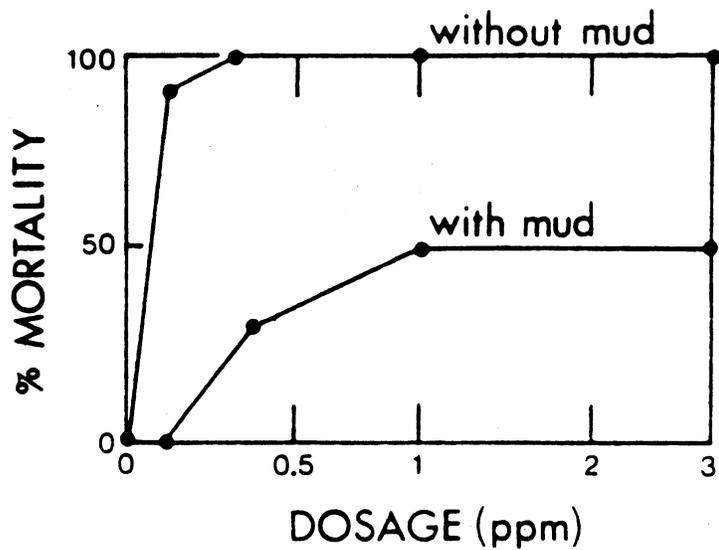


Figure 19 Effect of bottom muds on the survival of midge larvae (Chironomus plumosus) in aquariums subjected to various dosages of rotenone (ChemFish Special). Source: Lindgren 1960.

Table M Short and long-term effects of rotenone treatment on the benthos of various lakes and ponds.

Test water location	Surface acreage	Dosage (Formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of abundance	Time to recover to former abundance levels	Any species fall to abundance reappear	Fish introduced after rotenone (release date)	Increase in benthic abundance from pre-rotenone levels	Comments	Reference
Ponds C & D Missouri	0.1 each	0.5 (Noxfish)	3 mo.	1 year	none	0% within 14 days	Immediate	NO	none	no significant increase	-ponds heavily vegetated, muddy -results compared with untreated control ponds	Houff & Campbell 1977
Pond E Missouri	0.1	2.0 (Noxfish)	3 mo.	1 year	none	0% within 14 days	Immediate	NO	none	no significant increase		
Pond I Georgia	0.1	2.0 (Pro-Noxfish)	1 day	69 days	none	29.5% ^{a/} within 7 days	< 37 days	NO	none	121% (69 days later)	-few aquatic plants	Burress 1982
Pond II Georgia	0.1	5.0 (Pro-Noxfish)	1 day	69 days	none	59.0% ^{b/} within 7 days	< 37 days	NO	none	223% (69 days later)	-only significant decreases in Caenis (mayfly) and 5 genera of Odonata	
7 lakes Finland	2-11	up to 0.8 (synergized 2.5% rotenone)	1 year	4 years	perch	---	---	NO	brown trout, rainbow trout (~9 mo.)	0-400% (1-4 yrs later)	-"after the poisoning, the bottom animal density increased in all lakes."	Tuurnainen 1970
Bug Lake Wisconsin	11	2.5 (Pro-Noxfish)	3 mo., 24 days	2 years	shiners, minnows, LM bass, pumpkinseed, rock bass	---	---	NO	brook trout, (7 mo.) fathead minnow (1 yr., 7 mo.)	---	-no significant change in density of dipterans, oligochaetes, Odonata, and gastropods -trichopterans in shallow water never completely recovered, but this not due to rotenone	Sains 1979

Table M Continued

Test water location	Surface acreage	Dosage (formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of abundance	Time to recover to former abundance levels	Any species fail to reappear	Fish introduced after rotenone (release date)	Increase in abundance from pre-rotenone levels	Comments	Reference
Bill's Lake 11 New Brunswick, Canada		0.25 (derris (5% rotenone))	2 days	9 days	eels, lake chub, perch, SM bass, stickle-back	6% within 8 days	---	---	---	---	-rotenone had "no apparent effect on this community of bottom organisms with the possible exception of <u>Hyallela knickerbockeri</u> ."	Smith 1940
Potter's Lake, New Brunswick, Canada	113	0.5 (derris (5% rotenone))	2 months	11 months	eels, sucker, brown bullhead, SM bass, pickerel, perch, sunfish	0% within 10 days	Immediate	YES	brook trout (~11 months)	no significant increase	-Chaoborus killed in large numbers, as well as an unidentified leech.	Smith 1941
Velká Arazimova Czechoslovakia	0.1	"Lonchocarpus extract"	2 months	2 years, 3 months	perch, roach, bitterling, carp	58% within 3 weeks	< 2 months	---	carp, LM bass, roach, bream (1 month-2 years)	170% (1 year later)	"the mass development of the bottom fauna was caused by exceptional supply of food from the water column to the bottom."	Lellák 1965
Emaline Lake Colorado	3	"rotenone" 1.0 (Pro-Noxfish)	3 months	4 years	brook trout	---	---	---	cutthroat trout (3 years, 9 months later)	~500% (4 years later)	-small chironomids replaced by larger ones in absence of fish	Walter & Vincent 1973 Wrenn 1965

Table M Continued

Test water location	Surface acreage	Dosage (formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of abundance	Time to recover to former abundance levels	Any species fail to reappear	Fish introduced after rotenone (release date)	Increase in benthic abundance from pre-rotene levels	Comments	Reference
Carls Lake Minnesota	110	3.0 (Chem-Fish, 2 treatments)	1 year, 3 months	2 years	black bullhead	---	---	NO	LM bass, walleye, channel catfish, brown trout, pike, bluegills, black bullhead (within 1 year)	no significant increase	"rotenone... might have caused a temporary reduction in some species and in the total number of macroinvertebrates..."	Bandow 1980
Middle Pond Montana	20	0.7 (Chem-Fish Special)	1 year, 10 days	9 months	LM bass, black crapple, perch, sucker, carp, black bullhead	42% within 6 days	< 1 month	---	---	274% (37 days later)		Mollitz 1962
West Pond Montana	13	0.7 (Pro-Nox/fish)	1 year, 13 days	9 months	perch, bluegill, LM bass	71% within 7 days	40 days	---	---	no significant increase		
Third Sister Lake Michigan	10	0.5 (derris (5% rotenone))	2 years, 1 month	1 year	bluegill, LM bass, yellow bullhead, shiners	23% ^{c/} within 3 weeks	< 1 month	---	none	~100% (8 months later)	-almost all Chaoborus larvae killed -leeches, aescoline dragonflies severely affected	Ball & Hayne 1952

Table M Continued

Test water location	Surface acreage	Dosage (formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of abundance	Time to recover to former abundance levels	Any species fail to reappear	Fish introduced after rotenone (release date)	Increase in benthic abundance from pre-rotenone levels	Comments	Reference
Irwin Lake Michigan	10	0.55 (5% emulsified rotenone)	3 days	1 year, 2 wks.	perch	10% within 4 days	---	NO	brook trout (1 year later)	---	only Cheoborus significantly affected	Trude et al. 1954
Dawning Lake Minnesota	13	0.5 (derris root)	9 days	11 days	---	OK within 11 days	Immediate	---	---	---	---	Hooper 1948

- a/ Actual decrease was 66.5%, but a 37% decrease occurred in the untreated control pond during the same time; decrease due to rotenone was found by subtraction (66.5% - 37% = 29.5%).
- b/ Actual decrease was 96%. See note a/ above.
- c/ Authors did not mention a decrease, but their data show a decrease of 23% when compared to the mean decreases in May-June for the three years when the lake was not poisoned.

benthic abundance within three weeks or less of treatment. These data were either compared with previous bottom grabs in the same lake, or with untreated "control" waters.

There is no clear correlation between rotenone dosage and the number of benthic animals lost shortly after treatment. The factors that most likely influenced the varied results were the differing environmental conditions (especially the amount of submerged vegetation and the bottom type) in the lakes and ponds tested. For example, Houf and Campbell (1977) reported no loss of benthos following a heavy application of 2 ppm Noxfish, but their results may be influenced by the fact that their experimental ponds were heavily vegetated and had very muddy bottoms. Both these factors play important roles in detoxifying rotenone and providing a safe haven for benthic animals. The same dosage (2 ppm) in a similar-sized pond with very little aquatic vegetation destroyed almost 30% of the benthos when compared with the untreated control pond (Burruss, 1982). Unfortunately, there are not enough compatible data on these environmental variables from all the studies to fully explain the different results.

Although they did not provide enough quantitative data to be included in Table M, a number of other researchers have reported the short-term effects of rotenone treatment on benthos.

Most have reported that rotenone's impact is mild: Hongve (1977) stated that benthic insects were not affected by a dosage of 0.5 Pro-Noxfish in a Norwegian lake. Neves (1975) found that most benthic invertebrates were not distressed by a 0.6 ppm Noxfish treatment of a lake cove, although some dead mayfly and biting midge larvae appeared in subsequent plankton hauls. After poisoning two Canadian lakes with 0.75 ppm derris, Anderson (1970) concluded that benthic oligochaetes, dipterans, caddisflies, and damselflies appeared unaffected by the rotenone; leeches and snails, however, showed high mortalities. Cushing and Olive (1957) reported that oligochaetes were not affected by 1.0 ppm derris in Smith Lake, Colorado, and that reductions in the midge larvae were apparent for only three days following poisoning. Wright (1957) found that 1 ppm Noxfish and Pro-Noxfish did no harm to midge larvae. Zilliox and Pfeiffer (1960) found that rotenone products at 0.5 ppm did not adversely affect the fish-food organisms in New York lakes.

Some authors have reported drastic reductions in benthos following rotenone: Berzins (1958) found that 0.5 ppm rotenone destroyed most of the benthos of two lakes in southern Sweden. Oglesby (1964) reported that a freshwater polychaete, *Nereis limnicola*, was almost entirely exterminated following a 0.5 ppm treatment of Lake Merced, California, with 5% rotenone.

Taube et al. (1954) documented catastrophic reductions of benthos in five Michigan lakes treated with Fishtox (a 5% emulsifiable

liquid) and one treated with 1.7 ppm emulsifiable rotenone. Even a year after treatment, benthic animal density was down 73% - 97% over previous levels. These lakes remained toxic to fish for an unusually long time - between 19 and 33 months after treatment, even though qualitative tests for rotenone proved negative. The authors therefore suspected that their emulsions had been contaminated with a chemical dispersing agent which was responsible for both the extended toxicity and the benthic kill.

Susceptibility of Different Benthic Animals - Laboratory tests have shown that certain types of benthic fauna are more tolerant of rotenone than others. Research in the field has generally corroborated these laboratory findings. Crayfish proved highly tolerant in Bluewater Lake, New Mexico (Huntingdon, 1956), where they were not affected by 1.5 ppm. Lindgren (1960) noted that the genus *Cambarus* was very tolerant of rotenone, and Bocardy and Cooper (1963) reported that crayfish were unaffected in a Pennsylvania stream treated with rotenone. Dead crayfish were reported on the bottom of Liberty Lake, Washington following rotenone treatment (Funk, WSU, pers. comm.).

Gastropods (snails), also shown by laboratory tests to be relatively tolerant, have survived rotenone treatments in the field as well (Smith, 1941; Hooper, 1948; Serns, 1979), although Anderson (1970) reported that snails were among the first benthic animals to show high mortality following a 0.75 ppm treatment in a Canadian lake, and Smith (1941) noted disappearance of a snail, *Campeloma decisum*, after rotenone.

While no laboratory tests are available for comparison, investigators in the field have usually cited oligochaetes (aquatic earthworms, Tubificidae) as being among the most tolerant benthic organisms (Cushing and Olive, 1957; Anderson, 1970; Hooper, 1948; Serns, 1979; Bandow, 1980; Lindgren, 1960), with only one author reporting large kills of oligochaetes following rotenone poisoning (Wollitz, 1962).

Mayfly larvae, shown in laboratory tests to be very sensitive, have been killed in large numbers in several lakes (Neves, 1975; Burrell, 1982) while other benthic animals were unaffected or reduced at a lesser rate. Midge larvae (chironomids) also proved fairly sensitive to rotenone in the laboratory (see Figure 18), and field investigators have reported heavy losses following lake and pond treatments (Bandow, 1980; Wollitz, 1962). Anderson (1970), Serns (1979) and Taube et al. (1954), did note that dipteran larvae (largely midges) were unaffected by rotenone treatments.

Leeches were not extensively tested in the laboratory, but Brown and Ball (1943a), and Anderson (1970), Smith (1941), Ball and Hayne (1952), and Meehan (1942) all reported them to be very sensitive to rotenone.

The larval form of the phantom midge is unusual for insects in that it is largely planktonic (Merritt and Cummins, 1978); without the protection of the bottom sediments, and in view of its relatively high sensitivity in the lab (see Figure 18), it might be concluded that they would suffer heavy losses in poisoned lakes. This has been reported in at least four cases (Ball and Hayne, 1952; Smith, 1941; Meehean, 1942; Taube et al., 1954). The latter authors recorded an 82% reduction in *Chaoborus* within five days of poisoning on a Michigan lake. Contradictory reports have come from Hongve (1977) and Wright (1957), both of whom noted chaoborid larvae surviving rotenone treatments in large numbers.

Effects of Insect Emergence - Only one study (Houf and Campbell, 1977) has addressed the direct, short-term effects of rotenone treatment on the emergence of aquatic insects. These authors found no differences in emergence patterns between treated and untreated ponds, and concluded that rotenone at 0.5 ppm and 2.0 ppm did not interfere with insect emergence.

Long Term Effects - Recovery of the Benthic Community - In the eleven studies that quantitatively followed benthic abundance over the long term (i.e., all research cited in Table M except Smith, 1940 and Hooper, 1948), benthos recovered to at least prerotene levels of abundance at some time after poisoning. However in one of these studies (Serns, 1979), "recovery" was somewhat ambiguous; Serns reported that caddisfly larvae at a shallow-water sampling site never reached their former levels, but he blamed sampling variance and subsequent fish introductions rather than the rotenone itself.

Table M shows the results of six studies in which bottom grabs were taken often enough to determine how long recovery took. In two cases (Houf and Campbell, 1977; Smith, 1941), there was never a reduction in total benthic abundance following poisoning, so recovery was essentially "immediate". In the remaining four studies (representing six bodies of water), where between 23% and 71% of the benthic fauna was initially destroyed, recovery took between 1 and 2 months. Schnick (1974) concurred with this stating, after a review of the literature to date that: "benthic organisms reach equilibrium in a few months after treatment".

In many cases, the benthic fauna not only repopulated the lakes following rotenone, but their numbers increased dramatically over pretreatment levels. Table M shows that this occurred in 6 of 10 studies (13 of 18 test waters).

In four of the six studies where benthos increased significantly (Tuunainen, 1970; Wollitz, 1962; Ball and Hayne, 1952; Walters and Vincent, 1973), the authors claimed that reduced fish predation was the overriding cause.

A loss of predatory fish cannot explain the huge increases noted by Burress (1982), since his experimental ponds never contained fish. Burress himself does not venture a guess, but Lellak (1965) has a hypothesis which may explain the post-rotenone explosion of benthos when fish are not a factor. While admitting that the increase in bottom animals in Velka Arazimova was due in part to the elimination of predatory fish, Lellak claims that the most important factor was the "rain" of dead plankton that occurred shortly after poisoning. On reaching the lake bottom, this formed a new supply of food for the benthic fauna. Lellak supports this hypothesis by pointing out that in bottom areas of untreated ponds closed off to fish, benthic biomass doubled; but in poisoned ponds, the biomass increased sometimes 50-70 fold, or definitely more than would be expected as a result of merely removing the fish.

While this nutrient "rain" undoubtedly boosts benthic production, Walters and Vincent (1973) noted that in Emmaline Lake, Colorado, this increase was only temporary; the excess of bacteria and plankton that accumulated there after poisoning was soon depleted by the growing population of benthic animals.

Disappearance of Species - Smith (1941) reported that the snail *Campeloma decisum* never reappeared in bottom grabs on Potter's Lake, Canada as long as 11 months after poisoning. In the other five studies in which data were suitably detailed for analysis (Houf and Campbell, 1977; Burress, 1982; Serns, 1979; Bandow, 1980; Tuunainen, 1970), all taxa present before rotenone reappeared in samples after rotenone.

Effect on Species Diversity - Species diversity has traditionally been used as a monitor of benthic community stability. Pollution and other environmental disturbances tend to produce a community that is rich in terms of total benthic abundance, but poor in terms of the number of species.

Houf and Campbell (1977), Burress (1982) and Bandow (1980) are the only investigators who have used a quantitative diversity index (Wilhm and Dorris, 1968) to thoroughly examine the long-term effects of rotenone on the species diversity of benthic communities. Houf and Campbell (1977) reported that neither 0.5 ppm nor 2.0 ppm dosages of rotenone changed benthic diversity (d) in their experimental ponds. Burress (1982) noted pronounced reductions in diversity after poisoning ponds with 2.0 and 5.0 ppm rotenone. Diversity returned to prerotenone levels 69 days later in the pond given the lighter dosage, but in the heavily poisoned pond, benthic diversity was still reduced at that time. There was, however, a "strong trend toward recovery". Bandow's (1980) results are somewhat complicated by a winterkill, but post-rotenone diversity on Carls Lake, Minnesota was the same or greater than before treatment.

Changes in Community Structure - In addition to increases in total benthic standing crop following recovery from rotenone treatment in several lakes, some investigators have reported increases in the numbers of particular benthic animals within the community.

Oligochaete worms increased dramatically after poisoning in the lakes studied by Hooper (1948), Cushing and Olive (1957), Lellak (1965), and Bandow (1980). In these cases, oligochaete worms were not initially affected by the rotenone. Wollitz (1962), saw a doubling of the tubificid worm population in Middle Pond, Montana even after a drastic initial reduction. In all these cases, the shift was temporary; populations had restabilized at their former levels before the studies ended.

Aquatic snails and clams increased in several lakes following poisoning: Wollitz (1962) reported that the snails *Gyraulus* and *Lymnaea* increased tenfold over prerotenone numbers, while *Physa's* population doubled in a Minnesota lake. Aquatic snails increased in numbers following rotenone in Potter's Lake, Nova Scotia (Smith, 1941). Tuunainen (1970) noted much larger populations of the clam *Pisidium* in most of the seven Finnish lakes he poisoned. It is not clear from the literature whether these shifts to increased numbers of mollusks were temporary or permanent.

The midge population increased dramatically in the two Montana lakes studied by Wollitz (1962) and the Czechoslovakian oxbow poisoned by Lellak (1965). In both these cases, the shifts appeared temporary.

It is tempting to attribute all these shifts in community structure to rotenone tolerance. Oligochaete worms, snails, clams and crayfish are generally regarded as being the benthic animals most resistant to the poison. It may be hypothesized that these groups take advantage of the temporary absence of other more sensitive benthic animals to become dominant. Yet the post-rotenone dominance of midge populations in some instances (Wollitz, 1962; Lellak, 1965) does not fit this hypothesis; not only are midges usually rotenone sensitive in the lab and field tests, but Wollitz recorded a drastic initial reduction of midge larvae before the increase. The elimination of predatory fish may at least partially explain these shifts.

Apart from shifts in numerical abundance of certain benthic animals, only one other change in community structure has been observed following rotenone; Walters and Vincent (1973) found that large-sized midge larvae became dominant after poisoning. This shift has been attributed to a decrease in fish predation.

Fish/Benthos Interactions - When rotenone was used to eliminate fish, benthic animals populations increased in most of the test waters cited in Table M. Most authors credited the sudden reduction in fish predation as the main cause. The "rain" of dead

plankton, bacteria, and epiphyton was also mentioned as a probable catalyst for short-term benthic increases.

When these increases occur, the standing crop of benthos remains at the new, higher level if predatory fish are not restocked. This was demonstrated by Ball and Hayne (1952) when they poisoned Third Sister Lake, Michigan, and purposely avoided restocking so that they could follow the effects. They found that the number of benthic animals doubled; at that point, the benthic community reached a dynamic equilibrium whose limits were determined by factors other than fish predation. Annual cycles of abundance were undisturbed (Figure 20). A doubling of the benthic standing crop following fish removal was also recorded in experimental enclosures on a Swedish lake (Andersson et al., 1978).

Walters and Vincent (1973) ran a similar experiment in Emmaline Lake, Colorado and Figure 20 compares their results with Ball and Hayne (1952). They poisoned the lake's brook trout population and did not stock fish again until almost four years later, near the end of the study. Their results were similar to Ball and Hayne's, with the benthic population increasing about 3.5 times over prerotenone levels. These authors found that, in the absence of fish predation, benthic population regulation at the new, higher level occurred through density-dependent larval mortality.

When fish are restocked into a lake where post-rotenone benthic increases have occurred, the benthic standing crop generally returns to prerotenone levels. Lellak (1965) observed a dramatic increase in pond benthos following poisoning, but two years later (after their gradual introduction of new fish), both the abundance and biomass of the bottom fauna stabilized within the prerotenone limits (Figure 21).

In what is probably the best and most detailed of the studies, Tuunainen (1970) observed a clear relationship between the bottom animals and fish in seven Finnish lakes; after perch were poisoned with rotenone, benthic diversity increased in all the lakes. This increase was most obvious in the year following poisoning. After releasing new fish, brown trout (*Salmo trutta*) and rainbow trout (*Salmo gairdneri*), into the lakes, the benthic standing crop decreased again, although it remained at a somewhat higher level than before rotenone. Thereafter, benthos increased whenever there was a decrease in fish biomass; in some cases, this increase was even greater than the increase just after poisoning. The typical case of Lake Sahalampi is plotted in Figure 21.

While killing all the fish in the test lakes usually resulted in an increase in benthos, there were important exceptions: Table M showed that no increases occurred in the lakes studied by Bandow (1980), Smith (1941), and in one of the ponds studied by Wollitz (1962). Whether or not benthos increases following a fish-kill program depends a great deal on the types of fish killed and their

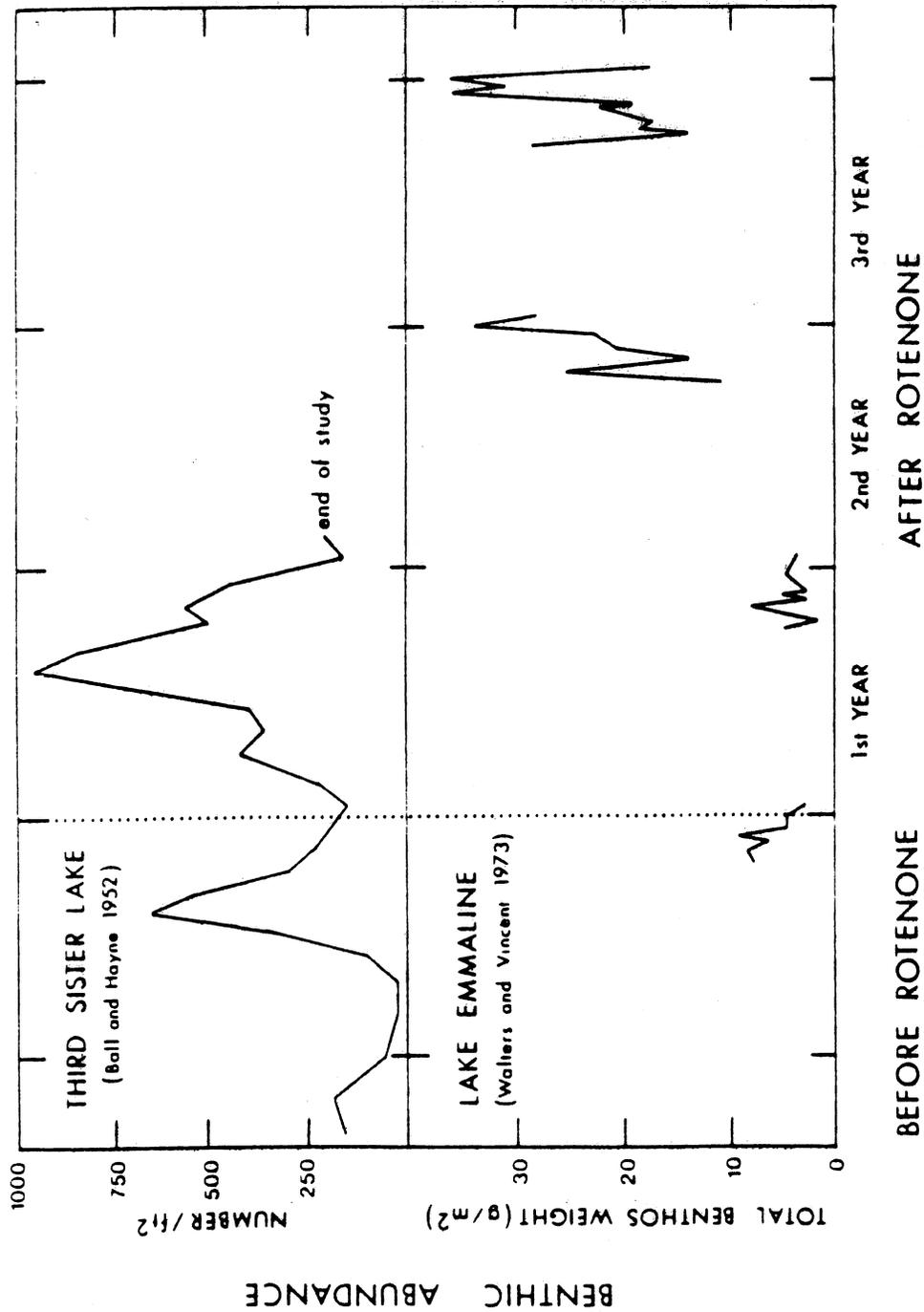


Figure 20 Effect of fish removal on benthos in two lakes where fish were not restocked following rotenone treatment. Dotted vertical line indicates the date of rotenone treatment.

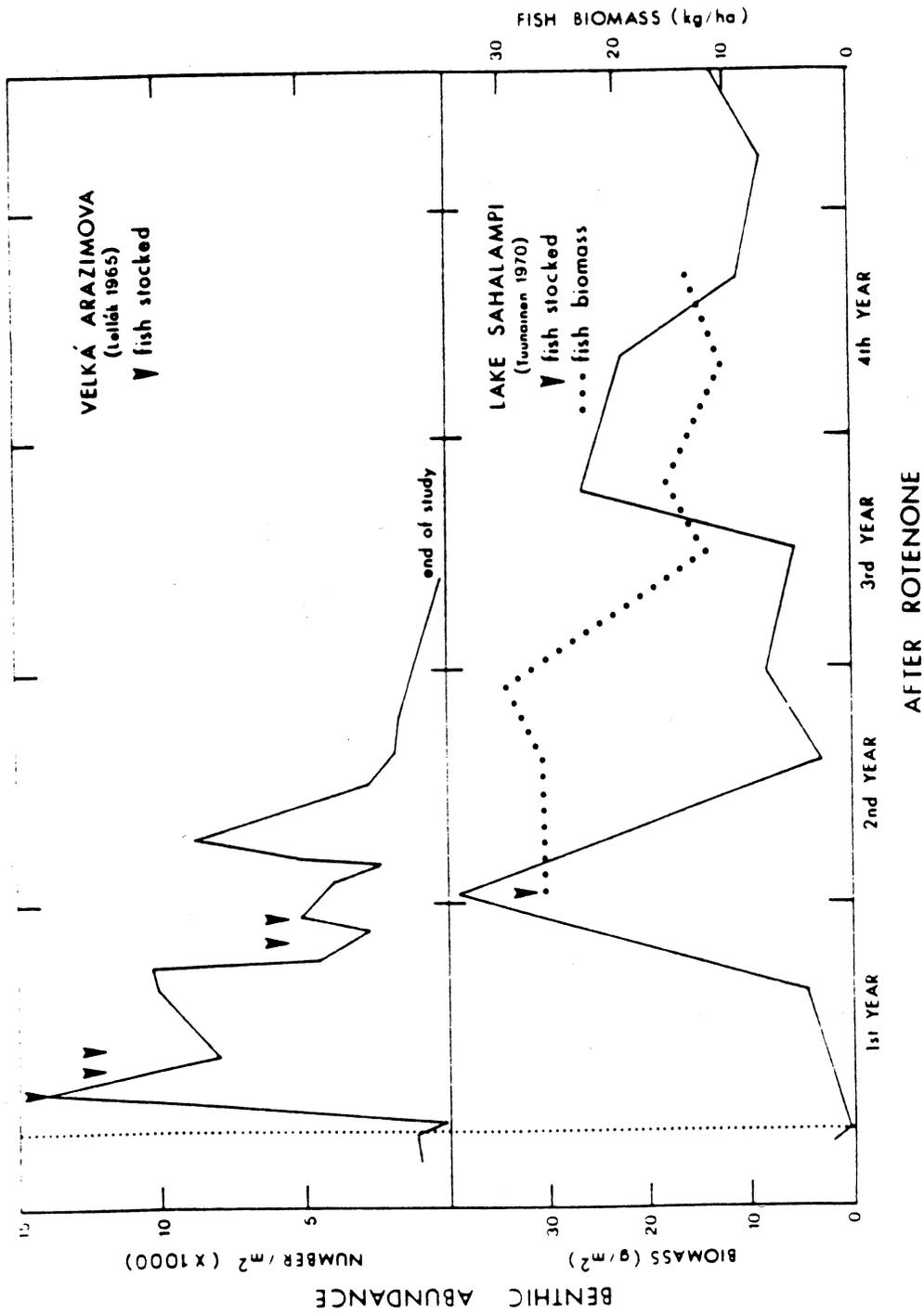


Figure 2) Effect of fish removal and subsequent restocking on benthos in two lakes. Dotted vertical line indicates date of rotenone treatment.

reliance on the lake's bottom animals as a food source. This factor may explain why the standing crop of benthos in Carls Lake, Michigan (Bandow, 1980) was not significantly affected following rotenone; Bandow reported that the most common fish prior to poisoning was the black bullhead (*Ictalurus melas*), and these were heavily dependent on *Daphnia* for food.

No such explanation is readily apparent for the other two cases (Smith, 1941; Wollitz, 1962) in which benthos was unaffected. There are other factors that may influence the way in which a benthic community reacts to fish removal; Tuunainen (1970) claims that lake size alone may be such a factor. With respect to rotenone removal of fish, he states that "small lakes or ponds with quite a small water volume are more susceptible to environmental changes than large ones". Other limnologists have concurred with this statement in regard to fish introductions (Li and Moyle, 1981; Magnuson, 1976). It is probably no coincidence, then, that the two largest lakes studied (Carls Lake and Potter's Lake) showed no long-term changes in the benthic standing crop after fish were killed with rotenone; much smaller lakes always exhibited large increases; with the exception of West Pond (Wollitz, 1962).

A final factor that probably influences the magnitude of fish/benthos interactions in rotenone-poisoned lakes is trophic state. Tuunainen (1970) claims that the effect of removing and restocking fish on the benthic community is much greater in oligotrophic lakes than in eutrophic lakes. As evidence, he compared his oligotrophic Finnish lakes with those eutrophic ponds poisoned by Lellak (1965): the magnitude of the benthic response to fish stocking and changes in fish biomass were dramatic in the nutrient-poor Finnish lakes, while in the eutrophic waters, the bottom fauna restabilized after the initial increase. Li and Moyle (1981) have confirmed that the impact of fish introductions is much greater, and more unpredictable, in oligotrophic lakes than in eutrophic ones.

Emmaline Lake, Colorado was one of the smallest lakes studied, and is also a highly oligotrophic alpine lake: benthos increased most dramatically in Emmaline Lake (~350%) following fish removal, possibly illustrating the combined influence of lake size and trophic state.

Apart from these quantitative changes in the benthic community, only one other aspect of fish/benthos interactions in rotenoned lakes has been studied: Walters and Vincent (1973) noted a shift to larger-sized midge larvae in the absence of fish. Unfortunately, their study did not run long enough following restocking to determine if the situation reversed with fish present.

Effects on Stream Benthos - Although WDW very rarely uses rotenone in running waters, brief mention should be made of the published papers on rotenone's effect on stream benthos. Bridges and Cope (1965), Claffey and Ruck (1967), and Engstrom-Heg et al. (1978) have all performed laboratory bioassays with rotenone on stream insects. Rotenone's short- and long-term effects on stream benthos in the field were investigated by Dexter (1965), Swan (1965), Binns (1967), Cook and Moore (1969) and Helfrich (1978).

In general, these studies demonstrated that rotenone has a far more drastic initial impact on stream benthos than on lake benthos. And while stream invertebrate communities do recover from rotenone, it takes more time than in standing water. The three main reasons for the increased sensitivity of stream benthos are:

- 1) On the whole, stream-dwelling insects themselves are far more sensitive to rotenone than those that live in lakes (Helfrich, 1978; Engstrom-Heg et al., 1978). Considering rotenone's status as a respiratory poison, this stands to reason; most stream invertebrates have very high dissolved oxygen requirements (Engstrom-Heg et al., 1978), and are less tolerant of a wide variety of pollutants than lake-dwelling insects (Hynes, 1970).
- 2) Stream applications, to be effective in killing fish usually require much higher rotenone concentrations than do lakes (Binns, 1967; Engstrom-Heg et al., 1978).
- 3) Streams generally provide less of the organic debris and mud that detoxify rotenone and protect lake-dwelling insects (Lindgren, 1960).

Fish

Short-Term Effects - The median lethal concentrations (LC50) of rotenone formulations for a variety of fish are displayed in Table N. More data are available in the literature, but much of the early work followed no standard procedure; dose-effect experiments have been standardized as 24 to 96-hour LC50's (Marking, 1975), and only these data are reported in Table N.

The upper range of the 96-hour LC50's for all species tested was 0.497 ppm. This is a far lighter dosage than the 1.23 ppm mean dosage used in Washington state lakes. Furthermore, dosages of at least 1 ppm and up to 5 ppm are repeatedly recommended for lake treatments nationwide (Schnick, 1974; Spitler, 1970).

Table N Toxicity of rotenone to fish in laboratory bioassays. All dosages are expressed as median lethal concentrations (LC50).

Species	Dosage (ppm)	Exposure	Temp. C°	Water chemistry	Formulation	Reference
AMIIDAE (bowfins)						
bowfin	0.0575	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0300	96 hr.				
ICTALURIDAE (catfishes)						
channel catfish	0.400	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.161	96 hr.				
	0.033	24 hr.	24°	35 mg/l alkalinity	4.85% rotenone powder	Bridges and Cope 1965
	0.029	48 hr.		pH 7.1		
	0.028	96 hr.				
black bullhead	0.665	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.389	96 hr.				
brown bullhead (1.0-1.4 inches)	0.247	72 hr.	21°	pH 7.8-9.4	Noxfish	Hester 1959a
	0.346			30-50 ppm CaCO ₃	Cubé (7.3% rotenone)	
	0.410				Pro-Noxfish	
brown bullhead (6-8 inches)	0.844	72 hr.	21°	pH 7.8-9.4	Noxfish	Hester 1959a
	0.794			30-50 ppm CaCO ₃	Cubé (7.3% rotenone)	
	1.033				Pro-Noxfish	
SALMONIDAE (trout and salmon)						
rainbow trout	0.031	24 hr.	13°	pH 7.1	4.85% rotenone powder	Bridges and Cope 1965
	0.027	96 hr.		35 mg/l alkalinity		
	0.028	48 hr.				
	0.0689	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0460	96 hr.				
	0.057	96 hr.	12°	*	Chem-Fish Regular	Howland 1969
	0.057	48 hr.	12°	*		
brook trout	0.0470	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0443	96 hr.				

Table N Continued.

Species	Dosage (ppm)	Exposure	Temp. C°	Water chemistry	Formulation	Reference
brook trout (fingerling)	0.0470	96 hr.	12° *		Noxfish	Olson and Marking 1975
lake trout	0.0269	24 hr.	12° *		Noxfish	Marking and Bills 1976
lake trout (fingerling)	0.0269	96 hr.	12° *		Noxfish	Olson and Marking 1975
Atlantic salmon	0.0350	24 hr.	12° *		Noxfish	Marking and Bills 1976
	0.0215	96 hr.				
chinook salmon	0.0490	24 hr.	12° *		Noxfish	Marking and Bill: 1976
	0.0369	96 hr.				
chinook salmon (fingerling)	0.0490	96 hr.	12° *		Noxfish	Olson and Marking 1976
coho salmon	0.0716	24 hr.	12° *		Noxfish	Marking and Bills 1976
	0.0620	96 hr.				
CATOSTOMIDAE (suckers)						
longnose sucker	0.0672	24 hr.	12° *		Noxfish	Marking and Bills 1976
	0.0570	96 hr.				
white sucker	0.0719	24 hr.	12° *		Noxfish	Marking and Bills 1976
	0.0680	96 hr.				
CYPRINIDAE (minnows)						
goldfish	0.175				Noxfish	
	0.218	72 hrs.	21°	pH 7.8-9.4	cube (7.3% rotenone)	Hester 1959a
	0.242			30-50 ppm CaCO ₃	Pro-Noxfish	

Table N Continued

Species	Dosage (ppm)	Exposure	Temp. C°	Water chemistry	Formulation	Reference
goldfish (cont'd.)	0.497	96 hr.	12° *		Noxfish	Marking and Bills 1976
carp	0.081	72 hr.	21°	pH 7.8-9.4 30-50 ppm CaCO ₃	Noxfish	Hester 1959a
	0.115				cubé (7.3% rotenone)	
	0.163				Pro-Noxfish	
fathead minnow	0.0840	24 hr.	12° *		Noxfish	Marking and Bills 1976
	0.0500	96 hr.				
	0.066	96 hr.	25° ---		2.5% rotenone, 5% cubé extractives, 2.5% sulfoxide	Cohen et al. 1960
golden shiner	0.400	24 hr.	12° *		Noxfish	Marking and Bills 1976
	0.142	96 hr.				
	0.159	72 hr.	21°	pH 7.8-9.4 30-50 ppm CaCO ₃	Noxfish	Hester 1959a
white amur	0.200	72 hr.	21°	30-50 ppm CaCO ₃	cubé (7.3% rotenone)	Hester 1959a
	0.191				Pro-Noxfish	
	0.470				Noxfish	
ESOCIDAE (pikes) northern pike	0.620	72 hr.	21°	pH 7.8-9.5 30-50 ppm CaCO ₃	cubé (7.3% rotenone)	Hester 1959a
	0.555				Pro-Noxfish	
	0.0630	96 hr.	17° ---		Noxfish	Marking 1972
CENTRARCHIDAE (sunfishes) green sunfish	0.0449	24 hr.	12° *		Noxfish	Marking and Bills 1976
	0.0330	96 hr.				
	0.165	72 hr.	21°	pH 7.8-9.4 30-50 ppm CaCO ₃	Noxfish	Hester 1959a
green sunfish	0.246	72 hr.	21°	30-50 ppm CaCO ₃	cubé (7.3% rotenone)	Hester 1959a
	0.238				Pro-Noxfish	
	0.218				Noxfish	
0.141	24 hr.	12° *		Noxfish	Marking and Bills 1976	
	96 hr.					

Table N Continued

Species	Dosage (ppm)	Exposure	Temp. C°	Water chemistry	Formulation	Reference
bluegill	0.026	24 hr.	24°	pH 7.1	4.85% rotenone	Bridges and Cope 1965
	0.023	48 hr.		35 mg/l alkalinity		
	0.023	96 hr.			Chem-Fish Regular	Howland 1969
	0.114	96 hr.	12°	*	Noxfish	
	0.179				cubé (7.3% rotenone)	Hester 1959a
	0.268	72 hr.	21°	pH 7.8-9.4	Pro-Noxfish	
largemouth bass	0.255			30-50 ppm CaCO ₃	Noxfish	Marking and Bills 1976
	0.149	24 hr.	12°	*	Noxfish	
	0.141	96 hr.			Noxfish	
	0.147				Noxfish	
	0.164	72 hr.	21°	pH 7.8-9.4	cubé (7.3% rotenone)	Hester 1959a
	0.081			30-50 ppm CaCO ₃	Pro-Noxfish	
smallmouth bass	0.200	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.142	96 hr.			Noxfish	
PERCIDAE (perches)	0.0932	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0790	96 hr.			Noxfish	
yellow perch	0.0920	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0700	96 hr.			Noxfish	
walleye	0.0165	24 hr.	12°	*	Noxfish	Marking and Bills 1976

* fish were tested under a range of water chemistries; see Lennon and Walker (1964) for laboratory procedures.

Markings and Bill cited four reasons for the apparent discrepancy between recommended field dosages and dosages known to be lethal in the lab:

- 1) Laboratory results (LC50's) indicate dosages that kill 50% of the fish, whereas the ideal field dosage is one that kills 100% of the target fish.
- 2) Organisms, particulate matter, and sunlight in natural waters tend to detoxify rotenone faster than in laboratory aquaria.
- 3) Uniform concentrations are far more difficult to achieve in the field, so that higher dosages are needed.
- 4) Individual fish of a species may be exceptionally resistant, so that a higher dosage is needed.

Markings and Bill (1976) concluded, along with Burress (1975), that field concentrations should be based on the results of on-site toxicity test rather than on laboratory or field data. Laboratory data can serve as guidelines in selecting field dosages (Gilderhus, 1972).

Susceptibility of Different Fish Species - Laboratory tests can also serve as indicators of the relative susceptibility of different fish species. Figures 23 and 24 display the results of the most thorough study on this subject (Markings and Bill, 1976). Of the twenty species tested under standardized conditions, goldfish (*Cyprinus carpio*) and black bullheads (*Ictalurus melas*) were the most resistant - 10 times as resistant as most other species.

These results are in general agreement with earlier, less detailed studies: Leonard (1939) stated that the least resistant species included the common shiner (*Notropis cornutus*), golden shiner (*Notemigonus crysoleucas*), bluegill (*Lepomis macrochirus*), pumpkinseed (*Lepomis gibbosus*), and brook stickleback (*Culaea inconstans*), while the mudminnow (*Umbra spp.*), and goldfish, (*Carassius auratus*), were the most resistant; Burdick et al. (1955) placed the following fish in order of their increasing resistance: brown trout (*Salmo trutta*), rock bass, (*Ambloplites rupestris*), creek chub (*Semotilus atromaculatus*), smallmouth bass (*Micropterus dolomieu*), common sucker (*Catostomus commersoni*), and brown bullhead (*Ictalurus nebulosus*). Jenkins (1956) ranked the following from least to most resistant: gizzard shad (*Dorsoma cepedianum*), carp (*Cyprinus carpio*), largemouth bass (*Micropterus salmoides*), redear sunfish (*Lepomis microlophus*), black crappie, (*Pomoxis nigromaculatus*), bluegill, white crappie (*Pomoxis annularis*), green sunfish (*Lepomis cyanellus*), warmouth (*Lepomis gulosus*) and black bullhead (*Ictalurus melas*).

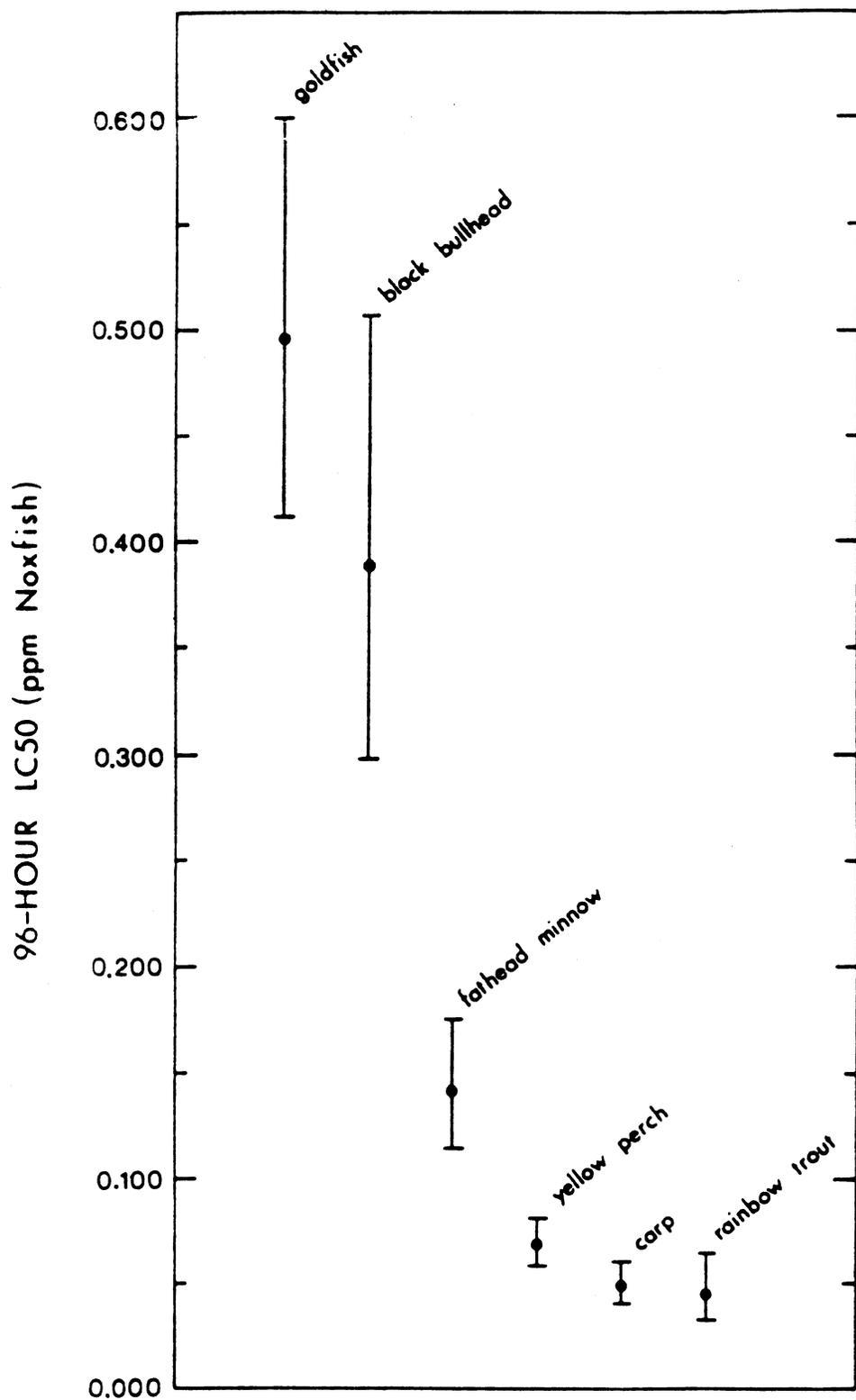


Figure 23 96-hour median lethal concentration (LC50) of Noxfish for several fish held under standardized laboratory conditions. I. Vertical bars represent 95% confidence intervals. Data from Marking and Bills 1976. See Figure 2 for additional data.

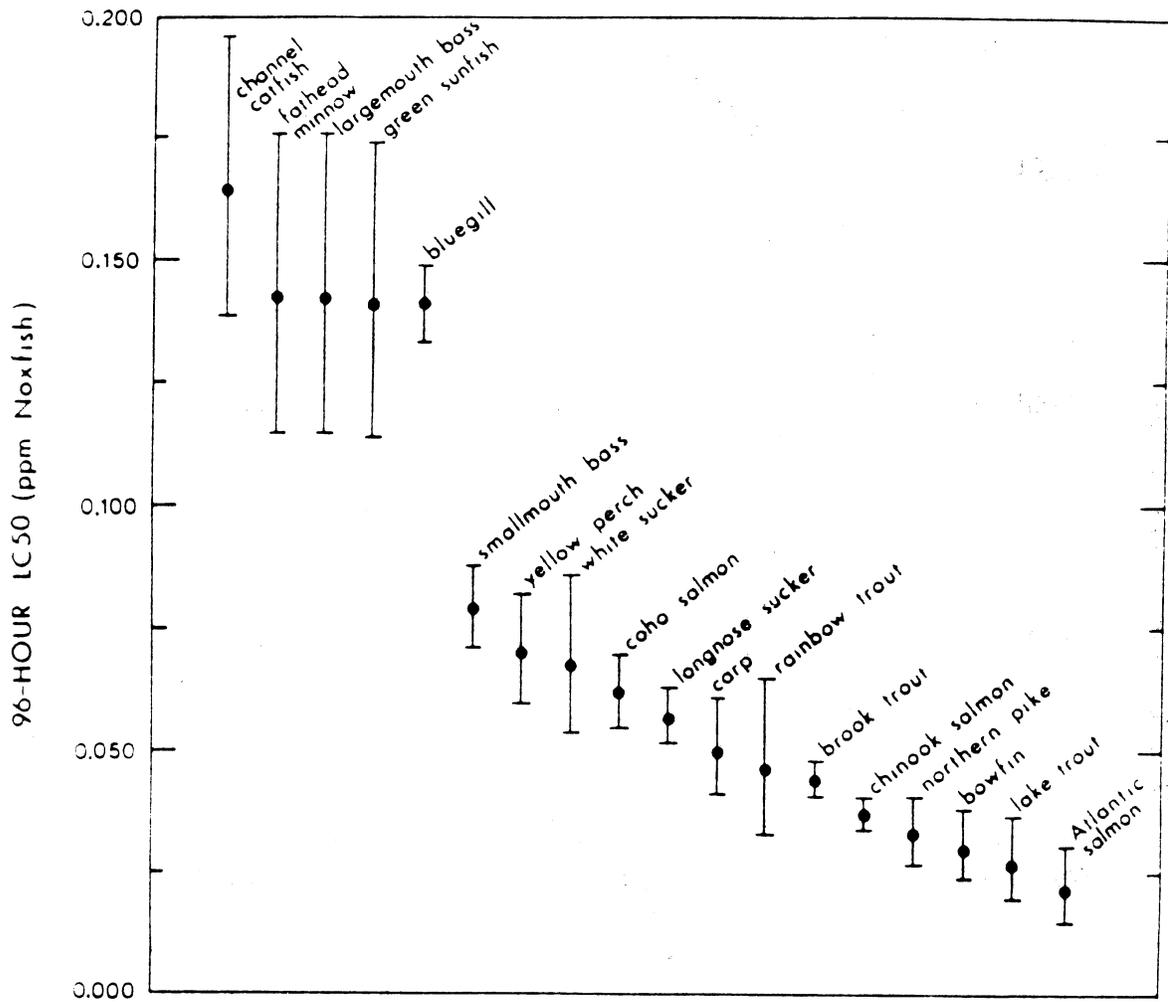


Figure 24 96-hour median lethal concentration (LC50) of Noxfish for several fish held under standardized laboratory conditions. II. Vertical bars represent 95% confidence intervals. Data from Marking and Bills 1976. See Figure 1 for goldfish and black bullhead LC50's.

Effect on Fish Eggs - Table O displays the results of laboratory toxicity tests on fish eggs. All researchers working with salmon eggs found that they were more resistant to rotenone than fry or fingerlings of the same species. Olson and Marking (1975) compared fingerling brook trout, lake trout (*Salvelinus namaycush*), and chinook salmon (*Oncorhynchus tshawytscha*) with eggs of those species; they concluded that the eggs were more resistant. Markings and Bill (1976) found that newly fertilized eggs of rainbow trout were 47 to 106 times more resistant than rainbow fingerlings to Noxfish. The actual degree depended on water hardness. Garrison (1968) reported that salmon eggs were 10 times as resistant to Pro-Noxfish than were salmon fry. He suggested that salmonid embryos would survive a fish-killing dose of rotenone. Leonard (1939) found that eyed brown trout eggs survived a 0.5 ppm dosage of derris powder, but that the fry died as soon as they broke the shell.

Leonard (1939) and Clemens and Martin (1953) reported that problem species have repopulated in lakes where they have been completely poisoned out, and where no illegal stocking or invasion from nearby waters occurred. They suggested that resistant eggs which hatched after detoxification could have been the reason. Some support for this hypothesis comes from Markings et al. (1983), who found that eyed carp eggs were about 50 times as resistant to rotenone as were carp larvae based on LC50 values. Rainbow smelt (*Osmerus mordax*) eggs were about 10 times as resistant as the larval form. Hester (1959b), reported that the LC50's of both carp eggs and fathead minnow eggs were very similar to those obtained with fingerlings of the same species. Either his results were in error, or carp and fathead minnow eggs behave differently than salmonid eggs when exposed to rotenone.

Effect on Non-Target Native Fish - Fish native to Washington state waters are seldom the target of rotenone treatments. It is reasonable to assume that native non-game fish (such as sculpins, suckers, dace, chubs, squawfish and shiners), as well as residual stocked trout, are killed along with target species in a rotenoned lake. Of the nonsalmonid fish native to Washington, only suckers have been tested for their tolerance to rotenone; Figure 24 shows that they succumb to smaller dosages of rotenone than most target species (e.g., perch, sunfish, catfish).

Zilliox and Pfeiffer (1960) reported that native fish in Adirondack Lakes - white suckers, brown bullheads, whitefish (*Coregonus spp.*), and several minnows - were temporarily eliminated along with the non-native target species, usually yellow perch (*Perca flacescens*).

Effectiveness of Treatment - In the past the most common way to judge the effectiveness of a rotenone treatment was on the basis of a "complete kill" of all target fish. Several more or less practical definitions of a "complete kill" have been offered (Clemens and Martin, 1953; Lennon et al., 1970; Zilliox and

Table O Toxicity of rotenone formulations to fish eggs. All dosages are expressed as median lethal concentrations (LC50).

Species	Dosage (ppm)	Exposure	Temp C°	Water Chemistry	Formulation	Reference
rainbow trout (newly fertilized eggs)	5.60	96 hr.	12°	very soft*	Noxfish	Marking and Bills 1976
	4.42			soft*		
	3.20			hard*		
	2.50			very hard*		
chinook salmon (green eggs)	> 3.00	96 hr.	12°	**	Noxfish	Olson and Marking 1975
brook trout (green eggs)	3.40	96 hr.	12°	**	Noxfish	Olson and Marking 1975
lake trout (green eggs)	> 1.00	96 hr.	12°	**	Noxfish	Olson and Marking 1975
carp (newly fertilized eggs)	0.091	192-	24°	---	Noxfish	Hester 1959b
	0.178	216 hr.			Pro-Noxfish	
carp (eyed eggs)	0.025	96 hr.	12°	soft*	Noxfish	Marking et al. 1983
rainbow smelt (eyed eggs)	0.015	96 hr.	12°	soft*	Noxfish	Marking et al. 1983
fathead minnow (newly fertilized eggs)	0.142	216-	21°- 24°	---	Noxfish	Hester 1959b
	0.233	247 hr.			Pro-Noxfish	

* hardness expressed as mg/l CaCO₃; very soft, 10-12; soft, 40-44; hard, 160-180; very hard, 290-310.

** eggs were tested under a range of water chemistries; see Lennon and Walker (1964) for laboratory procedures.

Table O Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to birds.

Animal	LD50	Formulation	Reference
White rock chickens	6 ml/kg	Chem-Fish Regular Noxfish	Brooks 1961
	8 ml/kg	Chem-Fish Special Pro-Noxfish	
Chickens (4-week)	>270 mg/kg	pure rotenone	Cutkomp 1943b
Chickens (5-day)	996 mg/kg	pure rotenone	
Chickens (5-day)	247 mg/kg	derris extract (25% rotenone)	
Eastern chipping sparrow (nestling)	113 mg/kg	pure rotenone	
Eastern song sparrows (nestlings)	130 mg/kg	pure rotenone	
Eastern robins (nestlings)	195 mg/kg	pure rotenone	Cutkomp 1943a
English sparrows (nestlings)	199 mg/kg	pure rotenone	
English sparrows (adults)	853 mg/kg	pure rotenone	
pheasants (5-day)	850 mg/kg	pure rotenone	
pheasants (4-week)	1190 mg/kg	pure rotenone	
pheasants (3-4 month)	>1414 mg/kg	pure rotenone	Tucker & Crabtree 1970
prairie horned larks (adult)	450-500 mg/kg	pure rotenone	Cutkomp 1943a
mallards (3-4 month)	>2000 mg/kg	pure rotenone	Tucker & Crabtree 1970

Table O Toxicity of rotenone to amphibians in laboratory bioassays.

Animal	Concentration (ppm)	Exposure	Formulation	Water Chemistry	Comments	Reference
Southern leopard frog larvae (<u>Rana sphenocéphala</u>)	0.5	96 hr.	Noxfish	16° C; see Lennon & Walker (1964) for test conditions	LC50	Chandler & Marking 1982
Leopard frog (<u>Rana pipiens</u>)	7.3	24 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness		
	7.9	24 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness	LC50	Farringer 1972
	4.6	96 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness		
	3.2	96 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
Leopard frog tadpoles (<u>Rana pipiens</u>)	0.1	8-24 hr.	5% rotenone	---	100% mortality	
tiger salamander, with gills (<u>Ambystoma tigrinum</u>)	0.017	8-24 hr.	5% rotenone	---	toxic but not necessarily fatal	Hamilton 1941
tiger salamander, metamorphosed (<u>Ambystoma tigrinum</u>)	0.1	8-24 hr.	5% rotenone	---	100% mortality	
frogs	4.0 mg/kg body weight	---	pure rotenone	---	oral LD50	Haag 1931

Table O Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to animals.

Animal	LD50	Formulation	Reference
Rabbits	1.7 ml/kg	Chem-Fish Special Pro-Noxfish	Brooks 1961
White mice	350 mg/kg	pure rotenone	Kenaga and Allison 1971
Rats	1.5±0.1 ml/kg 170 mg/kg	Pro-Noxfish cubé (4.7% rotenone) in aqueous solution	Brooks 1961 Haag & Taliaferro 1940
	132 mg/kg 1500 mg/kg 1.5 cc/kg	pure crystalline rotenone derris Chem-Fish Special	Lehman 1951 Lehman 1951 Blue Spruce Co. 1973
Guinea pigs	60 mg/kg 55-60 mg/kg	pure rotenone pure rotenone	Cohen et al. 1960 Cutkomp 1943b

Pfeiffer, 1956). The last authors gave the following commonly-cited criteria for a complete kill: "Failure of observation, angling and netting for two successive years following reclamation to indicate any species of fish present in a reclaimed pond, except stocked trout, would appear to be a reasonable indication of a complete kill". The authors later excluded native species from this definition, since they frequently reappear within two years even in "successfully" treated lakes (Zilliox and Pfeiffer, 1960).

Judged by the criteria of Zilliox and Pfeiffer (1956, 1960), WDW Biologist Bob Pfeifer stated that it was unlikely that complete kills were achieved in recent years in a number of Seattle-area lakes (Pfeifer, 1985).

Clemens and Martin (1953) pointed out that the only way to be entirely sure of a complete kill is to drain the lake or pond. This has been done on occasion: Cumming et al. (1975) drained a 0.1 acre Arkansas pond following a 2 ppm Noxfish application and found that a complete kill of channel catfish and grass carp had indeed occurred. But Clemens and Martin (1953) drained two ponds after rotenone treatment and found fish in both; one pond had been judged a "complete kill" before draining. On six other ponds which Clemens and Martin had initially termed "complete kills", intensive seining revealed some target fish still present in at least five of them.

In larger lakes, the possibility of ever exterminating 100% of the target fish with rotenone is small, and is probably an unrealistic goal (Klingbeil, 1975). Klingbeil notes that massive efforts to kill the last 0.1% of a target population in Wisconsin are usually followed immediately by illegal stocking of the same or different problem species.

Klingbeil (1975) and Zilliox and Pfeiffer (1960) have disregarded the concept of a "complete kill" altogether, offering another criterion by which to judge the effectiveness of a lake poisoning: the return of quality fishing. This would seem to be far more viable measure for two reasons:

- 1) the ultimate purpose of most treatments is to produce better fishing, not necessarily to eliminate X number of target fish (Prevost, 1960); and
- 2) quantifying "better fishing" (in terms of catch-per-unit-effort, CPUE, fingerling growth and survival, etc.) is far more practical than determining a "complete kill". These data are already collected on a yearly basis on virtually all Washington state "trout-only" lakes. Cost-benefit analyses can also be readily applied to these lakes.

Biologists have long been interested in what proportion of the fish poisoned in a lake eventually come to the surface, mostly out of a desire to make population estimates more reliable, (Brown and Ball, 1943b; Carlander and Lewis, 1948; Krumholz, 1950b; Lambou and Stern, 1957). It has been suggested that the decay of unrecovered fish that did not surface, might produce nuisance algae blooms in some lakes (Funk and Moore, 1984). There are five main factors that influence the surfacing of dead fish in rotenoned lakes:

- 1) Water temperature . Parker (1970) made both laboratory and field tests and found that in warm water, dead fish surfaced much more quickly than in cold water. Bartoo (1977) and Krumholz (1950b) also cited water temperature as a major factor in surfacing rates of rotenone poisoned fish.
- 2) Water depth . Parker (1970) found that deep water slowed the surfacing of dead warmwater fish.
- 3) Fish species . Parker (1970) reported that dead bullheads surface more slowly than centrachids (sunfish) and dead minnows faster than either. The data of Kempinger and Christenson (1978) indicate that a greater portion of dead walleye come to the surface compared to other warmwater species.
- 4) Fish size . Smaller (younger) fish surface at a much slower rate than larger fish of the same species (Parker, 1970; Brown and Ball, 1943b; Kempinger and Christenson, 1978).
- 5) Presence of aquatic rooted plants . When fish have access to extensive beds of underwater vegetation, they often become tangled and fail to surface after they die (Parker, 1970, Ball, 1945; Zook, 1978).

Parker (1970) found that the following factors, within the limits indicated, did not affect surfacing rate: dissolved oxygen (3.8-13.8 ppm); total alkalinity (40.0-140.0 ppm as CaCO₃); pH (7.7-8.5), total hardness (110.00222.3 ppm), transparency (clear - 8 inches), and rotenone dosage (0.5-6.3 ppm 5% dust).

Table P displays the data from rotenoned lakes and ponds in which mark-recapture experiments were made using various fish. In every case except Ford Lake (Ball, 1945), the authors were certain of a complete kill. Also, all authors felt that tagging mortality was insignificant and did not bias the results.

Considering the importance of water temperature in the surfacing of dead fish, it is unfortunate that no temperature data exist for some of the test waters. Figure 25 shows the percentage of fish surfacing within 24 hours of rotenone treatment on lakes and ponds where temperature data were available. Although data from studies involving different warmwater fish species, fish sizes, and lake

Table P Percentage of dead fish surfacing following rotenone treatment in mark-recapture experiments.

Test water, location	Species	Water Temp. (°F)	Depth Range (Ft.)	Time to Surface	% of Dead Fish That Surfaced	Reference
North Lake, Western Washington	Yellow perch	50°	1-35	24 hours	15.5%	Bartoo 1977
				96 hours	17.5%	
Laboratory & 4 ponds, Ohio	Bluegills (& other centrarchids)	80° 72° 63° 59° 50° 40°	1-15	24 hours	95-100%	Parker 1970
				48 hours	" "	
				72 hours	" "	
				120 hours	" "	
				32 days	" "	
30 days	" "					
-----	Largemouth Bass	68°	---	60 hours 48 hours 24 hours	96% 91% 62%	Krumholz 1950a
Shoofly Lake, eastern Washington	Largemouth bass	65°	1-12	24 hours	46.6%	Zook 1978
Farm pond, Iowa	Bluegill	---	----	120 hours	38%	Carlander and Lewis 1948
	White crappie				14%	
	Largemouth bass				33%	
	Black bullhead				80%	
	Golden shiner				91%	
Nebish Lake, Wisconsin	Walleye	56°	1-45	24 hours	44.0%	Kempinger and Christenson 1978
	Smallmouth bass				16.6%	
	Northern pike				17.0%	
	Yellow perch				4.2%	
	Rock bass				22.4%	
	Bluegill				24.1%	
	Pumpkinseed				23.0%	
	Green sunfish				7.0%	
	Largemouth bass				25.0%	
	Mean (all species)				20.4%	
Ford Lake, Michigan	Bluegill Brook trout	---	1-33	144 hours	59% 45%	Ball 1945

Test water, location	Species	Water Temp. (°F)	Depth Range (Ft.)	Time to Surface	% of Dead Fish That Surfaced	Reference
Farm pond, Indiana	Green sunfish	---	1-11	24 hours	70.1%	Krumholz 1950b
				48 hours	85.4%	
				72 hours	88.0%	
				120 hours	90.4%	
				168 hours	90.8%	
				192 hours	91%	
Farm pond, Indiana	Largemouth bass	---	1-11	48 hours	87.1%	Krumholz 1950b
Barkley Lake, Kentucky	Mostly sunfish, bluegill, largemouth bass	---	---	72 hours	89%	Axon et al. 1979

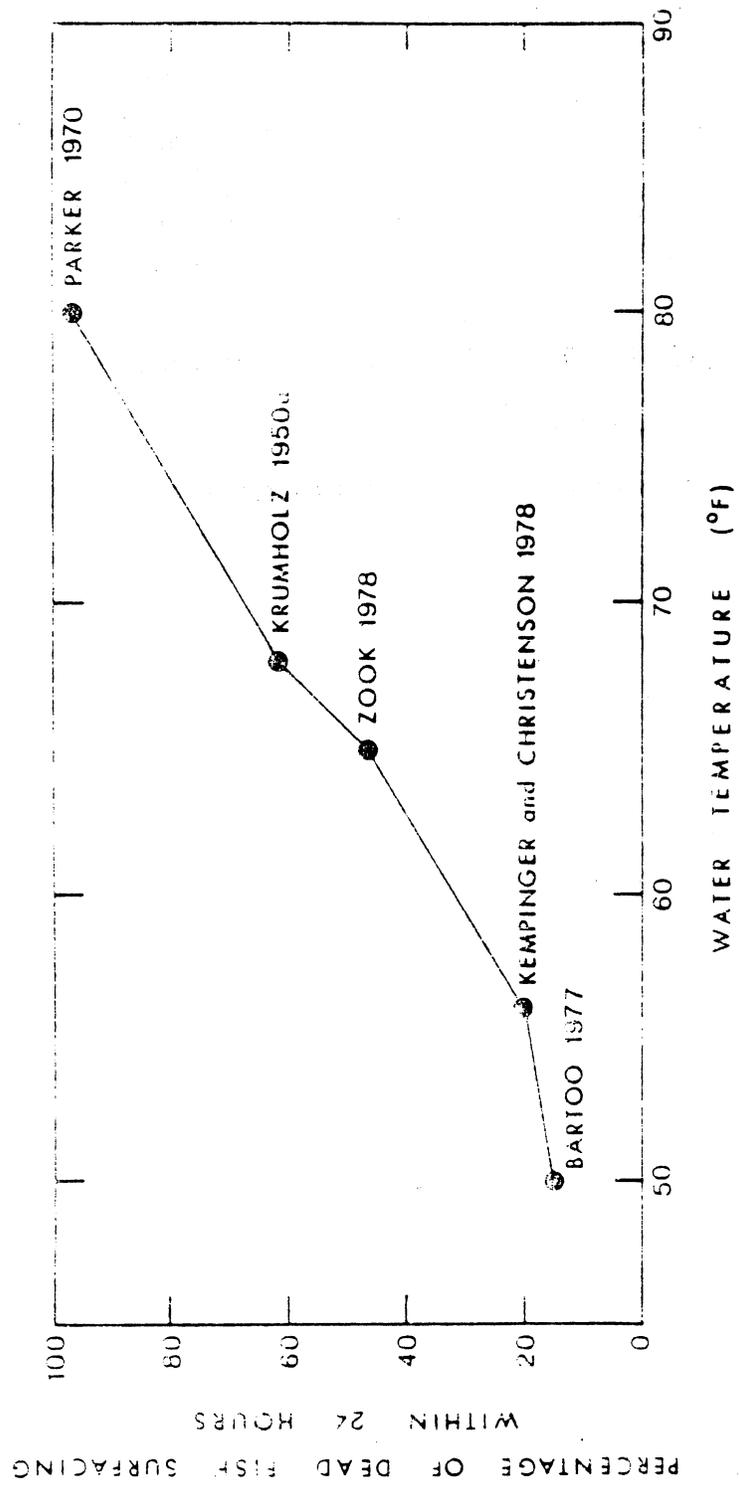


Figure 2 Relationship between water temperature and the percentage of dead fish that surface following rotenone treatment. Data from Table 12.

characteristics have been grouped together, it is still clear that in cold water, a much smaller percentage of dead fish surface than in warm water. Bartoo (1977) found that after the initial 24 hours, few fish surfaced in the relatively cold waters of North Lake, Washington; in the following three days, they were able to recover an additional 2%.

The relationship between fish size and the percentage of dead fish that float to the surface is well demonstrated by the data from Nebish Lake graphed in Figure 26. Regardless of species, the smaller fish showed a tendency to remain on the bottom.

Some investigators have stated that almost all dead fish can eventually be recovered from a lake, since even fish on the bottom will bloat over time and rise to the surface. Hoffman and Payette (1956) report this occurring eight days after rotenone treatment of a San Diego reservoir. This second harvest of bloated fish was actually much greater than the initial collection of dead fish made within five days of poisoning. Brown and Ball (1943a) had SCUBA divers observe individual dead fish lying on the bottom of Third Sister Lake, Michigan; a week after poisoning, these fish were still on the bottom and decaying.

On Washington lakes, the surface water temperatures at the time of treatment in the fall range from 44°-80° F, averaging 57°-58° F. Based on this mean and Figure 25, we would expect that only about 30% of the dead fish could be recovered. The bulk of the dead fish would never surface, eventually decaying in the lake.

Long Term Effects - Effect on Non-Target Native Fish - No quantitative studies have been made of the long term effects of rotenone poisoning on native fish. Zilliox and Pfeiffer (1960) reported on 12 Adirondack lakes which were rotenoned to eliminate yellow perch, an introduced species: in 1954, all these lakes were judged "complete kills", yet within five years, at least half were repopulated with brown bullheads, white suckers, and several minnow species, all native fish. The author's data indicated that the native species had survived poisoning, rather than merely being reintroduced.

It is reasonable to assume that native, nontarget populations eventually recover in the same way that target fish do: some fish survive either due to individual tolerance (Meyer, 1966; Tompkins, 1953) or, more likely, because a truly "complete kill" has not occurred. Quantitative data on recovery are lacking in the literature.

Complicating the situation is the fact that rotenone target species such as goldfish have a disastrous impact not only on trout, but on other native fish populations as well (Wydoski and Whitney, 1979; Gothschalk, 1966). The question of whether or not rotenone

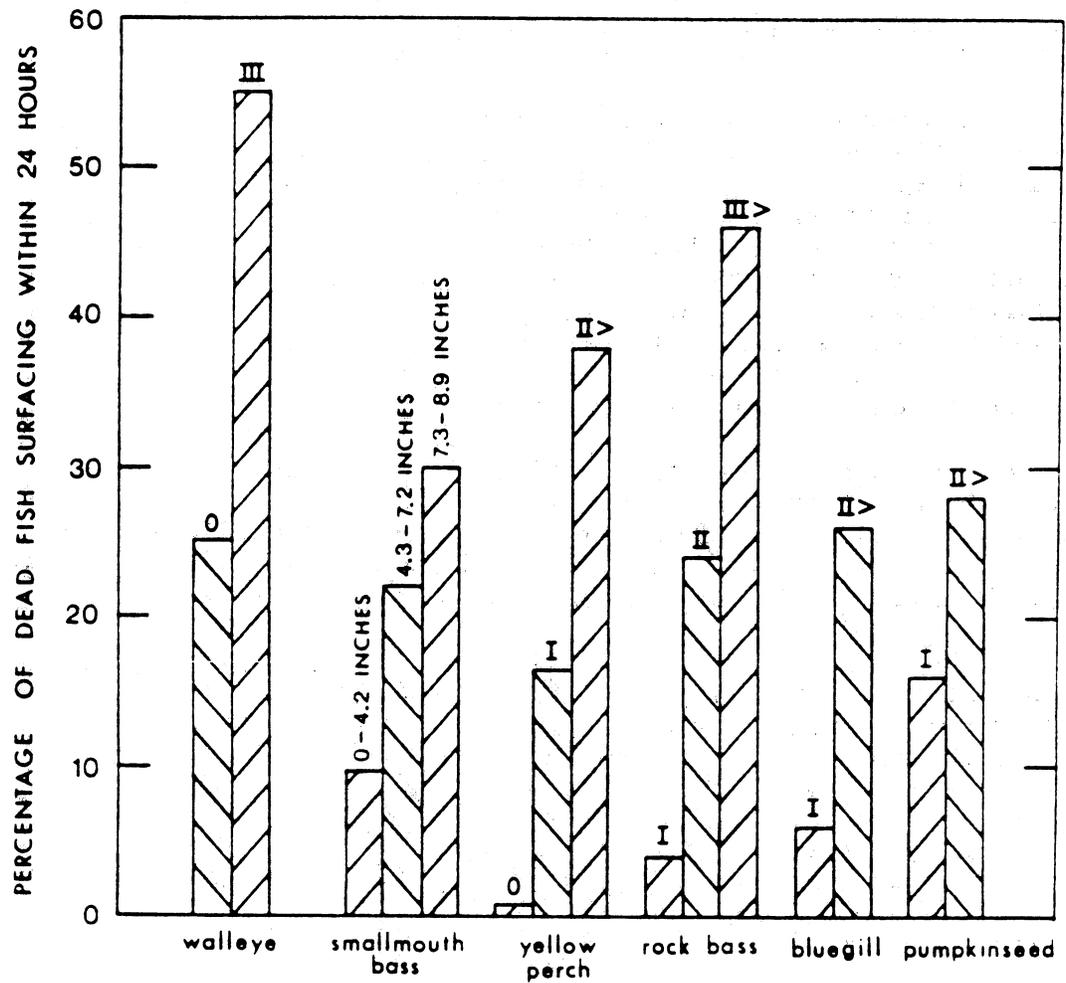


Figure 26 Relationship between fish size (age) and surfacing rate for various species in Nebish Lake, Wisconsin. Roman numerals indicate age group of fish. Surface temperature was 56° F at the time of poisoning. Source: Kempinger and Christenson 1976.

benefits native nongame fish in the long run (by eliminating competitive exotic species) is open to speculation.

Effect on Growth and Survival of Planted Trout - Trout are only restocked in a rotenoned lake after bioassays indicate that the water is completely detoxified, so that rotenone has no direct toxic effect on growth or survival. The indirect, long-term effects of rotenone treatment are increased growth and survival of fingerling trout; this occurs because predators and/or competitors are eliminated, and is the goal of most treatments in Washington state and nationwide (Lennon et al., 1970).

Some authors have cautioned fishery managers not to restock trout while zooplankton and/or benthic populations are still reduced following rotenone (Bennett, 1985; Kiser et al., 1963). Kiser and his colleagues stated that Fern Lake, Washington was stocked by WDW immediately after the lake detoxified about five weeks before zooplankton populations had recovered to prerotenone levels. They noted that successful fingerling stocking depends on an abundance of natural food, and that fish may have been stocked too soon following the spring treatment. There was no followup research on survival or growth.

Almost 80% of Washington state treatments occur in the fall, and trout are restocked the following spring. This far exceeds the time generally required for zooplankton and benthic animals to recover to prerotenone levels. In the case of spring treatments, there are two options: either a prestocking zooplankton sample, or a post-stocking measurement of fingerling growth. The later empirical approach seems more practical and more reliable.

Rotenone Tolerance in Fish - Repeated use of pesticides on crops has led to the well-documented phenomenon of resistant insects that become harder to control. Vertebrates such as fish usually breed too slowly for such resistant populations to develop (Fabacher and Chambers, 1972), but they do occur: Vinson et al. (1963) and Culley and Ferguson (1969) found mosquitofish (*Gambusia affinis*) that had apparently acquired a tolerance to DDT and a wide variety of other pesticides in a heavily-sprayed agricultural area in Mississippi. Hubbs (1963) was the first to theorize that undesirable fish might become tolerant to rotenone, requiring ever more frequent poisonings. Two instances have been reported where fish apparently acquired a tolerance to rotenone through exposure to rotenone or other pesticides: Fabacher and Chambers (1972) found that the insecticide-resistant mosquitofish from Mississippi showed a 1.8-fold tolerance to rotenone over mosquitofish from pesticide free waters. While rotenone was not one of the insecticides used in the area, Fabacher and Chambers demonstrated that heavy, repeated spraying of other organo-chlorine insecticides in the area produced a "cross-tolerance" in these fish.

Orciari (1979) demonstrated an acquired tolerance due to repeated use of rotenone itself: Ball Pond, Connecticut was treated with 1.0 ppm synergized rotenone six times during a 17 year period to rid the 90 acre lake of golden shiner (*Notemigonus crysoleucas*). They always reappeared, and tolerance to the poison was suspected as at least part of the problem. Paired bioassays showed that the Ball Pond shiners were 4.0-7.1 times more tolerant than golden shiners from six ponds that had never been rotenoned.

Klingbeil (1975) discussed a related problem following rotenone treatment: any fish which remain in a lake that is not a "complete kill" may take advantage of the sudden reduction in competition for food and space, rapidly filling the ecological void left by the poisoned fish. The original survivors may be individuals that have a natural tolerance to rotenone (Tompkins, 1953; Meyer, 1966; Marking and Bill, 1976), individuals that have an acquired tolerance (Orciari, 1979), fish of an especially rotenone-resistant species, or simply individuals that found refuge from the poison in thick weeds, springs, etc. (Kiser et al., 1963; Prevost, 1960). Whatever the reason for their survival, there is the possibility that these fish may not only repopulate to former levels, but become an even greater nuisance than before poisoning. While this scenario differs in the strict sense from true acquired tolerance, the net result from a practical standpoint would be the same: more frequent and possibly higher-dosage treatments would be required to maintain a fishery.

Hubbs (1963) hypothesized that such a situation could occur in Texas waters, especially with partial rotenone treatments. Scholz (1983) believed that goldfish populations in eastern Washington lakes were increasing because rotenone treatments allowed surviving goldfish to expand into newly-vacant ecological niches. Whether or not this actually occurred is problematic, since there are no reliable fish population estimates for these lakes, and since the dates of first introduction are unknown. Even if such data were available, continued illegal stocking would tend to confuse any analysis unless there were some way to separate the descendants of freshly stocked fish from those of actual rotenone survivors.

Nuisance-fish increases have been documented in at least two lakes where rotenone treatments were unsuccessful: Jenkins (1956) reported that the carp population in Ardmore City Lake, Oklahoma exploded after a partial treatment. The goldfish population in California's Big Bear Lake likewise exploded following two unsuccessful treatments (Johnson, 1966; Hoover, CDF&G, pers. comm.). Klingbeil (1975) felt that the same thing might happen in Wisconsin lakes, and recommended restocking with gamefish as quickly as possible after poisoning to avoid such a takeover. He also suggested predator stocking.

On a statewide basis empirical evidence from Washington's 50-year history of fish poison use suggest that the above scenarios are not yet a problem: in the lakes that have been poisoned most frequently, the time between treatments has not decreased over the years.

Amphibians and Reptiles

Table Q lists toxicity data for amphibians. No laboratory data are available for reptiles. These tests suggest that larval amphibians such as tadpoles are far more susceptible to rotenone than metamorphosed adults. This stands to reason when we consider rotenone's high toxicity to gill-breathing forms.

The young of many amphibian species have completely metamorphosed and lost their gills by fall, when most rotenone treatment occurs. Others metamorphose during the fall, so that at least some individuals could be affected by rotenone treatment. In Washington, this category includes the spotted frog (*Rana pretiosa*), the red-legged frog (*Rana aurora*), the Northern leopard frog (*Rana pipiens*), the long-toed salamander (*Ambystoma macrodactylum*), and the roughskin newt (*Taricha granulosa*). Still others overwinter with gills: the Pacific giant salamander (*Dicamptodon ensatus*), the Cascades frog (*Rana cascadae*), and the bullfrog (*Rana catesbeiana*). The tiger salamander (*Ambystoma tigrinum*) never loses its gills, while the Northwestern salamander (*Ambystoma gracile*) is variable: some metamorphose in the fall, some overwinter with gills, and some retain gills for their entire life (Weschler, WDW, pers. comm). Larvae and gill-breathing adults of the above species could potentially suffer from routine fall rotenone treatments. Spring treatments could affect all species, since young amphibians are always in the gilled stage during that time of year.

Laboratory tests indicate that gill breathing amphibians have a relatively high tolerance to rotenone. Chandler and Marking (1982) reported that larval leopard frogs were 3-10 times more tolerant of rotenone than most of the 21 fish species tested by Markings and Bill (1976), and had about the same tolerance as the hardy goldfish. They noted that these animals were more sensitive to rotenone in the lab than in the natural environment, and concluded that they would probably be safe during lake treatments.

Denis and Devlin (1968) found that rotenone inhibited cell respiration and development in amphibian eggs. Lamy and Melton (1972) noted that rotenone produced unusual cleavage in leopard frog embryos. The laboratory procedures used in both these studies make extrapolation to the lake environment impossible. Again, however, frog and salamander eggs are not present in the fall when most rotenone treatments occur.

Table Q Toxicity of rotenone to amphibians in laboratory bioassays.

Animal	Concentration (ppm)	Exposure	Formulation	Water Chemistry	Comments	Reference
Southern leopard frog larvae (<i>Rana sphenocéphala</i>)	0.5	96 hr.	Noxfish	16° C; see Lennon & Walker (1964) for test conditions	LC50	Chandler & Marckling 1982
Leopard frog (<i>Rana pipiens</i>)	7.3	24 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness		
	7.9	24 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness	LC50	Farringer 1972
	4.6	96 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness		
	3.2	96 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
Leopard frog tadpoles (<i>Rana pipiens</i>)	0.1	8-24 hr.	5% rotenone	---	100% mortality	
tiger salamander, with gills (<i>Ambystoma tigrinum</i>)	0.017	8-24 hr.	5% rotenone	---	toxic but not necessarily fatal	Hamilton 1941
tiger salamander, metamorphosed (<i>Ambystoma tigrinum</i>)	0.1	8-24 hr.	5% rotenone	---	100% mortality	
frogs	4.0 mg/kg body weight	---	pure rotenone	---	oral LD50	Haag 1931

Actual field data involving amphibians and reptiles are scarce and qualitative. When Brown and Ball (1943a) applied 0.5% ppm rotenone dust to a Michigan lake in early May, tadpoles were "greatly affected". Three months later, however, tadpoles were "extremely numerous", and the authors attribute it to post-rotenone breeding and the lack of predation by fish. High concentrations (~10 ppm) of Noxfish applied to ponds in Florida made alligators visibly ill, forcing them to leave the water (Fletcher, WDW pers. comm.).

In other field applications, Meehean (1942) noted that numerous salamanders (*Pseudobranchius striatus*) were killed by 0.5 ppm derris in five Florida lakes. The same author reported that 1.0 ppm derris killed the soft-shelled turtle (*Amyda ferox*).

Both adult and larval amphibians, as well as reptiles, may be indirectly affected by rotenone treatment. Most of Washington state's riparian herpetiles include fish and/or aquatic insects in their diets (Hodge, 1983; Stebbins, 1966), though none depend exclusively on these items. Aquatic insect reduction due to rotenone is rarely more than 71% in studied waters, and full recovery usually occurs within a month or two. Alternative food sources can probably support these animals during post-rotenone shortage of fish and benthos (State of California, 1983).

Birds

Oral toxicity for birds is listed in Table R.

The chipping sparrow is the most susceptible of the birds tested, with an LC50 of 113 mg pure rotenone per kg body weight. A six ounce chipping sparrow would require 19.2 mg pure rotenone, or 384 mg of the 5% fish-killing dust for a lethal dose. Similar calculations based on Brooks' (1961) work show that the lethal dose for a 6 ounce white rock chicken would be 1.02 ml Noxfish.

There would be no direct toxic effect of rotenone on birds and although no chronic, long-term toxicity studies have been performed on birds, the quick breakdown of rotenone and infrequent treatment of lakes and streams would decrease the likelihood of such effects.

As with mammals, only those birds which depend on fish or benthos for food such as: bald eagles (*Haliaeetus leucocephalus*), ospreys (*Pandion haliaetus*), loons (*Gavia spp*), kingfishers (*Megaceryle alcyon*), rails, grebes, and diving ducks - notably mergansers, buffleheads (*Bucephala albeola*), and goldeneyes (*Bucephala spp*) - could be affected indirectly by rotenone treatment of a lake. Except for the kingfisher, all these birds normally forage as adults over many miles and would probably not be harmed by the temporary loss in fish or benthic food following rotenone (Leschner, WDW, pers. comm.; State of California, 1983).

Table R Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to birds.

Animal	LD50	Formulation	Reference
White rock chickens	6 ml/kg	Chem-Fish Regular	Brooks 1961
	8 ml/kg	Chem-Fish Special Pro-Moxfish	
Chickens (4-week)	>270 mg/kg	pure rotenone	Cutkomp 1943b
Chickens (5-day)	996 mg/kg	pure rotenone	
Chickens (5-day)	247 mg/kg	derris extract (25% rotenone)	
Eastern chipping sparrow (nestling)	113 mg/kg	pure rotenone	
Eastern song sparrows (nestlings)	130 mg/kg	pure rotenone	
Eastern robins (nestlings)	195 mg/kg	pure rotenone	Cutkomp 1943a
English sparrows (nestlings)	199 mg/kg	pure rotenone	
English sparrows (adults)	853 mg/kg	pure rotenone	
pheasants (5-day)	850 mg/kg	pure rotenone	
pheasants (4-week)	1190 mg/kg	pure rotenone	
pheasants (3-4 month)	>1414 mg/kg	pure rotenone	Tucker & Crabtree 1970
prairie horned larks (adult)	450-500 mg/kg	pure rotenone	Cutkomp 1943a
mallards (3-4 month)	>2000 mg/kg	pure rotenone	Tucker & Crabtree 1970

Ospreys leave the Pacific Northwest beginning in September, returning in April, and thus would not be present during most treatments.

Kingfishers are highly territorial, so that the temporary disappearance of fish could force them off a lake and into competition with birds on other waters (Weschler, WDW, pers. comm.). Ducklings on a spring-rotenoned lake would be unable to forage on other waters, and may suffer reduced growth as an indirect result of rotenone treatment.

Mammals

Data on the acute toxicity of orally administered rotenone to mammals are listed in Table S. Only oral LD50's using aqueous solutions are shown, since these mirror the "real-life" situation. Schnick (1974) also conducted studies involving IP, IV, and IM injections of rotenone, as well as oral doses using unusual solvents.

The lowest LD50 of pure rotenone found in the literature on mammals is 55 mg/kg body weight for guinea pigs (Cutkomp, 1943b). To kill a small mammal weighing approximately half a pound would therefore require 12.5 mg pure rotenone, or 250 mg of the commonly used 5% dust. The smallest mammalian LD50 of rotenone formulation found in the literature is 170 mg/kg body weight of cube' powder (4.7% rotenone) reported by Haag and Taliaferro (1940) using male rats.

To produce subacute effects such as weight loss or liver damage also requires very high dosages fed continuously in the diet for many months. Rotenone is not likely to have a direct toxic effect on mammals in either the short or the long run. The reasons for the high mammalian tolerance to rotenone were discussed in the section describing the History of Rotenone. The EPA (1981) considers it safe to water livestock with rotenone-treated water.

Indirect effects might occur when rotenone disrupts the food supply for small mammals that feed on fish or benthos. In Washington this category includes mink (*Mustela vison*), river otter (*Lutra canadensis*), and water shrew (*Sorex palustris*).

Mink feed primarily on small mammals, with fish a secondary food source (Banfield, 1974). Additionally, they move frequently, all dens being temporary (Whitaker, 1980). River otters rely almost entirely on fish for food, and the temporary loss of prey following rotenone treatment may disturb them. But otters forage widely, sometimes travelling 50-60 miles during a year (Banfield, 1974), and would may not be displaced permanently. Water shrews may be indirectly affected by the temporary reduction in benthos (Weschler, WDW, pers. comm.).

Table S Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to animals.

Animal	LD50	Formulation	Reference
Rabbits	1.7 ml/kg	Chem-Fish Special Pro-Noxfish	Brooks 1961
White mice	350 mg/kg	pure rotenone	Kenaga and Allison 1971
Rats	1.5±0.1 ml/kg 170 mg/kg 132 mg/kg 1500 mg/kg 1.5 cc/kg	Pro-Noxfish cubé (4.7% rotenone) in aqueous solution pure crystalline rotenone derris Chem-Fish Special	Brooks 1961 Haag & Taliaferru 1940 Lehman 1951 Lehman 1951 Blue Spruce Co. 1973
Guinea pigs	60 mg/kg 55-60 mg/kg	pure rotenone pure rotenone	Cohen et al. 1960 Cutkomp 1943b

Human Health

Paths of Human Exposure to Rotenone - Figure 27 shows the uses of rotenone and how people may be exposed to it.

Direct contact with the dust used in fish control is a hazard faced mostly by fish biologists or other persons directly involved in the application. The ways in which the public could be exposed to the rotenone used in fish control are:

- 1) by eating fish killed with rotenone; or
- 2) by drinking water contaminated with rotenone.

Cohen et al. (1960) stated that the danger of ingesting rotenone by eating fish from poisoned lakes was very slight, since no significant amount would enter the fleshy part of the fish.

More recently, these residues have been quantified: following exposure to 2 ppm Noxfish, dead channel catfish, largemouth bass, bluegills, and redear sunfish contained from 0.045 to 0.101 ppm pure rotenone in their muscle fillets. Black bullheads which survived 1 ppm Noxfish for one hour contained 0.05 ppm pure rotenone immediately following treatment, and less than 0.020 ppm pure rotenone after 12 hours in fresh water (State of California, 1985). Based on the maximum residue figure and an estimated lethal dose of 18 g pure rotenone, researchers stated that a 130-pound person would have to eat a minimum of 397 pounds of fish at once to receive a lethal dose.

The California Department of Health Services suggested an acceptable daily intake (ADI) for humans of 0.0004 mg pure rotenone/kg body weight/day, applying a safety factor of 1,000 to the 0.4 mg/kg/day no-observable-effect levels (NOEL) determined by the Midwest Research Institute (1980). A 130-pound person would have to eat daily about one-half pound of fish containing 0.100 ppm pure rotenone to reach the ADI, not allowing for probable losses of rotenone through natural degradation and cooking (State of California, 1985). Canada allows a residue of 0.1 ppm pure rotenone in food (Khera et al., 1982).

The original use of rotenone-bearing plants in South America was the collection of fish for the table (Teixeira et al., 1984; Moretti and Grenand, 1982).

The main path by which people may come into contact with rotenone from fish applications is through drinking water (Gosa'lvez and Di'az-Gil, 1978). Cohen et al. (1960) concluded that the use of rotenone to kill fish in public reservoirs was consistent with the objective of safe and potable water. Where natural processes did not thoroughly detoxify rotenone by the time it reached the treatment station, they suggested the use of activated carbon to remove the residue.

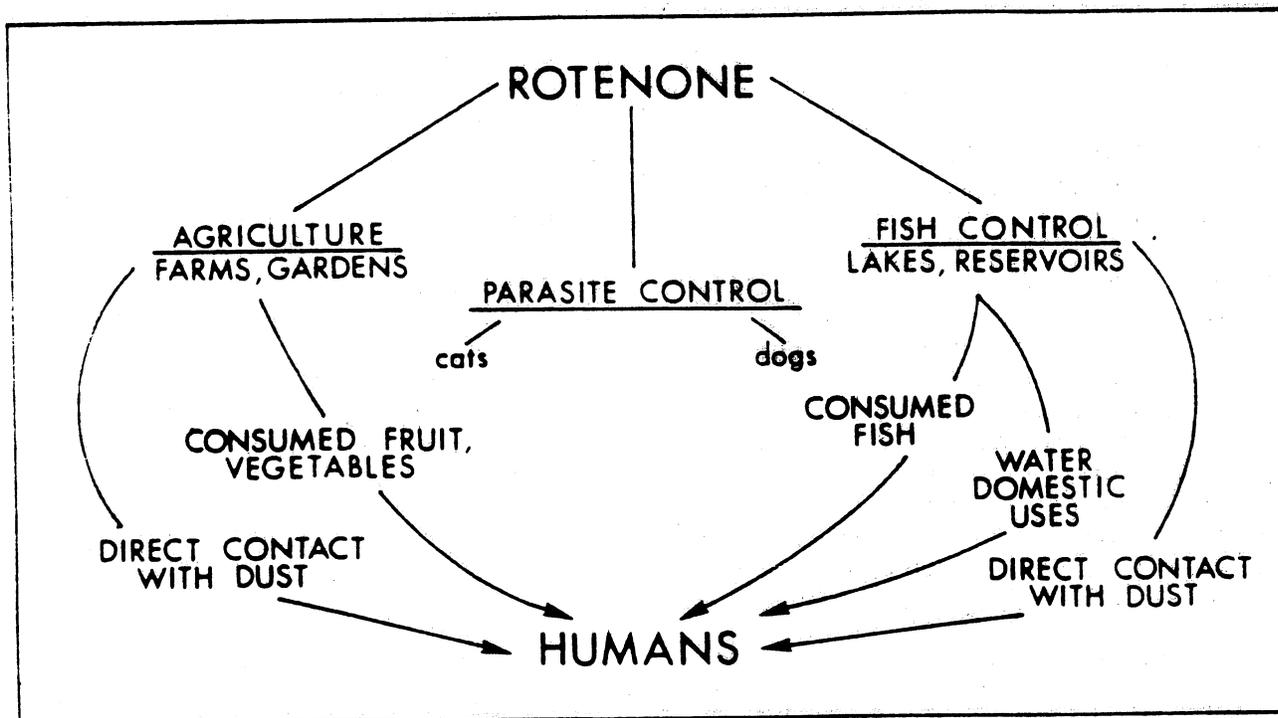


Figure 27 Paths of possible human exposure to rotenone. Source: Gosálvez and Díaz-Gil 1978.

Acute Oral Toxicity - There has never been a human death attributed to rotenone (Gosselin et al., 1984; Schnick, 1974; Thienes and Haley, 1972). The lethal oral dose for humans has been estimated from laboratory test with other mammals, mostly rats, and these estimates are shown in Table T. Lethal doses for pure rotenone range from 0.1 g per kg of body weight to 1.4 g/kg.

Santi and Toth (1965) warned that, contrary to most current literature, rotenone could be highly toxic to humans. Their experiments gave oral LD50's for rats that went as low as 0.049 g/kg, which is half of the smallest lethal dose listed in Table T. Yet their experimental solvent was acetone, and Santi and Toth admitted that to "render this (high) toxicity evident, it is necessary to choose proper solvents (ethanol and acetone) . . ."

Finally, while no record exists of a human fatality due to rotenone, there are several anecdotal reports of deaths due to the plants from which rotenone is extracted: Moretti and Grenand (1982) mention the use of *Lonchocarpus* by natives in French Guiana to commit suicide; Gimlette (1929) cites the use of "tuba root" *Derris elliptica* in Malaya for abortions, ritual suicide, and even attempted murder; Campbell (1916) describes a suicide in Singapore due to oral ingestion of *D. elliptica*. None of these references make mention of the dosages, and fresh derris root has a much higher toxicity than the dried powdered root from which rotenone is extracted (Gosselin et al., 1984).

Acute Respiratory Toxicity - Rotenone is more toxic when inhaled than when eaten (Windholz, 1983; Ambrose and Haag, 1937), though no estimates of the lethal respiratory dose for humans have been published. Santi and Toth (1965) tested a spray-mist of pure rotenone and ethanol on rats, and concluded that when inhaled "in proper vehicles or in association to other drugs, rotenone might cause unpleasant surprises". The "proper vehicle" they refer to would be a solvent such as ethanol or acetone. It is therefore highly unlikely that acute respiratory poisoning could occur in routine fisheries work.

Symptoms of Acute Rotenone Poisoning - Symptoms of acute oral rotenone poisoning are largely inferred from animal studies. Onset of the symptoms occurs within minutes to 5-6 hours after first coming in contact with the poison (Lehman, 1951). Poisoning results in numbness of the oral mucous membranes, nausea, vomiting, gastric pains, and muscle tremors. Respiration is at first stimulated, then depressed. Convulsions and coma are followed by death. The immediate cause of death is asphyxia from respiratory arrest (Gosselin et al., 1984; Thienes and Haley, 1972; Windholz, 1983; Sax, 1984). Symptoms of acute respiratory poisoning are the same except that there is also some lung irritation (Gosselin et al., 1984).

Table T Estimated lethal oral doses of rotenone for humans.

Lethal oral dose of pure rotenone (g/kg body weight)	Reference	Lethal oral dose of pure rotenone for a 150 lb. human	Lethal oral dose of 5% rotenone formulation for a 150 lb. human
0.3 - 0.5 g/kg	Gosselin 1984	20 - 34 g	400 - 680 g (14 - 24 oz)
0.3 - 0.4 g/kg*	Arena 1979	20 - 30 g	400 - 600 g (14 - 21 oz)
0.132 g/kg	Dreisbach 1983	9 g	180 g (6 oz)
0.14 - 1.4 g/kg**	Arena 1979	10 - 100 g	200 - 2000 g (7 - 70 oz)
0.20 g/kg	Sax 1984	14 g	280 g (10 oz)
---	Deichmann & Gerarde 1969	10 g***	200 g (7 oz)
0.132 g/kg	Lehman 1951	9 g	180 g (6 oz)
0.100 - 0.199 g/kg	U.S. EPA 1970	7 - 14 g	140 - 280 g (5 - 10 oz)

* actually reported as "20 - 30 g for a 150 - lb. man."

** actually reported as "10 - 100 g/70 kg."

*** actually reported as "the probable lethal dose (oral) for an adult."

Although rotenone has the potential to be highly toxic to humans when combined with certain solvents (Santi and Toth, 1965), there are certain properties of rotenone used in fisheries applications that reduce this potential:

- 1) the low percentage (1 to 5%) commonly used in commercial preparations (Gosselin et al., 1984);
- 2) it has an extremely low solubility in water (Santi and Toth, 1965);
- 3) it is unstable in nature, detoxifying quickly in both light and air (Haley, 1978);
- 4) it is an irritant when eaten, causing prompt vomiting (Haag, 1931);
- 5) it is inefficiently absorbed in the gastrointestinal tract (Gosselin et al, 1984);
- 6) the human body contains an effective oxidizing enzyme system (Schnick, 1974; Haag, 1931; Santi and Toth, 1965).

Subacute Toxicity - Direct Contact - WDW fisheries biologists handling rotenone dust during the course of routine lake poisonings usually report one or more of the following symptoms: a numb sensation in the mouth and lips, a mild sore throat, mild headache, eye irritation, and a runny nose (pers. comm.). Fisheries biologists in California, exposed to rotenone dust more or less continuously for periods up to three weeks developed all the above symptoms as well as sores on mucous membranes, eczema-like rashes, sloughing of the skin in some areas, severe week-long eye inflammations, and loss of appetite and the ability to taste (Pintler and Johnson, 1958).

Exposure to derris powder resulted in violent dermatitis of the genital region, irritation of the tongue and lips, and nasal passage inflammation (Racouchot, 1939). Both these studies recommended the use of face masks or protective clothing to reduce symptoms.

There has been no long-term study on the subacute effects of direct contact with rotenone dusts or liquids. The U.S. Environmental Protection Agency (1981) considers it safe to swim in water treated with rotenone. Dawson (1991) concluded that based on low mammalian toxicity and rapid rate of decomposition (especially at warmer temperatures that might be appropriate for swimming), the margin of safety is so great that water would be safe for swimming and other recreational use immediately following treatment.

Subacute Oral Toxicity - As with acute poisoning, the long-term toxic effects of rotenone on man must be inferred from experiments on other mammals. Table U presents the results of long-term oral dosages of rotenone on rats, dogs, and hamsters.

As shown in Table U, the most commonly noted effects of long-term rotenone feeding were:

- 1) Liver changes . Where noted, this usually involved a fatty metamorphosis of the liver. the lowest dosage that ever produce these changes was a continuous diet of food containing 130 ppm derris powder (9.6% rotenone) for 190 days. The same authors found no liver changes in dogs fed three times that amount for 240 days. No investigators since 1942 have reported these liver changes, although close histological inspection of all internal organs was part of all the later studies.
- 2) Growth inhibition . Either a major or minor decrease in weight gain, when compare to control animals, was reported in 10 fo the 13 studies. In some cases this may be a result of the unpalatability of the rotenone formulation, but Haag (1931) fed his dogs rotenone in capsule form and Freudenthal et al. (1981) took care to make the hamster diet equally palatable for both test and control animals. In both these studies significant growth inhibition was reported.
- 3) Other effects . Midwest Research Institute (1980) found that dogs fed and 10 mg of pure rotenone per kg of body weight developed gastrointestinal problems. The high dose also caused mild anemia and small but consistent decreases in blood glucose, total lipids, and cholesterol.

Studies have also involved pregnant mammals to determine if oral doses of rotenone would affect the fetuses of newborns. Hazelton Raltech, Inc., (1983; 1982; 1981) conducted three studies with pregnant rats, and determined that rotenone neither killed fetuses nor produced abnormal young when fed to the mothers on days 6 through 19 of gestation at doses ranging form 0.75 to 15 mg pure rotenone/kg body weight/day. The 1983 study involved feedings of up to 75 ppm pure rotenone tot two successive generations of rats on a daily basis; there was no effect on reproductive performance of either sex. Khera et al. (1982), in a 9-day study with pregnant rats, found that daily oral doses of 5 and 10 mg pure rotenone/kg body weight were responsible for a higher rate of nonpregnancies and resorptions, while 2.5mg/kg had no effect on the mothers or the young. Freudenthal et al. (1981) noted that a continuous diet of 500 ppm pure rotenone fed to a pregnant hamster for three months was toxic to the embryos and resulted in cannibalism of the young by the mothers.

Table V Results of long-term oral dosages of rotenone on dogs, rats, and hamsters.

Test animal	Period of daily treatment	Daily dosage, formulation	Liver change	Major growth inhibition	Minor growth inhibition	Other changes	No effect	Reference
dogs	102 days	10 mg (pure rotenone)	x	x				Haag 1931
dogs	180 days	0.4 mg/kg 2.0 mg/kg 10.0 mg/kg (pure rotenone)			x	x	x	Midwest Research Institute 1980
dogs	240 days	400 ppm (derris powder, 9.6% rotenone)			x			Ambrose and Haag 1938
dogs	190 days	130 ppm (derris powder, 9.6% rotenone)	x					Ambrose and Haag 1938
dogs	840 days	50 ppm 150 ppm 400 ppm (cube powder, 5.8% rotenone)					x x x	Hansen et al. 1965
rats	150 days	156 ppm 312 ppm 625 ppm (derris powder, 9.6% rotenone)	x x x		x			Ambrose and Haag 1938
rats	150 days	10 ppm 25 ppm 75 ppm 150 ppm 300 ppm (cube powder, 4.7% rotenone)					x x x x x	Haag and Talliaferro 1940

Table U Continued

Test animal	Period of daily treatment	Daily dosage, formulation	Liver change	Major growth inhibition	Minor growth inhibition	Other changes	No effect	Reference
rats	200 days	600 ppm	x	x				Ambrose et al. 1942
		900 ppm	x	x				
		1200 ppm (derris powder, 0.6-9.6% rotenone)	x	x				
rats	200 days	1200 ppm (cubé powder, 2.9% rotenone)			x			Ambrose et al. 1942
rats	730 days	50 ppm						Hansen et al. 1965
		100 ppm			x			
		250 ppm			x			
		500 ppm			x			
		1000 ppm (cubé powder, 5.8% rotenone)			x			
rats	490 days	100 ppm (Pro-Noxfish)		x			Brooks and Price 1961	
rats	365 days	100 ppm (detoxified Pro-Noxfish)			x		Brooks and Price 1961	
hamsters	90 days	500 ppm						Freudenthal et al. 1981
		1000 ppm (pure rotenone)		x			x	

Carcinogenicity - The results of a number of studies on the long-term effects of rotenone dusts (between 0.6 and 9.6% pure rotenone) were published from 1931 to 1942 (Haag, 1931; Ambrose and Haag, 1938; Haag and Taliaferro, 1940; Ambrose et al., 1942; Ambrose and Haag, 1936). While their results varied (see Table V), no tumors were observed by any of the researchers.

The first mention of tumors possibly caused by rotenone appeared in Lehman, 1952. He reported an increased incidence of peculiar cell masses - classified between hyperplasia and tumor - in the livers of rats fed rotenone continuously. These growths appeared in the rats fed between 2 and 10 ppm pure rotenone in the diet, but not at higher levels.

In 1959, another study by the U.S. Food and Drug Administration concluded that there was an abnormal incidence of liver tumors in rats fed 2, 5, and 10 ppm pure rotenone in the diet for two years. These tumors did not appear at higher levels (unpublished internal report, U.S. FDA, Division of Pharmacology, 1959; reported by Gosalvez, 1983).

Studies since that time on the cancer-causing potential of long-term exposure to rotenone are shown in Table V. All except two studies used pure rotenone in the tests; Hansen et al. (1965) fed cube' powder with 5.80% rotenone, similar to the commercial dusts used for fish control, and Brooks and Price (1961) fed Pro-Noxfish. In addition to the fresh powder, these last authors also tested Pro-Noxfish that had been completely detoxified, to see if the residues left in the water had any long-term effects.

Gosalvez and Merchan (1973) published a study in which rats injected with rotenone developed mammary tumors (Table V). Although these tumors were benign, they were transplantable, and showed an average doubling time of 2-3 months. The tumors were in many ways morphologically similar to human breast cancer (Gosalvez et al., 1977).

The same authors reported that these tumors could also be produced by low-level oral doses of rotenone on a daily basis for 45 days. They suggested a possible hormonal mechanism for the inducement of the tumors caused by rotenone (Gosalvez et al, 1979), and warned that rotenone could be "reaching the human female in certain countries" in amounts sufficient to cause mammary tumors, mostly by way of garden vegetables and drinking water (Gosalvez and Diaz-Gil, 1978).

As a result of this research, the U.S. EPA scheduled a reevaluation of rotenone and placed it on the Rebuttal Presumption Against Registration listing (RPAR) in 1976 (Anon, 1976). The agency commissioned a three year study, the results of which are shown in Table V (Freudenthal et al., 1981). The researchers concluded that neither direct oral administration, inclusion in the

Table V Studies on the cancer-causing potential of long-term exposure to rotenone.

Animal	Dosage	Vehicle	Diet	Fed or treated for	Observed for	Tumors	Reference
ROTENONE FED IN DIET OR WATER							
Syrian golden hamsters	125, 250, 500, & 1000 ppm (pure rotenone)	diet	Purina hamster chow	18 months (daily)	18 months	NO	Freudenthal et al. 1981
mice (two lab strains)	3 ppm (pure rotenone)	diet	not reported	18 months (daily)	18 months		Innes et al. 1969
Osborne-Mendel rats	50, 100, 250, 500 & 1000 ppm (cubé powder, 5.80% rotenone)	diet	Purina laboratory chow	24 months (daily)	24 months	NO	Hansen et al. 1965
Carworth rats	100 ppm (Pro-Noxfish)	drinking water	Gaines dog meal	17.5 months (daily)	17.5 months	NO	Brooks and Price 1961
Carworth rats	100 ppm (detoxified Pro-Noxfish)	drinking water	Gaines dog meal	13 months (daily)	13 months	NO	Brooks and Price 1961
ROTENONE FORCE-FED							
Wistar rats	2-3 mg/kg (pure rotenone)	sunflower oil	"deficient diet"	60 days (daily)	14 months	YES	Gosálvez et al. 1977
Wistar rats	1.7 & 3.0 mg/kg (pure rotenone)	Mazola corn oil	Purina rat chow	42 days (daily)	14 months	NO	Freudenthal et al. 1981
ROTENONE INJECTED							
Sprague-Dawley albino rats	1.7 & 3.0 mg/kg (pure rotenone)	Mazola corn oil	Purina rat chow	42 days (daily)	17 months	NO	Freudenthal et al. 1981
Wistar rats	1.7 mg/kg (pure rotenone)	sunflower oil	"deficient diet"	42 days (daily)	18 months	YES	Gosálvez and Merchán 1973
Wistar rats	9.0 mg total dose (pure rotenone)	---	"enriched" rat diet	---	18 months	NO	Gosálvez 1983

diet, or IP injection of rotenone caused tumors. As a result, the EPA dropped rotenone from its RPAR list in 1981 (Anon., 1981; 1983).

Marking (1988) also performed studies on chronic oral toxicity in rats, effects on reproduction in rats, and subchronic oral toxicity in dogs and concluded from the results of these studies and those in the literature that even high doses of rotenone do not cause tumors or reproductive failure, nor adversely affect fetal development.

APPENDIX A

FORMS

PRE-REHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

1. Demonstrate declines in the catch, survival, and/or size of trout fry (or other game fish).
2. Estimate number of recreational days lost due to poor trout fry (or other game fish) survival.
3. Demonstrate declines in past waterfowl use (if applicable).
4. For new waters, demonstrate the water's potential to produce a viable game fish fishery.

B. Physical Description of the Water Proposed for Rehabilitation

Provide the best map available with the following details:

1. Name of water (and county).
2. Location using township coordinates of proposed water.
3. Surface acres of water.
4. Depth range (and contours if available). If the water is greater than 100 acres, a bathymetric map will be produced if not available.
5. Volume of water.
6. Outlet statistics - Permanent, intermittent, dry.
7. Stream miles, stream flow.
8. Number of developed public access areas.
9. Land ownership (%) Public ____ Private ____
10. Established resorts.

C. Proposed Management Actions

1. Target species.
2. Date of last rehabilitation.
3. Proposed treatment date.
4. Estimated restocking date.
5. Species to restock.
6. Number of catchables, fry to stock.
7. Proposed toxicant name, type (liquid or powder) concentration, and amount required.
8. Method of application.
9. Size of crew and number and name of crew leaders needed.

II. PURPOSE

Detail the purpose of the rehabilitation and how this action relates to the management plan for this water.

III. INTENDED OUTCOME\MEASURE OF SUCCESS

Estimate duration of beneficial effects and how this will be measured.

IV. RESOURCE IMPACTS

1. Detail potential impacts to non-targeted resources, using survey data of individual waters (including outlets), information from non-game and waterfowl programs, and documented levels of impacts from published studies (use Bradbury for references).
2. Detail potential impacts to human related uses of the water or shoreline (i.e. irrigation, drinking water, beach combing, temporary loss of fishing, etc.) Identify the existence of water intakes.
3. List any endemic species, and/or species which are rare, endangered, threatened or otherwise listed which may be impacted by the proposed rehabilitation.

V. MITIGATING FOR ADVERSE IMPACTS

1. Describe how adverse impacts can be mitigated, or softened (i.e. time of year, removal of dead fish from shoreline, etc.)
2. Describe measures to protect downstream resources (list detoxicant used if applicable).
3. Describe measures to protect endemic species, and/or species which are rare, endangered, threatened and/or otherwise listed which may be impacted by the proposed rehabilitation.
4. Describe the safety precautions for pesticide applicators which will prevent health hazards.
5. Describe how the public will be discouraged from collecting dead or dying fish.

VI. RECREATIONAL IMPACT

Estimate increased angler success and number of recreational days generated from the proposed rehabilitation.

VII. ECONOMIC IMPACT

Given the above increased days in recreation, estimate impact to local businesses, and costs and benefits to our program. (Use Bradbury 1986 for reference).

VIII. RELATED MANAGEMENT ACTION

Detail management actions which are related to the proposed rehabilitation (e.g. stocking sizes and levels of fish, pre-rehab removal of selected fish, etc.)

IX. PUBLIC CONTACT

Detail how and when the public was contacted and what was the public's general response to the proposal.

POST REHABILITATION FORM

1. Lake or Stream _____ County _____
Section _____ Township _____ Range _____, WM
2. Lakes - surface acres _____ Miles of inlet or outlet treated _____
3. Steams - miles treated _____ Miles of tributaries treated _____
4. Maximum depth _____ Average depth _____
5. Weight (lbs) of water treated _____ Toxicant used _____
6. Amount used _____ lbs.; _____ % active ingredient
_____ gals.; _____ % active ingredient
7. Concentration applied _____ ppm, Date treated _____
8. Man hours expended in preparation, treatment and cleanup _____
Air time used _____

9. Conditions in the lake on date of treatment:

Depth in feet	Temperature	pH	Dissolved Oxygen
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

10. Species of fish eradicated in order of relative abundance:
- | | |
|----------|----------|
| 1. _____ | 5. _____ |
| 2. _____ | 6. _____ |
| 3. _____ | 7. _____ |
| 4. _____ | 8. _____ |

11. Possibility of a complete kill: _____

12. Detoxicant used _____
If any, report on effects recorded on downstream fishery.

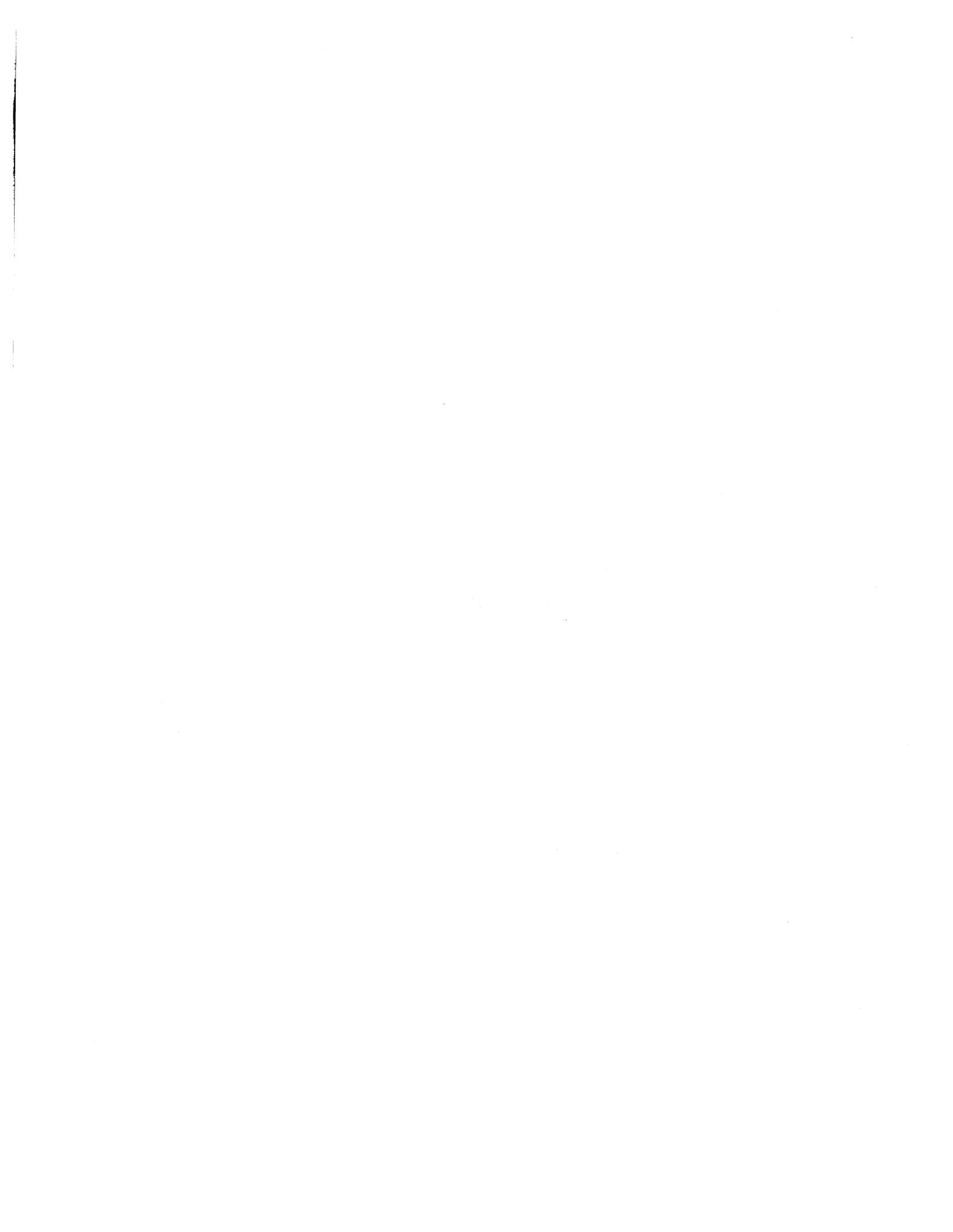
13. Period of toxicity: _____

14. Description of treatment and other comments: _____

Fishery Biologist
Region _____

Date

APPENDIX B
HISTORY OF ROTENONE



HISTORY OF ROTENONE

Rotenone is a white crystalline ketone with the chemical formula $C_{23}H_{22}O_6$. It is found in the roots of several tropical plants grown in Malaya, the East Indies, and Central and South America. For centuries, natives in these areas have killed fish for the table by poisoning lakes, ponds, and streams with rotenone preparations.

Besides rotenone itself, the so-called fish poison plants contain other active ingredients called rotenoids which are chemically related, but generally less toxic.

Rotenone and its parent plants have hundreds of common names, but the most widespread are derris, tuba (both names used to describe Asian genus *Derris*, especially *D. eliptica*), timbo', cube', and barbasco (the last three referring to the South American genus *Lonchocarpus*, especially *L. utilis*, *L. urucu*, and *L. nicou*).

Rotenone is used primarily as an agricultural insecticide and in household gardens. Its use as a fisheries management tool began in 1934, when Dr. Carl Hubbs attempted to poison carp and goldfish in two small Michigan ponds. By 1970, all states except Hawaii had used rotenone to kill fish, and most were using it routinely.

By far the most common aquatic use of rotenone today is the improvement of sport fishing via the elimination of other non-game or competitor species. Some other aquatic applications in the U.S. and Canada have been reported in the literature: Weier and Starr (1950) improved waterfowl refuges by poisoning carp from pond where they had uprooted the natural vegetation used by ducks for food and shelter; rotenone has been used to sample fish populations in lakes (Krumholz, 1950a), streams (Boccardy and Cooper, 1963), and coral reefs (Smith, 1973); M'Gonigle and Smith (1938) used rotenone to create a disease-free water source for a hatchery; and municipal water supplies have been treated with rotenone to reduce turbidity and algae caused by bottom-feeding fish (Hoffman and Payette, 1956; Bonn and Holbert, 1961; Barry, 1967).

The United States which consumes about 15 million pounds of rotenone per year, is supplied mostly by South America. Commercial preparations used in agriculture and fisheries are made primarily from the resins and dried and ground roots of *Derris* (and Asian genus) and *Lonchocarpus* (a South American genus) which are cultivated for that purpose. These dusts therefore contain not only rotenone itself (usually about 5% of the total content) but also varying amounts of the other rotenoids, as well as biologically inert material.

Synergists are sometimes added. Pure rotenone for laboratory purposes is extracted from the resins with solvents such as chloroform and benzene.

Technical literature on the sources, chemistry, history and use of rotenone abounds. The preceding is only a brief summary from the following detailed sources: Haley, 1978; National Academy of Science, 1983 (literature reviews of rotenone's chemistry, extraction, toxicology, biotransformation, and carcinogenicity); Schnick, 1974 (exhaustive literature review on all fisheries uses); Lennon, et al., 1970 Eschmeyer, 1975 (fish toxicants in general, with numerous references to rotenone and its history in fisheries); Gosa'lvez and Di'az-Gil, 1978 (scope of commercial use); Moretti and Grenand, 1982; Teixeira, et al., 1984 (botany and use of fish poison plants).

How Rotenone Works

Regardless of the organism, rotenone's primary toxic action is at the cellular level, where it blocks oxidative phosphorylation (Fukami, et al., 1967; Lindahl and Oberg, 1961; Ernster, et al., 1963; Figueras and Gosa'lvez, 1973). the specific site of action is localized in the electron transport system, where it becomes tightly bound (Oberg, 1961; Horgan, et al., 1968). Teeter, et al. (1969) demonstrated that high concentrations of rotenone can inhibit electron transfer in more than one region of the respiratory chain. Both the lethal and numerous pharmacological effects of rotenone can be ascribed to its inhibitory effect on cellular metabolism (Santi and To'th, 1965).

Rotenone's ability to inhibit cellular respiration has been well documented in cells of mammals, fish and insects (e.g., Fukami, et al., 1970), as well as amphibians (Denis and Devlin, 1968), and even plants (Ikuma and Bonner, 1967). Why then is rotenone extremely toxic to some life forms (fish and insects), relatively nontoxic to others (mammals, including humans) and virtually nonphytotoxic (being used extensively on crops and garden plants)?

Fukami, et al. (1969, 1970) concluded that the selective toxicity of rotenone between mammals, fish and insects was due to the differences in the site of entry and/or ease of rotenone detoxification rather than any cellular differences in the oxidation chain of these animals. There are some minor variations, however, in the mitochondria of different animals (and organs within a species) that may also contribute to these differences in toxicity (Ilivicky and Casida, 1969).

Although rotenone is toxic to isolated mammalian mitochondria, mammals - including humans - are not highly susceptible to the poison because they are protected by effective oxidizing enzyme systems (Shnick, 1974; Haag, 1931; Santi and To'th, 1965) and because of slow, inefficient gastrointestinal absorption (Gosselin, et al., 1984). If rotenone is enabled to reach its site of action through the use of solvents such as ethanol or acetone, however, there is no real difference in the sensitivity to the poison

between fishes and warm-blooded animals (Santi and To'th, 1965; Schmidt and Weber, 1975). Also while absorption in mammals is very inefficient, extremely high or continuous dosages may allow enough rotenone to reach the site of action for toxic effects to appear.

The high susceptibility of fish to rotenone is mostly due to its efficient entry through the gills (Schmidt and Weber, 1975; Oberg, 1964, 1967b). Oberg (1967a) demonstrated that the specialized structure of gills and lipid solubility favored the entrance of rotenone from water - where it is virtually insoluble - into the gill cell membrane. Once in the bloodstream rotenone is quickly carried to vital organs (such as the brain), where it inhibits cellular respiration (Oberg, 1964). The fact that fish immersed in rotenone solutions are protected if their gills are in contact with pure water is further proof that the gills are the main entry site in fish (Oberg, 1964). Orally administered rotenone does have a toxic effect on fish, but not nearly so much as topically applied rotenone (Hashimoto and Fukami, 1969).

Previously, rotenone was thought to kill fish by either destroying the gill tissues (Danneel, 1933) or by constricting the tiny gill capillaries (Hamilton, 1941). Microscopic examination of the gills of both fish and aquatic insects revealed that death usually occurred without any gill vasoconstriction or deterioration (Oberg, 1959; Lindahl and Oberg, 1961; Claffey and Ruck, 1967). However, gill epithelium may be damaged by high concentration of rotenone as a side effect, and when this occurs, the fish may die even when cellular respiration is restored by placing the fish in fresh, untreated water (Oberg, 1967b).

As in fish, the high susceptibility of insects to rotenone is primarily due to easy entry via the gill-like tracheae and the cuticle, although rotenone can also enter effectively through the mid-gut (Tischler, 1935; Fukami, et al., 1970).

In both aquatic insects and fish, rotenone tolerance tends to vary inversely with oxygen requirements, as would be expected for a poison that inhibits respiration (Engstrom-Heg, et al., 1978).

Rotenone's toxic effects are reversible, depending on the amount absorbed by the animal. Natural detoxification of sublethal rotenone dosages in insects, fish and mammals is primarily via oxidation by microsomal mixed function oxidase (mfo) enzymes (Fukami, et al., 1969; Fabacher and Chambers, 1972; Ludke, et al., 1972). In fact, certain chemicals (such as Sesamex) known to inhibit these mfo enzymes are sometimes added to insecticidal rotenone preparations as a synergist to increase its toxicity. At least in mammals, the inhibitory effect of rotenone on mitochondria is overcome by adding vitamin K (menadione), which activates a bypass of the rotenone-sensitive site (Santi and To'th, 1965; Gosselin, et al., 1984).

In fish, these natural mechanisms are sometimes able to effectively counter rotenone poisoning if the fish is removed to fresh, untreated water. While Leonard (1939) and Brown and Ball (1943a) were unable to revive rotenone-poisoned fish that had lost their equilibrium, Smith (1940) found that brook trout recovered in a fresh water bath, even when rotenone had affected their ability to swim upright. Gilderhus (1972) performed laboratory tests demonstrating that fish which had been floating on their sides in lethal concentrations of rotenone for as long as four hours often recovered if they were placed in fresh, untreated water. Oberg (1967b) revived rotenone-poisoned cod in untreated water and suggested the metabolic pathways involved.

In addition to fresh water baths, biologists have apparently succeeded in reviving fish with at least two other techniques. Bouck and Ball (1965) revived a variety of warmwater fish in methylene blue solutions. They tried the stain after Oberg (1961) showed that it reduced respiratory inhibition due to rotenone in the mitochondria of rat livers and fish gills. In one of their tests, Bouck and Ball were able to show that neither fresh water alone nor very low concentrations of methylene blue revived fish. The technique was not effective on rainbow trout, and the authors also cautioned that the stain was toxic to higher aquatic plants and that it encouraged bacterial growth on fish.

Fletcher (1976) successfully revived rotenone-poisoned bass on four Washington state lakes using a potassium permanganate dip. These fish were then moved by hatchery trucks to other lakes where they were released. Many of the fish that later recovered showed no signs of life when initially placed in the hatchery trucks. Fletcher hypothesized that the 20-second permanganate dip worked by neutralizing residual rotenone on the gills and body surface of the fish. Hepworth and Mitchum (1966), who also revived fish with permanganate dips and fresh water, concurred that the chemical neutralized residual rotenone on the gills. Fletcher also suggested that the extremely cold, hyperoxygenated fresh water in the hatchery trucks aided recovery. But since all fish in both Fletcher's and Hepworth and Mitchum's tests received the dip, there is no way to tell which factor was responsible for the recovery. It is possible that the cold, oxygenated fresh water alone would have revived the fish. Bouck and Ball (1965) stated that while permanganate detoxified rotenone in water, it was of no value in reviving fish..

Rotenone is unstable, degrading rapidly with exposure to light, heat, oxygen and alkalinity (Lennon, et al., 1970; Schnick, 1974). The degradation products were originally identified as dehydrorotenone (which is non-toxic to fish) and water (Subba-Rao and Pollard, 1951). Cheng, et al. (1972) later identified 20 degradation products, mainly rotenoids.

In natural waters, a variety of other factors contributes to the rate of degradation. These include the presence of organic debris, turbidity, lake morphology, dilution by inlets and runoff, and the dosage used (Shnick, 1974).

Post (1958) was the first to quantify the rate of rotenone detoxification in water. He concluded that water temperature was the most significant factor in the breakdown of rotenone; total dissolved solids, pH, alkalinity, dissolved oxygen, and various other cations and anions did not change the rate of breakdown to any great extent, and were not useful as predictive tools. He derived two empirical equations based on temperature for determining the time to detoxification.

More recent field and laboratory research has shown deviations from Post's predictive equations; these turned out to be related to the amount of sunlight reaching the toxic water. As noted above, rotenone is photochemically unstable, degrading rapidly in sunlight, and this reaction is accelerated at higher temperatures. Rotenone was shown to detoxify quickly in shallow warm lakes and slowly in deep or ice-covered lakes (Meyer, 1966; Engstrom-Heg and Colesante, 1979). The darker waters of the hypolimnion also detoxify more slowly than the well-lit epilimnetic water in a given lake (Engstrom-Heg and Colesante, 1979).

With these additional factors in mind Engstrom-Heg and Colesante (1979) developed the most complete set of equations for predicting rotenone breakdown in a wide variety of lakes and ponds. Their results in epilimnetic waters coincided closely with Post's (1958) earlier findings, and two of their predictive equations for use in clear, shallow, unstratified ponds are simple modifications of Post's formulas. But they added that the reduced sunlight in the hypolimnetic waters played an important role in the slow breakdown of rotenone in other lakes, and they developed three additional equations that take this into account. These equations are practical for field use, requiring only standard limnological data that area already available for most lakes. Engstrom-Heg and Colesante's detoxification rates coincided very closely with the results of Markings and Bills (1976), who arrived at their rate constants using a totally different approach.

While toxic periods vary greatly depending on the factors mentioned above, most lakes treated with rotenone are completely detoxified within five weeks of treatment (Shnick, 1974). Lakes in Washington state are usually non-toxic to fish about four to five weeks after treatment.

It is possible to accelerate the natural breakdown of rotenone in water by using certain oxidizing chemicals such as chlorine or potassium permanganate (Dawson, 1975). Considering the high rate of natural rotenone breakdown and the quantity of water involved,

these chemicals have little practical value in lakes. No lake detoxification with chemicals has been recorded in the literature (Lennon, et al., 1970).

Potassium permanganate is sometimes used, however, to detoxify outlet streams that flow from treated lakes (Engstrom-Heg, 1972). Pfeifer (1985) describes its use in detail and cites two case histories in western Washington. Both chlorine and activated carbon have been used to detoxify and deodorize treated lake water as it entered municipal water supplies (Cohen, et al., 1960; 1961a; 1961b).

Commercial fish-killing preparations of rotenone fall into three basic categories (Schnick, 1974):

- 1) 5% powder;
- 2) 5% emulsifiable concentrate;
- 3) 2.5% synergized emulsifiable concentrate.

Emulsifiable concentrates were developed to make application easier and to aid in dispersing the product (Meyer, 1966). Synergists (usually organic solvents such as sulfoxide) were later added to some formulations. These synergists aid absorption of the poison so that a 2.5% synergized mixture can be as effective as the more costly mixtures containing 5.0% rotenone (Price and Calsetta, 1957). Marking and Bills (1976) made extensive laboratory tests and found that the 25% synergized formulation Pro-Noxfish somewhat more toxic than a 5.0% nonsynergized formulation (Noxfish) to rainbow trout. The synergist sulfoxide, the emulsifying agents, and the solvents used in these preparations have been tested and found innocuous themselves (Penick and Co., 1959).

Bassett (1956) tested to see if there were significant toxicity differences between 2.5% preparations (Pro-Noxfish and Chem-Fish Special) and a 5% preparation (Chem-Fish); he found that in terms of toxicity, they were basically the same. Shannon (1969) tested nine commercial formulations ranging from 2.5% to 7.5% rotenone content. His laboratory bioassays with sunfish showed little variation in the amounts of formulation needed to produce a 24-hour LC50; he concluded that cost, mixing ability, and ease of handling should therefore determine the formulation used. Marking and Bills' (1976) laboratory tests showed no significant difference in toxicity between Noxfish and 5% rotenone powder. (Commercial preparations mentioned by trade name are shown in Table B.)

Although there are some conflicting reports, most investigators reported that rotenone was more toxic at high than at low temperatures, in acid than in alkaline waters, and in soft than in hard water. Many of these were field studies, however, where a great many other unmeasured variables could have affected the results. Furthermore, efficacy in many of the early laboratory and

caged-organism studies was based on survival time of the test organism rather than on concentration of the toxicant (Marking and Bills, 1976).

In the most recent, extensive and statistically thorough research on this topic, Marking and Bills (1976) found only slight changes in the toxicity of rotenone at differing temperature (44-72°F), pH (6.5-9.5), and water hardness (10-300 mg/l CaCO). These test were performed under standardized laboratory conditions using rainbow trout, channel catfish, and bluegills.

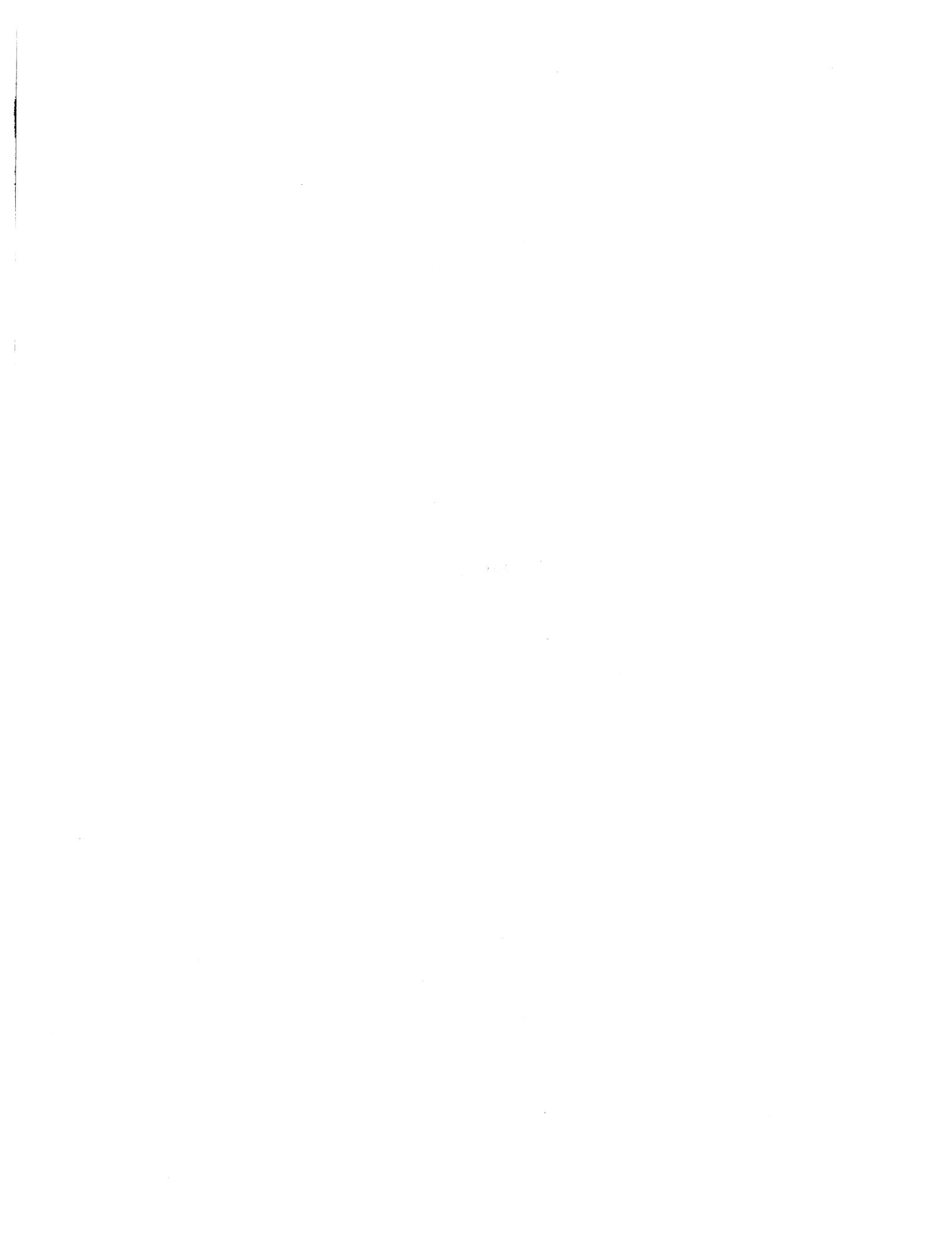
Burdick, et al. (1955) also concluded from bioassays that pH's between 6.28 and 8.10 made no difference in the toxicity of rotenone; they found, however, that toxicity increased as temperature rose. These conflicting reports on the effect of temperature may be due to the fact that rotenone degrades more rapidly in warm water than in cold.

In the lake environment, there are a number of other variables that act to either increase or decrease the effective toxicity of rotenones. Turbidity, soft, mucky bottom areas, weed beds, and organic sediments all appear to decrease the killing power of rotenone. The presence of a thermocline may prevent rotenone from reaching all areas of a lake, thus reducing efficiency. Underwater springs and surface outlets sometimes provide refuge for fish and invertebrates.



APPENDIX C

GLOSSARY



GLOSSARY

ADI	Acceptable Daily Intake of a material which should protect human health. Given as a mg of material per kg of body weight per day.
anoxic	deprived of oxygen
CPUE	Catch per unit effort
cube'	Common name for ground, dried roots (especially of <i>Lonchocarpus</i> sp.) containing rotenone.
DDT	an insecticide, dichlorodiphenyltrichloroethane, $(ClC_6H_4)_2CHCl_3$
derris	Common name for ground, dried roots especially of <i>Derris elliptica</i>) containing rotenone.
eutrophic	designating or of a lake, pond, etc. rich in plant nutrient minerals and organisms but often deficient in oxygen in midsummer.
hypolimnion	the lower most, noncirculating layer of cold water in a thermally stratified lake, usually deficient in oxygen.
IP	intraperitoneal
LC50	median lethal concentration; the concentration of a toxin in water that kills 50% of the test animals in the water within a specified time (usually 24, 48, or 96 hours). Usually expressed in ppm.
LD50	median lethal dosage; the dosage of a toxin that when fed or injected kills 50% of the test animals. Usually expressed as mg of toxin per kg of the test animal's body weight.
macrophytes	plant forms, individuals of which can be observed with the unaided eye.
mfo	mixed function oxidase
MSY	maximum sustainable yield
NOEL	No Observable Effect Level for a material exposed to test organisms.

oligotrophic designating or of a lake, pond, etc. poor in plant nutrient minerals and organisms and rich in oxygen at all depths.

ppm parts per million, usually by weight. 1 ppm equals 1 mg/l.

RPAR Rebuttal Presumption Against Registration list for protests against chemicals that the US Environmental Protection Agency has registered and labeled for use.

SNARL Suggested No Adverse Response Level.

APPENDIX D

REFERENCES

- Almquist, E. 1959. Observations on the effect of rotenone emulsives on fish food organisms. Inst. Freshwater Res. Drottningholm Rep. 40:146-160.
- Ambrose, A.M., F. Deeds, and J.B. McNaught. 1942. Chronic toxicity of derris. Ind. Eng. Chem. 34(6):684-689.
- Ambrose, A.M., and H.B. Haag. 1936. Toxicological study of derris. Ind. Eng. Chem. 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. Ind. Eng. Chem. 29:429-431.
- Ambrose, A.M., and H.B. Haag. 1938. Toxicological studies of derris: chronic toxicity of derris. Ind. Eng. Chem. 30(5):592-595.
- American Public Health Association. 1971. Standard methods for examination of water and wastewater, 13th ed. APHA, Washington, D.C. 874 pp.
- Anderson, R.S. 1970. Effects of rotenone on zooplankton communities and a study of their recovery patterns in two mountain lakes in Alberta. J. Fish. Res. Board Can. 27:1335-1356.
- Anderson, R.S. 1972. Zooplankton composition and change in an alpine lake. Verh. Internat. Verein. Limnol. 18:264-268.
- Andersson, G., H. Berggren, G. Cronberg, and C. Gelin. 1978. Effects of planktivorous and benthivorous fish on organisms and water chemistry in eutrophic lakes. Hydrobiologia 59:9-15.
- Anon. 1976. EPA lists pesticides that may be too dangerous to use. Chem. Eng. News, 14 June:18.
- Anon. 1981. EPA drops rotenone from its RPAR list. Chem. Mark. Rep. 220(3):16.
- Anon. 1983. Rotenone re-registration. Bull. Sport Fish. Inst. 347:4-5.
- Arnold, D.E. 1971. Ingestion, assimilation, survival, and reproduction by *Daphnia pulex* fed seven species of blue-green algae. Limnol. Oceanogr. 16:906-920.
- Ball, R.C. 1945. Recovery of marked fish following a second poisoning of the population in Ford Lake, Michigan. Trans. Am. Fish. Soc. 75:36-42.

- 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.
- Bandow, F. 1980. Effects of winterkill and chemical eradication of fish on a lake ecosystem. Minn. Dep. Nat. Resour., Div. Fish Wildl. Invest. Rep. No. 369. Completion Rep., Study 303, D.J. Project F-26-R. 41 pp.
- Banfield, A.W.F. 1974. The mammals of Canada. University of Toronto Press, Toronto. 438 pp.
- Barry, J.J. 1967. Evaluation of cree census, rotenone embayment, gill net, traps and electro-fishing gear samples, by complete drainage of Lenape and Bischoff Reservoirs. Indiana Dep. Nat. Resour., Div.. Fish Game, Fish. Res. Section. 35 pp.
- Bartoo, N.W. 1977. Proportions of a yellow perch population surfacing after a rotenone treatment. *Prog. Fish-Cult.* 39(1):27-28.
- Bassett, H.M. 1956. The economic evaluation of several fish toxicants. Colo. Fish. Res. Unit, Quart Rep. Vol. 2(3 and 4):6-16.
- Beard, T.D. 1971. Panfish literature review. Wis. Dep. Nat. Resour. Res. Rep. 71. 44 pp.
- Bennett, D.H. 1985. Fish community rehabilitation with rotenone: effects on zooplankton and benthic organisms. Pages 12-19 in Liberty Lake Sewer District 1985.
- Bennett, G.W. 1943. Management of small artificial lakes. Ill. Nat. Hist. Surv. Bull. 22:357-376.
- Bennett, G.W. 1962. Management of artificial lakes and ponds. Reinhold Publishing Corporation, New York. 283 pp.
- Berzins, B. 1958. Om fiskerard med rotenon. *Sodra Sveriges Fiskeriforening, Arsskrift* 1957:41-53. Referred to in Almquist, 1959.
- Bhagat, S.K., G.C. Bailey, W.H. Funk, J.E. Ongerth, and J.F. Orsborn. 1975. Study of Silver Lake eutrophication: current problems and possible solutions. Washington State University, Water Res. Center Rep. 19. 298 pp.
- Binns, N.A. 1967. Effects of rotenone treatment on the fauna of Green River, Wyoming. Wyo. Game Fish Comm., Fish. Res. Bull. 1. 114 pp.

- Blue Spruce Company. 1973. Toxicological and ecological information on the effects of rotenone products. Blue Spruce Company, Basking Ridge, N.J. 11 pp. (Now Tifa Limited, Millington, N.J.)
- Boccardy, J.A., and E.L. Cooper. 1963. The use of rotenone and electrofishing in surveying small streams. Trans. Am. Fish Soc. 92(3):307-310.
- Bonn, E.W., and L.R. Holbert. 1961. Some effects of rotenone products on municipal water supplies. Trans. Am. Fish. Soc. 90(3):287-297.
- Bouck, G.R., and R.C. Ball. 1965. The use of methylene blue to revive warmwater fish poisoned by rotenone. Prg. Fish-Cult. 27(3):161-162.
- Braaten, D.O. 1970. Characteristics and angling desires of western Washington trout anglers, and a simulation of the fishery management system so as to optimize angler enjoyment. Ph.D. Thesis. University of Washington, Seattle, Wash. 155 pp.
- Bradbury, W.A., and R.L. Pfeifer. 1986. Fisheries investigations of Lakes Washington and Sammamish, 1980-1983. Final Report, Part 6: a survey of public attitudes towards the fisheries of Lakes Washington and Sammamish. Wash. Dep Wildlife, in prep.
- Brezonik, P.L. 1978. Effect of organic color and turbidity on secchi disc transparency. J. Fish. Res. Board. Can. 35:1410-1416.
- Bridges, W.R., and O.B. Cope. 1965. The relative toxicities of similar formulations of pyrethrum and rotenone to fish and immature stoneflies.
- Brooks, I.C. 1961. Research methods and findings on fish toxicants and their application. S.B. Penick and Co. Research Dep., New York, N.Y. 10 pp.
- Brooks, I.C., and R.W. Price. 1961. Studies on the chronic toxicity of Pro-Noxfish, a proprietary synergized rotenone fish-toxicant. Toxicol. Appl. Pharmacol. 3:49-56.
- Brooks, J.L., and S.T. Dodson. 1965. Predation, body size, and composition of plankton. Science 150:28-35.
- Brown, C.J., and R.C. Ball. 1943a. An experiment in the use of derris root (rotenone) on the fish and fish food organisms of Third Sister Lake. Trans. Am. Fish. Soc. 72:267-284.

- Brown, C.J., and R.C. Ball. 1943b. A fish population study of Third Sister Lake. *Trans. Am. Soc.* 72:177-186.
- Brown, L. 1978. Opening Day creel census models for trout management lakes. *Wash. Dep. Wildlife Fish. Res. Rep.* 122pp.
- 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. 49 pp.
- Brynildson, O.M., and J.J. Kempinger. 1973. Production, food and harvest of trout in Nebish Lake, Wisconsin. *Wis. Dep. Nat. Resour. Tech. Bull.* 65. 20pp.
- Bull, C.J., and W.C. Mckay. 1976. Nitrogen and phosphorus removal from lakes by fish harvest. *J. Fish. Res. Board Can.* 33:1374-1376.
- Burdick, G.E., H.J. Dean, and E.J. Harris. 1955. Toxicity of emulsifiable rotenone to various species of fish. *N.Y. Fish Game J.* 2(1):36-67.
- Burns, C.W. 1969. Relation between filtering rate, temperature, and body size in four species of *Daphnia*. *Limnol. Oceanogr.* 14:693-700.
- Burress, R.M. 1975. Development and evaluation of onsite toxicity test procedures for fishery investigations. *U.S. Fish Wildl. Serv. Invest. Fish Control* 68. 8 pp.
- Burress, R.M. 1982. Effects of synergized rotenone on nontarget organisms in ponds. *U.S. Fish. Wildl. Serv. Invest. Fish Control* 91. 7pp.
- Carlander, K.D. 1969. Handbook of freshwater fishery biology, Vol. 1. Life history data on freshwater fishes of the United States and Canada, exclusive of Perciformes. Iowa State University Press, Ames, Iowa. 752 pp.
- Carlander, K.D., and W.M. Lewis. 1948. Some precautions in estimating fish populations. *Prog. Fish-Cult.* 10:134-137.
- Chandler, J.H., and L.L. Marking. 1982. Toxicity of rotenone to selected aquatic invertebrates and frog larvae. *Prog. Fish-Cult.* 44(2):78-80.
- Cheng, H.M., I. Yamamoto, and J.E. Casida. 1972. Rotenone photodecomposition. *J. Agric. Food. Chem.* 20(4):850-856.

- Claffey, F.J., and J.E. Ruck. 1967. The effect of rotenone on certain fish food organisms. Proc. Annu. Conf. Southeast. Assoc. Game Fish Comm. 20:278-283.
- Clemens, H.P., and M. Martin. 1953. Effectiveness of rotenone in pond reclamation. Trans. Am. Fish. Soc. 82:166-177.
- 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. J. Am. Water Works Assoc. 52(12):1551-1566.
- Cohen, J.M., G.A. Rourke, and R.L. Woodward. 1961a. Effects of fish poisons on water supplies. Part 2. Odor problems. J. Am. Water Works Assoc. 53(1):49-62.
- Cohen, J.M., Q.H. Pickering, R.L. Woodward, and W. Van Heuvelen. 1961b. Effect of fish poisons on water supplies. Part 3. Field study at Dickinson. J. Am. Water Works Assoc. 53(2):233-246.
- Cook, G.D., M.R. McComas, D.W. Waller, and R.H. Kennedy. 1977. The occurrence of internal phosphorous loading in two small, eutrophic, glacial lakes in northeastern Ohio. Hydrobiologia 56(2):129-135.
- Cook, S.F., Jr., and R.L. Moore. 1969. The effects of rotenone treatment on the insect fauna of a California stream. Trans. Am. Fish. Soc. 98:539-544.
- Culley, D.D., Jr., and D.E. Ferguson. 1969. Patterns of insecticide resistance in the mosquitofish, *Gambusia affinis*. J. Fish. Res. Board Can. 26:2395-2401.
- Cumming, K.B. 1975. History of fish toxicants in the United States. Pages 5-21 in P.H. Eschmeyer, ed. 1975.
- Cumming, K.B., R.M. Burress, and P.A. Gilderhus. 1975. Controlling grass carp (*Ctenopharyngodon idella*) with antimycin, rotenone, and thanite and by electrofishing. Prog. Fish-Cult. 37(2):81-84.
- Cummins, J.L. 1975. 1974-75 Morton Lake creel census project. Wash Dep. Wildl. 40 pp.
- Cushing, C.E., Jr., and J.R. Olive. 1957. Effects of toxaphene and rotenone upon the macroscopic bottom fauna of two northern Colorado reservoirs. Trans. Am. Fish. Soc. 86:294-301.
- Cutkomp, L.K. 1943a. Toxicity of rotenone and derris extract administered orally to birds. J. Pharmacol. Exp. Ther. 77:238-246.

- Cutkomp, L.K. 1943b. Toxicity of rotenone to animals. Soap Sanitary Chem. 19(10):107-123.
- Danneel, R. 1933. Die giftwirkung des rotenons und seiner derivate auf fische (II. Der angriffspunkt der gifte). Z. Vgl. Physiol. 18:524-535. Referred to in Schnick, 1974.
- Dawson, V.K. 1975. Counteracting chemicals used in fishery operations: current technology and research. Pages 32-40 in P.H. Eschmeyer, ed. 1975.
- Dawson, V.K., W.H. Gingerich, R.A. Davis, and P.A. Gilderhus. 1991. Rotenone Persistence in Freshwater Ponds: Effects of temperature and Sediment Adsorption. North Am. J. Fish. Manage. 11:226-231.
- Denis, S., and T.M. Devlin. 1968. The effect of oligomycin on the development of amphibian eggs. Exp. Cell Res. 52:308-318.
- Dexter, W.D. 1965. Some effects of rotenone treatment on the fauna of the Green River Wyoming. West. Assoc. Game Fish Comm. 45:193-197.
- Dunst, R.C., S.M. Born, P.D. Uttormark, S.A. Smith, S.A. Nichols, J.O. Peterson, D.R. Knauer, S.L. Serns, D.R. Winter, and T.L. Wirth. 1974. Survey of lake rehabilitation techniques and experiences. Wis. Dep. Nat. Resour. Tech. Bull. 179 pp.
- Edmondson, W.T. 1957. Trophic relations of the zooplankton. Trans. Am. Microsc. Soc. 76:225-246.
- Edmondson, W.T. 1980. Secchi disk and chlorophyll. Limnol. Oceanogr. 25(2):378-379.
- Edmonson, W.T., and A.H. Litt. 1982. *Daphnia* in Lake Washington. Limnol. Oceanogr. 27:272-293.
- Engstrom-Heg, R. 1972. Kinetics of rotenone-potassium permanganate reactions as applied to the protection of trout streams. N.Y. Fish Game J. 19(1):47-58.
- Engstrom-Heg, R., and R.T. Colesante. 1979. Predicting rotenone degradation in lakes and ponds. N.Y. Fish Game J. 26(1):22-36.
- Engstrom-Heg, R., R.T. Colesante, and E. Silco. 1978. Rotenone tolerances of stream-bottom insects. N.Y. Fish Game J. 25(1):31-41.
- Eschmeyer, P.H., ed. 1975. Rehabilitation of fish populations with toxicants: a symposium. Am. Fish. Soc., North Central Div., Spec. Publ. 4. 74 pp.

- Eschmeyer, R.W. 1953. Oklahoma program. Bull. Sport Fish. Inst. 24:2.
- Fabacher, D.L., and H. Chambers. 1972. Rotenone tolerance in mosquitofish. Environ. Pollut. 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, WI. 32 pp.
- Fitzgerald, G.P. 1970. Aerobic lake muds for the removal of phosphorus from lake waters. Limnol. Oceanogr. 15:550-555.
- Fletcher, D.H. 1976. Salvage of bass affected by rotenone. Wash. Dep. Wildl., unpublished rep. 8 pp.
- Fowler, C.W. 1973. An ecosystem model of Fern Lake. Ph.D. Thesis. University of Washington, Seattle, Wash. 115 pp.
- Freudenthal, R.I., A.P. Leber, D.C. Thake, and R.L. Baron. 1981. Carcinogenic potential of rotenone: subchronic oral and peritoneal administration to rats and chronic dietary administration to Syrian golden hamsters. U.S. Environ. Prot. Agency Rep. EPA-600.1-81-037. (NTIS Tech. Rep. PB 81-190936) 47 pp.
- 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. J. Agric. Food Chem. 17(6):1217-1226.
- Fukami, J.I., T. Mitsui, K. Fukunaga, and T. Shishido. 1970. The selective toxicity of rotenone between mammal, fish and insect. Pages 159-178 in R.D. O'Brien and I. Yamamoto, eds. Biochemical toxicology of insecticides. Academic Press, New York. 218 pp.
- Funk, W.H., and B. Moore. 1984. Statement in regard to Liberty Lake rotenone treatment. Presented at Big Game Commission Hearing, Yakima, Wash., on July 9, 1984. In Washington Department of Wildlife, Final supplemental environmental impact statement, Lake Rehabilitation Program 1984-1985.
- Galbraith, M.G. 1967. Size-selective predation on *Daphnia* by rainbow trout and yellow perch. Trans. Am. Fish. Soc. 96:1-10.
- Galbraith, M.G. 1974. Dynamics of *Daphnia* in relation to fishing quality for rainbow trout and in virgin fish-free lakes. Mich. Dep. Nat. Resour. Job. Progress Rep., Project F-28-R-8. 8 pp.

- Garrison, R.L. 1968. The toxicity of Pro-Noxfish to salmonid eggs and fry. *Prog. Fish-Cult.* 30(1):35-38.
- Gilderhus, P.A. 1972. Exposure times necessary for antimycin and rotenone to eliminate certain freshwater fish. *J. Fish. Res. Board Can.* 29:199-202.
- Gliwicz, Z.M. 1975. Effect of zooplankton grazing on photosynthetic activity and composition of phytoplankton. *Verh. Internat. Verein. Limnol.* 19:1490-1497.
- Gimlette, J.D. 1929. Malay poisons and charm cures, 3rd ed. J. and A. Churchill, London. 301 pp.
- Goad, J.A. 1982. Biomanipulation and its potential in the restoration of Green Lake. M.S. Thesis. University of Washington, Seattle, Wash. 55 pp.
- Gosalvez, M. 1983. Minireview: carcinogenesis with the insecticide rotenone. *Life Sci.* 32(8):809-816.
- Gosalvez, M., and J.J. Diaz-Gil. 1978. Rotenone: a possible environmental carcinogen? *Europ. J. Cancer* 14:1403:1404.
- Gosalvez, M., J. Diaz-Gil, J. Coloma, and L. Salganicoff. 1977. Spectral and metabolic characteristics of mitochondrial fractions from rotenone-induced tumors. *Brit. J. Cancer* 36:243-253.
- Gosalvez, M., J. Diaz-Gil, J. Alcaniz, and J. Borrell. 1979. A possible pathogenic mechanism for the induction of rotenone tumors. *Biochem. Soc. Trans.* 7:113-115.
- Gosalvez, M., and J. Merchan. 1973. Induction of rat mammary adenomas with the respiratory inhibitor rotenone. *Cancer Res.* 33:3047-3050.
- Gosselin, R.E., R.P. Smith, and H.C. Hodge. 1984. Clinical toxicology of commercial products, 5th ed. Willams and Wilkins, Baltimore, MD. 2000 pp.
- Gustafson, K.J., N.A. Nilsson, and E. Olofsson. 1981. Oevre Saervsjoen: Konsekvenser av rotenonbenhandling av en stor norrlaendsk sjoe (in Swedish, English abstract). *Inf. Soetvattenslab. Drottningholm.* 20 pp.
- Haag, H.B. 1931. Toxicological studies of *Derris elliptica* and its constituents. I. Rotenone. *J. Pharmacol. Exp. Ther.* 43:193-208.
- Haag, H.B., and I. Taliaferro. 1940. Toxicological studies on cube'. *J. Pharmacol. Exp. Ther.* 69:13-20.

- Haley, T.J. 1978. A review of the literature of rotenone. J. Environ. Pathol. Toxicol. 1:315-337.
- Hamilton, H.L. 1941. The biological action of rotenone on freshwater animals. Proc. Iowa Acad. Sci. 48:467-479.
- Hansen, W.H., K.J. Davis, and O.G. Fitzhugh. 1965. Chronic toxicity of cube'. Toxicol. Appl. Pharmacol. 7:535-542.
- Hashimoto, Y., and J.I. Fukami. 1969. Toxicity of orally and topically applied pesticide ingredients to carp, *Cyprinus carpio* Linne. Bochu Kagaku 34:63-66.
- Hazelton Raltech, Inc. 1981. Teratology study with rotenone in mice. Summary statement. Hazelton Raltech, Inc., Madison, WI. 1 pp.
- Hazelton Raltech, Inc. 1982. Teratology study with rotenone in rats. Summary statement. Hazelton Raltech, Inc., Madison, WI. 1 pp.
- Hazelton Raltech, Inc. 1983. Reproduction study for the safety evaluation of rotenone using rats. Summary statement. Hazelton Raltech, Inc., Madison, WI. 1 pp.
- Helfrich, L.A. 1978. Effects of rotenone on macrobenthic invertebrates of a Pennsylvania stream. Proc. Annu. Conf. Southeast. Assoc. Fish Wildl. Agencies 32:401-408.
- Hepworth, W.G., and D.L. Mitchum. 1966. Study of fish toxicants. Wyo. Game Fish Comm. Job Completion Rep., Project FW-3-R-13. 19 pp.
- Hester, F.E. 1959a. The tolerance of eight species of warmwater fishes to certain rotenone formulations. Proc. Annu. Conf. Southeast. Assoc. Game Fish Comm. 13:121-133.
- Hester, F.E. 1959b. The toxicity of Noxfish and Pro-Noxfish to eggs of common carp and fathead minnows. Proc. Annu. Conf. Southeast. Assoc. Game Fish Comm. 13:325-331.
- Hodge, R.P. 1983. Washington amphibians and reptiles: check list and guide. Wash. Dep. Wildl., Nongame Program brochure, 8 pp.
- Hoffman, D.A., and J.R. Olive. 1961. The effects of rotenone and toxaphene upon plankton of two Colorado reservoirs. Limnol. Oceanogr. 6(2):219-222.
- Hongve, D. 1977. Virkninger av rotenonbehandling pa zooplanktonfaunaen i et lite vann. (in Norwegian). Vatten 33(1):39-42.

- Hooper, F.F. 1948. The effect of derris root (rotenone) upon plankton and bottom fauna organisms of a small Minnesota lake. Proc. Minn. Acad. Sci. 16:29-33.
- 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. J. Biol. Chem. 243(4):834-843.
- Houf, L.J., and R.S. Campbell. 1977. Effects of antimycin A and rotenone on macrobenthos in ponds. U.S. Fish Wildl. Serv. Invest. Fish Control 80. 29 pp.
- Howland, R.M. 1969. Interaction of antimycin A and rotenone in fish bioassays. Prog. Fish-Cult. 31(1):33-34.
- Hrbacek, J. 1964. Contribution to the ecology of water-bloom-forming blue-green algae, *Aphanizomenon flos aquae* and *Microcystis aeruginosa*. Int. Ver. Theor. Angew. Limnol. Verh. 15:837-846.
- Hrbacek, J., M. Dvorakova, V. Korinek, and L. Prochazkova. 1961. Demonstration of the effect of the fish stock on the species composition of zooplankton and the intensity of metabolism of the whole plankton association. Verh. Internat. Verein. Limnol. 14:192-195.
- Hrbacek, J., and M. Novotna-Dvorakova. 1965. Plankton of four backwaters related to their size and fish stock. Rozpr. Cesk. Akad. Ved 75:1-66.
- Huntington, E.H. 1956. Bluewater Lake rehabilitation. N.M. Dep. Game Fish, Job Completion Rep., Project F-13-D. 10 pp.
- Hutchinson, G.E. 1957. A treatise on limnology, volume 1 part 2: chemistry of lakes. John Wiley and Sons, New York. 1015 pp.
- Hynes, H.B.N. 1970. The ecology of running waters. University Toronto Press. Toronto. 555 pp.
- Ikuma, H., and W.D. Bonner, Jr. 1967. Properties of higher plant mitochondria. III. Effects of respiratory inhibitors. Plant Physiol. 42:1535-1544.
- Ilivicky, J., and J.E. Casida. 1969. Uncoupling action of 2,4-dinitrophenols, 2-trifluoromethylbenzimidazoles and certain other pesticide chemicals upon mitochondria from different sources and its relation to toxicity. Biochem. Pharmacol. 18:1389-1401.

- 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. Bioassays of pesticides and industrial chemicals for tumorigenicity in mice: a preliminary note. *J. Nat. Cancer. Inst.* 42:1101-1114.
- Jackson, S.Y. 1984. Preliminary study of the upper Cow Creek watershed rehabilitation project. Wash. Dep. Wildl., unpublished report. 21 pp.
- Jenkins, R.M. 1956. Some results of the partial fish population removal technique in lake management. *Proc. Okla. Acad. Sci.* 37:164-173.
- Johnston, J.M. 1973. An evaluation of the lowland lake management program in Region 10 and Kitsap County, based on 1972 and 1973 creel census data. Wash. Dep. Wildl. 26 pp.
- Kempinger, J.J., and L.M. Christenson. 1978. Population estimates and standing crops of fish in Nebish Lake. Wis. Dep. Nat. Resour. Rep. 96. 12 pp.
- Kenaga, E.E., and W.E. Allison. 1971. Commercial and experimental organic insecticides (1971 revision). Reprinted from the *Bull. Entomol. Soc. Am.* 15(2):85-148, 1969, and revised according to changes in 16(1):68, 1970.
- Kerfoot, W.C., ed. 1980. Evolution and ecology of zooplankton communities. University Press of New England, Hanover, N.H. 793 pp.
- Khera, K.S., C. Whalen, and G. Angers. 1982. Teratogenicity study on pyrethrum and rotenone (natural origin) and ronnel in pregnant rats. *J. Toxicol. Environ. Health* 10:111-119.
- Kiser, R.W., J.R. Donaldson, and P.R. Olson. 1963. The effect of rotenone on zooplankton populations in freshwater lakes. *Trans. Am. Fish. Soc.* 92:17-24.
- Kitchell, J.A., and J.F. Kitchell. 1980. Size-selective predation, light transmission, and oxygen stratification: evidence from the recent sediments of manipulated lakes. *Limnol. Oceanogr.* 25(3):389-402.
- Klingbeil, J.H. 1975. Use of fish toxicants in Wisconsin, 1941-1973. Pages 54-59 in P.H. Eschemeyer, ed. 1975.
- Knapp, S.M., and R.A. Soltero. 1983. Trout-zooplankton relationships in Medical Lake, WA, following restoration by aluminum sulfate treatment. *J. Freshwater Ecol.* 2:1-12.

- Krska, R.J., and R.L. Applegate. 1984. Food of bluegills (*Lepomis macrochirus*) from heated and unheated areas of a power plant cooling reservoir. *J. Freshwater Ecol.* 2(4):325-334.
- Krumholz, L.A. 1950a. Some practical considerations in the use of rotenone sampling data. *Proc. Annu. Conf. Southeast. Assoc. Game Fish Comm.* 11:91-98. Referred to in Bartoo 1977.
- Krumholz, L.A. 1950b. Some practical considerations in the use of rotenone in fisheries research. *J. Wildl. Manage.* 14:413-424.
- Lamarra, V.A. 1975. Digestive activities of carp as a major contributor to the nutrient loading of lakes. *Verh. Internat. Verein. Limnol.* 19:2461-2468.
- Lamarra, V.A. 1976. Experimental studies of the effect of carp (*Cyprinus carpio* L.) on the chemistry and biology of lakes. Ph.D. Thesis. University of Minnesota, Minneapolis, MN 326 pp. University Microfilm abstr. No. 76-27, 811.
- Lambou, V.W., and H. Stern, Jr. 1957. An evaluation of some of the factors affecting the validity of rotenone sampling data. *Proc. Annu. Conf. Southeast. Assoc. Game Fish Comm.* 11:91-98.
- Lampert, W. 1981. Toxicity of the blue-green *Microcystis aeruginosa*: effective defense mechanism against grazing pressure by *Daphnia*. *Int. Ver. Theor. Angew. Limnol. Verh.* 21:1436-1440.
- Lefevre, M. 1950. *Aphanizomenon gracile* Lem. cyanophyte defavorable au zooplankton. *Ann. Sta. Cent. Hydrobiol. Appl.* 3:205-208. Referred to in Edmondson, 1957.
- Lehman, A.J. 1951. Chemicals in foods: a report to the Association of Food and Drug Officials on current developments. Part II. Pesticides. *Quart. Bull. Assoc. Food Drug Officials U.S.* 15:122-133.
- Lellak, J. 1965. The food supply as a factor regulating the population dynamics of bottom animals. *Mitt. Internat. Verein. Limnol.* 13:128-138.
- Lennon, R.E., J.B. Hunn, R.A. Schnick, and R.M. Burrell. 1970. Reclamation of ponds, lakes, and streams with fish toxicants: a review. *FAO Fish. Tech. Pap.* 100. 99 pp.
- Leonard, J.W. 1939. Notes on the use of derris as a fish poison. *Trans. Am. Fish. Soc.* 68:269-279.
- Li, H.W., and P.B. Moyle. 1981. Ecological analysis of species introductions into aquatic systems. *Trans. Am. Fish. Soc.* 110:772-782.

- Libey, G.S., and L.E. Holland. 1980. The use of periodic light applications of rotenone as a management technique for small impoundments. Purdue Univ., Water Resour. Res. Center, Tech. Rep. 132. (NTIS microfilm PB 81-118770). 39 pp.
- Lindahl, P.E., and K.E. Oberg. 1961. The effect of rotenone on respiration and its point of attack. *Exp. Cell Res.* 23:228-237.
- Lindgren, P.E. 1960. About the effect of rotenone upon benthic animals in lakes. *Inst. Freshwater Res. Drottingholm Rep.* 41:172-184.
- Ludke, J.L., J.R. Gibson, and C.L. Lusk. 1972. Mixed function oxidase activity in fresh water fishes: Aldrin epoxidation and parathion activation. *Toxicol. Appl. Pharmacol.* 21:89-97.
- Lynch, M. 1980. *Aphanizomenon* blooms: alternative control and cultivation by *Daphnia pulex*. Pages 229-304 in W.C. Kerfoot, ed. 1980.
- Lynch, M., and J. Shapiro. 1981. Predation, enrichment, and phytoplankton community structure. *Limnol. Oceanogr.* 26(1):86-102.
- Marking, L.L. 1972. Sensitivity of the white amur to fish toxicants. *Prog. Fish-Cult.* 34(1):26.
- Marking, L.L. 1975. Toxicological protocol for the development of piscicides. Pages 26-31 in P.H. Eschmeyer, ed. 1975.
- Marking, L.L. 1988. Oral toxicity of rotenone to mammals. U.S. Fish Wildl. Serv. Invest. Fish Control 94.
- Marking, L.L., and T.D. Bills. 1976. Toxicity of rotenone to fish in standardized laboratory tests. U.S. Fish Wildl. Serv. Invest. Fish Control 72.
- Marking, L.L., T.D. Bills, and J.J. Rach. 1983. Chemical control of fish and fish eggs in the Garrison Diversion Unit, North Dakota. *N. Am. J. Fish. Manage.* 3:410-418.
- Meadows, B.S. 1973. Toxicity of rotenone to some species of coarse fish and invertebrates. *J. Fish. Biol.* 5(2):155-163.
- Meehan, O.L. 1942. Fish populations of five Florida lakes. *Trans. Am. Soc.* 71:184-194.
- Merritt, R.W., and K.W. Cummins, eds. 1978. An introduction to the aquatic insects of North America. Kendall/Hunt Publishing Co., Dubuque, IA.

- Meyer, F.A. 1966. Chemical control of undesirable fishes. Pages 498-510 in A. Calhoun, ed. Inland fisheries management. Calif. Dep Fish Game, Sacramento, CA.
- M'Gonigle, R.H., and M.W. Smith. 1938. Cobequid hatchery - fish production in Second River, and a new method of disease control. Prog Fish-Cult. 38:5-11.
- Midwest Research Institute. 1980. Subchronic oral dosing study for safety evaluation of rotenone using dogs. Summary statement, U.S. Fish Wildl. Serv. Contract 14-16-009-79-115. Midwest Research Institute, Kansas City, MO. 1 pp.
- Mires, J.M., R.A. Soltero, and G.R. Keizur. 1981. Changes in the zooplankton community of Medical Lake, WA, subsequent to its restoration by a whole-lake alum treatment and the establishment of a trout fishery. J. Freshwater Ecol. 1:167-178.
- Mongillo, P.E. 1983. Lake and stream rehabilitation statewide. Wash. Dep. Wildl., Progress Rep., Project F39D-25 (1982-1983). Fish Manage. Div. Rep. 83-7.
- Moretti, C., and P. Grenand. 1982. Les nivrees ou plantes ichtyotoxiques de la Guyane Francaise. (in French). J. Ethnopharmacol. 6(2):139-160.
- Moyle, J.B. 1968. Notes on some characteristics of Minnesota lakes having blue-green algal blooms. Minn. Dep. Conserv. Spec. Publ. 52.
- Municipality of Metropolitan Seattle. 1981. Pine Lake restoration analysis. Final Environmental Impact Statement. 43 pp.
- National Academy of Science. 1983. Rotenone. Pages 63-70. in Drinking water and health, volume 5. Safe Drinking Water Committee, Board of Toxicology and Environmental Health Hazards, Commission on Life Sciences, National Research Council, National Academy Press, Washington, D.C.
- Needham, R.G. 1966. Effects of toxaphene on plankton and aquatic invertebrates in North Dakota lakes. U.S. Fish Wildl. Serv. Invest. Fish Control 4. 16 pp.
- Negherbon, W.O. 1959. Handbook of toxicology. Vol. III: insecticides. W.B. Saunders Co., Philadelphia and London. 854 pp. referred to in Schnick, 1974.
- Neves, R.J. 1975. Zooplankton recolonization of a lake cove treated with rotenone. Trans. Am. Fish. Soc. 104:390-393.

- Oberg, K.E. 1959. The structure of gill epithelium and the circulation in gills of fishes poisoned with rotenone. Ark. Zool. 12(25):383-386.
- Oberg, K.E. 1961. Site of action of rotenone in the respiration chain. Exp. Cell. Res. 24:163-164.
- Oberg, K.E. 1964. the inhibition of the respiration of the brain mitochondria of rotenone-poisoned fish. Exp. Cell Res. 36:407-410.
- Oberg, K.E. 1967a. On the principla way of attack of rotenone in fish. Ark. Zool. 18(1):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. Exp. Cell. Res. 45:590-602.
- Olson, D.E., and L.J. Koopman. 1976. Interactions of three species of bullheads (under commercial exploitation) and associated panfishes in a eutrophic lake. Minn. Dep. Nat. Resour. Sect. Fish. Invest. Rep. 337. 87 pp.
- Olson, L.E., and L.L. Marking. 1975. Toxicity of four toxicants to green eggs of salmonids. Prog. Fish-Cult. 37(3):143-147.
- Orciari, R.D. 1979. Rotenone resistance of golden shiners from a periodically reclaimed pond. Trans. Am. Fish. Soc. 108(6):641-645.
- Parker, R.O., Jr. 1970. Surfacing of dead fish following application of rotenone. Trans. Am. Fish. Soc. 99:805-807.
- Penick and Co. 1959. An interim report on the toxicity of Pro-Noxfish. S.B. Penick and Co., New York. Unpublished report. 32 pp.
- Perkins, M.A. 1982. Limnological characteristics of Green Lake. Draft report, phase I restoration analysis. URS Company, Seattle, WA 92 pp.
- Pfeifer, R.L. 1985. Potassium permanganate detoxification following lake rehabilitation: procedures, costs, and two case histories. Wash. Dep. Wildl., Fish Manage. Div.
- Porter, K.G. 1973. Selective grazing and differential digestion of algae by zooplankton. Nature 244:179-180.
- Post, G. 1958. Time versus water temperature in rotenone dissipation. Proc. Western Assoc. Game Fish Comm. 38:279-284.

- Prevost, G. 1960. Use of fish toxicants in the province of Quebec. *Can. Fish Cult.* 28:13-35.
- Price, R.W., and D.R. Calsetta. 1957. Pro-Noxfish: a new synergized rotenone formulation for fish control. *Proc. Annu. Conf. Southeast. Assoc. Game Fish Comm.* 10:68-75.
- Ricker, W.E., and J. Gottschalk. 1940. An experiment in removing coarse fish from a lake. *Trans. Am. Fish. Soc.* 70:382-390.
- Santi, R. and C.E. Toth. 1965. Toxicology of rotenone. *Farm. Ed. Sci.* 20:270-279.
- Schindler, D.W. 1974. Eutrophication and recovery of experimental lakes: implications for lake management. *Science* 184:897-899.
- Schindler, D.W., and G.W. Comita. 1972. The dependence of primary production upon physical and chemical factors in a small, senescing lake, including the effects of complete winter oxygen depletion. *Arch. Hydrobiol.* 69:413-415.
- Schindler, D.W., and E.J. Fee. 1974. Experimental lakes area, whole lake experiments in eutrophication. *J. Fish. Res. Board Can.* 31:937-953.
- Schmidt, D.C., and L.J. Weber. 1975. The effect of surgical impairment of biliary excretion on the toxicity of rotenone to trout and rats. *Gen. Pharmacol.* 6:229-233.
- Schnick, R.A. 1974. A review of the literature on the use of rotenone in fisheries. U.S. Fish Wildl. Serv. Rep. LR-74-15. (NTIS microfilm PG-235-454). 130 pp.
- Scholz, A.T. 1983. The seasonal distribution and aggregation behavior of goldfish (*Carassius auratus* L.) in eastern Washington lakes: new technology for control of goldfish populations based on their behavioral ecology. Washington State University., Water Res. Center and U.S. Dep. Interior Project A-112-WASH, completion rep. 21pp.
- Serns, S.L. 1979. Effects of Pro-Noxfish on the benthos and zooplankton of Bug Lake, Forest County, Wisconsin. *Water Resour. Bull.* 15(5):1385-1393.
- Serns, S.L., and M.H. Hoff. 1984. Food habits of adult yellow perch and smallmouth bass in Nebish Lake, Wisconsin, with special reference to zooplankton density and composition. *Wis. Dep. Nat. Resour. Tech. Bull.* 149. 24 pp.

- Shannon, E.H. 1969. The toxicity and detoxification of the rotenone formulations used in fish management. N. Carolina Wildl. Resour. Comm., Div. Inland Fish. Annual Progress Rep., Project F-19, Study IV, Job IV-B. 9 pp.
- Shapiro, J. 1970. A statement on phosphorous. J. Water Pollut. Control Fed. 42(5):772-775.
- Shapiro, J. 1973. Blue-green algae: Why they become dominant. Science 179:382-384.
- Shapiro, J. 1979. The importance of trophic level interactions to the abundance and species composition of algae in lakes. SIL Workshop on Hypertrophic Ecosystems, Vaxjo, Sweden, Sept 10-14, 1979.
- Shapiro, J. 1982. Effect of rotenone in Wirth Lake. Pages 195-207 in Shapiro, et al. 1982.
- Shapiro, J., B. Forsberg, V. Lamarra, G. Lindmark, M. Lynch, E. Smeltzer, and G. Zoto. 1982. Experiments and experiences in biomanipulation: studies of biological ways to reduce algal abundance and eliminate blue-greens. U.S. Environ. Protection Agency, Corvallis Environmental Research Lab, Rep. 600/3-82-096. 250 pp.
- Shapiro, J., and E. Smeltzer. 1982. The effects of fish toxicants on phytoplankton: file data. Pages 190-194 in Shapiro et al., 1982.
- Smeltzer, E. 1982. The influence of fish on the abundance of algae in Clear Lake, Minnesota. Pages 208-209 in Shapiro et al., 1982.
- Smeltzer, E., and J. Shapiro. 1982. Biological effects on the size of the nutrient pool. The role of benthivores in Lake Marion, Minnesota. Pages 12-29 in Shapiro et al., 1982.
- Smith, C.L. 1973. Small rotenone stations: a tool for studying coral reef fish communities. Am. Mus. Novit. 2512:1-21.
- Smith, M.W. 1940. Copper sulphate and rotenone as fish poisons. Trans. Am. Fish. Soc. 69:141-157.
- Smith, M.W. 1941. Treatment of Potter's Lake, New Brunswick, with rotenone. Trans. Am. Fish. Soc. 70:347-355.
- Soltero, R.A., D.G. Nichols, A.F. Gasperino, and M.A. Beckwith. 1981. Lake restoration: Medical Lake, Washington. J. Freshwater Ecol. 1(2):155-165.

- Sorokin, J.I. 1968. the use of ^{14}C in the study of nutrition of aquatic animals. Mitt. Internat. Verein. Limnol. 16. 41 pp.
- Spitler, R.J. 1970. An analysis of rotenone treatments for elimination of fish populations in southern Michigan lakes, 1957-1967. Mich. Acad. 3(1):77-82.
- Sprague, J.W. 1961. Report of fisheries investigations during the seventh year of impoundment of Fort Randall Reservoir, South Dakota, 1959. South Dakota D-J Project F-1-R-9, Job Nos. 5,6,7, and 8.
- State of California. 1983. An assessment of the use of chemical fish toxicants in California. Reour. Agency, Dep. Fish Game, Inland Fish. Admin. Rep. 83-2. 21 pp.
- State of California. 1985. Draft Programmatic Environmental Impact report: Rotenone use for fisheries management. Resour. Agency, Dep. Fish Game. 186 pp.
- Stebbins, R.C. 1966. A field guide to western reptiles and amphibians. Houghton Mifflin Company, Boston, MA. 279 pp.
- Stecher, P.G., ed. 1952,1960,1968,1976. The Merck Index of chemicals and drugs, 6th, 7th, 8th, and 9th eds. Merck and Co., Inc., Rahway, N.J.
- Stenson, J.A.E., T. Bohlin, L. Henrickson, B.I. Nilsson, H.G. Nyman, H.G. Oscarson, and P. Larsson. 1978. Effects of fish removal from a small lake. Verh. Internat. Verein. Limnol. 20:794-801.
- Straskraba, M., and V. Straskraba. 1969. Eastern European lakes. Pages 65-97 in Eutrophication: causes, consequences, correctives. Proceedings of a symposium. Nat. Acad. Sciences, WA, D.C. 661 pp.
- Stroud, R.H. 1956. Progressive attitude. Bull. Sport Fish. Inst. 51:4.
- Swan, R.B. 1965. Fall Creek Reservoir: pre-impoundment chemical treatment to remove undesirable fish. Completion report, U.S. Army Corps Eng. Contract DA-35-026-CIVENG-66-19, No. 17. Oregon State Game Comm.
- Tanner, H.A., and M.L. Hayes. 1955. Evaluation of toxaphene as a fish poison. CO Coop. Fish. Res. Unit Quart. Rep. 1:31-39.
- Taube, C.M., K.G. Fukano, and F.F. Hooper. 1954. Further studies on the use of fish poisons in Michigan lakes. Mich. Acad. Sci., Arts, Letters, Rep. 1414:1-29.

- Teixeira, J.R., A.J. Lapa, C. Souccar, and J.R. Valle. 1984. Timbos: ichthyotoxic plants used by Brazilian Indians. *J. Ethnopharmacol.* 10:311-318.
- Thienes, C.H., and T.J. Haley. 1972. *Clinical toxicology.* Lea and Febiger, Philadelphia. 459 pp.
- Tischler, N. 1935. Studies on how derris kills insects. *J. Econ. Entomol.* 28:215-220.
- Tucker, R.K., and D.G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. U.S. Bureau Sport Fish. Wildl., Resour. Publ. 84. 131 pp.
- Tuunainen, P. 1970. Relations between the benthic fauna and two species of trout in some small Finnish lakes treated with rotenone. *Ann. Zool. Fenn.* 7:67-120.
- U.S. Environmental Protection Agency. 1970. Water quality criteria data book, vol. 1: organic chemical pollutants of freshwater.
- U.S. Environmental Protection Agency. 1981. Completion of pre-RPAR review of rotenone. Agency memorandum from Marcia Williams (Director, Special Pesticide Review Division) to Douglas Campt (Director, Registration Division), June 22, 1981.
- 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. Dep. Interior, Fed. Water Pollut. Control Admin., WA, D.C. 234 pp.
- Vinogradov, A.P. 1953. The elementary chemical composition of marine organisms. Mem. Sears Foundation for Marine Research, Yale University, New Haven, CN 647 pp.
- Vollenweider, R.A. 1968. The scientific basis of lake and stream eutrophication with particular reference to phosphorus and nitrogen as eutrophication factors. *Organ. Econ. Coop. Dev., Tech. Rep.* DAS/SCR/68:27.
- Walters, C.J., and R.E. Vincent. 1973. Potential productivity of an alpine lake as indicated by removal and reintroduction of fish. *Trans. Am. Fish. Soc.* 102(4):675-697.
- Washington Department of Wildlife. 1968. Rainbow fingerling survival estimates - 1968. Wash. Dep. Wildl. App. Res. Div., mimeo. 1 pp.

- Washington Department of Wildlife. 1981. Lake and stream rehabilitation statewide. Wash. Dep. Wildl. Fish. Manage. Div. Rep. 81-21.
- Washington Department of Wildlife. 1983. Draft Environmental Impact Statement, Lake Rehabilitation Program 1983-84. 136 pp.
- Washington Department of Wildlife. 1984. Supplemental Draft Environmental Impact Statement, Lake Rehabilitation Program 1984-85. 52 pp.
- Watkins, W.D., and D.C. Tarter. 1974. Acute toxicity of rotenone on the anial stage of the dragonfly. Proc. W. VA Acad. Sci. 46:141-145.
- 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. J. Wildl. Manage. 14(2):203-205.
- Welch, E.B. 1980. Ecological effects of waste water. Cambridge University Press, Cambridge. 337 pp.
- Welch, E.B., M.A. Perkins, G. Pelletier, and R.R. Zisette. 1981. The trophic state and phosphorus budget of Pine Lake, Washington. Municipality of Metropolitan Seattle. Seattle, WA. 96 pp.
- Wetzel, R.G. 1983. Limnology, 2nd ed. Saunders College Publishing, Philadelphia. 767 pp.
- Whitaker, J.O., Jr. 1980. The Audobon Society field guide to North American mammals. Alfred A. Knopf, New York. 745 pp.
- Wilhm, J.L., and T.C. Dorris. 1968. Biological parameters for water quality criteria. Bioscience. 18:477-481.
- Windholz, M., ed. 1983. The Merck Index of chemicals and drugs, 10th ed. Merck and Co., Inc., Rahway, N.J. 2179 pp.
- Wollitz, R.E. 1962. Effects of certain commercial fish toxicants on the limnology of three cold-water ponds, Montana. Proc. Mont. Acad. Sci. 22:54-81.
- Wrenn, W.B. 1965. Effect of removal of the fish population on the invertebrate fauna and phytoplankton of Emmaline Lake, Colorado. M.S. Thesis. Colorado State University, Fort Collins, CO. 40 pp.
- 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, AL 47 pp.

- Wydoski, R.S., and R.R. Whitney. 1979. Inland fishes of Washington. University of Washington Press, Seattle, WA 220 pp.
- Zicker, E.L., K.C. Berger, and A.D. Hasler. 1956. Phosphorus release from bog lake muds. *Limnol. Oceanogr.* 1:296-303.
- Zilliox, R.G., and M. Pfeiffer. 1956. Restoration of brook trout fishing in a chain of connected waters. *N.Y. Fish Game J.* 3(2):167-190.
- Zilliox, R.G., and M. Pfeiffer. 1960. The use of rotenone for management of New York trout waters. *Can. Fish. Cult.* 28:3-12.
- Zischkale, M. 1952. Effects of rotenone and some common herbicides on fish-food organisms. *Field and Laboratory* 20:18-24.
- Zisette, R.R. 1981. Prerestoration phytoplankton dynamics in Pine Lake. M.S. Thesis. University of Washington, Seattle, WA 91 pp.
- Zook, W.J. 1978. Warm water fisheries research in Washington State - 1978. Wash. Dep. Wildl., annual report submitted to U.S. Fish Wildl. Serv., Project F-68-R-3. 223 pp.

APPENDIX E
LAKES PROPOSED FOR REHABILITATION
1992

Quincy and Burke Lake Management Plans

Waters: Quincy and Burke Lakes

Location: Quincy Wildlife Area, Secs. 14 and 15, T19N, R23E, approximately five miles SW of Quincy, Grant County, Washington

Size: 62 and 57 surface acres, respectively

Maximum Depth: 26 and 27 feet, respectively

Water Source: Subsurface seep springs

Outflow: Westerly several hundred feet to disappear into basaltic fissures and rubble

Management History: Have been managed as trout waters for about 30 years. Annual stocking approximately 30,000 fry at 80 - 100 per pound. Stocking density averaged 500 - 550 fish per surface acre. Season originally opened in April and closed the end of September and produced opening day harvests of 50,000 trout of 11-inch size. This high early harvest made for a rapid fishout and an effective or productive season of about two weeks. The first chemical rehabilitations on these lakes occurred in 1966. Since that time the lakes have been subject to repeated illegal introductions of perch, pumpkinseed sunfish, largemouth bass and crappie. Tui chub have also been a problem in Quincy.

Rehabilitation was carried out as soon after presence of an unwanted species was detected as possible. Quincy Lake has been rehabilitated four times and Burke five times.

Serious upland habitat damage, litter accumulation and fire hazard resulted from the hundreds of campers and day-trippers visiting these waters in the usually sunny April days. In 1983 the season opening was changed to March 1 with a limit reduction to five fish and a subsequent earlier closing date of July 31.

Largely because of adverse weather and ice on the lakes, opening day crowds diminished and catch rates became highly variable. About 50 percent of the years finds ice still on during the March opener. Size of fish diminished as a consequence of shortened growing time. An earlier spawning rainbow stock (Goldendale) was used to partially compensate, i.e., obtain an average size of 10 - 10.5 inches.

Current Management Objectives: Manage as a trout-only water with a five fish limit and a season of March 1 - July 31. Provide an average harvest of 3 - 4 fish per person on opening day, realizing weather and ice conditions will cause some yearly variation in catch/effort. Stocking will continue at about 500 fish per acre, or 30,000 fry annually in each water. Maintain a carryover harvest of 13 - 17 inch rainbows that comprises 5 - 10 percent of the opening day catch. Monitor opening day effort and harvest as per the Brown Model. Check randomly for fishing success after the opening week. Sample prior to opening to estimate relative survival rate as weather conditions permit.

Sample also once each year for presence of non-trout species. Continue rehabilitations with rotenone as soon as possible after detection of unwanted fish species.

PREREHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

Burke and Quincy lakes are statewide resources. A WDW survey during the 1981-82 seasons indicated only 18% of those fishing Burke Lake were from Grant County. Over 45% were from western counties. A similar survey conducted in 1992 for all March 1 Opening Day waters indicated 20% participation from Grant County anglers and 47% of the total residing on the westside. Biologists in 1983 conservatively estimated 9,000 angler trips per season on Burke Lake, contrasted with an average estimate of 200 angler days on comparable warmwater lakes. Data collected at Quincy Lake in 1988 provides an estimate of over 6,400 angler trips per season.

After the 1977 rehab of Burke Lake, fish/angler reached a high of 8.8, declining to 1.1 just before the 1983 rehab. Catch limits on Burke were reduced to 5 fish per day in 1984. Catches after 1983 peaked out at 4.6 fish/angler and declined to 0.8 before the 1987 rehab. The Burke Lake rainbow fishery failed completely in 1991; no trout were checked the entire season. Quincy Lake was last rehabed in 1986 and catches peaked at 4.2 fish/angler in 1988. Only 1.7 fish/angler were recorded spring 1991 opening day. The 1992 fishery for both waters depended completely on catchables due to the complete failure of the fry plant.

Dr. Jim Walton and students from Peninsula State College investigated the fish populations of Burke Lake in 1991. The bulk of fish biomass was found to be yellow perch and pumpkinseed sunfish. Yellow perch made up 68.5% of the catch and the population was dominated by 2-3 year old fish. Sunfish comprised 31.5% of the total, and 2-4 year old fish dominated. Because of the weeds, collections of 0+ and 1+ fishes were difficult, and these age classes are probably vastly underestimated in the sample. Both species exhibited better than average growth their first year and less than average growth in later years. Of over 9,000 fish captured by a variety of methods, only three trout and one bass were taken. The study concluded that perch and sunfish were over abundant and too small to provide a fishery.

Gill net samples taken 1991 in Quincy Lake indicated largemouth bass were the most prevalent species in the lake, and 4-5 year old fish most abundant. Perch and bluegill were also present with 4 year olds again the dominant age class. No rainbow trout were captured. Growth for the bass and bluegill was average or better than average. Perch growth for the 4 year olds was slightly less than average.

Opening Day Catch and Effort Estimates:

Year	Angler Trips	Total Catch	Catch Hour	Catch Angler	Hours/Trip	Yrly Size	Comments
<u>Quincy Lake -</u>							
1984			3.2		11.1		
1985			3.0		12.3		80 % Iced
1986							Spring Rehab
1987			3.5		9.5		Illegal Plant?
1988	1344	5647	3.6	4.2	1.9	11.2	
1989	104	219	1.3	2.1	1.6		Ice, Very Cold
1990			0.9	1.6		9.0	
1991			0.6	1.7		11.5	
1992	520	116	0.1	0.2	3.0	10.5	Catchables Planted
<u>Burke Lake -</u>							
1984			4.6		10.6		
1985			1.5		12.3		80 % Iced
1986			1.7		10.0		
1987	496	406	0.3	0.8	2.6	9.5	Fall Rehab
1988	254	131	0.3	0.5	1.8	8.2	Catchables Planted
1989	150	260	1.4	1.7	1.3		Illegal Plant?
1990	352		454	0.4	1.3	3.5	Ice, Very Cold
1991	88	0		0	0	2.3	0 24 Checks
1992	781	1263	0.6	1.6	2.9	9.5	Catchables Planted

B. Physical Description of Water Proposed for Rehabilitation

1. Quincy Lake, Grant County
 2. Sec 14 & 15, T19N R23E
 3. 62
 4. 26 ft.
 5. 1,813,002,048 lbs.
 6. Permanent
 7. Miles: N/A, Flow: (cfs) N/A
 8. Entire Lake
 9. Public 100%, Private 0%
 10. None
-
1. Burke Lake, Grant Co.
 2. Sec 14,15,23 T19N R23E
 3. 57
 4. 27 ft.
 5. 1,791,256,000 lbs.
 6. Permanent
 7. Miles: N/A, Flow: (cfs) N/A
 8. Entire Lake
 9. Public 100%, Private 0%
 10. None

C. Proposed Management Actions

1. Quincy Lake
2. Largemouth bass, yellow perch, bluegill
3. March 1986
4. March 1993
5. Feb.-May '93
6. Rainbow trout

7. Catchables: 10,000, fry: 30,000
8. Rotenone, powder and liquid, 4 ppm
(Rotenone at 5% act. ingred.): 7,200 lbs., 30 gal.
9. Tow sack or slurry and spray
10. Jeff Korth (leader) and six to ten personnel

1. Burke Lake
2. Yellow perch, pumpkinseed sunfish, largemouth bass
3. October 1987
4. March 1993
5. Feb.-May '93
6. Rainbow trout
7. Catchables: 10,000, fry: 30,000
8. Rotenone, powder and liquid, 4 ppm
(Rotenone at 5% act. ingred.): 5,400 lbs., 30 gal.
9. Tow sack or slurry and spray
10. Jeff Korth (leader) and six to ten personnel

II. PURPOSE

Quincy and Burke lakes are the middle two of four adjacent waters. They have been managed as trout fisheries since the mid-fifties and continue to be popular opening day fisheries. The two lakes north and south (Stan Coffin and Evergreen Reservoir) are managed as warmwater fisheries. The greatest complicating factor in the management of Quincy and Burke lakes is recurring illegal introductions of yellow perch, largemouth bass, black crappie, bluegill and pumpkinseed sunfish. Both lakes have a long and colorful history of public involvement in management. The Department was actually brought to court in 1983 by several Quincy area sportsmen over the planned rehabilitation of Burke Lake. WDW prevailed, and the following excerpt from testimony still applies today:

"There are 20 waters around the Quincy area. Thirteen are trout fisheries (190.2 surface acres) and seven are warm water fisheries (341.6 surface acres). Four lakes of 61 acres in surface area are located within one mile of Burke Lake. These four lakes, Coffin, "H", Judith Pool and Ancient, are managed for warm water fishing. Burke Lake is 57 surface acres in size and is an acceptable candidate for lake rehabilitation.

"Burke Lake's inlet flows are intermittent and seepage in origin, isolating the lake from any recurring contamination of unwanted fish species. The outlet is short and flows spill over a natural impassable barrier to upstream movement of any unwanted fish species. Very little marsh exists and submergent weedy areas are minimal in the spring months....

"Burke Lake has been managed for trout since 1955. Yellow perch suddenly appeared for the first time in 1964. The 1966 treatment removed the perch successfully, since none were present in the 1970 treatment. Nevertheless in 1967, after the complete kill in 1966, different species, largemouth bass and pumpkinseed sunfish, suddenly appeared for the first time. After the 1970 treatment, the perch, bass and sunfish suddenly reappeared. The 1975 treatment removed the perch successfully since none were present in the 1977 treatment. Nevertheless, the bass and sunfish suddenly reappeared after the 1975 treatment and had to be removed by the next treatment in 1977. Once again all these species are now present. And, for the first time, black crappie have appeared. All of these species are rather readily eliminated with low concentrations of rotenone.

"Furthermore, Columbia Basin lakes do not naturally repopulate with perch, bass, sunfish and crappie without a trace of other fish species which are more likely to occur, such as carp, bullheads, tui chub, suckers, and cottids. Yet this lake has repopulated without these other species which also are not desirable for warm water fishermen.

"It is unlawful to plant any fish species without authorization from the Game Department. See RCW 77.16.150. The Department has never authorized the planting of the above mentioned warm water species in Burke Lake. This rehabilitation history makes it clear that unknown and unauthorized parties have continued to illegally plant the lake...."

Jackson Affidavit
Office of the Attorney General
Temple of Justice
Olympia, WA 98504

These lakes have a long history of being managed for trout and together account for approximately 15,000 angling trips per season when trout fishing is in its prime. A warmwater fishery will not create the same amount of recreation. The cost for producing a mixed species fishery is an order of magnitude greater for the larger trout necessary to compete with other species and will not produce the same quality fishery that trout only management can achieve. Proposed WDW policy states that lake rehabilitation is an option for eliminating illegally planted fish.

III. INTENDED OUTCOME/MEASURE OF SUCCESS

We intend to restore Quincy and Burke lakes to their former glory as trout only waters. Success of this measure will be apparent during annual creel surveys. Given a reasonable chance of eliminating the undesirable species and provided further illegal plants are curtailed, the beneficial effects should be everlasting. Aside from reasons listed under Resource, Recreational and Economic Impacts, to abandon these lakes as trout fisheries is to invite other incursions across the state.

IV. RESOURCE IMPACTS

1. Regional Habitat, Wildlife and Non-Game biologists have been appraised of our rehabilitation plans. No concerns were expressed on the potential impacts to non-targeted species for Quincy or Burke Lakes. Impacts to nontargeted resources in the lake are consistent with those covered in the Programmatic Environmental Impact Statement. The effects of rotenone on benthos are variable, depending on the concentrations and species. Crustaceans are most tolerant while the smaller insects are most affected. Immediate reduction of populations averages 25%, and survival doubles when access to bottom sediments exists. Benthic communities generally recover to at least pretreatment levels within two months. Zooplankton is more severely impacted, and communities generally take two to twelve months to fully recover. While relatively tolerant of even heavy doses of rotenone, amphibians (especially larval) and herptiles are at risk. However, the chances of eliminating the entire population are minimal.
2. Quincy and Burke Lakes and their outflows are not used for domestic water, livestock watering, irrigation or skiing and related water sports. Swimming may occur infrequently during summer, but not during fall or spring when rehabilitations occur. A fishery is the primary use and the lakes support occasional waterfowl hunting in late fall.

Loss of the popular early spring fishery is likely during 1993 as the lakes will probably not detoxify in time for planting catchables. The existing warmwater fishery created by illegal planting will be eliminated.
3. No known endemic, rare, threatened or otherwise listed species will be impacted by the rehabilitation.

V. MITIGATING FOR ADVERSE IMPACTS

1. Trout fry survival and growth will be greatly enhanced, and future trout fisheries will attain their previous status. Catchable trout will be planted later in the spring to provide angling for the remainder of the season. Bluegill and largemouth bass will be saved, if possible, for replanting in other area lakes. The 1992 season will also be extended to provide greater opportunity for the harvest of the target species.

There will be no measurable impacts to waterfowl and waterfowl hunting. For a few weeks after the early spring rehab, invertebrate densities will be low, especially for zooplankton. This will have some adverse impacts to predatory invertebrates and ultimately to some wetland birds. Literature on this aspect and our own experience on these lakes and other nearby waters have shown little measurable changes in aquatic and terrestrial fauna beyond a few weeks. No mitigation for these impacts is deemed necessary as recovery is always rapid.
2. Downstream resources will also be treated as they may harbor remnants of the target populations. Those waters downstream not to be treated are protected by subterranean flows.

3. N/A
4. Protective wear for the eyes, face and hands will be supplied on-site for all purveyors of rotenone.
5. Lakes will be posted to discourage the public from consuming dead fish. In addition, enforcement agents will be on hand the day of the treatment to control public access and inform the public of the Department's action.

VI. RECREATIONAL IMPACT: ALSO SEE PROPOSAL I.A.

Fry plants are no longer an option for either Quincy or Burke, thus the only valid comparisons are with a warmwater or a mixed species fishery. If both lakes produced good warmwater fisheries (Burke has already overpopulated and produces no appreciable warmwater fishery), 400-500 trips per season are estimated. This is roughly 3 % of the 15,000 trips per season produced by a good trout only fishery. The results of catchable fish plants are so variable that angler interest will probably wane somewhat. Catchables planted for the 1992 opener produced roughly one third to one half the trips anticipated when fry are planted in competition free waters.

VII. ECONOMIC IMPACTS:

Using angler days estimated for Burke Lake and the 1983 WDG estimate of \$31.71 generated per angler day, that fishery had an annual value of \$285,390 to the state's economy. Quincy Lake estimates for 1988 of 6,400 trips produces \$202,944. Current estimates would be higher. The fishery as it now exists generates far less as participation decreases with the declining trout catch. Rehabilitation would bring back the fishery and associated economic activity.

Current total costs to Columbia Basin Hatchery to plant both lakes with 30,000 fry each is less than \$4,800. The cost of planting with advanced fry or legal, which are necessary to compete in a mixed species water, would exceed \$14,800. These rehabilitations will cost the Department conservatively \$20,000 (including time, travel, etc.). If rehabilitations continue to occur every four years, the cost of fry plants (4 yrs.), one catchable plant (optional-sustains the fishery on a rehab year), and the rehab totals \$54,000. Provided illegal activity does not resume and further rehabilitations are no longer necessary, the cost to manage for the same four year period equals \$19,200 for fish plus the cost of an opening day creel survey and analysis.

Maintaining a mixed species fishery and planting advanced fry (planted in the fall, rather than spring) every year for four years costs \$44,600, with as yet unknown results. Planting catchables every year for four years costs \$66,700 for fish alone. Hatchery space and water are fully utilized in accomplishing the current area program, and other waters would suffer cutbacks if greater numbers of larger fish were to be raised. In addition, Department time and equipment dollars to manage this type of fishery may be considerable in the long term.

Estimates for the cost of the enforcement action necessary to curtail the activity of the individuals responsible are not available. However, this cost might be looked upon as a statewide expenditure since some preventive benefit would certainly occur as perpetrators find out the Department takes illegal transport and planting of fish very seriously.

VIII. RELATED MANAGEMENT ACTION

See I.C.6. for fish planting data

Emergency regulation changes should be enacted to lengthen the season (currently March 1 to July 31). A season extension will allow increased harvest of available fish. Recommend until October 1, 1992. These waters will also be closed to the taking of fish for the period of the rehabilitation.

We are also proposing rehabilitation of nearby warmwater fisheries (see Coffin, H, Ancient Lakes proposal). Fish salvaged from Quincy will be used to repopulate these waters. Hopefully, we will convince the public that WDW is sincere in our commitment to manage area waters for both warmwater and trout fisheries.

Dr. Jim Walton and his students have conducted a thorough study of Burke Lake during 1991 and intends a follow-up after the rehabilitation. Region Two will investigate Quincy Lake. We intend to treat both lakes in two stages, approximately one month apart, to assure ourselves, once again, of a complete kill.

Increased penalties and enforcement activities are desirable if WDW is ever going to dissuade illegal plantings of state managed waters. Education of the public as to the costs in Department dollars and time, emphasis on what WDW might be able to accomplish with those resources, is advisable. This might help in terms of stemming recruitment to this ill advised group and turning local opinion against the offenders. This type of action would be a very worthwhile activity for I & E.

IX. PUBLIC CONTACT

With approximately 80% of the lake's users living outside Grant County, actual percentages pro and con are difficult to obtain. Public support may be best judged by the number of participants in the fishery (vis a vis Recreational Impacts).

Anglers at Quincy and Burke Lakes were queried as to being in favor of or against plans to rehab those lakes. Of the total, 71% were in favor of rehabilitation. Among anglers from the westside, 82% were in favor, while only 63% of eastside anglers favored keeping Quincy and Burke trout only lakes. Grant Co. anglers were split almost 50-50. My observations indicated that those opposed to the rehab wanted these waters managed as mixed species lakes (i.e. trout plus spiny rays). Only 1% of the total were at these lakes to fish for species other trout. Additional creel survey days are planned for the summer to evaluate the warmwater anglers.

A public meeting was held May 21, 1992 in Ephrata to explain the rehabilitation plans for these waters and address local concerns. The meeting was announced in three area papers a week to three days in advance. Only three non-Department people attended, one being a newspaper reporter. Although many questions were raised, all in attendance seemed to understand and support the Departments plan, perhaps with some reservation. Either interest is lacking energy or the concerned parties have already decided their course of action. Proponents are probably the former and opponents the later.

Initiated by: Region Two Fisheries Management

c:\hueckel\quin-bur.rhb

Stan Coffin, "H", and Ancient Lake Management Plans

Waters: Stan Coffin, H and Ancient Lakes

Location: Quincy Wildlife Area, Secs. 9, 10, and 11, T19N, R23E, approximately five miles SW of Quincy, Grant County, WA

Size: 54, 8 and 30 surface acres, respectively

Maximum Depth: 20, 17 and 25 feet, respectively

Water Source: Subsurface seep springs

Outflow: Average 1 cfs from Stan Coffin draining into H Lake and in turn into one of the Ancient Lakes group. Two of the Ancient Lakes are isolated and fed by subsurface seep springs.

Management History: Over 20 years ago the system was used to divert excess water from irrigation canals to the Columbia River. Ancient Lake then was a single, large lake of several hundred acres in size. It has since drained via natural fissures in the basalt underlayment to four small ponds totalling about 30 acres.

In recent years, irrigation managers have not used the system for canal diversion, but still retain the right to spill canal water through the system in an emergency. Management has had to contend with entry of several species of fish coming in from the canals. Development of sustainable warmwater or trout fishery was never satisfactorily achieved. The potential became evident with cessation of irrigation spills and the system was first rehabilitated in 1984. The plan was to develop a fishery centered on largemouth bass and bluegill. Following the rehab, broodstock bass and bluegill were released into Stan Coffin, H and three of the four Ancient lakes. The fourth pond in the Ancient pod was too shallow to support a fishery. Regulations were enacted to protect the few mature bass and maintain a sizeable population of large predators to counteract population growth of carp and other species. A slot limit sought to protect bass between 12" and 17". The plan was not effective because carp and pumpkinseed sunfish survived the chemical treatment in high numbers and quickly repopulated the system.

Rainbow trout were also planted after the 1984 rehab and provided a good fishery until competition by other species made trout releases ineffective.

Current Management Objectives: Manage as a warmwater lake with emphasis on largemouth bass and bluegill under present regulations of a daily catch limit of five bass, not more than 3 over 15 inches in length, and no slot limit on length. Season length will be year around.

Rehabilitation in the fall of 1992 will be necessary to build a satisfactory warmwater fishery. Broodstock will be collected from Quincy Lake prior to a planned spring, 1993 rehab for Quincy. Trout fry will also be planted annually at a density of 200 - 300 fish per acre until bass and bluegill populations expand to attractive harvest levels. This approach will provide angling opportunity during the first few years. We hope to develop

informational signs outlining the management plan and install these at each lake. It is hoped this step will discourage people from restocking these and the Quincy/Burke group themselves. In addition, this group of lakes may likely require periodic, partial rehabilitations to maintain predator/prey balance and/or to reduce numbers of undesired species which might re-enter the system.

PREREHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

Stan Coffin, H, and Ancient Lakes lie north of Quincy and Burke lakes on the Quincy Wildlife Area. Until the late 1960's, this chain of lakes was used as a spillway for the irrigation project, rendering maintenance of a scrapfish-free fishery impossible. As of 1977, the Bureau of Reclamation agreed not to release water through this drainage, barring an emergency. Rehabilitation was attempted in 1978 at 1 ppm rotenone without success. In 1984, 2 ppm rotenone was applied, yet carp and possibly other undesirable species persist. This is probably due to the physical attributes of the system (shallow, suspended organics, water temperature, littoral zones, etc.) creating a need for higher than normal concentrations of rotenone to achieve toxic levels.

Stan Coffin and the associated lakes currently provide virtually no fishery due primarily to the presence of carp and to a lesser extent sunfish. Populations of perch are severely stunted, and very few bass large enough to entice anglers remain. Warmwater fisheries of this size should produce 200-300 angler trips per year. Currently, so few anglers fish these waters that no angler contacts could be made.

Past attempts at this management scheme have failed for reasons already explained, but this is not reason to discontinue our efforts. Discouraging results should only illustrate that we have more to learn. We propose to try once again to rehabilitate these waters, employing greater concentrations of rotenone and varying techniques. These lakes will be re-stocked with largemouth bass and bluegill. This proposal is closely associated with the proposed Quincy/Burke rehabilitation.

B. Physical Description of Water Proposed for Rehabilitation

1. Stan Coffin, Grant County
2. Sec 10 & 11, T19N R23E
3. 54
4. 20 ft.
5. 799,134,336 lbs.
6. Permanent
7. Miles: .125, flow: (cfs) N/A
8. Entire Lake
9. Public 100%, Private 0%
10. None

B. Physical Description of Water Proposed for Rehabilitation (con't)

1. "H" Lake, Grant County
2. Sec 10 T19N R23E
3. 8
4. 17 ft.
5. 220,016,906 lbs.
6. Permanent
7. Miles: N/A, Flow: (cfs) N/A
8. Entire Lake
9. Public 100%, Private 0%
10. None

1. Ancient Lakes, Grant County
2. Sec 9 T19N R23E
3. 30
4. 25 ft.
5. 1,223,164,800 lbs.
6. None
7. Miles: N/A, flow: (cfs) N/A
8. Entire Lake
9. Public 100%, Private 0%
10. None

C. Proposed Management Actions

1. Stan Coffin Lake
 2. Carp, yellow perch
 3. March 1984
 4. October 1992
 5. Apr.-Aug. '93
 6. Largemouth bass, bluegill
 7. Broodstock: 100, fry: 30,000
(dependent on capture and production success)
 8. Rotenone, powder and liquid, 4 ppm
(Rotenone at 5% act. ingred.): 3,200 lbs., 60 gal.
 9. Tow sack or slurry and spray
 10. Jeff Korth (leader) and six to ten personnel
-
1. "H" Lake
 2. Carp, yellow perch
 3. March 1984
 4. October 1992
 5. Apr.-Aug. '93
 6. Largemouth bass, bluegill
 7. Broodstock: 100, Fry: 30,000
(dependent on capture and production success)
 8. Rotenone, powder and liquid, 4 ppm
(Rotenone at 5% act. ingred.): 900 lbs., 15 gal.
 9. Tow sack or slurry and spray
 10. Jeff Korth (leader) and two to three personnel

C. Proposed Management Actions (con't)

1. Ancient Lakes
2. Carp, yellow perch, pumpkinseed sunfish
3. March 1984 4. PROPOSED TREATMENT DATE: October 1992
5. Apr.-Aug. '93
6. Largemouth bass, bluegill
7. Broodstock: 100, Fry: 30,000
(dependent on capture and production success)
8. Rotenone, powder and liquid, 4 ppm
(Rotenone at 5% act. ingred): 4,850 lbs., 15 gal.
9. Tow sack or slurry and spray
10. Jeff Korth (leader) and two to three personnel

II. PURPOSE

Removal of undesirable species and restocking with proper ratios of spinyray fishes is the best available option of managing these relatively small lakes for warmwater fisheries. Barring interference from over-zealous "helpers", these waters should produce fine warmwater fisheries.

III. INTENDED OUTCOME/MEASURE OF SUCCESS

The successful recovery of this warmwater fishery will be measured by random creel checks and biological sampling. Treatment will occur in two stages, to assure ourselves of a complete kill. The fishery will not actually reach its prime for 3-4 years and then continue to function for another 2-3 years before further management is required. The long term duration of beneficial effects will depend on how well Fisheries Management learns to manipulate these small warmwater fisheries, a new task for most of us.

If fish are available, we propose to stock rainbow trout for 3 - 4 years (fry origin) to provide an interim fishery until bass and bluegill reach harvestable numbers and size.

IV. RESOURCE IMPACTS

1. Regional Habitat, Wildlife and Non-Game biologists have been appraised of our rehabilitation plans. Non-Game found records of night heron nesting activity in the western marshes of Stan Coffin and was concerned for the disturbance of such during the rehab.

Impacts to nontargeted resources in the lake are consistent with those covered in the Programmatic Environmental Impact Statement. The effects of rotenone on benthos are variable, depending on the concentrations and species. Crustaceans are most tolerant while the smaller insects are most affected. Immediate reduction of populations averages 25%, and survival doubles when access to bottom sediments exists. Benthic communities generally recover to at least pretreatment levels within two months. Zooplankton is more severely impacted, and communities generally take two to twelve months to fully recover. While relatively tolerant of even heavy doses of rotenone, amphibians (especially larval) and herptiles are at risk. However, the chances of eliminating the entire population are minimal.

2. These lakes are not used for domestic water, or by livestock, or for irrigation. Fishing is the primary use. A small amount of waterfowl hunting occurs also.

Loss of spinyray fishes will occur, but these are not currently supporting much of a fishery due to either small size or lack of abundance, depending on the species.

3. No known endemic, rare, threatened or otherwise listed species will be impacted by the rehabilitation.

V. MITIGATING FOR ADVERSE IMPACTS

1. Both fry survival and growth of largemouth bass and bluegill populations will be greatly enhanced. Night heron nesting activity will be unaffected as rehabilitation will occur during the fall. There will be no measurable impacts to waterfowl and hunting. The same is true for other non-targeted terrestrial species which use this water during spring through fall. The lakes are frozen in winter. By spring, aquatic invertebrates will have returned to former, or even greater, abundance to provide forage for non-target wildlife.
2. Downstream resources will also be treated as they may harbor remnants of the target populations. Those waters downstream not to be treated are protected by subterranean flows.
3. N/A
4. Protective wear for the eyes, face and hands will be supplied on-site for all purveyors of rotenone.
5. Lakes will be posted to discourage the public from consuming dead fish. In addition, enforcement agents will be on hand the day of the treatment to control public access and inform the public of the Department's action.

VI. RECREATIONAL IMPACT: ALSO SEE PROPOSAL I.A.

Quantification of increased use is currently impossible due to lack of data. These waters should be capable of producing 200-300 angler trips per season on each water. Provided trout fry become available and are stocked as an interim fishery, several hundred more angler trips each year can be assured for the first 3 - 4 years.

VII. ECONOMIC IMPACTS:

Using the 1983 WDG estimate of \$31.71 generated per angler trip, these waters should have an annual value of \$6,000 to \$10,000 to the state's economy. Warmwater fisheries of this size do not favorably compete with trout fisheries in terms of angler days and subsequent revenue generation. However, the maintenance of a good warmwater fishery in this area is inextricably tied to the success of other area fisheries. Maintaining variety in angling opportunity will also serve to encourage maximum participation.

VIII. RELATED MANAGEMENT ACTION: See I.C.6.

Bluegill and largemouth bass will be saved, if possible, for replanting in other area lakes. The 1992 season will also be extended to provide greater opportunity for the harvest of non-target species. Recommend until October 1, 1992. These waters will also be closed to the taking of fish for the period of the rehabilitation.

We are also proposing rehabilitation of nearby trout fisheries (see Quincy and Burke Lakes proposals). Fish salvaged from Quincy will be used to repopulate these waters. Hopefully, we will convince the public that WDW is sincere in our commitment to manage area waters for both warmwater and trout fisheries.

While the merits of this project stand on their own, the timing of the proposal itself is a related management action. If approved, this will be the first time that both warmwater and trout lakes in the area will be included in a single rehabilitation project. The Department will be showing "good faith" in treating both fisheries equally, and hopefully proponents of both will learn something of the management endeavors necessary to each type of program.

The warmwater fishery will likely require periodic partial rehabs or other means to control overabundance and maintain age class composition. In the future, special regulations might also be employed if conditions warrant.

As for the Quincy/Burke proposal, enforcement and education related to illegal stocking activities is very desirable.

IX. PUBLIC CONTACT

Unlike the opening day trout waters, most of the users of this warmwater fishery are probably local (Grant County). Opposition to this particular part of the overall plan is not expected. Public support may be best judged by the number of participants in the fishery (vis a vis Recreational Impacts). Additional creel checks (random) are planned for the 1992 summer to evaluate the warmwater anglers.

A public meeting was held May 21, 1992 in Ephrata to explain the rehabilitation plans for these waters and address local concerns. The meeting was announced in three area papers a week to three days in advance. Only three non-Department people attended, one being a newspaper reporter. Although many questions were raised, all in attendance seemed to understand and support the Departments plan.

Initiated by: Region Two Fisheries Management

Upper Caliche Lake Management Plan

Water: Caliche Lake, Upper

Location: Quincy Wildlife Area, Sec. 27, T18N, R23E, approximately five miles west of George, Grant County, WA

Size: 21 surface acres

Maximum Depth: 25 feet

Water Source: Surface and subsurface seep springs

Outflow: Average 2 - 3 cfs draining West into Lower Caliche and West Caliche, to eventually disappear into the ground about one mile from Upper Caliche

Management History: A series of lakes (four in group) have been managed for trout since their formation by elevated ground water from irrigation development over 20 years ago. Irrigation diversions and/or periodic breaks in canal systems allowed carp to enter these waters several years ago and four rehabs, the first in 1975, have been unsuccessful in total eradication. Trout production has been very good and anglers use this small lake heavily with harvests generally high at about 4.5 fish per person on openers. Annual stocking densities have ranged as high as 850 rainbow fry per surface acre. In recent years stocking rates have dropped to 500 fish per acre. Growth has been excellent with fish averaging 11 inches on the opener. A fish barrier (permeable rock gabion) was installed on the dike built by WDW to form this lake, but has deteriorated to the extent that carp may be entering from Lower Caliche. Lower Caliche and the remaining waters in this drainage also supported good trout fisheries in their early years, but have not been managed for many years because of access by carp from private lands.

Current Management Objectives: Continue management of this popular water as a trout-only lake. Retain present season of March 1 - July 31 and daily catch limit of five trout per angler. Provide an average harvest of 3 - 4 fish per person on opening day, realizing weather and ice conditions will cause some yearly variation in catch/effort. Stocking will continue with 10,000 Goldendale rainbow fry at a density of 500 per acre. Maintain an a small carryover harvest of 13 - 15 inch rainbows that comprises 3 - 5 percent of the opening day harvest. Monitor opening day harvest and effort as per the Brown Model. Check randomly for fishing success after the opening week. Sample prior to opening to estimate relative survival of fry plant and growth/condition as weather permits. Sample once each year for presence of non-trout species. Continue rehabilitations with rotenone as soon as possible after detection of unwanted fish species. Reconstruction of the outlet fish barrier is currently funded and would be desired to prevent upstream migration of carp.

PREREHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

The persistence of carp in the Caliche Lakes has had a detrimental effect on the fishery. Rehabilitated in 1983, catch rates reached 4.9 fish per angler two years in a row in this 5 fish limit water before carp again flourished. Rehabilitation was again necessary in 1988. Low catch rates and unusually large yearlings in 1990 was the harbinger of bad news: poor fry survival. The 1991 catch rate reached 3.2 fish per angler, however yearling size was unusually small. Net samples taken that same summer indicated complete failure of the 1991 spring fry plant and a plethora of three year old carp. Failure of the outlet isolation barrier or an incomplete kill probably occurred immediately after the last rehab. The 1992 fishery was propped up with a catchable plant which yielded 4.0 fish per angler.

This March 1 opening day water is very popular because of its location near several communities and the easy accessibility. Catchables are usually planted whenever problems occur since large crowds are expected regardless of mediocre angling the previous year. Opening day 1992 provided an estimated 353 angler trips and 1,413 fish caught. By contrast, neighboring Lower Caliche, no longer planted and abandoned for the near future, had about 16 trips with 1 trout caught.

B. Physical Description of Water Proposed for Rehabilitation

1. Caliche Lake, Upper, Grant Co.
2. Sec 27, T18N R23E
3. 21
4. 25
5. 679,536,000 lbs
6. Permanent
7. .25 miles, Flow (cfs) 1.0
8. Entire Lake
9. Public 100%, Private 0%
10. None

C. Proposed Management Actions

1. Caliche Lake, Upper
2. Carp
3. October 1988
4. October 1992
5. April-May 1993
6. Rainbow trout
7. Catchables: 5,000 Fry: 10,000
8. Rotenone, powder and liquid, 4 ppm
(Rotenone at 5% act. ingred): 2,800 lbs., 90 gal.
9. Tow sack, spray
10. Jeff Korth (leader) and four to six personnel

II. PURPOSE

Upper Caliche Lake was once connected to the irrigation canals, whereby carp had established their minions in this water. Extensive marshes, springs and problems with isolation make this a difficult rehabilitation. Funding has been established for repairs to the outlet barrier and for the purchase of equipment which will render this rehabilitation more effective. An ATV with spray attachment will be used to treat the extensive marshes and pumps will be used to slurry powdered rotenone and reach the springs. Treatment will occur in two stages, at least two weeks apart, to assure ourselves of a complete kill.

III. INTENDED OUTCOME/MEASURE OF SUCCESS

This effort intends the restoration of the fry managed trout fishery for recreational as well as economic reasons. Annual creel surveys on opening day will be the measure of success. The complete elimination of carp from a system of this type is a challenge and certainly no certainty. Without a complete kill we can expect 3 - 5 years of good to excellent angling before rehabilitation is again necessary.

IV. RESOURCE IMPACTS

1. Regional Habitat, Wildlife and Non-Game biologists have been appraised of our rehabilitation plans. No concerns were expressed on the potential impacts to non-targeted species for Caliche Lakes. Impacts to non-targeted resources in the lake are consistent with those covered in the Programmatic Environmental Impact Statement. The effects of rotenone on benthos are variable, depending on the concentrations and species. Crustaceans are most tolerant while the smaller insects are most affected. Immediate reduction of populations averages 25%, and survival doubles when access to bottom sediments exists. Benthic communities generally recover to at least pretreatment levels within two months. Zooplankton is more severely impacted, and communities generally take two to twelve months to fully recover. While relatively tolerant of even heavy doses of rotenone, amphibians (especially larval) and herptiles are at risk. However, the chances of eliminating the entire population are minimal.
2. Loss of the opening day fishery will ensue unless funding and hatchery space are authorized for the rearing of catchable trout.
3. No known endemic, rare, threatened or otherwise listed species will be impacted by the rehabilitation.

V. MITIGATING FOR ADVERSE IMPACTS

1. Trout fry survival and growth will be greatly enhanced, and future trout fisheries will attain their previous status. Catchable trout will be planted, if possible, for opening day anglers and to provide recreation for the remainder of the season. The 1992 season will also be extended to provide greater opportunity for the harvest of the remaining trout.
2. Those waters downstream not to be treated currently are also plagued by the target species and are future rehabilitation candidates.
3. N/A
4. Protective wear for the eyes, face and hands will be supplied on-site for all purveyors of rotenone.
5. Lakes will be posted to discourage the public from consuming dead fish. In addition, enforcement agents will be on hand the day of the treatment to control public access and inform the public of the Department's action.

VI. RECREATIONAL IMPACT: ALSO SEE PROPOSAL I.A.

Fry plants are no longer an option for Caliche Lake. Catchable trout plants have so far lured anglers to this water in normal numbers, but the results of catchable fish plants are so variable that angler interest may eventually wane somewhat. Catchables are also usually smaller and considered by many anglers to be of poorer quality than lake reared fish. Catchables planted for the 1992 opener produced roughly the same number of trips anticipated when fry are planted in competition free waters.

VII. ECONOMIC IMPACTS

Using angler trips estimated for Caliche Lake and the 1983 WDG estimate of \$31.71 generated per angler trip, that fishery had an annual value of \$11,194 to the state's economy. Current estimates would be higher. The fishery as it now exists may eventually generate far less as participation decreases with the variable trout catch. Rehabilitation would ensure the associated economic activity.

The 10,000 fish fry plant currently costs the Columbia Basin Hatchery about \$800. The cost of planting catchables would exceed \$3,000. This rehabilitation will cost the Department approximately \$10,000 (including time, travel, etc.). If rehabilitations continue to occur every four years, the cost of fry plants (4 yrs.), one catchable plant (optional-sustains the fishery on a rehab year), and the rehab totals \$16,200. Planting catchables every year for four years costs \$12,400 for fish alone. This also assumes the Department is able to provide the facilities to raise catchables for this water every year. Provided a complete kill is achieved, and rehabilitations are no longer necessary, the cost to manage for the same four year period equals \$3,200 for fish plus the cost of an opening day creel survey and analysis.

VIII. RELATED MANAGEMENT ACTION

See I.C.6. for fish planting data

Emergency regulation changes should be enacted to lengthen the season (currently March 1 to July 31). A season extension will allow increased harvest of available fish. Recommend until October 1, 1992. These waters will also be closed to the taking of fish for the period of the rehabilitation.

If available, catchable trout will be planted prior to opening day.

The complete elimination of carp from this part of the system is essential to reclaiming the downstream resources. Depending on our success at Upper Caliche, Lower Caliche and possibly one other lake downstream would be proposed for rehabilitation in the future. These projects are also dependent on isolation structures and/or landowner cooperation.

IX. PUBLIC CONTACT

A public meeting was held May 21, 1992 in Ephrata to explain the rehabilitation plans for this water and address local concerns. The meeting was announced in three area papers a week to three days in advance. Only three non-Department people attended, one being a newspaper reporter. Although many questions were raised, all in attendance seemed to understand and support the Department's plan. With approximately 80% of the lake's users living outside Grant County, actual percentages pro and con are difficult to obtain. Public support may be best judged by the number of participants in the fishery (vis a vis Recreational Impacts).

Initiated by: Region Two Fisheries Management

c:\hueckel\caliche.rhb

BINGEN LAKE WETLANDS HABITAT IMPROVEMENT PROJECT

By The Columbia Gorge Audubon Society

The Columbia Gorge Audubon Society would like to present a plan for improving the wetland habitat of Bingen Lake, located along the Columbia River at Bingen, Washington. We propose to implement these ideas in conjunction with, and complementing, the Port of Klickitat's acceptance of Bonneville Spoils to raise the elevation of land surrounding the lake in preparation for development. We initiated this project at the invitation of the Port of Klickitat, and look forward to working together.

Wetlands and ponds are one of the planet's most productive and diverse ecosystems. Unfortunately, the US Fish and Wildlife Service has reported to Congress that 53% of the wetlands in the lower 48 states had been lost by 1990 (Dahl, 1990). Washington state lost 31% of its wetlands (Dahl, 1990). Although most of this loss has been attributed to draining and filling for agriculture and urban development, many remaining areas have been degraded by sedimentation and contamination (Turner 1990).

These losses place an ever greater importance on maintaining our existing wetlands, and ensuring that they provide high quality habitat for floral and faunal communities. We believe that project work at Bingen Lake offers us a unique chance to enhance the habitat quality of the lake and wetlands, while providing aesthetically pleasing educational and recreational opportunities for the public.

DESCRIPTION OF THE LAKE AND ITS USE

Bingen lake is located along the Columbia river at Bingen, Washington. The lake is approximately 20 acres, including the associated wetlands. The flora of the lake and a description of the method used to determine the wetland boundary are presented in a letter from IES Associates, (appendix A). The predominant emergent vegetation surrounding the lake are cattails, reed canarygrass, and willows.

The Columbia Gorge Audubon conducted a bimonthly waterfowl survey, from November 1990 through March 1991, to determine the winter usage of the lake. We discovered that the lake is used primarily by puddle ducks (mallards, green-winged teal, American widgeon, and wood ducks) and Canada geese, with some common mergansers, great blue heron, and coot. The vegetation surrounding the lake was used extensively by other birds, which were noted for species, but not counted. This information has been given to the Washington Department of Wildlife for analysis, and will be attached to this report when available.

There is no significant inflow of water into the lake, nor any outflow. Water levels are manipulated by a pump at the west end of the lake, which pumps water into and out of the Columbia river.

The area surrounding the lake has been farmed for several years for vegetables. We are concerned about the water quality of the lake, which may contain substantial levels of fertilizers and possibly pesticides. The results of a preliminary water quality analysis of the lake by Marc Harvey on August 2, 1990 at 10:15am were: lake depth = 14 inches, water temperature = 18.2c, dissolved oxygen at 12 inches = 8.9, pH = 8.8, nitrate (NO3) = 2mg/l, nitrite (NO3) = 1mg/l, ammonium nitrogen = <0.1 mg/l. We have not yet interpreted this data.

A fisheries survey of the lake will be conducted by John Weinheimer, with results attached to this report when available. Carp are clearly inhabiting the lake, and are strongly suppressing the development of submergent and emergent vegetation. They keep the water muddy and the lake bottom stirred up.

Bingen lake and the surrounding property is owned by the Port of Klickitat. The Port has agreed to accept up to 1.5 million cubic yards of rock and soil "spoils" from the Bonneville dam, for the purpose of raising the ground level surrounding the lake in preparation for commercial or industrial development. This material is projected to arrive approximately August 1992.

In August and September 1991, the Washington Department of Transportation deposited approximately 50K yards of excavated material at Bingen lake. The material was used to create a berm around the south and west end of the wetlands. The berm is sloped 3:1, and will be covered with topsoil and seeded with annual grass when completed. Installation of a silt fence at the bottom of the berm has been recommended.

It is the Farmers Home Administration understanding that the "first level of berm around the lake will be set back 50 and then raised 6 or 8 feet then go back another 50 feet. This second level will not be developed beyond trails, paths, interpretive areas and etc." (See appendix B).

PROJECT PREPARATION PHASE

The Columbia Gorge Audubon Society conducted several meetings at the lake, during which we solicited the advice and concerns of the Port of Klickitat, the Washington Department of Wildlife, US Fish and Wildlife Service, the Environmental Protection Agency, the Washington Environmental Council, the Underwood Conservation District, and Friends of the Columbia Gorge. The following issues, concerns, and opportunities were identified:

- 1) **Lack of water flow into and out of lake, and resultant water quality.** Water quality and any effects on flora and fauna need to be determined. Perhaps the Washington Department of Wildlife or the Institute of Environmental Toxicology and Chemistry (Western Washington University) could be of assistance. There is a potential opportunity to develop a source of inflowing water by creating a wetland in the northeast corner of the land surrounding the lake. This created wetland might serve to expand the secondary sewage treatment plant across

the street. It is our understanding that the water discharged from the sewage treatment plant already meets quality standards, since it is currently discharged directly into the Columbia River. Concern about this program include the potential for a treatment failure to contaminate the lake water, although perhaps a safety valve might be arranged to prevent contamination. This idea merits further investigation.

If oxygen levels are insufficient, perhaps removal of the carp, followed by plantings of sago pondweed, arrowhead wapato duck potato, and other plants in the lake might suffice to improve the water quality and oxygen levels. Otherwise, it might be possible to purchase an oxygen aerator to install in the lake.

- 2) There is a concern about potential conflicts of interest between the Port's plans for development and the life history requirements of target wildlife species. Farmers Home Administration has asked the Port to slope the lands created by the spoils away from the lake, so that runoff should not pose a problem. We are concerned that noise levels associated with post-development not be overly disruptive. Visual disturbance should be mitigated by strategic plantings. In areas where physical access to the lake might not be desired, thickets of blackberry or other vegetation could be planted.
- 3) We are concerned about the carp population in the lake. Carp destroy submergent and emergent vegetation, and keep the waters turbid. We believe that removal of the carp would significantly increase the productivity of the lake for breeding puddle ducks and provide significantly greater winter forage for all wildlife. John Weinheimer will survey the lake and give us his recommendations.
- 4) There was a concern as to how this wetland fits into the larger picture of wetland habitats in the Columbia Gorge area and how we might tailor our actions to complement the habitat network of native flora and fauna. Tara Zimmerman indicated that this lake was probably one of only two shallow water foraging areas for waterfowl in the Columbia Gorge area. David Anderson felt that the lake would provide foraging opportunities for great blue heron that nest on nearby Wells island. Harold Cole felt that an interpretive trail around the lake would be complementary to the Conboy wildlife refuge, in that it would allow people a close up view of wetland ecology that is not accessible to the public at the refuge.
- 5) We will be looking to the Port of Klickitat and the Washington Department of Wildlife for aid in submitting appropriate permits, and for help in completing the enhancement projects.
- 5) We believe that there are several potential funding sources to complete the work, or to provide equipment and materials,

(4)

potentially: Washington Department of Wildlife Habitat Improvement Program, the Volunteer Cooperative Fish & Wildlife Enhancement Program, Ducks Unlimited, Port of Klickitat, US Forest Service, and Columbia Gorge Audubon Society.

- 7) Maintaining the habitat and the interpretive trail will be the cooperative responsibility of the Port of Klickitat and the Columbia Gorge Audubon Society.

OUR GOAL

To enhance the wetland ecosystem at Bingen Lake so that it will consist of more typical and more productive wetland floral and faunal communities, and will provide a greater diversity of habitats. We would also like to develop a trail around the lake to provide educational opportunities for wetland interpretation, and to provide an aesthetically pleasing wetland focal point for surrounding development and the local community.

ENHANCEMENT PROPOSALS

1. **REMOVE CARP FROM THE LAKE.** There are two possible ways to accomplish this: dry up the lake, or apply rotenone to poison the fish. John Weinheimer will advise and assist us. Following the removal, measures will need to be taken to reduce the likelihood of reintroduction of carp to the lake. This will probably result in the need for some type of screening or filtering of river water that is pumped into the lake. Additionally, we might utilize public education opportunities along the trail.

Once the lake has been cleared of carp, we would like to see it restocked with other suitable fish species. Gambouzi were recommended to feed upon mosquitos. At this time, it is not our intention to encourage a sport fishery at the lake.

2. **BUILD THREE EARTH ISLANDS IN THE LAKE.** Assuming that the lake can be dried up temporarily, bulldozing equipment that will be moving the Bonneville spoils might be utilized to push up rock and lake-bottom material into three islands. Two islands are designed with indented and asymmetrical shapes, and with gently sloping edges (3:1) for basking, nesting and cover. They should have an elevation of 3-4 feet above the water level. The north side of the islands would be planted with shrubs, maybe some trees, and the south side would be designed to retain a mud or grit beach. Logs would be placed on the islands, for perching or sunning. Two islands would be placed in the north half of the lake, about 2 acres and 1 acre in size. These sizes would probably necessitate the addition of spoil material to create the islands. The exact location might take advantage of any naturally high spots in the lake bottom. They would be placed and designed to provide visual screening and refuge for ducks. The third island would be in the south end of the lake and would emphasize mud flat habitat. It

would be about 0.25 acre in size, and would have an elevation of about 1 foot above water level.

3. **BUILD AND INSTALL THREE NESTING BASKETS FOR CANADA GOOSE.** This is primarily to provide educational and aesthetic opportunities for the public. Baskets will be placed 300 feet apart, with the top edge 30 inches above water level. Baskets will be constructed of pipe and hardware cloth, and will be supplied with straw or hay nesting material seasonally by the Audubon Society. Alternatively, nest platforms of wood could also be designed.

4. **DREDGE AREAS OF THE LAKE TO FIVE FEET.** This would be to provide habitat for diving waterfowl, such as mergansers. It would also help provide a diversity of habitat in the lake, by controlling the establishment of certain plants. A varied contour of lake bottom would an interspersed of open water and emergent plants. If the lake is drained, this could be done in conjunction with building the islands.

5. **CREATE EIGHT POTHoles.** These are designed to provide territorial sites for breeding waterfowl. Potholes allow breeding pairs to disperse and maintain a measure of isolation from other members of the same species. Potholes would be located in the beds of cattails and canarygrass along the edges of the lake. If the lake is drained, preferably potholes would be scraped or excavated by construction equipment in order to create a wedge shaped bottom contour, which would provide a shallow sloped edge for ducks, regardless of water level. Bulldozing equipment could be used to also scrape meandering ditches connecting the potholes with the open water. Alternatively, potholes could be created by blasting a hole with ammonium nitrate fuel oil mixture. While possibly less expensive, these type of potholes have much steeper, almost vertical edges, less favorable for puddle ducks.

6. **STOCK THE LAKE WITH PRODUCTIVE AND AESTHETIC PLANTS.** Sago pondweed is one of the best all around duck foods available. Ducks feed on the seeds, then later on the tubers. It is also highly beneficial for fish, as it purifies and clarifies the water by taking up poisonous gases and releasing oxygen. Sago pondweed also shades and keeps the water below it cooler. Arrowhead wapato duck potato is also prized by ducks, geese and swans. This plant will filter polluted water, since it feeds heavily on phosphorus, potash, and other nutrients (which might be in high concentration due to the history of agriculture on the surrounding lands). Nodding smartweed and three-square rush also provide ample seed production for ducks. Several other plants, such as American lotus waterlily and blue and yellow water iris can be planted along strategic viewing areas from the trail. Plant tubers might be transferred from other wetlands or purchased from Kester's Wild Game Food Nurseries.

7. **DETERMINE ANY WATER QUALITY NEEDS.** (See concern #1 above.)

8. DEVELOP AN INTERPRETIVE TRAIL AROUND THE LAKE. In northeast corner of the lake, the trail will access a wooden viewing platform built over the water's edge. In the southeastern corner of the lake, the trail will consist of a boardwalk to actually allow visitors to walk through wetland vegetation. Along the northern and western ends of the lake, the trail would stay a distance from the lake, and along the southern edge of the lake the trail would pass relatively close to the wetland boundary, with an established viewing area. Interpretive signs would be developed to provide public educational opportunities. Benches would be constructed at the southern and northeastern viewing points. Shrubs which provide wildlife foods and pleasing aesthetics would be planted strategically along the trail to minimize wildlife disturbance, and to attract wildlife to the viewers. Nest boxes and bird feeders might be placed along the trail, depending upon commitment to their maintenance and public use. Educational opportunities might include self-guided brochures and occasional Audubon-led tours. A program will be developed and distributed to local schools encouraging classes to visit the wetland ecosystem. Trail should be designed to be easily maintained, and to minimize littering opportunities.

LITERATURE CITED

- Dahl, T. E. 1990. Wetlands Losses in the United States 1780's to 1980's. U.S. Department of the Interior, Fish and Wildlife Service, Washington, D.C. 21 pp.
- Turner, J. F. 1990. Fish & Wildlife '90, A Report to the Nation, U.S. Department of the Interior, Fish and Wildlife Service, Washington, D.C. 48 pp.



UNITED STATES
DEPARTMENT OF
AGRICULTURE

Farmers
Home
Administration

Room 319 Federal Building
P.O. Box 2427
Wenatchee, WA 98807-2427
Tele: (509) 662-3202

June 18, 1991.

Laurie Smith, President
The Columbia Gorge Audubon Society
P.O.Box 512
Hood River, OR 97031

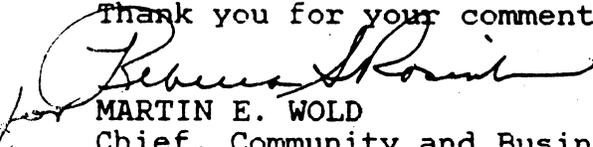
Dear Laurie:

We appreciate your input to the developments concerning Bingen Lake and the Port of Klickitat Industrial Park. In our discussions with the port they have expressed a desire to work closely with your organization as they plan the developments at the industrial park.

We too are concerned about drainage from the streets and roads into Bingen Lake. We are planning to require mitigation to the extent possible with slope requirements, drains to the Columbia and etc.

It is our understanding that the first level of berm around the lake will be set back 50 feet and then raised 6 or 8 feet then go back another 50 feet. This second level will not be developed beyond trails, paths, interpretive areas and etc. This appears to be a practical alternative to a 100 foot set back with a 15-18 ft slope to the top. It was our understanding that this concept was developed with at least partial input from you.

Thank you for your comments and suggestions.


MARTIN E. WOLD
Chief, Community and Business Programs

PREREHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

This is the Washington Department of Wildlife prerehabilitation plan for Bingen Lake in Bingen, Washington. The purpose of this rehab is to rid this shallow 20 acre lake/wetland area of carp to provide high quality waterfowl habitat. The carp are currently suppressing the development of submergent and emergent vegetation and keeping the water muddy and the bottom stirred up. This rehab is being done in conjunction with the Columbia Gorge Audubon Society and the Port of Klickitat County.

The fishery of the lake as it exists now is primarily carp and, to a lesser extent, bullhead. The lake is only 1.5 feet deep and water temperatures become severe during the winter and summer for other fish. The lake receives very little or no fishing pressure and many other local waters contain the same fish such as the mainstem Columbia and its numerous backwater areas north of Highway 14.

If the carp are eliminated, the lake/wetland area has the potential to become an excellent waterfowl habitat based on the survey work done by the Audubon Society and our own Wildlife Management Division. In addition to enhancing the habitat, the Audubon Society feels this rehab in conjunction with other work to be done will offer educational and recreational opportunities for the public about waterfowl.

B. Physical Description of the Water Proposed for Rehabilitation

1. Bingen Lake, Klickitat County
2. Township 3 North, Range 11 East, Section 29
3. 20 acres including vegetated area
4. 1.5 feet deep at deepest point, lake is very shallow
5. Estimated volume at full pool = 1,306,800 cubic ft.
Estimated volume at rotenone treatment = 326,700 cubic ft. =
2,456,391 gallons of water.
6. There is no significant natural inflow or outflow of water. Water levels are manipulated by a pump operated by the Port of Klickitat at the west end of the lake.
7. Stream miles = 0
8. Developed public access = 0
9. The lake and the surrounding area are owned by the Port of Klickitat County. The Port has agreed to accept up to 1.5 million cubic yards of rock and soil "spoils" from the Bonneville Dam for preparation for commercial and industrial development of the land around the lake. This is scheduled to start August, 1992.
10. There are no developed resorts or recreation areas.

C. Proposed Management Actions

1. Carp are the target species
2. Lake has not been rehabilitated before.
3. Proposed treatment is September, 1992.
4. Restocking of fish is not being recommended at this time.
5. None.
6. 0
7. Proposed toxicant is Cube Powder fish toxicant powder, 5% rotenone. 41 lbs. are estimated to be needed.
8. Lake will be pumped down as low as possible starting in June. Hopefully most of the remaining water will evaporate during the summer and rotenone treatment will start in September.
9. Estimate three WDW employees and two to three volunteers from the Audubon Club.

II. PURPOSE

The purpose of this rehab is to rid the lake of carp to optimize its potential as waterfowl habitat. The proposed enhancement of the waterfowl habitat by the Audubon Society is dependent on the elimination of the destruction of the aquatic vegetation caused by the carp population. See the Columbia Gorge Audubon Management Plan for Bingen Lake, "Bingen Lake Wetlands Improvement Project".

III. INTENDED OUTCOME/MEASURE OF SUCCESS

The intended outcome is an increase of submergent and emergent vegetation in the lake which will enhance the food availability and cover for waterfowl. Unless carp are reintroduced, this condition should last at least a decade.

IV. RESOURCE IMPACTS

The lake has been surveyed by our Wildlife Management Division for sensitive species, none were found. The rehab may make the lake a possible candidate for western pond turtle introduction. The lake is not used for any human uses anymore. Impacts to nontargeted resources in the lake are consistent with those covered in the Programmatic Environmental Impact Statement.

V. MITIGATION FOR ADVERSE IMPACTS

1. The Lake will be dried up as much as possible during the summer to reduce the amount of rotenone needed to treat the lake. Dead fish will be gathered up and taken to a renderer.
2. The lake has no downstream resources.

3. No rare species of plant or animal are found in the lake.
4. Applicants will comply with all safety rules.
5. Area will be posted to discourage public from collecting dead fish.

VI. RECREATIONAL IMPACT

The rehab will aid in the development of excellent waterfowl habitat and interpretive sites and trail for viewing by non-consumptive users. This site will be used by schools, clubs, and the general public as a site to view wetland ecology.

VII. ECONOMIC IMPACT

The area will be valuable to the community and the port.

VIII. RELATED MANAGEMENT ACTION

This rehab is in cooperation with the Columbia Gorge Audubon Society at their request to help with their proposed enhancement plans for waterfowl habitat. See "Bingen Lake Wetlands Habitat Improvement Project" by the Columbia Gorge Audubon Society.

IX. PUBLIC CONTACT

The Columbia Gorge Audubon Society has conducted several meetings with Washington Department of Wildlife, U.S. Fish and Wildlife Service, the Environmental Protection Agency, the Washington Environmental Council, the Underwood Conservation District, the Port of Klickitat County, and the Friends of the Columbia Gorge, about the enhancement of the lake which included the use of rotenone to get rid of the carp. All were favorable to the above actions. Several letters from the above agencies and clubs are available.

c:\hueckel\bingen.rhb

PREREHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

The lake was enhanced for waterfowl habitat value by constructing dikes to separate/isolate it from Winchester Wasteway in 1984 and removal of carp and other fish in March 1985. Construction of dikes was funded jointly by WDW (Wildlife Management Division) and Washington Duck Hunters, Inc.

After rehabilitation, the lake appeared fish-free and remained very production for waterfowl through summer of 1989. Presence of carp was suspected (murky water) in 1989 and confirmed in the fall of 1990. The method of reintroduction is unknown but was likely that an incomplete kill occurred or carp were transplanted by humans or fish-eating birds (e.g., terns, herons, gulls).

Duck broods have been counted annually in the lake since 1983 (Fig. 1). In 1983 and 1984 (prerehab) a total of three broods (2 in 1983 and 1 in 1984) were observed on the lake during brooding counts. From 1985 to 1989 (postrehab) the number of broods observed on the lake averaged 106 annually, with a peak count of 142 broods in 1986.

B. Physical Description of the Water Proposed for Rehabilitation

1. Unnamed lake in Desert Unit of the Columbia Basin, WA, Grant County
2. TWN (18N), RGE (26E), SEC (11,14)
3. 75 surface acres
4. Maximum depth, approximately 6 feet. Average depth, approximately 3 feet.
5. 225 acre feet
6. Lake has no outlet and has no surface water connection to Winchester Wasteway and adjacent lakes, ponds, and wetlands.
7. N/A
8. Lake is approximately 1 mile from WDW access area on Dodson Road.
9. One hundred percent publicly owned (U.S. Bureau of Reclamation).
10. No resorts.

C. Proposed Management Actions

1. Carp
2. March, 1985
3. September 30, 1992 or March, 1993
4. No fish are to be restocked.

5. N/A
6. N/A
7. Rotenone, powder, 1 ppm, 450 pounds at 5 percent concentration, and 5 percent liquid concentration, 3 ppm 1,822 pounds powder and 20 gallons liquid.
8. Dispense rotenone from bags behind boats. Spray shoreline emergent zone with liquid.
9. Four people and one crew leader

II. PURPOSE

The primary objective of the carp removal is to improve quality of duck brood-rearing habitat. The pond is large and near optimum depth for maximum value to most species of ducks. Increased production and availability of submergent aquatic vegetation (primarily sago pondweed) and invertebrates after carp removal will also support more waterfowl use during other seasons of the year. Several species of aquatic wildlife will benefit from the rehab.

In its present state, the lake's primary limiting factor to waterfowl production and use is the presence of carp. However, the presence of other species of fish that would compete with ducks for invertebrates and the presence of fishermen disturbing waterfowl would also substantially limit waterfowl habitat value. In order to maximize benefit to waterfowl and other aquatic wildlife, the lake will be managed to remain fish-free.

III. INTENDED OUTCOME/MEASURE OF SUCCESS

Removal of carp is expected to result in increased production and use by ducks similar to the increase observed after the initial rehab in 1985.

Duck pair counts (May) and brood counts (July) are made annually. Nongame species are counted in conjunction with duck counts on the lake.

The lake can be expected to remain productive for at least 5 years postrehab, similar to that observed after the initial rehab and provide high-quality waterfowl habitat.

IV. RESOURCE IMPACTS

- A. Prior to the initial rehab the lake's fish population was dominated by carp but also had smaller numbers of pumpkin seed sunfish, bluegill, crappie, yellow perch, largemouth bass, bullhead, and sucker. Carp made up approximately 75 percent of the total fish number and about 95 percent of biomass. Species composition in the lake now is likely similar to that before the initial rehab, but no sampling has been done. A large number of carp, approximately 2-3 pound in size were observed in the lake in May, 1991.

- B. Impacts to nontargeted resources in the lake are consistent with those covered in the Programmatic Environmental Impact Statement.

V. MITIGATING FOR ADVERSE IMPACTS

- A. The lake is remotely located (approximately 1 mile from the nearest road) and is visited rarely by people except during waterfowl hunting season. It is not likely there would be a need to remove dead fish from the site or schedule the rehab for a specific time to reduce potential inconvenience to the public.
- B. The lake has no outlet and thus would not require protection of downstream resources.
- C. N/A
- D. Applicators will wear protective clothing and masks to reduce contact with rotenone.
- E. Signs will be posted at the lake to discourage public from collecting dead and dying fish.

VI. RECREATIONAL IMPACT

Prior to the initial rehab in 1985 the lake appeared to have a very low intensity fishery with less than 50 man-days annually of fishermen seeking primarily largemouth bass. No fishermen or evidence of fishing have been seen at the lake since 1985. The lake has been visited by the area wildlife biologist (Jim Tabor), approximately 6-8 times annually. On this basis, no loss of fishing recreation would occur as a result of the proposed rehab.

Increased waterfowl production and use at the lake would provide increased hunter opportunity/satisfaction and possibly recreation days for waterfowl hunters. The lake is also an important hunting site because in a carp-free condition it is attractive habitat for ducks during the hunting season.

VII. ECONOMIC IMPACT

The proposed rehab would be expected to provide a favorable cost/benefit ratio. Assuming a 5-year project life, the lake can be expected to produce about 100 duck broods (600 ducklings) annually, similar to that observed after the initial rehab, for a total of 3,000 ducklings. Cost of the rehab would be about \$3,000, for a cost of \$1.00 per duckling. In addition to ducklings produced, the improved habitat quality would support a large increase in waterfowl and other aquatic wildlife use-days.

Funding for the proposed rehab is the waterfowl stamp/artwork program.

VIII. RELATED MANAGEMENT ACTION

None are anticipated.

IX. PUBLIC CONTACT

This rehab would be expected to produce little if any public controversy or concern, primarily because no fishery has existed in the pond since May 1985 and a very low-level one existed before 1985.

This proposed rehab will be included in the public meeting presented by Region Two Fisheries Management Division on May 21, 1992.

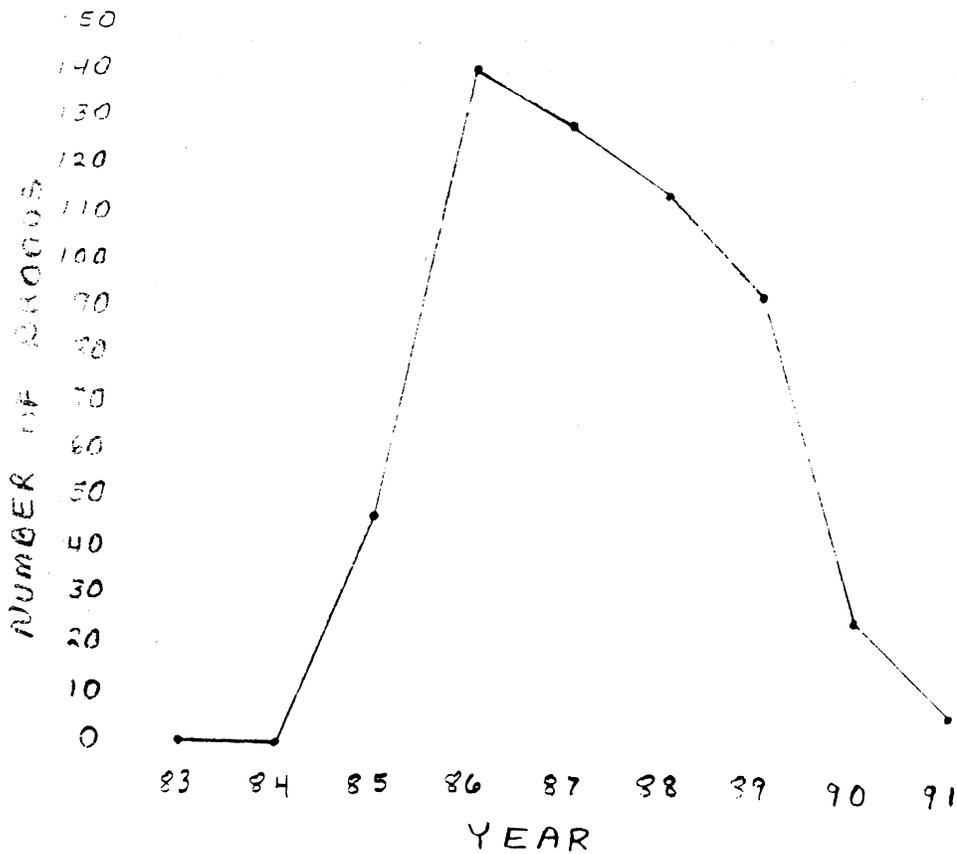
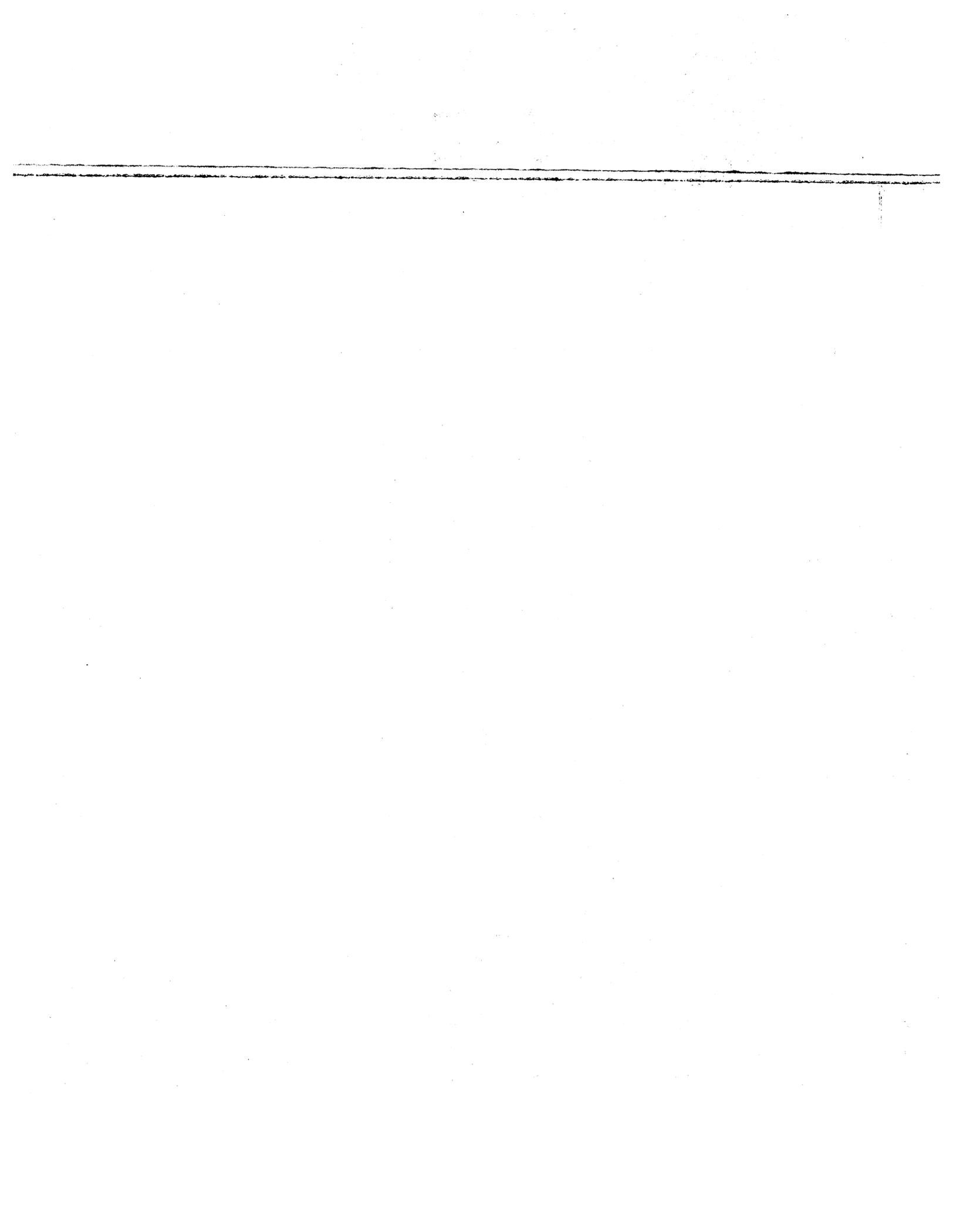


Figure 1. Number of duck broods counted in lake proposed for rehabilitation. This lake was rehabed in March 1985 (for first time). Presence of carp were suspected in 1989 and confirmed in 1990.



Buck Lake Management Plan

1) Location - Description: Kitsap County (Twp 28n Rge 2E Sec 21) located 1 1/2 miles southwest from Hansville. Volume of water is 157 acre feet and is 20 surface acres in size. It has an intermittent outlet stream and the north end shoreline is primarily covered with cattails.

2) Past and Present Recreational Fisheries: Recreational fisheries both past and present on Buck Lake have been dominated by rainbow trout anglers. The primary catch has been from fingerling stocked rainbow that reach an average of 11 inches by spring of the following year. Catchables are also stocked to supplement the recreational fishery due to the lake's remote location and popularity for trout fishing. See attached table for documented opening day catch statistics.

3) Fish Stocking Records:

<u>Date</u>	<u>Species</u>	<u>Number Fish</u>	<u>Size (#/lb.)</u>
April 92	Rainbow	2,000	5.0 (catchable)
May 92	Rainbow	1,000	4.0 (catchable)
March 91	Rainbow	2,550	5.0 (catchable)
May 91	Rainbow	1,200	4.0 (catchable)
May 91	Rainbow	8,010	89.0 (fingerling)
April 90	Rainbow	1,320	4.4 (catchable)
May 90	Rainbow	1,450	5.0 (catchable)
June 90	Rainbow	5,025	75.0 (fingerling)
June 90	E. Brook	498	6.0 (catchable)
May 89	Rainbow	1,802	5.3 (catchable)
June 89	Rainbow	5,025	75.0 (fingerling)
March 88	Rainbow	1,500	5.3 (catchable)
June 88	Rainbow	8,400	80.0 (fingerling)
March 87	Rainbow	2,502	5.7 (catchable)
June 87	Rainbow	7,865	65.0 (fingerling)
April 86	Rainbow	2,002	4.5 (catchable)
March 85	Rainbow	1,500	4.4 (catchable)
May 85	Rainbow	8,800	44.0 (fingerling)

4) Current Management Objectives: The present management objective of Buck Lake is to restore and maintain an active trout fishery from stocking fingerling rainbow trout annually and a supplemented catchable plant after opening day.



BUCK LK—Kitsap Co—T28N—R2E—Sec 21—Looking N'ly
—Game Dept. 1911—Open to the public

Kitsap

Section

25-B

21-M

27-H

33-J

21-D

BUCK LAKE — KITSAP COUNTY
T 28 N — R 2 E — Sec 21
22 Surface Acres
Surv. by State Dept of Game
Volume—157 Acre Feet



PREREHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

1. See graph that shows catch rates prior to last rehabilitation and after.
2. Seventy-five (75) to 100 angler days based on opening day creel information.
3. N/A
4. N/A

B. Physical Description of the Water Proposed for Rehabilitation

1. Buck Lake, Kitsap County
2. TWN (28N), RGE (2E), SEC (16,21)
3. 22 surface acres
4. See map
5. 157 acre feet
6. Intermittent outlet stream
7. N/A
8. Two developed access areas. Department of Wildlife boat access and County park.
9. Ten (10) percent public land ownership and 90 percent private.
10. County park used for swimming and fishing.

C. Proposed Management Actions

1. Largemouth bass
2. September 30, 1986
3. ~~September 30, 1992~~
4. April, 1993
5. Rainbow trout
6. 3,000 catchables and 8,000 fingerling
7. Rotenone, powder, 1 ppm, 450 pounds at 5 percent concentration
8. Dispense rotenone from bags behind boats
9. Four people and one crew leader

I. PURPOSE

The purpose of the proposed rehabilitation is to eradicate the largemouth bass fish population in order to reduce competition for food and eliminate predation of rainbow fingerling plants. Buck Lake is managed for trout only, employing primarily fingerling plants at approximately 400 fish per surface acre.

Buck Lake has been rehabilitated through the use of rotenone in 1950 and 1986 to control and eradicate the undesirable and illegally introduced speices of warmwater fish, primarily largemouth bass.

5) Regulations: Buck Lake is open for the general lake season.

c:\hueckel\buck-lk.mgt

III. INTENDED OUTCOME/MEASURE OF SUCCESS

Duration of beneficial effects should be five to ten years, and success will be measured by opening day creel checks and annual settings of gill nets.

IV. RESOURCE IMPACTS

- A. Impacts to nontargeted resources in the lake are consistent with those covered in the Programmatic Environmental Impact Statement.
- B. Potential impacts to human related uses include loss of recreational fishing opportunity during the month of October, and some swimming days at the county park.
- C. None that are known.

V. MITIGATING FOR ADVERSE IMPACTS

- A. Department of Wildlife personnel will remove dead fish from shoreline at county park to reduce any impacts to swimmers.
- B. None
- C. None
- D. Standard method of application and safety precautions will be employed.
- E. Department of Wildlife personnel will be present at the time of rehabilitation and signs will be posted around the lake.

VI. RECREATIONAL IMPACT

Angler participation will be increased by approximately 75 to 100 on opening day and a 25 to 50 percent increase annually. Angler success will probably double on fingerling stocked fish.

VII. ECONOMIC IMPACT

Economic benefits to our program include a reduction of catchable plants (3,000 fish) made annually to Buck Lake and a possible increase in license sales from increased angler participation. The local community will also benefit economically from increased angler participation.

VIII. RELATED MANAGEMENT ACTION

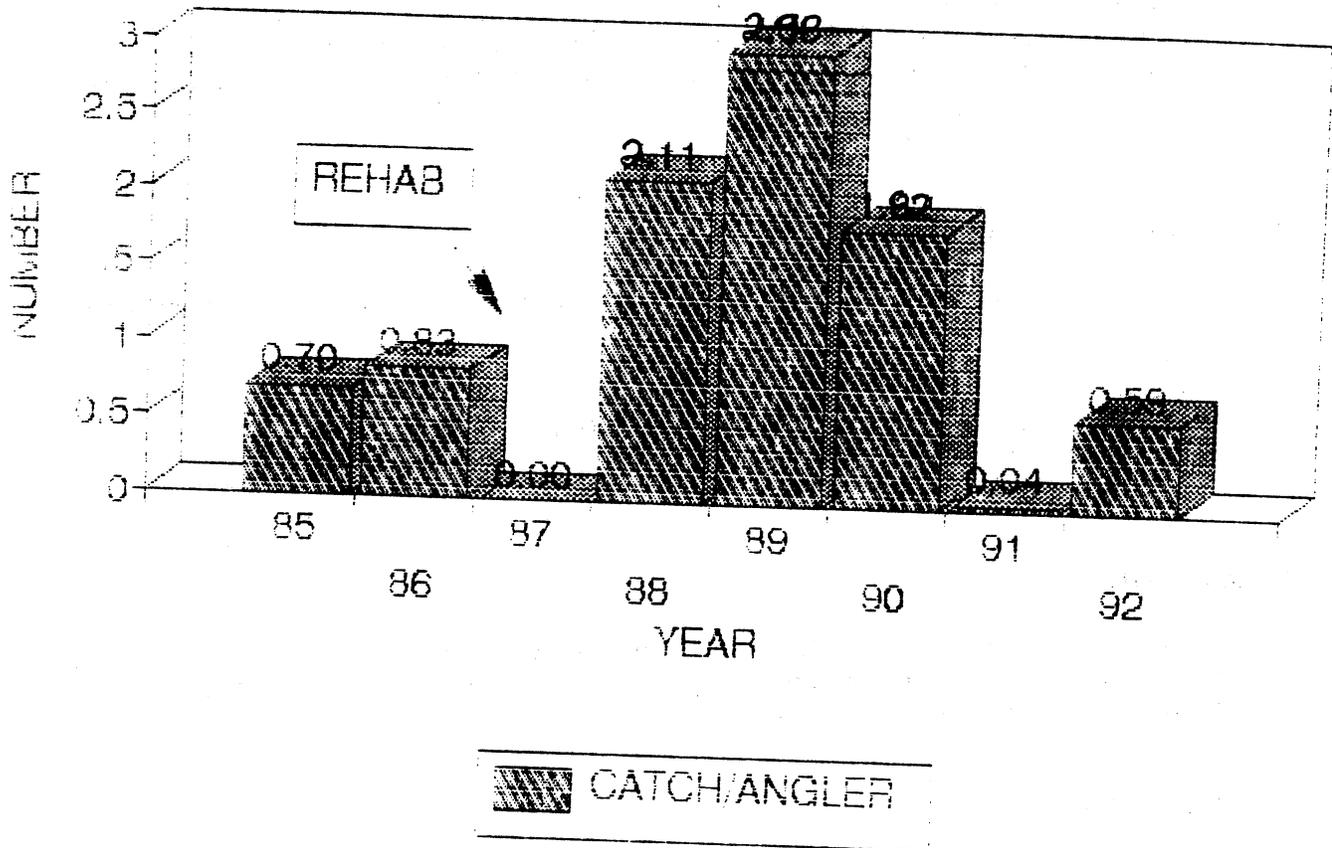
Buck Lake will be stocked annually with fingerling rainbow trout at approximately 400 fish per surface acre.

IX. PUBLIC CONTACT

A public meeting will be held in the vicinity of Buck Lake in June.

BUCK LAKE

OPENING DAY CATCH (FINGERLING)



APPENDIX F

DRAFT EIS
COMMENTS AND RESPONSES

MEMORANDUM

TO: Mr. Greg Hueckel
Fish Management Division
Washington Department of Wildlife
600 North Capitol Way
Olympia, Washington 98504

FROM: Mr. Ben Schroeter
Ben & Jerry's Paralegal Services
P.O.Box 2856
Olympia, Washington 98507-2856
(206) 866-3965

DATE: July 30, 1992

RE: Comments on the Draft - Programmatic Environmental Impact Statement Lake and Stream Rehabilitations - 1992-93. (PEIS)

RECEIVED
JUL 31 1992
DEPARTMENT OF WILDLIFE
OLYMPIA FRONT DESK

I am extremely disappointed in the Washington Department of Wildlife's (WDOW) decision to spend a lot of time and taxpayer money on trying (again) to ram through this ridiculous and illegal program of nuking our lakes and waters with dangerous pesticides.

At last years meeting of the Wildlife Commission when the department withdrew it's proposal for the Lake and Stream Rehabilitation Plan (LSRP) because the EIS wasn't worth the paper it was printed on, I was assured by Patricia McClean that the department would work with us instead of trying to figure out some furtive way around the issues.

Needless to say, promises are meant to be broken and have been. Rather than again go through a long laundry list of deficiencies which at this time I have no time for, due to my involvement in another ongoing "pesticides in the water" action, I will simply refer interested parties to my comments from last year's Environmental Impact Statement Final Supplemental.

#1
I still maintain that the Legislature specifically stripped WDOW of the option to introduce pesticides into the water as a means of eradicating fish. This year's PEIS again misstates WDOW's legal standing on page 11. I will repeat myself - RCW 77.12.420 no longer empowers the Wildlife Commission to eradicate "undesirable types of fish by means of poisoning".

RCW 77.12.420 now reads: "The eradication of fish shall be authorized by the commission". To again misstate the law, especially when I advised you of the changes in the law in my comments last year, leaves me with no alternative but to accuse WDOW of purposely lying about the issue.

Since WDOV is now purposely lying about their legal standing, it makes you wonder how much more information contained in the PEIS is also lies.

I also find it amusing that the WDOV has had some incredible success with predator stocking and yet your PEIS summarily dismisses the option as too costly, totally disregarding the successes as well as the actual means of doing predator stocking.

The PEIS while trying real hard to "justify" the nuking of our lakes, still does not show any evidence of any wonderful successes. One noted lake that got botched real good was Caliche. After nuking it with rotenone, the catch averages for the opener following treatment were so poor that people actually stopped fishing the lake. Some way of improving fishing eh?

I've done some of my own surveys of Washington fishermen. My results show that over 75% would rather catch one or two large German Browns than a bunch of little genetically inferior stocked rainbow.

Despite your attempts to spruce up your EIS and make it a legal document, you still have left out a lot of crucial information necessary for making a proper assessment, cite rather bunk scientific data which does not properly address the impacts, and again fail to provide baseline studies of the current status of the waters you wish to rehabilitate.

For example, if you have small amounts of an industrial solvent already in one of these proposed lakes, what would be the synergistic effects of the solvent with the rotenone? Don't know? Neither do I. Therefore wouldn't it be prudent to find out first, before you create a major disaster and kill off some eagles and other animals?

I wish to be notified by WDOV of when you apply to DOE for your Water Quality Modification and NPDES permits. I need to be notified so I can legally challenge the issuance of these permits through the Pollution Controls Hearing Board (PCHB). I am already preparing to make this challenge.

This whole program is an antiquated stupid way of wasting state and federal monies. Why don't we put our money to good use building desirable fisheries?

Any person or organization who wishes to join me in a legal injunctive action against WDOV's Lake and Stream Rehabilitation Program, please contact me at my listed address or phone.

Sincerely,

Ben Schroeter



Washington Wildlife Commission
600 Capitol Way North
P.O. Box 3200
Olympia, WA 98501-3200

RECEIVED

AUG 17 1992

FISHERIES MGMT DIV

Greg Hueckel
Fish Management Division
Washington Department of Wildlife
600 Capitol Way North
Olympia, WA 98504

August 13, 1992

SUBJECT: Lake and Stream Rehabilitation Program

Dear Commissioners and Mr. Hueckel:

We are writing to express our deep concerns about the Department of Wildlife's Lake and Stream Rehabilitation Program and about what we believe was a badly implemented public input process.

As explained in our letter of August 8 to Commissioner Dean Lydig, we both submitted comments on the EIS for last year's program, and were assured at that time that we would be put on the mailing list for future program actions. However, neither of us received any information about this year's program until we learned from a friend last week that the Programmatic EIS had come out on July 1. Over the course of the past week we have learned that at least three other respondents (Renee Reed, Garret Jackson, Don Miller) whose letters, like ours, were printed in last year's FEIS, were not included on this year's mailing list; two environmental groups to which we belong (Greater Ecosystem Alliance, Washington Toxics Coalition) were also excluded, although we had requested they be sent copies.

We were anxious to get a copy of the EIS, but when we each individually requested a copy from Program Manager Greg Hueckel, he refused to send us one. After expending an entire day on phone calls, trying (unsuccessfully) to locate the EIS here in Seattle, making repeated requests from Wildlife, and also requesting an extension of the comment period, the Commission office agreed to send us a copy and Connie Iten, the acting SEPA official, granted us an additional four days to comment. However, Commissioner Lydig advised us to submit our comments as soon as possible, since the Commission is scheduled to meet on August 15th.

We are sending our comments to the Commission as well as Mr. Hueckel, because (1) Mr. Hueckel has been generally unresponsive to our concerns, and (2) we are unclear about the program's decision-making process. On page ii of the EIS, it is stated that a public hearing occurs on August 15th, with a final decision rendered on September 15th. On page 8, it is stated that the Commission approves or denies treatment of individual lakes at the August hearing.

"Rehabilitation" is a Misnomer for this Program

#1 **rehabilitate:** *to put back in good condition; reestablish on a firm, sound basis; to bring or restore to a normal or optimum state of health.*

poison (v.): *[to administer] any agent which, introduced into an organism, may chemically produce an injurious or deadly effect. (Webster's New World Dictionary)*

To begin with the basics, this program neither attempts nor achieves lake rehabilitation; its purpose is not to rehabilitate lakes as lakes, but to turn them into controlled ponds acting as "habitat" for selected species. These lakes are not being managed on a firm basis if they must be treated repeatedly, nor, obviously, are they being brought to a "normal or optimum" state of health when the organisms living in them are being periodically, and almost totally, eradicated.

While poison is a distasteful word (a word actually crossed out by hand on page 11 of the DEIS) poison is what this program is about. Calling it a rehabilitation program is also potentially very misleading in terms of public awareness. If the program were called the Undesirable Fish Poisoning Program, the Lake and Stream Rotenone Treatment Program or even the Fish Elimination and Exchange Program, it would not only be more accurate, but might draw the attention and input of more citizens.

Long-Term Effects of Rotenone on the Lake Community Are Not Understood

#2 This year's document contains an exhaustive review of literature on the effects of rotenone, but major gaps in knowledge remain. For example, page 117 tells us that there have been no long-term studies on the effects of rotenone use on native fish; page 123 says there have been no studies on the long-term effects on birds. There are detailed descriptions of how many of the fish will float, how often "complete kill" is achieved, and generally how effective rotenone is for the purpose of killing undesirable fish, while questions regarding long-term and cumulative effects are simply left unanswered.

Page 121 states that effects on reptiles have not been studied; page 123 cites high mortality of salamanders and turtles, and also says that aquatic insect reduction due to rotenone is rarely more than 71%, and full recovery "usually occurs with a month or two". The comparative impacts matrix (pp. 3-5) states that lakes can recover from loss of benthic fauna, phyto- and zooplankton in "two to twelve months", while birds and mammals which depend on fish or benthic organisms "may be temporarily impacted", and mitigated through timing of the application. If some effects last twelve months, it would seem that timing is not going to make a lot of difference. It is easy to dismiss temporary, partial impacts in this way when the data are merely being used to support the false premise that rotenone treatment is relatively benign to a lake but the targeted fish; however, we don't know what the long-term impacts are of this repeated disruption of food sources, especially for already-marginal species or populations. With the kind of mortalities cited in the EIS, the long-term effects on the lake community must be assumed to be devastating.

#4 Even if long-term studies were available to assure us that rotenone itself has no long-term adverse effects, the rotenone formulation also needs to be examined. According to some literature on pesticides and the

formulations (see Attachment A), inert ingredients may be the worst offenders in these chemical treatments. On page 15 of the EIS, a discussion of odor associated with rotenone treatment mentions a kerosene odor attributed to the hydrocarbon solvents in the formulations. What are the "inert" ingredients in formulations used by WDW? These ingredients, too, may have long-term effects, particularly worrisome in cases such as McIntosh Lake, which drains into the Deschutes River, Lawrence Lake, a known bald eagle site, and Bingen Lake, where the actual long-term management goal is restoration of habitat.

The Program is Described But Not Justified: Genuine Restoration and Sustainable Management are Needed

In our last conversation with Mr. Hueckel, he energetically defended the EIS (of which he would not send us a copy), citing the thorough analysis provided. Yet this detailed review of past studies on rotenone, techniques of application, and percentage of "kill" achieved is ultimately irrelevant, because it does not explain the need for the program.

Reference to the Department's mandate to provide sport fishing does not indicate the need for massive and repeated poisoning of lakes and planting with a few "desirable" species. The mandate could have been (and probably should be) interpreted to mean that the Department should return the State's lakes to natural, balanced, ecosystems. The fact that many lakes are not now natural is not justification to leave them in their impaired state. In fact, the program itself is responsible for the unnatural state of many lakes.

The existence of "600,000 anglers" does not provide justification for the program; it is merely a head count of the fishing licenses issued by the State. It is doubtful that many of these anglers actually want complete kills of all life, fish and non-fish, in their lakes. Department effort and funding might be better spent helping its constituents appreciate and work toward more natural sporting opportunities.

Why are certain species considered "desirable"? Why are others considered "undesirable"? Where do the "desirable" fish come from? Are they being used because a "need" for them needs to be found, and because the Department needs work? Are the fish merely being moved from one side of the state to the other? Why are bass removed from Quincy and Burke Lakes and planted in Stan Coffin, H. and Ancient Lakes? Does the Department know that those who fish the latter like bass more than those who fish the former?

Is this the best way in the long run to spend state and federal funds for wildlife, or do the funds simply "need" to be spent, and this is the customary way to do it?

Fifty years ago, perhaps this program made sense. Now, it sounds like something that would only have been conceived of fifty years ago. It is not only archaic, but with what we now know about the comparative stability of natural systems, it is downright dangerous to continue to manipulate these lake systems. It is the same as turning forests to tree farms and rivers to reservoirs: it may serve a certain purpose, and it may seem like a good idea at the time.

But now we know that things have gone seriously awry with a lake when we are poisoning the undesirable non-native fish using it; writing off the temporary eradication of numerous other living things in that lake replacing the fish with other non-native fish (that were undesirable in the lake from which *they* were removed); and repeating this process every seven years or so. The Department is perpetuating systems that are inherently unstable, simply because they are the systems that are in place. The program is not necessary, it is merely customary.

What the Department should be doing is truly rehabilitating these lakes, restoring them as much as possible to the state they enjoyed many years ago before all this manipulation began, and reintroducing native stocks. Does WDW even know what the native species were in these lakes? If so, the EIS should say so; if not, the Rehabilitation Program should be focusing on understanding and rebuilding the native communities that existed in these lakes before humans started reinventing them. The program should be working toward the goal of finding the best management scheme to ensure ecosystem health and provide fishing opportunities.

We request that:

1. The Commission cease authorization of the poisoning or artificial stocking of lakes or streams.
2. The Commission direct the Department to develop a comprehensive, long-term program for true rehabilitation of lakes and streams, with the goal of restoring the health and sustainability of these ecosystems. This program will benefit not only sports fishers, but the entire natural community.
3. That this program encourage the full involvement of the public and the environmental community.

Sincerely,



George Draffan
P.O. Box 95316
Seattle, WA 98145-2316



Janine Blaeloch
7040 14th NW
Seattle, WA 98117-5308

cc: Gov. Booth Gardner
Curt Smith
Gordon Zillges
Connie Iten
Rep. Dick Nelson
Washington Toxics Coalition

Federal Regulation of Pesticide Inert Ingredients

By Martha McCabe

A significant weakness of federal pesticide regulation is the lack of information the U.S. Environmental Protection Agency (EPA) requires about the inert ingredients, metabolites, degradation products and impurities that are present in every pesticide product (see glossary below). Each one may be hazardous to human health, wildlife, or the ecosystem where it will be used. Each may increase the toxic properties of the active ingredient under certain conditions. Each may play a role in making the pesticide product to which people are actually exposed significantly more hazardous than the active ingredient standing alone.

Yet the way Congress has written the controlling law (the Federal Insecticide, Fungicide and Rodenticide Act or FIFRA¹) and the way EPA has enforced it leads the agency to ignore pesticide ingredients that may pose an "unreasonable risk to man [sic] or the environment" and thus fail to observe FIFRA's "risk benefit" registration standard.²

This article focuses only on the inert ingredients, though many of the limitations of federal regulation of in-

Martha McCabe is an Assistant Attorney General in the Environmental Protection Bureau of the New York State Department of Law in Albany, New York. She coordinates litigation and legislative and educational work on pesticides for New York Attorney General Robert Abrams.

Martha gratefully acknowledges the research assistance of Kevin Hogan, a student at Vermont Law School and a legal intern in the Environmental Protection Bureau in the summer of 1989.

This article reflects the author's personal views and not necessarily those of the Department of Law.

erts affect EPA's treatment of metabolites, impurities, and degradation products as well.

General Registration Requirements

In general, no one can distribute or sell a pesticide in the United States unless the product has been registered by EPA. FIFRA Section (§) 3 outlines the basic steps it takes to get a product registered; § 4 governs registration of pesticides containing active ingredients first registered before November 1, 1984. Besides submitting a copy of the chemical formula and proposed labeling, the applicant has to submit information about the product.

"Both Congress and the Executive share responsibility for what an increasing number of critics believe is an excessively narrow focus on active ingredients to the exclusion of other potentially hazardous components of pesticides."

Congress has told EPA to "publish guidelines specifying the kinds of information... required to support the registration."³ Those guidelines, the "Data Requirements for Registration," are contained in the Code of Federal Regulations (CFR).⁴ As recently as 1988, Congress again amended FIFRA without improving regulation of inert ingredients.⁵

Both Congress and the Executive share responsibility for what an in-

creasing number of critics believe an excessively narrow focus on active ingredients to the exclusion of other potentially hazardous components of pesticides.

This narrow focus is most easily described by comparing the amount of data required for inerts with amount of data required for active ingredients, manufacturing use products and end use products (see glossary). Most data points used by EPA to assess a product's ecological and toxicological impacts require applicants to test only active ingredient manufacturing use products and, to a much lesser extent, end use products (full formulations).

Inert ingredients alone have generally been subjected to the same requirements for any health or environmental impacts.⁶ In those cases where the test substances are manufacturing or end use products, tests will reflect the presence of inerts. Because the end use product must usually be tested for acute toxicological effects, EPA has been gathering no data on the inerts' subchronic (short term chronic, or genetic toxicity).⁷

One regulation permits, although does not require, EPA to require more testing on inerts than would otherwise be required by the general registration regulations. The Data Requirements for Registration include a policy statement on additional testing providing that, where EPA determines the required data are not sufficient to determine whether the pesticide poses the "unreasonable adverse risk to man or... the environment proscribed by FIFRA §§ 2(bb) and 3(a), the Administrator will, on a case-by-case basis, require additional testing. An explicit and definitive standard by which the EPA decide whether data are sufficient is unavailable (see 40 CFR § 158.75).

EPA may also require testing of intentionally added inert ingredients, impurities of an active or inert ingredient, plant or animal metabolite or degradation product.⁸

"Because the end use product must only be tested for acute toxicological effects, EPA has been gathering no data on the inerts' subchronic, chronic, or genetic toxicity."

Regulation of Inert Ingredients

EPA regulates inerts both under standard registration requirements and under newer policies prepared in response to specific criticisms that the inerts present significant risks of adverse health effects and environmental harm. Those are separately discussed below.

In all cases, the following information is required for each inert ingredient (if any) in the product:

1. *Chemical name of the ingredient according to the Chemical Abstracts Society (CAS) nomenclature, the CAS Registry Number, and any common names. If the identity or composition is unknown to the applicant because it is proprietary information known only by the producer of the ingredients, the applicant must ensure that the producer submits this information to the EPA;*

2. *The nominal concentration in the product;*

3. *The upper and lower certified limits;⁹ and*

4. *The purpose of the ingredient in the formulation.¹⁰*

If an inert ingredient is used to produce the product, EPA requires the following information:

1. Each brand name, trade name or other commercial designation of the ingredient; and

2. All information the applicant knows about the composition of the ingredient including specifications, data sheets, or other documents.¹¹

3. If requested by EPA, the name and address of the producer of the ingredient or, if that information is not known to the applicant, the name and address of the supplier of the ingredient.¹²

No other data, testing, or informa-

tion are required for ingredients currently registered as inerts.

Recent Developments at EPA

For over a decade, EPA has been reviewing and to some extent strengthening its regulation of inerts. In 1977, it identified 52 inert ingredients posing health or environmental threats.¹³ Seven years later, EPA admitted that it lacked the information necessary "to prioritize inerts for further review and regulation on the basis of risk," and lacked the resources to do so in any event.¹⁴

In 1987, EPA published a Policy Statement on Inert Ingredients in Pesticide Products in the *Federal Register*.¹⁵ Again, EPA conceded that "[i]nert ingredients in products registered only for non-food use... have received little review."¹⁶ EPA formally divided all 1,200 inerts as follows: List 1 (toxicological concern); List 2 (potentially toxic/high priority for testing); List 3 (unknown toxicity); and List 4, those of minimal concern.

EPA announced a data call-in for any product retaining a List 1 inert after April 22, 1987. EPA states that few, if any, List 1 inerts (e.g., benzene, cadmium, mercury) are still being used in products sold in the U.S., though it is unlikely that the Agency can prove that assertion.

Meanwhile, inerts on Lists 2 and 3 are increasingly recognized as posing potential problems. As early as 1984, EPA expressed concern about petroleum distillates, many of which are now on List 2.

The EPA notes, "The polynuclear aromatic components of petroleum distillates have a high potential for carcinogenicity and the aliphatic content may pose problems as well... [They] occur in about 80% of all pesticide formulations as inerts or actives and pose significant regulatory problems."¹⁸

As of June, 1989 EPA lists toluene as an inert in 112 registered pesticide products; xylenes in another 1,948.¹⁹

In the April 22, 1987 policy statement, EPA indicated a number of toxicology tests may be required of "new" inerts, including 90-day feeding studies, a rodent teratology (birth defects) study, genetic damage assays, and a 96-hour fish lethality test.²¹ These tests may be waived, however. Similarly, these tests may (or may not) be required of some food

use inerts when use changes or additional exemptions from re- limits on food are requested.²¹

On November 22, 1989, the again published an inert ingredi- policy statement in the *Federal Register*.²² Revised Lists 1 and 2 re- tively include 40 and 64 inerts- tantly in use. In addition, EPA i- a rather incomprehensible twist- already confusing lists: There is- a List 4A and 4B. Inerts on Li- (the previous List 4) represent- generally regarded as safe. List- composed of inerts that may n- "safe," but their current use pa- supposedly will not adversely- public health and the environm-

Moreover, certain inerts may- appear on two lists simultane- Gamma butyrolactone, for inst- will be on List 4B because h- health effects are known and o- 3 because the ecotoxicity of this- is unknown.²⁴

"Until Congress and E- squarely address the n- to assess the risks of- exposure to inerts... t- whole data-gathering- effort on which the U- pesticide regulation re- is guaranteed to produ- inadequate answers to- serious questions."

Conclusion

EPA's regulation of pest- yields relatively little inform- about the human and ecotoxicol- effects of these integral compo- of pesticide products. Until Co- and EPA squarely address the n- assess the risks of exposure to- (quite apart from exposure to m- lites, impurities and degradate- whole data-gathering effort on- U.S. pesticide regulation re- guaranteed to produce inadequ- swers to serious questions. In FI- own terms, EPA must balance- cide benefits against "any- sonable risk,"²⁵ not just the- posed by the active ingredients.

References

- Known by its acronym "FIFRA," the law is found at 7 United States Code § 136. This is abbreviated as 7 U.S.C. § 136. Citations to the law refer to the sections of FIFRA as Congress passed it; thus, FIFRA § 3 is more formally cited as 7 U.S.C. § 136a.
- Contained in FIFRA §§ 3(a) and 2(b).
- FIFRA § 3(c)(2), 7 USC § 136a(c)(2).
- 40 CFR Part 158.
- Rogers, William H. 1988. *Environmental law: Pesticides and toxic substances*. St. Paul, MN: West Publishing Co.
- 40 CFR § 158.108.
- 40 CFR § 158.340.
- Policies of Flexibility and Waiver also exist in the Data Requirements for Registration, Sections 158.35 and 158.45. EPA considers its policy of flexibility a policy to increase as well as decrease requirements as necessary in specific situations in order to fulfill the purposes of this rule. 49 Fed. Reg. 42, 856. Through its waiver policy, the EPA may waive specific data requirements on a case-by-case basis and in response to specific written requests by the applicant. 49 Fed. Reg. 42, 858. Waiver decisions are supposedly based on scientific judgments and regulatory policy.
- In Section 158.175 "Certified Limits," a table for calculating standard certified limits is included. Applicants may propose certified limits or choose to have the EPA set them.
- Section 158.155.
- The manufacturer may not have first-hand knowledge of ingredients it uses to make a pesticide product. The EPA asks the manufacturer to submit only the information (of the types specified) that is available. No new tests for chemical composition are required. 49 Fed. Reg. 37, 929.
- Section 158.160.
- General Accounting Office, 1986. *Pesticides: EPA's formidable task to assess and regulate their risks*. Gaithersburg, MD.
- EPA Office of Pesticides and Toxic Substances, Memorandum, February 1984.
- 52 Federal Register 13305 (April 22, 1987). In 1984, EPA said that "an inert ingredient is defined as any ingredient in a pesticide product which is not pesticidally active. This definition includes... non-pesticidally active impurities in the technical grade of the active ingredient or formulation." U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances, February 11, 1984; Memorandum concerning criteria for determining which inert ingredients are of toxicological concern and should be given priority review. In 1987, EPA specifically excluded impurities from the regulatory definition of inerts. 52 FR 13305.
- 52 FR 13306.
- U.S. Environmental Protection Agency, Discussion paper on inerts prepared for the Administrator's Pesticide Advisory Committee, October 25, 198, p. 3.
- Freedom of Information Act File No. RIN 3825-89, author to U.S. Environmental Protection Agency, responded to August 21, 1989.
- 52 FR 13308.
- 54 FR 48314.
- 54 FR 48315.
- FIFRA § 2(bb) (emphasis supplied).

Glossary of Pesticide Terms: Legal and Standard Definitions¹

Active Ingredient: Any substance (or group of structurally similar substances, if specified by the Agency) that will prevent, destroy, repel, or mitigate any pest, or that functions as a plant regulator, desiccant, or defoliant within the meaning of FIFRA § 2(a). 40 C.F.R. § 158.153.

Biochemical Pesticide Data: Data concerning the fate and potential adverse effects of biochemical pesticides; biochemical pesticides include products such as insect pheromones, juvenile growth hormones and natural plant regulators. 49 Fed. Reg. 42, 856.

Certification of Limits: For all quantities of the product noted in § 158.110, the maximum (or upper) and minimum (or lower) value of concentration of the variability of that substance when normal quality assurance procedures are utilized in the production process. 40 C.F.R. § 158.110.

¹Most terms are followed by a definition as found in the federal pesticide law (FIFRA); Code of Federal Regulations, Section 158; Federal Register; or a standard reference. Others have been defined by the author.

Degradation Process: Substance produced when one compound is transformed into another substance through physical, chemical or biological processes.

Efficacy Data: Data that demonstrate whether a pesticide product will control the pests as specified in the claims on product labels. 49 Fed. Reg. 42, 856.

End Use Product: A pesticide product whose labeling "(1) Includes directions for use of the product (as distributed or sold, or after combination by the user with other substances) for controlling pests or defoliating, desiccating or regulating growth of plants, and (2) Does not state that the product may be used to manufacture or formulate other pesticide products. 40 C.F.R. 158.153.

Environmental Fate Data: Data that demonstrate the fate of pesticides in the environment through degradation, metabolism, mobility, dissipation, and accumulation. 49 Fed. Reg. 42, 856.

General Use Pattern: Nine categories of pesticides that distinguish between the concepts and intended uses of pesticides; a Use Pattern Index is included in Appendix A of 40 C.F.R. § 158 (Data Requirements for Registration) to aid in classifying unique and ambiguous cases. 40 C.F.R. § 158.55.

Formulation: (1) The process of mixing, blending or diluting of one

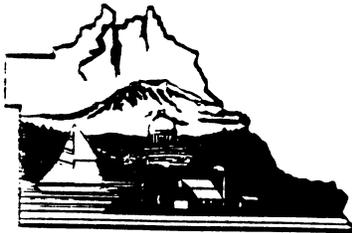
or more active ingredients with one or more other active or inert ingredients, without an intended chemical reaction, to obtain a manufacturing use product or an end use product, or (2) The repackaging of any registered product. 40 C.F.R. § 158.153.

Impurity: Any substance (or group of structurally similar substances if specified by the Agency) in a pesticide product other than an active ingredient or an inert ingredient, including unreacted starting materials, side reaction products, contaminants and degradation products. 40 C.F.R. § 158.153.

Impurity Associated With An Active Ingredient: (1) Any impurity present in the technical grade of active ingredient; and (2) Any impurity which forms in the pesticide product through reactions between the active ingredient and any other component of the product or packaging of the product. 40 C.F.R. § 158.153.

Inert Ingredient: Any substance (or group of structurally similar substances if designated by the Agency), other than an active ingredient, which is intentionally included in a pesticide product. 40 C.F.R. § 158.153.

Integrated System: A process for producing a pesticide product that: (1) Contains any active ingredient derived from a source that is



THURSTON COUNTY
WASHINGTON
SINCE 1852

RECEIVED

JUL 13 1992

FISHERIES MANAGEMENT DIV

Office of Management
Structure
County of Thurston
FISHES MANAGEMENT DIV
1000 1st Street
P.O. Box 1000
Olympia, WA 98501

BOARD OF COUNTY COMMISSION

July 8, 1992

Greg Hueckel
State Resident Trout Manager
Washington Department of Wildlife
600 N. Capitol Way
Olympia, WA 98501

Dear Mr. Hueckel:

SUBJECT: PROPOSED ROTENONE TREATMENT OF LAWRENCE AND MC INTOSH LAKES

The Board of Thurston County Commissioners is opposed to the proposed use of rotenone in Lawrence and McIntosh Lakes. This is an invasive technique which sacrifices a multitude of resident fish, amphibians, insects and other species to create a planted trout fishery. We do not believe there is justification for such drastic measures in these lakes.

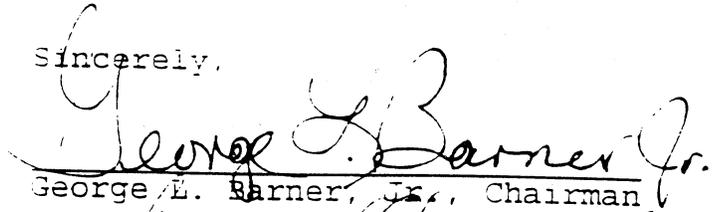
We are also concerned about the lack of integration with the Lake Lawrence management plan. The comprehensive study of this lake revealed very high nutrient loading rates - which could be severely exacerbated by the rotenone treatment.

Thurston County encourages the development of comprehensive lake management programs. In the case of Lawrence and McIntosh Lakes, we encourage the Department to work with lake residents, fishing groups and other agencies to develop clear objectives for fishery management which are compatible with multiple-use lake management. Techniques could then be selected which would best meet fishery management and other objectives in the long term, with least disruption of the environment. Thurston County - and many lake residents - would be very willing to assist with developing and implementing such a program.

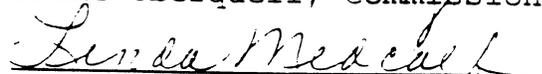
We welcome the Department of Wildlife's role in providing productive sport fisheries for our State's residents. However, we are very concerned that lake fishery management be conducted using a wholistic, ecological approach. We strongly oppose the repeated use of rotenone to turn these natural systems into virtual trout ponds. We also urge that you carefully consider comments made by lake residents at your recent public hearing.

Please contact our office or Tom Clingman at Thurston County Public Works, 786-5485, if you have any questions or would like to have a meeting on this issue.

Sincerely,


George E. Barner, Jr., Chairman


Diane Oberquell, Commissioner


Linda Medcalf, Commissioner

C:
Bill Freymond, WDW
Lorena Lindley, Sec't, Lake Lawrence Improvement Club
Mark Swartout, Office of Community and Environmental Programs



STATE OF WASHINGTON
DEPARTMENT OF ECOLOGY

Mail Stop PV-11 • Olympia, Washington 98504-8711 • (206) 459-6000

RECEIVED
AUG 10 1992
FISHERIES MGMT DIV

August 7, 1992

Mr. Greg Hueckel
Department of Wildlife
P.O. Box 43200
Olympia, WA 98504-3200

Dear Mr. Hueckel:

We have completed our review of the Draft Programmatic Supplemental Environmental Impact Statement for Lake and Stream Rehabilitation (PSEIS) and have the following comments. We will be providing comments on the specific project proposals under separate cover as part of the permit review process. However, we have highlighted for your convenience those comments that may impact the permitting process.

In general, we were very pleased to find that this document provides an in-depth, scientific and objective analysis of the rehabilitation program. Ecology is still fundamentally opposed to the use of aquatic pesticides, including piscicides, other than as part of an integrated, watershed or waterbody management plan. However, this document alleviates many of our previous concerns regarding the use of piscicides through the addition of several planning and public review enhancements to the rehabilitation program.

Some specific comments regarding the draft PSEIS include the following:

- #1 - (pg. 9) Agree with the concept of netting, reviving and relocating unwanted species. This discussion could be elaborated on regarding how long after treatment is reviving likely to be successful, what volume or number of fish could realistically be revived and relocated, and how would revival and relocation be accomplished.
- #2 - (pg. 9) Should note that the use of potassium permanganate will also require a short-term modification (permit) to the Water Quality Standards. Some additional discussion of the properties, behavior, toxicity and potential impacts of potassium permanganate (or other proposed oxidizing agents) should be included.
- #3 - (pg. 9) Would encourage the use of water column chemical analysis for rotenone in addition to the in situ trout bioassay. In addition, given the potential impacts of a rotenone treatment on

Mr. Greg Hueckel

Page 2

August 7, 1992

the planktonic community and the dependence of re-introduced species on an adequate food supply, we would suggest monitoring the abundance and composition of the planktonic community as part of the post-treatment procedures and report.

- #4 - (pg. 9) Nutrient levels, particularly phosphorus, should be monitored and reported as part of the post-treatment procedures. The potential for a pulse of nutrients following a treatment to result in accelerated macrophyte growth or an algal bloom, and particularly blooms of the potentially toxic algae anabaena, makes this important information to resource managers.
- #5 - (pg. 12) Having only one study, and that being in Texas, is inadequate for assessing impacts to water quality. This section does a good job of analyzing and discussing the scattered data, but points out the need for more detailed monitoring and comprehensive studies of rotenone's impacts on water quality.
- #6 - (pg. 13-15) Only a passing reference is made regarding the impact that increased nutrients and enhanced clarity may have on macrophyte growth. If information exists, additional discussion would be helpful. If information does not exist, some monitoring of macrophyte composition and abundance would seem necessary.
- #7 - (pg. 14-15) Depth and substrate composition of the various study lakes are key factors influencing turbidity and transparency which are not discussed. If this information is unknown for the study lakes, at least some discussion of the relative role of depth and substrate composition is warranted (i.e., in a deep lake with gravelly substrate, turbidity from stirred up mud is not likely to be a problem. However, nutrient re-suspension resulting in bloom conditions and reduced transparency could be a factor).
- #8 - (pg. 15-16) The potential presence of hydrocarbon solvents in the liquid formulation of rotenone is of major concern to us. Until additional information is provided regarding the chemistry, quantity, and toxicity of such solvents, we will likely be unable to permit the use of liquid formulations.
- #9 - (pg. 17) Although we recognize that the Action Level (AL) and Suggested No-adverse Response Level (SNARL) are conservative levels and based on long-term exposure scenarios, we believe they are applicable and that California's policy of no measurable levels of rotenone in drinking water is prudent. This would only be an issue where water from a treated water body is used for domestic purposes, therefore, in completing item 2., section IV of the Pre-Rehabilitation Plan form, it will be necessary for WDW to identify whether water intakes exist (legal or illegal) for a lake proposed for treatment.

- #10 - (pg. 17) The last paragraph regarding "...no significant change in any water quality parameter..." appears to contradict the previous discussion in this section. Perhaps wording relating to "no changes beyond those which have historically occurred as part of previous rehabilitation and stocking of trout-only lakes" would be more appropriate.
- #11 - (pg. 18) Given that the proposed treatment doses are generally higher than the toxic effects levels reported by Wollitz and Almquist, some discussion of the conflict between these studies and those referenced as showing no direct affect of rotenone on phytoplankton would seem warranted.

The discussion on plants, particularly as relating to nutrients and algae, is very indepth and well done. As previously mentioned, additional consideration of nutrient loading on macrophyte growth is warranted (pg. 24, #2 and 3), and the addition of information relating to depth and sediment type (if known) would be useful (pg. 28).

- #12 (pg. 43) Question the statement that "...no phosphorus budgets exist for the other Western Washington Lakes..." If this statement refers to the lakes in Table E, then it may be accurate, however, if it refers to Western Washington lakes in general, researchers at Metro, the University of Washington, Ecology, and consultants involved with lake restoration grant projects have all been active in developing phosphorus budgets for various lakes.

- #13 - (pg. 47-50) As previously mentioned, concern exists regarding the potential for nutrient pulses to result in blooms of toxic or noxious strains of algae, primarily blue-green algae such as Anabaena. Blooms of specific toxic-producing strains of Anabaena have resulted in domestic animal deaths in American and Clear lakes in Pierce County, necessitating closures of these lakes to human use. Therefore, additional discussion of the composition of the algal populations relative to green and blue-green algae, and the potential implications of the various species and strains, would seem warranted, particularly given that many of these species may bloom as a result of a nutrient pulse but not be affected by grazing (per table F). Ecology may require algal composition analysis as part of the pre- and post-treatment plans and reports.

- #14 - The discussion of impacts to zooplankton (pg. 57-76) is very well done. Although we recognize the lake rehabilitation program is not likely to include oligotrophic alpine lakes, the long recovery time (2-3 years) for zooplankton and benthic communities (pg. 98) in oligotrophic lakes, particularly alpine oligotrophic lakes, suggests that rehabilitation of such lakes may be inappropriate.

Mr. Greg Hueckel

Page 4

August 7, 1992

Also, the variability in recovery time, when combined with the critical function of zooplankton in supporting fish populations, would suggest that WDW should do pre- and post-treatment monitoring of zooplankton populations.

- #15 - (pg. 77) The importance of access to bottom muds and the potential role of bottom vegetation in the survival of benthic fauna reinforces the need to include information on bottom substrate and aquatic macrophytes in the Pre-Rehabilitation Plan forms.
- #16 - (pg. 94) The potential for populations of aquatic snails to increase following rotenone applications raises a concern for potential increases in swimmer's itch, caused by a parasite associated with snails. The number of serious cases of swimmer's itch reported to Ecology, including reports of associated serious illnesses, have increased significantly this past year, raising our concern and that of other health and natural resource professionals regarding this affliction. Therefore, we may consider rotenone treatments to be inappropriate for lakes which also support recreational swimming as a major beneficial use and, at the very least, would require pre- and post-treatment monitoring of snail populations in such lakes.
- #17 - (pg. 99) The potentially severe impacts reported on stream benthos indicates that rehabilitation of streams using rotenone is ill-advised. Although we recognize the possibility of such a request is remote, Ecology would likely be opposed to issuing short-term modifications for such projects.
- #18 - (pg. 114-115) The variability in % of dead fish surfacing relative to water temperature re-enforces the need to include water temperature and discussions on fish collection, revival and relocation plans in the pre- and post-treatment reports.
- #19 - (pg. 119) A typo in the first sentence, last paragraph: need an "e" in "Repeated us of pesticides..."
- #20 - (pg. 121) The reference to potential toxicity to the spotted frog re-enforces the need to check and report listings of threatened or endangered species when completing the Pre-Rehabilitation Plan form as there is a threatened or endangered species of spotted frog in Washington.
- Although this document is very well written, we did note some spelling errors, most of which could be rectified by running the document through a spell checker.

Mr. Greg Hueckel

Page 5

August 7, 1992

Thank you for the opportunity to review and comment on this document, and our apologies for the delay in getting comments to you. The PSEIS is very well done and we commend WDW for undertaking this effort. Please contact me at (206) 438-7086 if you have questions or concerns regarding our comments.

Sincerely,



Stephen L. Saunders
Water Quality Program

\\bss\wqsu\5dpseis.let

RECEIVED

AUG 03 1992

FISHERIES MGMT DIV

6959 NE Buck Lake Rd
Hansville, WA 98340
July 30 1992

RE: Supplemental Programmatic Environmental Impact Statement
Lake and Stream Rehabilitation

Greg Hueckel
Fish Management Division
Washington Department of Wildlife
600 N. Capitol Way
Olympia, Washington 98504

Dear Mr Heuckel,

I attended the public hearing in Port Orchard regarding the use of rotenone in Buck Lake.

There didn't appear to be anyone there that was in favor of this treatment. Many questions were asked of the biologist present. The public was against such treatment of an ecosystem and was knowledgeable in the ecosystem concept of interrelationships. The biologist present has his mind made up to manage Buck Lake as a trout only lake. At one time during the meeting the Wildlife Dept. even questioned the value of the hearing since no one present was in favor of rotenone treatment.

I am one of the local area residents involved in an inventory of Buck Lake and the adjoining beaver ponds. We have been doing the survey since April and find a wide diversity of wildlife. Following is a list of my concerns.

- #1. 1. The decision to use rotenone is based on opening day creel counts. Was this the only data available? Is it just an assumption that the fingerling trout survival rate is due to bass predation. Could there be other factors that could affect fingerling survival?
- #2. 2. Using rotenone in Buck Lake has been done in the past, but bass are still present. Obviously bass are getting or remaining in the system.
- #3. 3. Were alternative methods considered? Many people at the hearing volunteered manpower to the use of an alternative.
- #4. 4. Attached is a list of some of the wildlife inventoried at Buck Lake. We have not done the month of proposed treatment so do not know what uses the lake during that time the lake is recovering from the use of rotenone. Does your department know what other wildlife will be affected during this time?

#5 5. A benefit of rotenone use is listed as increased angler participation. Buck Lake is a very small lake. How do you know that more anglers would be willing to fish in such crowded conditions? Is catching fish all that is important to fisherman.

X6 6. Another benefit listed is economic return to the area. Have the anglers on Buck Lake been surveyed to see where they come from? I think you would find the majority of them are from the local area. I do not know why the Wildlife Dept. thinks fishing on Buck Lake contributes many dollars to the local economy.

#7 7. Washington State Dept. of Ecology is asking private citizens to get lakes off drugs. Attached is copy. Why not have public agencies do the same?

#8 From your document pg 125-"Kingfishers are highly territorial so that temporary disappearance of fish could force them off a lake and into competition with birds on other waters." We see kingfishers regularly at Buck Lake.

From your document-Significant impact due to increased human activities. If there is increased use as the Dept. predicates, there will be impact on the wildlife that use Buck Lake.

CONSIDER: Maybe the best use for Buck Lake isn't as a fishery but as WILDLIFE HABITAT.

Sincerely

Barbara Fournier

Following as a partial list of the wildlife inventoried at Buck Lake. There have been other people participating in the inventory.

April:

Bald eagles
Bufflehead
Swallows
Pied bill grebes
Canadian geese
Bonaparte gulls
Belted kingfisher
Red shafted flicker
Pileated woodpecker
Song sparrow
Red winged blackbirds
Mallards
Crows
Osprey
Cinnamon teal
Audobon warblers
Brown headed cowbird
Great blue heron
Black capped chickadees
Common yellowthroat
Marsh wren
Frogs
Nuthatch
Hooded merganser

Wood ducks
Calif. quail

May:

Red wing blackbirds
Tree swallow
Marsh wren
Song sparrow
Barn swallow
Starling
Pacific slope Flycatcher
Ruby crowned kinglet
Great blue heron
Osprey
Red shafted flicker
Canada Geese
Mallard
Wood duck
Bald eagle
Crow

May (continued)

Hooded merganser
Red shafted flickers
Belted kingfisher
Crows
Otters
Racoons
Downy Woodpecker
Dragonflies

June:

Osprey
Redwinged blackbirds
Frogs
Dragonflies
Cedar waxwings
song sparrow
Crows
Marsh wren
Tree swallows
Common loon
Belted kingfisher

July:

Green backed heron
Bald eagles
Tree swallows
Song sparrow
Redwing blackbirds
Belted kingfisher
Mallard.

Getting lakes off drugs

Many Washington lakes are on drugs — herbicides — that are used by residents to control plants that interfere with lake access. Ecology's Water Quality Program is asking people who routinely use herbicides to pursue more natural approaches and reduce their lake's dependence on chemicals.

A newly released Environmental Impact Statement written by Ecology looks at ways to control lake weeds other than by using herbicides. The EIS looks at the effects that both chemical and non-chemical controls have on the environment. Chemicals are of particular concern because they can leach into groundwater, collect on the lake floor in sediments and cause other impacts.

"Any healthy lake has aquatic plants, just as a forest has trees. It is the over-abundance of these plants that creates problems," said Water Quality Program Manager Mike Llewelyn.

The irony is that excessive growth is often caused by lake-area residents in the first place. When people remove trees, brush and other plants from around the lake, they remove a natural filter, giving nutrients such as lawn and garden fertilizer a direct path to the water. Leaking septic tanks and inadequate drain fields close to a lake can add more nutrients. The nutrients spur the growth of aquatic plants and algae.

"The environmentally sound system for dealing with the problem

is to involve the community in an effort to make sure that water draining into the lake is not contaminated, so the lake can return to a more natural state," said Llewelyn.

Some grant money is available for lake protection efforts. In the meantime, the use of chemicals is becoming more difficult. A permit is required for any application of chemicals into the water. When applying for permits, residents are being asked to look at alternatives.

Using chemicals that are not permitted by Ecology could jeopardize the health of people, landscape plants, and the fish and wildlife that

need clean water to survive.

"We want people to look at ways to keep the plants from becoming a nuisance in the first place," said Kari Rokstad, environmental specialist with Ecology's Water Quality Program. "Waterbody plans may eventually be required before permits are issued."

For more information

For information on the new Environmental Impact Statement, call Kari Rokstad at (206) 459-6366. For more about permits for chemical applications, call Ecology's Chris Maynard at (206) 459-6360.



Photo: Brian Wals

Sue Koenig
P.O. Box 393
Indianola, WA 98342

July 29, 1992

Greg Hueckel
Fish Management Division
Washington Department of Wildlife
600 North Capitol Way
Olympia, WA 98504

RECEIVED
JUL 31 1992
FISHERIES MGMT DIV

Dear Greg:

I am writing to put on record the concerns I expressed to you per our phone conversation July 28, 1992. This is in regards to the proposed poisoning of Buck Lake.

I am a member of a group doing a detailed inventory of Buck Lake, the two adjoining beaver ponds and their stream. When completed in spring of 1993, the survey will be presented to the county for planning purposes. Wetlands will be professionally mapped, plants and wildlife listed.

The Buck Lake team consists of Barbara Fournier and I. Since we are the most familiar with the lake in our group, we will both be submitting letters. Since March, we have spent at least two hours per week canoeing the lake and listing the wildlife. Bald Eagles and Osprey have fished the lake heavily all spring and summer. Barbara has again sighted the Green-backed Heron, a repeat of last year. They are not common in our area. The Great Blue Herons have brought their young to the lake. Five were fishing the north end Saturday, one was obviously this year's fledgling, still somewhat fuzzy.

In early spring Wood Ducks, Mergansers, and Pied-billed Grebes used the lake for a month to feed, rest, and court before moving on to quieter nesting areas. The woods shelter a wide variety of warblers, woodpeckers, and thrushes. Otter, beaver, and turtles live and feed at the lake. Large colonies of Tree Swallows and Red-winged Blackbirds have had a good nesting year and there is a conspicuous absence of mosquitoes.

The prerehabilitation plan for Buck Lake answers "none that are known" when asked to list endemic, listed species that may be affected. The entire lake will be taken out of the system from October to April. It

is now serving as an important food source to species listed by D.O.W. as priority along with many others not listed but just as important in maintaining the balance.

Specific concerns:

1. I understand that rotenone is relatively harmless to warm-blooded animals. However, there will be a large windfall of dead fish (the plan states fish will only be retrieved from the park's shore, about one-tenth of the total shoreline). The eagles in our area tend to stay year-round. Does D.O.W. want to accept responsibility for the possibility these birds may ingest large numbers of poisoned fish? The E.I.S. also doesn't address possible genetic problems as a result of ingestion.

#3
2. As almost-year-round residents, how will the eagles be affected this winter? Two adults and at least one young from a previous year may be joined by two more nestlings since this has been a good year. The lack of the lake as a food source will push them into already overburdened surrounding areas. Will nesting season be affected next spring? A lower food supply for winter plus increased competition may trigger a lower nesting response in spring.

#4
3. With the insect population lowered or eliminated next spring, nesting of swallows, blackbirds and woodland birds will suffer. The colonies that return will have to "disperse" into already occupied territory. This is almost never successful.

#5
4. One painted turtle and one unidentified turtle may also suffer from lowered insect count. Otters will not be able to feed here.

#6
At the public meeting, the local bass club offered (under direction of D.O.W.) to overfish the bass at the end of the season to reduce predation. This would be free to the public and much kinder on the ecosystem of the lake. It would also allow a choice in fishing. Buck Lake is the only public fishing lake in the north end of the county. It seems very one-sided to manage it as a trout-only lake.

I believe more research should be done on the wildlife using the lake. It is my feeling that D.O.W. doesn't realize the importance of this lake as habitat to its own priority-listed species. Buck Lake is not for trout only.

Sincerely,

Gue Keenig

Gue Keenig



Greg - 11

Greg:

I AM INCLUDING MY WILDLIFE LISTS FROM 3/1 TO 7/25. I APOLOGIZE FOR THE SLOPPINESS BUT I DIDN'T HAVE TIME TO TYPE THEM OUT. I THOUGHT IT MIGHT GIVE YOU SOME IDEA OF WHAT WE HAVE OUT THERE. I'VE HIGHLIGHTED SPECIES FROM R.O.W.'S "PRIORITY HABITATS & SPECIES" LIST TO (HOPEFULLY) MAKE IT CLEARER.

I WILL ATTEND THE AUC IS MEETING & PRESENT SOME MORE GENERAL E.I.S. COMM

Sue Koenig



PACIFIC
WETLAND
NURSERY

7035 Crawford Drive
Kingston, WA 98346
206.297.7575

HANSHVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUE KOENIG

DATE: 3/1/92 Sunday

SITE: BUCK LAKE

TIME OF OBSERVATIONS:

UPPER BEAVER POND

8:15 Am - 10:45 Am

LOWER BEAVER POND

LOCATION: CIRCLED PERIMETER OF LAKE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
GREAT BLUE HERON	①, ① MARSHY	2	FROM SHORELINE - A FLEW INTO WOODS, FE IN TREES
RED-WING BLACKBIRD	①-⑦, ① MARSHY	10+	CALLING, FLYING, S
CROW	①-③ WOODS	3+	CALLING, FLYING
FROGS (HEARD, NOT SEEN)	③-⑥ MARSHY	LOW- (?) 2 HIGHT - TREE FROG 5+	CALLING
SONG SPARROW	③-4 "	5	CALLING, FIGHTING
RACCOONS	④ TREE @ WOODS EDGE	2	FORAGING, SINGING, CLIMBING, PLAYING LARGE CEDAR (LAST YE. YOUNG
RUFOUS-SIDED TOWHEE	④-5 SHRUBS @ MARSH EDGE	1 MALE	CALLING, FORAGING
DOWNY WOODPECKER	⑤, ② ALDERS	3	FIGHTING, FEEDING
BLACK-CAPPED CHICKADEE	⑤ SNAG	1	PREPARING NEST - SNAG EYES HOI
ROBINS	①, ② ALDERS SHRUBS	3	MALES FIGHTING SINGING, FEMALE WAT
CHESTNUT-BACKED CHICKADEE	② ALDERS	10+	FLOCK FORAGING
BROWN CREEPER	② ALDERS	1	FEEDING
GOLDEN-CROWNED KINGLET	② ALDERS	10+	FLOCK FORAGING
MALLARD	OPEN WATER	PAIR (2)	FLYING INTO LAKE
CORMORANT		1	FLYING OVER

Pencil &



PACIFIC
WETLAND
NURSERY

7035 Crawford Drive
Kingston, WA 98346
206.297.7575

HANSVILLE WETLANDS

WILDLIFE OBSERVATIONS

NAME:

DATE: 3/1/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS:

UPPER BEAVER POND

8:15 AM - 10:45 AM

LOWER BEAVER POND

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
BALD EAGLE	TREES, SHORELINE	1 ADULT 1-2 ⁺ YR. OLD IMMATURE	HUNTING, PERCHING
<p>FUSHED FROM WOODS BY HORSE & RIDER ON NORTHWEST SHORE. THE ADULT EXHIBITED INTEREST IN FEEDING BEHAVIOR - FLEW DOWN TO SHORELINE, WADED INTO SHALLOWS & OCCASIONALLY GRABBED AT SOMETHING. IT REPEATED THIS IN SEVERAL PLACES THEN FLEW INTO THE WOODS. THE YOUNG EAGLE JUST WATCHED THEN FOLLOWED THE ADULT</p>			
LONG-BILLED MARSH WREN	(6) CATTAILS	1	GUARDING TERR FUSSING AT ME
PILEATED WOODPECKER	(4) WOODS	1	HEARD HAMMERS (NOT SEEN)
<p>HIDDEN BEAVER LODGE - HEARD GRUNTING SOUNDS (BEAVER INSIDES?) RECENTLY CUT WILLOW STEMS NEXT TO LODGE.</p>			



HANSVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUE KOENIG

DATE: 3/21/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 8:00

UPPER BEAVER POND

10 A

LOWER BEAVER POND

LOCATION: CANOE PERIMETER

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
CANADA GOOSE	(4) MARSHY	3	FLYING FROM LAKE
BUFFLEHEAD	(7-8) OPEN WATER	5 MALE 3 FEMALE	SWIMMING, DIVING
PELAGIC CORMORANT	(7-8) "	1	"
REDWING BLACKBIRDS	(6, 1) TREES, CATTAILS	5 +	FLYING, SINGING, CALL
GREAT BLUE HERON	(4-7) SHORELINE	1	FLYING, PERCHING
MALLARD	(7, 4) SHORELINE	1 MALE 1 FEMALE	SWIMMING, CIRCULAR DIVE
WINTER WREN	(4) WOODS	1 (HEARD)	SINGING
RING-NECKED DUCK	(7) SHORELINE	2 MALE 1 FEMALE	SWIMMING
FROGS	(6) "	2 (HEARD)	LOW CROAKING
NOT OBS (THE BOG BUCKLEHEAD IS MAKING)		A BIG PATCH	THIS YEAR (4)
CROW	(6, 11) TREES	3 +	FLYING, CALLING
LONG-BILLED MARSH WREN	(4-5) CATTAILS	1	CATHERING CATTAIL F POTTING IN NEST FUDDING
BLACK-CAPPED CHICKADEE	(4) BLACKBERRIES	2	FORAGING, FLYING
STARLING	(5) SNAGS	2	SINGING, EITHER PADDING OR BUILD NEST IN SNAG #

Pencil &



HANSVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUB KOENIG
SITE: BUCK LAKE
UPPER BEAVER POND
LOWER BEAVER POND

DATE: 3/21/92
TIME OF OBSERVATIONS: 8 AM
10 AM

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
Tree Swallow	(6) SWAGS WATER	2	FEEDING OVER WATER
Chestnut-backed Chickadee	(6) FIR ALDERS	3-4	FORAGING
Bald Eagle	(7) SHORELINE	1 IMMATURE	FLEW IN TO DRINK W.
Wood Ducks	(1-11) SHOESUNG- MILLS	4 MALE, 1 FEMALE	SWIMMING, FEED
American Koenig	(10) TREES, LAWN	2+	FEEDING, SINGING
Song Sparrow	(3-4) WOODS	1 HEARD	SINGING

HANSHVILLE WETLANDS
 WILDLIFE OBSERVATIONS

NAME: SUE KOENIG

DATE: 4/11/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 9-11

UPPER BEAVER POND

LOWER BEAVER POND

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
REDWING BLACKBIRD	CATTAILS, TREES	5+ MALES/FEMALES	SINGING, FLYING, CALLING, FORAGING
CROW	TREES	4	CALLING, FLYING
AMERICAN ROBIN	LAWN, TREES	10+	SINGING, FLYING, CALLING, FORAGING
STARLINGS	SNAGS, TREES	10+	FLYING, CALLING
PUFFLEHEAD	OPEN WATER	1 FEMALE, 5 MALES	SWIMMING, FORAGING
FROGS	SHRUBS	10+	LOW CROAKING (HEARD)
TREE SWALLOWS	SNAGS, WILLOWS	10+	FLYING, CALLING, PERCH
PIED BILL GREBE	OPEN WATER, RUSHES	1 VISIBLE IN RUSHES	MAY BE NESTING PERCHES - 1 REMAINED HIDDEN BUT ANSWERED CALLS FROM VISIBLE
POG BUCK BEAN IN BLOOM			
POG-SHAPED FLICKER	SNAGS	2	CALLING, FLYING, FEED
BLACK-CAPPED CHICKADEE	SHRUBS	3+	CALLING (HEARD)
SONG SPARROW	SHRUBS	5+	SINGING, FORAGING
WINTER WREN	WOODS	1	SINGING (HEARD)
LONG-BILLED MARSH WREN	CATTAILS	1	FORAGING, CALLING
RUFOUS HUMMINGBIRD	CATTAILS	1 FEMALE	GATHERING CATTAIL FLUFF FOR NEST



HANSHVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUE KOENIG

DATE: 4/11/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 9-11A

UPPER BEAVER POND

LOWER BEAVER POND

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
RUFOUS-SIDED TOWHEE	SHRUBS, SWAG, TREES	3	FEEDING, PERCH
"MYRTLE" FORM			
YELLOW-RUMPED WARBLER	TREES	1	FEEDING, SINGING
TREE SWALLOW	WILLOWS, SWAGS, WATER		
RED-TAILED HAWK	(9) TREE	1 IMMATURE	PERCHING
CANADA GOOSE	(10) WATER LAWN	2	FEEDING, SWIMMING
GREAT BLUE HERON		2	FLYING OVER
BROWN CREEPER	(9) SHRUBS	1	CALLING (HEARD)



HAUSVILLE WETLANDS

WILDLIFE OBSERVATIONS

NAME: SUE KOLNIG

DATE: 4/27/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 8-10

UPPER BEAVER POND

WINDY, RAIN

LOWER BEAVER POND

DAY AFTER OPENING

LOCATION: CANOE

SEASON - FISHING,

5 OTHER BOATS PRE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
Red-winged Blackbird	(11, 16) TREES, CATTAILS	10+ FEMALES & MALES	FEEDING, NESTING, CALL
Song Sparrow	FLYING OVER	1	
Brown-headed Cowbird	(3, 6) SHRUBS, SNAG	2	CALLING, FEEDING
Mallard	(5, 10) CATTAILS	1 PAIR	FLYING
Belted Kingfisher	(5) SNAG	1	CALLING, FEEDING
Tree Swallow	(3, 6) WILLOWS, SWALS	10+	FEEDING, CALLING, FEEDING OVER WATER
Great Blue Heron	(4) WOODS	1	FLYING FROM WOODS
Song Sparrow	MOST OF PERIMETER - SHRUBS	10+	CALLING, SINGING, FEEDING
Yellow-rumped Warbler	(6) ALDERS	3	SINGING, FEEDING
Black-capped Chickadee	(4) WOODS	1	CALLING (HEARD)
Common Yellowthroat	(4-5) SHRUBS, CATTAILS	1 MALE	FORAGING, SING
Marsh Wren	(5) CATTAILS	1	CALLING
Frog	(4-5) POND EDGE	1	CALLING (LOW) (HE)
Nuthatch	(3) WOODS	1	HEARD CALLING
PATCHES OF MILFOIL ARE LARGE & SPREADING MOSTLY (4, 8, 10)			



HANSHVILLE WETLANDS

WILDLIFE OBSERVATIONS

NAME: SUB KENNIG

DATE: 5/15/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 2:30 -

UPPER BEAVER POND

4:30 PM

LOWER BEAVER POND

2 OTHER BOATS

LOCATION: CALDES

FROGONT

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
Red-wing Blackbird	(11) Cattails, Trees	4	Feeding, Singing
Tree Swallow	(6) Swags	7	Feeding
Meadow Wren	(5) Cattails	1	Singing
Song Sparrow	(3, 6) Shrubland	5	Singing, Foraging
Barn Swallow	(6) Open Water	1	Feeding, Bathing, In Flight
Robin	(1, 5) Lawn, Trees	5	Feeding, Singing
Starling	(3) Swag	1	Perching, Singing
Pacific Swift Flycatcher	(4) Dead Trees	1	Feeding from site
Ruby Crowned Kinglet	(4) Alder	1	Foraging
Great Blue Heron	(4)	1	Flying over
Coppy	(7) Swag Water	1	Feeding - caught fish
Red-throated Flycatcher	(5) Swags	2	Chasing, Feeding
Canada Goose	(11) Open Water	1 PAIR	Swimming
Mallard	Open Water	1 FEMALE w/ 3 YOUNG / 1 PAIR	Swimming / Flying
Wood Duck	(6-6) Water	1 MALE	Feeding
Red-tailed Hawk	(4) Trees	1 IMMATURE	Flying over
Crow	(7) Ground		Chasing, Flying

HAUSVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUB KOENIG

DATE: 5/25/92

SITE: BUCK LAKE

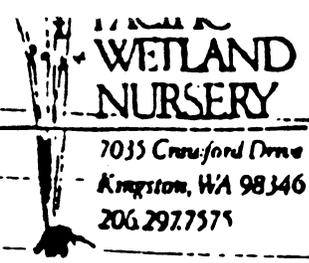
TIME OF OBSERVATIONS: 8-11

UPPER BEAVER POND

LOWER BEAVER POND

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
RED WING BLACKBERRY	(11, 6, 4) CATTAILS, BRUSH	14 MALES/FEMALES	NESTING, FEEDING, SINGING
ROBIN	(6, 10) TREES, GRASS	5	SINGING, FEEDING
TREE SWALLOW	(3, 10) OPEN WATER, SWALS	10+	FORAGING, NESTING
BARN SWALLOW	(3) NESTING IN OLD SHACK	1 PAIR	FEEDING, NESTING
CEDAR WAXWINGS	(2-6) TREES	30+	FORAGING IN FLOCKS
WILLOW FLYCATCHER	(3, 5) BRUSH, SWALS	3	SINGING, PERCH
SONG SPARROW	(3) SHORELINE BRUSH	4	SINGING, FORAGING
FIELD FAUNO GREENS	NORTH OF BUCK LAKE	1	HEARD CALLING
MARSH WREN	(5, 4) CATTAILS	2	SINGING
WILSON'S WARBLER	(3) TREES	1	SINGING (HEARD)
PACIFIC SHORE FLYCATCHER	(3, 7) TREES	3	SINGING (HEARD)
YELLOW THROAT	(8, 3) TREES	2	SINGING (HEARD)
BLACK-CAPED CHICKADEE	(3, 7) FOLIAGE	2	SINGING
CROW	(5) TREES	5	CALLING
SWAINSON'S THRUSH	(4) WOODS	1	SINGING (HEARD)



HANESVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUE KOENIG

DATE: 6/27/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 11-12

UPPER BEAVER POND

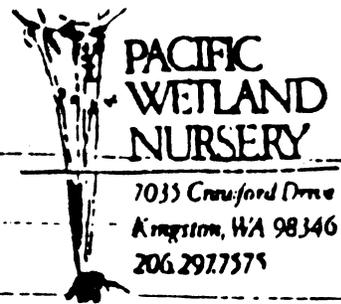
1 OTHER BOAT

LOWER BEAVER POND

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
RED-TAILED HAWK	④ SHORE	1	CIRCLING OVER POA
KILLDEER	⑨ SHORELINE	2	FLYING, CALLING
TREE SWALLOW	③⑥ SNAGS	10 ⁺	FLYING, NESTING, FEE
RED WINGED BLACKBIRD	①⑦ BRUSH, CATTAILS	10 ⁺	" " "
SWAINSON'S THRUSH	④⑧ WOODS, BRUSH	4	SINGING, FORAGING
SOOTY SPARROW	PERIMETER BRUSH-SHORELINE	6	" "
BARN SWALLOW	③ OVER OPEN WATER	2	FLYING - FEEDING
BLACK-CAPPED CHICKADEE	⑤⑧ WOODS, BRUSH	2	FEEDING, CALLING
ROBIN	③ TREES	3	SINGING, FEEDING
PURPLE FINCH	④ TREES, BRUSH	3	" "
ORANGE-CROWNED WREN	④ "	1	" "
BUSH TIT	④ "	2	FORAGING
COPPER WAXWING	④⑦ TREES, SNAGS	5	"
PURDUS SISKIYOU TOUHEE	⑥ WOODS	1	SINGING
YELLOW THROAT	③ WOODS	1	"
BALD EAGLE	⑤ SNAG	1 IMMATURE	FORAGING

REMARKS: FIELD GUIDES, TRAIL, DIRT SHEDS, TAPE MEASUREMENT, CLIPPED, RAIN GUTTER, BATS, FOOD & DRINK



HANSHVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUE KOENIG

DATE: 6/27/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 11 AM

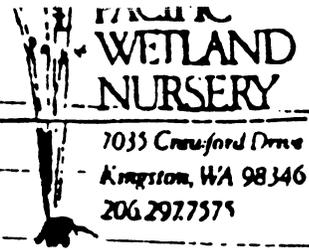
UPPER BEAVER POND

1 OTHER POND

LOWER BEAVER POND

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
AMERICAN GOLDFINCH	(7) TREES	1	CALLING
TURTLE (? SPECIES)	(8) LOG IN WATER	1-8" LONG	SUNNING
CROW	(8) TREES	6	PERCHING, FLYING, (1)
GREAT BLUE HERON		1	FLYING OVER
MARSH WREN	(5) CANALS	2	SINGING
NEW CUTTINGS	ON & NEXT TO OLD BEAVER LODGE		
WHITE WATERLILIES	IN BLOOM		} VERY ABUNDANT THIS YEAR
BRIGHT BLUE DAMSEL FLIES			
ORANGE FOOT DRAGONFLIES	(MEDIUM)		
DARK DRAGONFLIES	W/ WHITE WINGSPOTS (LARGES)		
MILFO	NOT AS NOTICEABLE		



HANSHVILLE WETLANDS
WILDLIFE OBSERVATIONS

NAME: SUE KOENIG

DATE: 7/10/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 5 PM - 7 PM

UPPER BEAVER POND

LOWER BEAVER POND

WITH BARBARA

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
BLACK-CAPPED CHICKADEE (10)	WILLOWS	7	FORAGING
SONG SPARROW (10)	SHRUBS	2	SINGING
DEAD CROW	CATCH IN TREE	BY FISHING LINE	
SWAINSON'S THRUSH (4)	WOODS	2	SINGING
RED-WINGED BLACKBIRD (1)	PODS, POND	5-6	FORAGING
CROW (2)	TREES	2	CALLING, FLYING
BARN SWALLOWS (2)	WOODS/MARSH	3	FORAGING
TREE SWALLOW (3)	SNAG	2	PERCHING
CECIL WAXWING (3)	TREES	6	FORAGING, CALLING
WILSON'S WACREER (4)	WOODS	1	SINGING
PILEATED WOODPECKER (4)	SNAGS	2	FORAGING
BELTED KINGFISHER (10)	SNAGS	1	CALLING, FLYING
GREAT BLUE HERON (4)	FRESH SWALS	1	PERCHING, FISHING
PURPLE LOOSTRIFE	IN 5-6 AREAS @		
WILLOW FLYCATCHER (7, 9)	SHRUBS	2	FORAGING

NORTH END - BARBARA WILL REMOVE

HANSVILLE WETLANDS
 WILDLIFE OBSERVATIONS

NAME: SUB KOENIG

DATE: 7/25/92

SITE: BUCK LAKE

TIME OF OBSERVATIONS: 5:30

UPPER BEAVER POND

7:30 PM

LOWER BEAVER POND

LOCATION: CANOE

SPECIES	HABITAT	# OF INDIVIDUALS	ACTIVITY
BARN SWAN	(2,0) MARSH, OPEN WATER	2-3	FEEDING, PERCH
TROUS SWAN	(2,6) MARSH, OPEN WATER	20+	FEEDING, PERCH
		THOSE 2 WERE FEEDING TOGETHER IN A LARGE FLOCK	
BLACK-CAPED CHICKADEE	(3) WOODS	3	CALLING
WILLOW FLYCATCHER	(3,6) SHRUBS	4	FORAGING, CALL
GREAT BLUE HERON	(4) SWAG, STRAWBERRIES	3 YOUNG 1 ADULT	FISHING, PREEN
RED-WINGED BLACKBERRY	(3,10) CATTAILS	5	FEEDING, CALLING
BOSTON KINGFISHER	(4,7) SWAGS	2	FLYING, PERCHING, CALL
SONG SPARROW	(3) BRUSH	1	SINGING
Hairy Woodpecker	(4) SWAG	1	PREENING
MALLARD	(4) IN WATER ON LOG	1	PREENING
WESTERN Tanager	(4) TREES	1	FLYING, CALLING, SINGING
ROCK DOVES	(2) ON OLD HOUSE	3	FLYING, PERCHING

Sue Koenig
P.O. Box 393
Indianola, WA 98342

August 15, 1992

Greg Hueckel
Fish Management Division
Washington Department of Wildlife
600 North Capitol Way
Olympia, WA 98504

RECEIVED
AUG 17 1992
FISHERIES MGMT DIV

Dear Greg:

Due to lack of transportation, I will be unable to attend the meeting today in Wenatchee to present my comments on the lake rehabilitation E.I.S. Several people also entrusted me with their letters containing their comments. I know July 31 was the last date to send comments but I was hoping you could "unofficially" read and consider these comments.

#1
I feel the lake rehab program focuses entirely on fish management with no scientific consideration of effects on the surrounding terrestrial communities. Impressive amounts of data are given for below-waterline lifeforms. Only two short pages in the E.I.S. address "probable" effects on birds, reptiles, and mammals. Targeted lakes should be thoroughly researched for the presence of endangered species, either by D.O.W. or local conservation groups. Assumptions should not be made as to what "might" happen. Solid research should be done to gather data necessary to confidently say species won't be affected. Increased human disturbance through development of surrounding areas and usage are already putting pressure on local species. To state that they will simply "move on" from disruption (through poisoning) of what little balance is left is very short sighted. It is time to consider these lakes as part of entire ecosystems, not just as large fish-rearing pools.

#2
I would like to suggest choosing some key lakes in varying habitats that will, because of public preference or true necessity, be poisoned, and doing detailed studies on them before and after. U.W. apparently conducted detailed research on Koeneman Lake in Kitsap County but no "above-water" results seem to be available. I realize resources

are limited but the data could be gathered utilizing graduate students in wildlife biology or ecology, internships, or local groups such as Audubon. Our study is showing a wide range of usage as the year progresses so a year would probably be the minimum amount needed to get needed data. Insect populations and bird feeding and nesting successes would be good indicators to stress.

Thank-you for bringing this to the public this year. I hope alot of useful local information will come out of this to help you in your decisions.

Sincerely,

Sue Koenig



Lake Lawrence Improvement Club

FISH
public
input

Save Lake Lawrence

16646 Pleasant Beach Dr. • Lake Lawrence, WA 98597 • (206) 894-361

July 22, 1992

State of Washington
Governor Booth Gardner
Olympia Wa 98504-0413

RECEIVED
JUL 3 1992

Board of County Commissioners
George L Barner, Jr
Diane Oberquell
Linda Medcalf
2000 Lakeridge Dr SW
Olympia Wa 98502-6045

WILDLIFE COMMISSION

Director of Wildlife Commission
Dean Lydig Terry Karro
Jim Walton John McGlenn
Mitch Johnson Norm Richardson
600 Capitol Way N
Olympia Wa 98501-1091

We are writing on behalf of Lake Lawrence Improvement Club a non-profit consolidated group representing all segments of the population surrounding Lake Lawrence. Lake Lawrence Improvement Club is committed to enhancing the quality of Lake Lawrence and preserving lake use for a broad base of diverse uses and enjoyment, both for present users and future generations.

We strongly protest the proposed use of Rotenone in Lake Lawrence by the Department of Wildlife. Strong expressions of opposition to this proposal were made at the public hearing on July 1, 1992 by individuals in our area. In a very short time, with very limited effort, petitions were solicited to people in our area and over 270 signatures were obtained to express opposition to this proposal. Our purpose in writing is to confirm a collective opposition to this proposal by a consolidated group representing Lake Lawrence. We have decided to write to you because of our feeling that the Department of Wildlife has been unresponsive to public opinion expressed at the public hearing and will make a decision on this matter contrary to the overwhelming opposition by those of us most directly impacted by the decision.

Such a conclusion on our part appears justifiable as it was very clear at the public hearing that the Department of Wildlife had made little or no effort to articulate with our Lake Management District and other county and state agencies involved in activities related to lake restoration and lake management in proposing the use of Rotenone.

Although we will not attempt to present all of our concerns relative to this issue, we would like to state some specific reasons for our strong opposition to the proposal.

(1) The diverse interest in fishing Lake Lawrence go well beyond the proposed "trout preserve" created by the proposal. Lake Lawrence is regarded as one of the finest bass fishing lakes in our state.

(2) Residents and public lake users will have to endure and clean-up rotting fish from the lake and beaches.

(3) The bald eagle habitat, with their protected rights and heron families living on our lake will have their food supply significantly impacted for a number of years.

(4) Our aquifers may be contaminated by the use of Rotenone.

(5) Algae blooms, already a major problem, will intensify with the decay of fish.

(6) The weed problem will increase without fish and wildlife eating the weeds. Our weed problem is already a significant problem. We have had to spend thousands of dollars.

(7) The finances used to poison the lake could be spent on re-stocking fish and other projects having a positive impact on lake use.

We are enclosing a copy of a letter sent to the Department of Wildlife by members of our Board of County Commissioners in which they state their strong opposition to the proposal. We urge you to read this document and to carefully consider the position of our local governing officials.

Thank you for this consideration. Please inform all those considering this issue of our concerns. We also request that we be informed of all developments relative to this proposal.

Lake Lawrence Improvement Club
David M. Olson, President

David M. Olson

Robert Lindley, Vice President

Robert Lindley

Joan Patrick, Secretary

Joan Patrick



THURSTON COUNTY
WASHINGTON
SINCE 1852

George L. Bamer, Jr.
District One
Diane Oberquell
District Two
Linda Medcalf
District Three

BOARD OF COUNTY COMMISSIONER

July 8, 1992

Greg Hueckel
State Resident Trout Manager
Washington Department of Wildlife
600 N. Capitol Way
Olympia, WA 98501

Dear Mr. Hueckel:

SUBJECT: PROPOSED ROTENONE TREATMENT OF LAWRENCE AND MC INTOSH LAKES

The Board of Thurston County Commissioners is opposed to the proposed use of rotenone in Lawrence and McIntosh Lakes. This is an invasive technique which sacrifices a multitude of resident fish, amphibians, insects and other species to create a planted trout fishery. We do not believe there is justification for such drastic measures in these lakes.

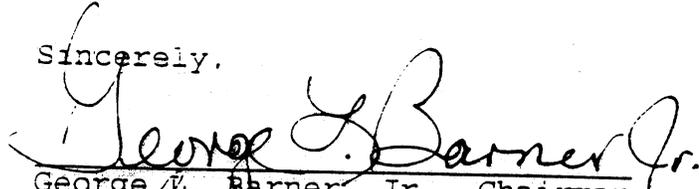
We are also concerned about the lack of integration with the Lake Lawrence management plan. The comprehensive study of this lake revealed very high nutrient loading rates - which could be severely exacerbated by the rotenone treatment.

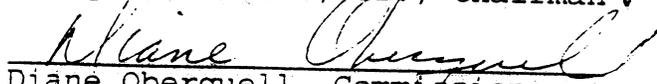
Thurston County encourages the development of comprehensive lake management programs. In the case of Lawrence and McIntosh Lakes, we encourage the Department to work with lake residents, fishing groups and other agencies to develop clear objectives for fishery management which are compatible with multiple-use lake management. Techniques could then be selected which would best meet fishery management and other objectives in the long term, with least disruption of the environment. Thurston County - and many lake residents - would be very willing to assist with developing and implementing such a program.

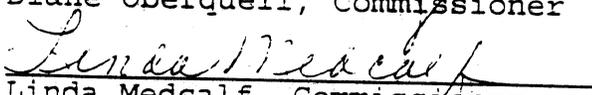
We welcome the Department of Wildlife's role in providing productive sport fisheries for our State's residents. However, we are very concerned that lake fishery management be conducted using a wholistic, ecological approach. We strongly oppose the repeated use of rotenone to turn these natural systems into virtual trout ponds. We also urge that you carefully consider comments made by lake residents at your recent public hearing.

Please contact our office or Tom Clingman at Thurston County Public Works, 786-5485, if you have any questions or would like to have a meeting on this issue.

Sincerely,


George E. Barner, Jr., Chairman


Diane Oberquell, Commissioner


Linda Medcalf, Commissioner

C:

Bill Freymond, WDW

Lorena Lindley, Sec't, Lake Lawrence Improvement Club

Mark Swartout, Office of Community and Environmental Programs

8-7-92

Dear Greg Tueckel,

I write to you as a concerned citizen regarding the proposed poisoning of Buck Lake and Koeneman Lake. The time has long passed that the practice of lake poisoning be removed from Department of Wildlife policy.

My training & degree is in natural sciences from the University of Washington. My readings and research conclude the entire food chain for plant and animal life is negatively impacted by lake poisoning.

#1 I urge you to decide against the proposed October poisoning of Buck and Koeneman Lakes.

#2 (at present I am a team leader in an eight month observation of a rich beaver pond biome south of Buck Lake. The Hansville Environmental Committee has sponsored the evaluation of Buck Lake and the 2 connected beaver ponds south of Buck Lake for the purposes of wetlands designation. We must do all that is possible to maintain the stable quality of the waters and adjacent habitats in their present balance.

#3 There are species designated by the state to be protected that are dependent on the waters of Buck Lake and the 2 beaver ponds for their fish and amphibian food sources. It is critical to the total food chain

the poisoning not be done.

Another coming trauma to the Buck Lake habitat is the clear cutting proposed for the west and south shores. Even though a buffer is proposed the lake will be adversely impacted.

Too much - for the large picture - is at stake to poison Buck Lake.

Sincerely,

Marion D. King

2800 N. E. Cedar Road

Hingston,

Washington

98346

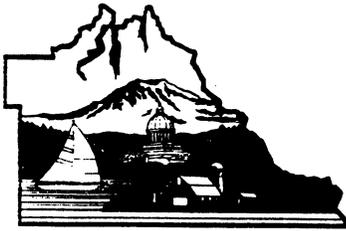
8-12-92

Dear Greg Hueckel,

#1 I am writing to you about the Dow plan to "sterilize" Buck Lake in Kitsap County. I think it is an error to manage a lake for "trout only." I think it is wise to view the lake as an entire ecosystem which includes the plankton & insects as well as the many species of birds. I fear ~~in~~ the plankton & the insects will also be killed which ~~is~~ will impact those animals which eat them.

#2 I want you to reverse your decision to poison the lake. Further, I want you to reevaluate Dow policy which would allow the poisoning of any lake. We simply must look at whole systems when we are making management decisions.

Sincerely,
Mike Eir Ouster
P.O. Box 224
INDIANOLA, Wa. 98342



THURSTON COUNTY
WASHINGTON
 SINCE 1852

COUNTY COMMISSIONER
 George L. Barner, Jr.
 District One
 Diane Oberquell
 District Two
 [Redacted] Linda Medcalf
 District Three

**PUBLIC HEALTH AND
 SOCIAL SERVICES DEPARTMENT**

Patrick M. Libbey, Director
 Diana T. Yu, MD, MSPH

July 30, 1992

Greg Hueckel
 Department of Wildlife
 600 North Capitol Way
 Olympia, WA 98504

RECEIVED

AUG 03 1992

FISHERIES MGMT DIV

Dear Mr. Hueckel:

Thank you for the opportunity to comment on the supplemental programmatic environmental impact statement (SPEIS) on lake and stream rehabilitation. The following comments are consolidated from three county departments: Health, Public Works, and Office of Community and Environmental Programs.

General Comments

1. Description of Proposed Action and Scope of EIS

The draft SPEIS is a significant improvement over the past SEPA document used for this program. However, the document contains a great deal of data about specific aspects of rotenone treatments while failing to explore other alternatives as intended by SEPA. The WDW and interested reviewers of the EIS might be better served by basing the document in a more comprehensive description of problems and objectives, followed by a more thorough exploration of alternatives.

#1 On page 1, the draft document uses the term "manage" to refer solely to lakes with periodic rotenone treatment by WDW. This would appear to be too narrow to accommodate the actual interest and scope of activities of WDW: The agency "manages" all lakes where planting or other action is conducted. Optimally, the SPEIS should assess all alternative actions which may be utilized to address the various types of fish species management problems encountered by WDW in various waterbodies.

#2 The problems which the SPEIS "proposed action" would address are not clearly defined in the Justification section (page 6). "...Overpopulation with fish species outside...management emphasis" (p. 6) doesn't adequately describe the problems for the reviewer. In appendices regarding specific lakes, the following



appear to be the types of problems which need to be addressed by WDW in managing fish species:

- a.) Predation on planted fingerling trout by piscivorous fish;
- b.) Competition by overpopulation of planktivorous fish which reduces food source too low for adequate fingerling trout growth; and
- c.) Carp overpopulation which:
 - (1) Affects success of trout plants;
 - (2) Triggers excessive nutrient cycling from bottom feeding, and;
 - (3) Damages dabbling duck habitat.

3)
The focus of the document is on "elimination" as the sole management strategy for addressing competing fish species. Alternatives to rotenone are found inadequate (after very brief review) for achieving elimination of target species, with rotenone the preferred alternative. However, on page 112 the SPEIS explains that complete eradication is not likely to be achieved, with "improved fishery" identified as a more accurate goal. This is a crucial difference in establishing the criteria for evaluating alternative actions. These alternatives would compare more favorably if the goal is reducing rather than eliminating target species.

If the above three problems are an accurate grouping of issues, perhaps the SPEIS could then examine alternatives for each one. Each problem could be defined utilizing Integrated Pest Management (IPM) principles, including clear definition of the "injury" (damage caused) and "action level" (point at which action is necessary to avoid reaching the injury level.) The following is not intended to be an exhaustive list but does illustrate an alternate approach which might better meet the intent of SEPA to explore alternatives:

- a.) Predation on trout fry problem (alternative actions):
 - (1) Plant catchables
 - (2) Shift to warmwater management
 - (3) In-lake rearing of fingerlings to acclimate and increase size
 - (4) Plant fingerlings mid-lake to reduce predation on stressed fry by littoral-dwelling bass;
 - (5) Reduce numbers of predator fish via rotenone treatment.
 - (6) Integrated approach
 - (7) No action
- b.) Food source competition from planktivorous fish:
 - (1) Increase predation by increasing number of brown trout, bass, etc.
 - (2) Reduce overabundance of aquatic plants which limit predation and favor overpopulation;
 - (3) Complete drawdown;
 - (4) Rotenone treatment to reduce number of competing fish.

- (5) Integrated approach
- (6) No action

c.) Carp:

- (1) Netting/killing schools in shallows;
- (2) Fish derby/bounty;
- (3) Partial drawdown;
- (4) Rotenone with strong and/or double treatment.
- (5) Integrated approach
- (6) No action

Some of these techniques would need to be initiated at an early stage ("action level") in contrast to periodic use of rotenone after conditions are highly degraded. Again, the focus on "elimination" (which may not be achievable) leads to dismissal of all alternatives rather than exploration of alternatives and disclosure of potential environmental impacts. The alternative of removing congregations of spawning fish (page 2) is noted as requiring repeated action. However, this is also the case for rotenone treatment - it must periodically be repeated.

#4
Exploration of the "no action" alternative also should be included: In many cases, the same factor which is degrading the sport fishery may also degrade the lake ecosystem (ex. over-predation on zooplankton) if no action is taken.

2. Evaluation approach

#5
The SPEIS should use a uniform evaluation approach throughout the document. While portions of the SPEIS, such as the sections on phytoplankton and zooplankton, present the reader with a large array of results and interpretations of many individual studies, the sections on environmental fate and toxicity rely heavily on other researchers' reviews. The level of detail and type of data presented (individual studies, studies with interpretations provided by WDW, tables, summaries, etc) should be consistent. Enough information should be presented to provide the reader with an understanding of the issues involved and bases for conclusions reached.

Further, the SPEIS should adopt a reasonable worse-case approach to explore possible adverse effects. The existing approach is based largely on "best-cases," resulting in a document in which the potential hazards are understated and minimized. For example, permanent species shifts, permanent species losses, and changes in community structure are documented in several of the studies cited. Without exception they are discounted due to poor sampling technique, sample design, or unusual circumstances. Using a reasonable worse-case approach, these effects would be considered rather than discounted.

3. Public Process

WV The "pre-treatment process" might be improved through a more direct approach including local groups and local governments involved with lake management: First build consensus on broad management objectives for the lake fishery using an IPM approach; then explore all options and define the best management actions. WDW management plans for the lake -- whether rotenone or other technique(s) -- would then hopefully have concurrence of the working group. See also the letter from Board of County Commissioners dated July 8.

4. Data gaps and uncertainty

Any discussion of significant data gaps -- the information needed but unavailable to make a thorough evaluation of the proposal -- is lacking. Data gaps should be clearly identified, and the cost and feasibility of obtaining the information should be evaluated. Uncertainties should also be identified, to assist decision-makers in distinguishing known and uncertain risks.

5. Rotenone product chemistry (contaminants, metabolites, degradation products, and inert ingredients)

#8 Additional information on rotenone should be included in the SPEIS: a) The toxicity, persistence, and environmental fate of degradation products. b) The toxicity of the metabolites. c) A discussion on possible contaminants. According to the Science Chapters for registration of rotenone, the manufacturing process uses trichloroethylene and carbon tetrachloride. Has the product been tested for residual levels of these compounds? The Science Chapters also noted rotenone crystals are 72 percent rotenone. What is known about the remaining 28 percent? d) The composition and toxicity of inert ingredients. What efforts has WDW made to try to identify the inerts?

Specific Comments

#9 p. 9 Description of rotenone treatment procedures. Typical Washington rotenone dosage rates should be described here, to assist reviewers with comparing dosages used in various studies to common dosages in WDW treatments.

#10 p. 12 Air. The discussion should be expanded beyond odor concerns. It should assess the potential for workers, nearby residents (including children), persons pursuing recreational activities in or near the lake, and wildlife being exposed to air-borne rotenone powders from typical application techniques and accidental spills.

p. 16 Residual Toxicity in Drinking Water. This section states that rotenone residues must be removed to produce a finished drinking water of good quality. Options to treat surface water supplies are mentioned. However, most residents in Thurston County obtain their drinking water from ground water. Many homes are on individual wells. Yet no mention is made of options available to treat ground water supplies. The likelihood of rotenone entering ground water in lakes which flow into aquifers should be evaluated. For example, what risk would residents using sand point wells have of drinking rotenone contaminated water (given the increased persistence of rotenone in anaerobic aquatic environments)?

#11
Instead of including a broad statement that rotenone "breaks down quickly in the environment," a more detailed discussion of the results from field use would be more useful in estimating risk. For example, what would be the expected range of the half-life of rotenone in the mud in the bottom of a lake? In aerobic versus anaerobic conditions? Does the half-life vary with the size of the lake, clarity of water, temperature, etc?

#12
p. 17, 2nd paragraph. Why is California's Action Level cited here? It seems a more appropriate level to mention is the Acceptable Daily Intake. There is also a typographical error. The factor of 1.000 should be 1,000.

#13
p. 18 Plants. Effects on macrophytes should be discussed (e.g., possible increases in macrophyte growth resulting from decreased turbidity, effects resulting from changing nutrient balance, etc.)

#14
p. 123 Birds. Temporary loss of forage for birds feeding on lake fish is mentioned. However, no mention is made of the possible direct toxicity to birds from eating fish with rotenone residues. In one study submitted for EPA's registration process, bluegill sunfish had a bioconcentration factor of 3,607x in viscera. While rotenone may only be slightly acutely toxic to the bird species studied, 3,607 x 0.25 ppm is a fairly high concentration. What are the LD50s for the piscivorous species mentioned in the last paragraph on page 123? If they are unknown, how can the hazard or the risk to these species be adequately assessed? The implications of bioconcentration factors should be included in estimating exposure.

#15
p. 125 Mammals. Similar to the above comments about birds, the discussion of mammalian oral toxicity may underestimate the actual exposure by not considering elevated concentrations in dead fish. The exposure should be recalculated and the risk re-evaluated.

#16
On page 125 is also the statement "To produce subacute effects such as weight loss or liver damage also requires very high dosages fed continuously in the diet for many months." Yet the associated table on page 133 lists weight loss or liver damage at

0.4 mg/kg, 10 mg, 50 ppm, 100 ppm, and 130 ppm. By what measure is 0.4 mg/kg considered a very high dosage?

#17
The statement "The EPA (1981) considers it safe to water livestock with rotenone-treated water" is misleading. FIFRA prohibits any manufacturer from making any claim that a pesticide is "safe." The registration process is not a measure of "safety" but rather benefits and risks.

#18
Last paragraph. More detail should be provided on the potential impact to mammalian species. (1) Are mink, otters, and water shrews the only mammals which rely on the fish and invertebrates of a lake? (2) How may water shrews be indirectly affected? (3) If otters rely almost entirely on fish for food, stating that the temporary loss of prey following treatment "may" disturb them seems an understatement -- especially when the lake is not restocked for several months. Unfortunately, the next sentence in the paragraph does not clarify. "But otters forage widely, sometimes travelling 50-60 miles during a year (Banfield, 1974), and would may not be displaced permanently." Does this refer to other otters coming in as replacements or resident otters moving to other waterbodies? If the latter, given that the niche may already be filled, the potential to displace other otters should be discussed. (4) What are the effects on the rest of the system when fish-eating mammals disappear?

#19
p. 127, Human Health. Almost no discussion of the risk to applicators is included. While the focus of this section is understandably the larger public, a discussion (similar to the one on page 131, 3rd paragraph) of the signs and symptoms experienced by applicators after using rotenone should be included.

#20
The residue levels in fish are likely underestimated because bioconcentration is not included in the calculations of exposure. Using the highest bioconcentration factor submitted to EPA, potentially a 0.25 ppm level in lake water could result in a 34 ppm level in fish, which is far in excess of California's suggested ADI level of 0.0004 mg/kg/day.

#21
If the statement "The original use of rotenone-bearing plants in South America was the collection of fish for the table..." is offered to assure the reader of the safety of rotenone, the supporting documentation of the lack of any health problems from this practice should be included. Just because a practice is historical does not mean it is safe. Tobacco has been used as a stimulant for centuries. That does not lessen its cancer-causing properties.

#22
Last paragraph. The SPEIS cites Cohen's conclusion in 1960 that "the use of rotenone to kill fish in public reservoirs was consistent with the objective of safe and potable water." Standard methods of weighing risks and benefits have changed significantly since 1960. Further, insufficient data is provided

to document the hazard, exposure, or risk to the public from drinking water containing rotenone. The SPEIS should re-evaluate this issue independently, rather than rely on a 1960 review.

#23
p. 129 The SPEIS states "Municipal water supplies have been treated...in at least six states with no harmful effects." How was it determined that no harmful effects occurred? It seems unlikely that epidemiological studies were performed. Did the water suppliers interview consumers? Make note of any complaints received? The statement is overly broad as it stands.

#24
p. 131. Similar to previous comment on livestock. The statement "The U.S. Environmental Protection Agency (1981) considers it safe to swim in water treated with rotenone" should be modified or deleted. The registration process is not a measure of "safety" but rather benefits and risks.

#25
The last paragraph of page 131 mentions the "low mammalian toxicity" of rotenone. Yet EPA's reregistration guidance document classifies rotenone in Toxicity Category I (most toxic) "because of its high toxicity" -- listed as 39.5 mg/kg for female rats and 102 mg/kg for male rats.

#26
Last paragraph. Relying on another reviewer's general conclusion that "the margin of safety is so great that water would be safe for swimming and other recreational use" is difficult to support when data are not presented to allow calculations of margins of safety.

#27
p. 132. Results from mutagenicity studies should also be reviewed and evaluated. The relative weights of the positive studies (such as DNA breakage, micronucleus test in mouse cells, gene mutation in mouse lymphocyte cells, sister chromatid exchange in hamster ovary cells) versus the negative studies (such as bacteria gene mutations, unscheduled DNA synthesis in human fibroblast cultures, rat hepatocyte assay) should be compared.

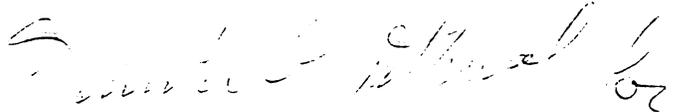
The discussion of developmental and reproductive effects should be broadened to include studies (see enclosed references) which have found adverse effects not mentioned such as: decreased live birth, nerve damage, neural tube defects, increased incidence of unossified sternebrae, urinary tract abnormalities, decreased litter size, and increased incidence of extra ribs. These studies do not support Marking's statement that "even high doses of rotenone do not cause tumors or reproductive failure, nor adversely affect fetal development."

#28
3. Appendix E page 37: Lake Lawrence Management Plan - Extensive data exists for Lawrence Lake which should be summarized in this document: fish population and growth rates, creel census, and fish/effort ratios (Thomas et al. 1990 Feasibility of Aquatic Plant Control in Lake Lawrence, Washington Using Triploid Grass Carp, UW School of Fisheries) Also, more

complete and recent phosphorus loading and water budget data are available (Gibbons et al. 1991 Lake Lawrence Phase I Lake Restoration Analysis, Thurston County.)

If you have any questions, please contact Marie Zuroske with Health (786-5457), Tom Clingman with Public Works (786-5485), or Mark Swartout with Community and Environmental Programs (754-4111). We look forward to your response.

Sincerely,



Paula Ehlers
Environmental Review Officer

Enclosures

cc: Tom Clingman
Marie Zuroske
Mark Swartout

**Response to Mr. Ben Schroeter, Ben and Jerry's Paralegal Services,
P.O. Box 2856, Olympia, WA 98507-2856 (letter dated July 30, 1992):**

1). Legal Authority - I consulted with the Office of Attorney General concerning the question of the Wildlife Commission's authority to approve the use of rotenone to rehabilitate lakes. Their response was that the wording changes made by the 1987 Legislature were housekeeping, non-substantive changes, and were not intended to restrict the Commission's ability to approve rotenone for this use. On page 9, the first sentence under Legal Standing now reads:

"RCW 77.12.420 empowers the Wildlife Commission to eradicate "undesirable types of fish."

2). Concerning your reference to the inert ingredients in liquid formulations of rotenone, the Minnesota Department of Health conducted a risk assessment of these ingredients in Nusyn-Noxfish from the Minnesota Department of Natural Resources. Their assessment determined that "There is negligible risk to human health from the contaminants found in the rotenone whether the exposure is from drinking, swimming, or eating fish from treated waters. Treatment with rotenone will introduce the contaminants into the lakes, but at concentrations considerable lower than a level that would harm human health."

3). You are on the mailing list to receive all publications of future Draft and Final Environmental Impact Statements on Lake and Stream Rehabilitations. We have requested from the Department of Ecology permits for Water Quality Modifications on Burke, Quincy, Upper Caliche, H, Ancient, and Stan Coffin Lakes and an unnamed lake in Desert Unit of the Columbia Basin TWN(18N), RGE(26E), SEC(11,14) in Grant County, Bingen Lake in Klickitat County, and Buck Lake in Kitsap County.

**Response to Mr. George Draffan, P.O. Box 95316, Seattle, WA 98145
and Ms. Janine Blaelock, 7040 14th NW, Seattle, WA 98117-5308
(letter dated August 13, 1992):**

1). Your names have been put on our mailing list to receive all future Supplemental Environmental Impact Statements on Lake and Stream Rehabilitations from the Washington Department of Wildlife. I apologize for you not receiving copies this year at the time of issue.

2). Terminology: WDW uses the term "rehabilitation" because we use rotenone to restore fisheries in the state's waters to optimum production. This term is used throughout the United States by other natural resource agencies who also treat their state's waters with rotenone.

3). Long-Term Effects of Rotenone on the Lake Community:

Comments noted. WDW believes there is sufficient information in the Environmental Impact Statement which documents the impacts of rotenone on the lake community in most cases. If there are data gaps concerning potential impacts in waters which may be of special concern, WDW will collect the necessary information to monitor those impacts, as time and resources allow.

4). Inert Ingredients:

The Minnesota Department of Health (MDH) analyzed the impurities of the rotenone stock from the Minnesota Department of Natural Resources (MDNR). The MDNR obtains their rotenone from the same supplier as the WDW. The MDH identified the following compounds in the liquid formulation of rotenone Nusyn-Noxfish: 1) Trichloroethene (740 mg/kg), 2) Tetrachloroethene (90 mg/kg), 3) n-Propylbenzene (430 mg/kg), 4) 1,3,5-Trimethylbenzene (890 mg/kg), 5) 1,2,4-Trimethylbenzene (2950 mg/kg), 6) Ethylbenzene (260 mg/kg), 7) m/p-Xylene (990 mg/kg), and 8) o-Xylene (560 mg/kg).

5). Justification of the Program:

Comments noted. Justification of the proposed action is found on pages 6-7. Fisheries management plans for the individual waters proposed for rehabilitation are detailed in Appendix E.

Response to Thurston County Commissioners, Building #1, 2000 Lakeridge Drive SW, Olympia, WA 98502-6045 (letter dated July 8, 1992):

Following the agency's internal review process, and the public meeting in Rainier, we have decided to withdraw our proposals to treat Lawrence and McIntosh Lakes.

We agree with you of the necessity to work with all interested parties on lake management plans. At this time, we will continue to manage these lakes as mixed species waters, supplementing the trout populations with catchable size fish from our hatcheries in early spring as hatchery space and funds will allow. Because of the extra costs required to raise trout to catchable size, the decision not to rehabilitate these lakes may have a significant impact on future trout fishing in these waters if our agency's budget continues to decline.

Response to Mr. Stephen L. Saunders, Washington State Department of Ecology, Mail Stop PV-11, Olympia, Washington 98504-8711 (letter dated August 7, 1992):

1). Comments noted. We have added to this section as follows: Fishing regulations are liberalized, when possible and upon approval by the Wildlife Commission, to utilize fish in waters scheduled for rehabilitation. Warmwater game fish, usually mature bass, are collected (depending on need) prior to rehabilitation, to be utilized as broodstock for waters nearby which are managed for warmwater fisheries. On some lakes, bass that have floated to the surface have been netted by WDW employees and bass club volunteers, revived by dipping the fish in potassium permanganate, and moved to mixed-species or spiny-ray lakes to augment or start a population (Fletcher, 1976). WDW has typically transplanted 200-300 fish from a single lake during this type of procedure.

2). We have added the following sentence to this section: "The use of potassium permanganate also requires a short-term modification (permit) to the Water Quality Standards issued by the Washington Department of Ecology."

We will further expand this section with the potential impacts of potassium permanganate when we propose to use it in conjunction with rotenone. We do not propose to use potassium permanganate for the rehabilitations we are proposing for 1992-1993.

3). WDW will work closely with DOE to collect abundance and composition information of phyto- and zoo-planktonic communities in waters which may be of special concern as time and resources allow.

4). WDW will work closely with DOE to measure phosphorus levels in waters which may be of special concern as time and resources allow.

5). WDW agrees, and we will continue our search of the scientific literature for more studies on how water quality is affected by lake rehabilitations.

6). WDW will work closely with DOE to measure macrophyte composition and abundance in waters which may be of special concern as time and resources allow.

#7). We have added the following sentences to the end of 3): "However, in a deep lake with a coarse or gravelly substrate, turbidity from bottom-scavenging fish is not likely to be a problem. It is possible that nutrient re-suspension resulting in bloom conditions following a rehabilitation can reduce water transparency, although no studies were found to substantiate this speculation."

#8). We understand this concern. However, the WDW proposes to use minimal amounts of the liquid formulation of rotenone only in densely vegetated areas where fish may hide. Using only the powder, fish can more easily escape the treatment, which increases

the frequency of rehabilitations we must propose. We are currently experimenting with applying the powdered formulation in a "slurry" (following procedures used in other states) to further minimize our use of the liquid formulation.

Additionally, the Minnesota Department of Health conducted a risk assessment of the inert ingredients found in the liquid formulation of rotenone Nusyn-Noxfish from the Minnesota Department of Natural Resources. Their assessment determined that "There is negligible risk to human health from the contaminants found in the rotenone whether the exposure is from drinking, swimming, or eating fish from treated waters. Treatment with rotenone will introduce the contaminants into the lakes, but at concentrations considerably lower than a level that would harm human health."

9). On the pre-rehabilitation planning form, the phrase "Identify whether water intakes exist (legal or illegal)" has been added to IV. RESOURCE IMPACTS, (2).

10). We have changed this sentence to read:

"In annual stocking of trout-only lakes in Washington state, no changes in any water quality parameter would be expected beyond those which have historically occurred as part of similar previous rehabilitation and stocking efforts."

11). We understand your concern because of this conflicting information. We will work closely with DOE to conduct pre- and post-treatment surveys of macrophytes in waters of special concern as time and resources allow.

12). This statement now reads:

"Since no phosphorus budgets exist for the other western Washington lakes in Table E, ..."

13): WDW shares DOE concerns. We will work closely with DOE to monitor algal composition in waters of special concern as time and resources allow.

14): WDW does not propose to rehabilitate any oligotrophic or alpine lakes during 1992-1993.

15): WDW will work closely with DOE to conduct pre- and post-treatment surveys of macrophytes in waters of special concern as time and resources allow.

16): WDW will work closely with DOE to conduct pre- and post-treatment surveys of snail populations in waters of special concern as time and resources allow.

17): WDW no longer rehabilitates streams which are not directly connected to targeted lake or pond waters. These streams are normally short "waterways". The last stream-only rehabilitation conducted in Washington was in 1988 when WDW, in conjunction with DOE, treated Rocky Ford Creek in Grant County.

18): WDW has expanded the pre-rehabilitation and management plans from previous years to include this information.

19): Typo corrected.

20): The Non-game Program of the Washington Department of Wildlife reviews all proposed rehabilitations. They check the statewide distribution list of threatened or endangered species as part of their review.

Response to Ms. Barbara Fournier, 6959 NE Buck Lake Rd, Hansville, WA 98340 (letter dated July 30, 1992):

1). The decision to use rotenone was based on decreased rainbow trout fry survival and the presence of a significant population of largemouth bass. Based on our experience with lowland lake fisheries management, the presence of largemouth bass, who feed voraciously on trout fry, is the probable cause for the decline in the trout fishery.

2). Bass are indeed returning to the system. Either they are being illegally planted (it is illegal to plant fish into the state's waters without first obtaining a fish planting permit from the Washington Department of Wildlife), or the past treatments have not completely eradicated the populations.

3). The Department of Wildlife uses alternative methods of management in over 1 million acres of water throughout the state. These methods are detailed in the Draft Supplemental Environmental Impact Statement. To manage Buck Lake as trout-only, we see no other feasible alternative than to use rotenone.

4). We are aware of the wildlife present in the Buck Lake area, and will do everything possible to minimize the impacts to these species. Our wildlife management biologists who have reviewed the pre-rehabilitation plan do not believe the rotenone treatment will create significant impacts on wildlife living on or near the lake. We will be applying the rotenone in October when the majority of birds have migrated from the area, and the populations of insects are at their lowest. We are replanting the lake with trout fry in the early spring, which will replenish the lake with fish lost during the rehabilitation. We anticipate insects will also be present once again in the lake during this time. Trout, as well as returning birds, depend upon these insects as a primary food source.

5). Based on our knowledge of trout fisheries, anglers never seem bothered with crowded conditions if fishing is good. Based on our angler surveys, catching fish is one of many reasons people choose to go fishing.

6). Based on our surveys of anglers, people will drive from all parts of the state to fish in waters which provide productive trout fishing.

7). Comment noted.

8). See comment listed under 4) above.

**Response to Ms. Sue Koenig, P.O. Box 393, Indianola, Wa. 98342
(to letter dated July 29, 1992):**

1). We are aware of the wildlife present in the Buck Lake area, and will do everything possible to minimize the impacts to these species. Our wildlife management biologists have reviewed the pre-rehabilitation plan and has assessed the rotenone treatment of Buck Lake would not create significant impacts to the wildlife which utilizes the lake and surrounding habitats. The fact that Buck Lake was rehabilitated in 1986 and the numerous wildlife species and populations you have sighted also indicates that any potential impacts are temporary.

2). We do not anticipate a large amount of fish being killed during this rehabilitation. We also feel the risk of oral toxicity is minimal, based on the quick breakdown of rotenone in the environment.

3-5). We are aware of the wildlife present in the Buck Lake area, and will do everything possible to minimize the impacts to these species. Our wildlife management biologists who have reviewed the pre-rehabilitation plan do not believe the rotenone treatment will create significant impacts on nesting eagles, swallows, blackbirds or woodland birds, and other wildlife living on or near the lake. We will be applying the rotenone in October when the majority of birds have migrated from the area, and the populations of insects are at their lowest. Eagles normally feed on salmon during this time. We are planting the lake with trout fry in the early spring, which will replenish the lake with fish lost during the rehabilitation. We anticipate insects will also be present once again in the lake during this time. Trout, as well as returning birds, depend upon these insects as a primary food source.

6). The Department of Wildlife manages the state's lowland lakes to maximize and diversify recreational opportunity. The Department manages over 1 million acres of mixed species (trout and warmwater fish) waters statewide. Trout-only waters, such as Buck Lake, produce at least four times more trout than those managed as mixed species. Additionally, mixed species waters require planting

catchable sized trout, which are more expensive to raise than the fry planted in trout-only waters. It is unlikely that trying to overfish Buck Lake with hook-and-line gear will allow the Department to manage this water as trout-only.

Response to Ms. Sue Koenig, P.O. Box 393, Indianola, WA 98342 (letter dated August 15, 1992):

1). Targeted lakes are researched for the presence of any state or federally listed species during the review process of the pre-rehabilitation and management plans. We have withdrawn our proposal to rehabilitate Koeneman Lake because of a Western pond turtle sighting by a Woodland Park Zoo official. We will substantiate this sighting by conducting surveys with our fish and non-game biologists prior to making a future proposal to rehabilitate this lake.

2). Your idea to choose some key lakes to conduct intensive research on the impacts of rehabilitations to fill data gaps on terrestrial animals is an excellent one, and one that I will pursue through the University of Washington Cooperative Fishery Research Unit at the School of Fisheries. In the meantime, we are committed to conduct before and after rehabilitation surveys on waters which may have special concerns as time and resources allow.

Response to Lake Lawrence Improvement Club, 16646 Pleasant Beach Dr., Lake Lawrence, WA 98597 (letter dated July 22, 1992):

Following the agency's internal review process, and the public meeting in Rainier, we have decided to withdraw our proposals to treat this lake.

The Department of Wildlife will continue to manage these lakes as mixed species waters, supplementing the trout populations with catchable size fish from our hatcheries in early spring as hatchery space and funds will allow. Because of the extra costs required to raise trout to catchable size, the decision not to rehabilitate these lakes may have a significant impact on future trout fishing in these waters if our agency's budget continues to decline.

Response to Ms. Marion Kling, 8800 N.E. Ohman Road, Kingston, WA. 98346 (letter dated August 7, 1992):

1). We have removed our proposal to rehabilitate Koeneman Lake for 1992-1993 because of a Western pond turtle sighting by a Woodland Park Zoo official. The Western pond turtle is a threatened species which may be detrimentally impacted by rotenone. Our fish and non-game biologists will conduct surveys throughout the next year to try to verify this sighting. Our future proposals to rehabilitate Koeneman will be dependent on these surveys.

2). We are aware of the wildlife present in the Buck Lake area, and will do everything possible to minimize the impacts to these species. Our wildlife management biologist has reviewed the pre-rehabilitation plan and has assessed the rotenone treatment of Buck Lake would not create significant impacts to the wildlife which utilizes the lake and surrounding habitats. The fact that Buck Lake was rehabilitated in 1986 and the numerous wildlife species and populations at the lake also indicates that any potential impacts are temporary.

3). We will be applying the rotenone in October when the majority of birds have migrated from the area, and the populations of insects are at their lowest. We are planting the lake with trout fry in the early spring, which will replenish the lake with fish lost during the rehabilitation. We anticipate insects will also be present once again in the lake during this time. Trout, as well as returning birds, depend upon these insects as a primary food source.

Response to Ms. Nike Eir Quester, P.O. Box 224, Indianola, WA 98342 (letter dated August 12, 1992):

1). The Department of Wildlife manages the state's lowland lakes to maximize and diversify recreational opportunity. The Department manages over 1 million acres of mixed species (trout and warmwater fish) waters statewide. Trout-only waters, such as Buck Lake, produce at least four times more trout than those managed as mixed species. Additionally, mixed species waters require planting catchable sized trout, which are more expensive to raise than the fry planted in trout-only waters.

2). We are aware of the wildlife present in the Buck Lake area, and will do everything possible to minimize the impacts to these species. Our wildlife management biologists have reviewed the pre-rehabilitation plan and has assessed the rotenone treatment of Buck Lake would not create significant impacts to the wildlife which utilizes the lake and surrounding habitats. The fact that Buck Lake was rehabilitated in 1986 and the numerous wildlife species and populations at the lake also indicates that any potential impacts are temporary.

We will be applying the rotenone in October when the majority of birds have migrated from the area, and the populations of insects are at their lowest. We are planting the lake with trout fry in the early spring, which will replenish the lake with fish lost during the rehabilitation. We anticipate insects will also be present once again in the lake during this time. Trout, as well as returning birds, depend upon these insects as a primary food source.

Response to Ms. Paula Ehlers, Environmental Review Officer, Social Services Division, Thurston County, 529 Fourth Avenue W., Olympia, WA 98501-1097 (to letter dated July 30, 1992):

1). This sentence is accurate as written. The WDW actively manages 5.9% of the states lowland lakes. The third sentence in the first paragraph further explains that "...elimination of non-game or competitor species in a portion of these lakes ...".

2). Comments noted. We have added the following to the Justification Section on page six to serve as examples of our goal to manage fisheries and waterfowl in the state's lowland lakes:

"Occasionally, these waters become overpopulated with fish species outside this management emphasis. This often results in increased predation and/or competition, hence poor growth and survival, of targeted game fish. If carp overpopulate, fish survival decreases and nesting bird habitat is degraded due to siltation and uprooting of emergent vegetation."

3). While the chances of complete eradication of fish decreases as lake size increases, our goal of complete eradication does not change. We do not treat lakes for complete eradication whose size would prohibit us from obtaining our goal.

4). We believe we have listed and discussed in sufficient detail on pages 1-5 the alternatives (many of which we currently use) to using rotenone for game fish management.

5). Comments noted. The level of detail varied throughout the document with respect to the number and detail of scientific publications we found which dealt with the particular issues.

6). Comments noted.

7). Comments noted. If there are data gaps in potential impacts in waters which may be of special concern, WDW will collect the necessary information to monitor those impacts as time and resources allow.

8). The Minnesota Department of Health (MDH) analyzed the impurities of the rotenone stock from the Minnesota Department of Natural Resources (MDNR). The MDNR obtains their rotenone from the same supplier as the WDW. The MDH identified the following

compounds in the liquid formulation of rotenone Nusyn-Noxfish: 1) Trichloroethene (740 mg/kg), 2) Tetrachloroethene (90 mg/kg), 3) n-Propylbenzene (430 mg/kg), 4) 1,3,5-Trimethylbenzene (890 mg/kg), 5) 1,2,4-Trimethylbenzene (2950 MG/KG), 6) Ethylbenzene (260 mg/kg), 7) m/p-Xylene (990 mg/kg), and 8) o-Xylene (560 mg/kg).

The MDH also conducted a risk assessment of the inert ingredients found in liquid formulation of rotenone Nusyn-Noxfish from the MDNR. Their assessment determined that "There is negligible risk to human health from the contaminants found in the rotenone whether the exposure is from drinking, swimming, or eating fish from treated waters. treatment with rotenone will introduce the contaminants into the lakes, but at concentrations considerably lower than a level that would harm human health."

9). The following sentence has been added to the section under Treatment Procedures:

"Common dosages of rotenone (5%) in lakes treated in Washington ranges between 1-4 ppm."

10): The following sentences has been added to the section under AIR:

"Rotenone droplets or mist may be carried in the air from the liquid applications. Powder rotenone is applied by towing an open sack underwater, so escape of particles in the air should be minimal."

11): A thorough, detailed discussion on the detoxification of rotenone appears in Appendix B, pages 4-5.

12): California has been the most conservative state with regards to the use of rotenone for fish management purposes over the past 10 years. Typographical correction noted and corrected.

13): Effects of fish removal on aquatic macrophytes is discussed on page 24. The WDW will monitor macrophytes in rehabilitated lakes which are of special concern as time and resources allow.

14): We have listed the LD50's for many different types of birds in Table R. We believe the information presented in this Table, along with the discussion presented on Page 123 to adequately address the potential impacts to birds which may feed on fish killed during lake rehabilitations.

15): We believe the information presented on the page adequately addresses the potential impacts to mammals which may feed on fish killed during lake rehabilitations.

16): The results of long-term oral dosages of rotenone on dogs you refer to in Table U, on Page 133, demonstrated that 0.4 mg/kg had no effect on dogs following 180 days of daily treatment. The EIS is referring to only those dosages which had an impact on the health of mammals. In Table U, the lowest dosage which had an impact was 2.0 mg/kg pure rotenone fed to dogs over a 180 period.

A 10 pound (22 kg dog) would have to eat 44 kg of pure rotenone over 180 days to have the same impacts. This dosage is well beyond that found in fish killed during a rehabilitation, as detailed on page 127.

17): We are citing a memorandum from the EPA on the completion of pre-RPAR review of rotenone from Marcia Williams (Director, Special Pesticide Review Division) to Douglas Campt (Director, Registration Division), June 22, 1981.

18): We believe the information presented on this page adequately addresses the potential impacts to mammals which may feed on fish killed during lake rehabilitations.

19): The section on Acute Respiratory Toxicity and Symptoms of Acute Rotenone Poisoning on pages 129-132 adequately covers, and also refer to, potential impacts to applicators of rotenone.

20): Comment noted. We feel these concentrations are appropriate to use, even without bioaccumulation (what if only one fish is eaten?) to speculate on risk, since we are not allowing for probable losses of rotenone through natural degradation and cooking.

21): The statement "The original use of rotenone-bearing plants in South America was the collection of fish for the table." is stated because it is fact, and sheds light on the historical use of rotenone.

22): Comment noted. We are actively working to expand on the literature supporting this, and future EIS's for Lake and Stream Rehabilitations.

23): This statement is made without supporting literature. We have removed it from the EIS.

24): We are citing a June 28, 1981 memorandum from the EPA on the pre-RPAR review on rotenone. We do not feel justified to change its contents.

25): Comment noted. However, this was what Dawson (1991) concluded.

26): Comment noted. However, this was what Dawson (1991) concluded.

27): Comment noted. We are actively working to expand on the literature supporting this, and future EIS's for Lake and Stream Rehabilitations.

28): We are no longer proposing to rehabilitate Lake Lawrence during 1992-1993.

Chairman, Ecological
Commission
Department of Ecology
MS: PV-11
Olympia, WA 98504

David Kile, Assistant
Director
Agricultural Development
406 General Admin. Bldg.
Olympia, WA 98504

Hugh Fowler, Assistant
Director
Dept. of Emergency Management
4220 E. Martin Way
Olympia, WA 98504

Steven Fansen
Skagit System Coop.
P.O. Box 368
Reservation Rd.
La Conner, WA 98257

Greg Sorlie, Supervisor
Environmental Review
Dept. of Ecology
MS: PV-11
Olympia, WA 98504

Environmental Coordinator
Environmental Review
Dept. of Ecology
MS: PV-11
Olympia, WA 98504

Environmental Coordinator
Dept. of Transportation
Hwys. Admin. Bldg.
MS: KF-01
Olympia, WA 98504

Environmental Coordinator
Dept. of Natural Resources
Public Lands Bldg.
MS: QW-21
Olympia, WA 98504

Sam Reed, Chief
Office of Environmental
Programs
DSHS
MS: LD-11
Olympia, WA 98504

Office of the Governor
Legislative Bldg.
MS: AS-13
Olympia, WA 98504

Washington Dept. of Fisheries
115 General Admin. Bldg.
MS: AX-11
Olympia, WA 98504

Planning and Comm. Affairs
Agency
400 Capitol Center Bldg.
MS: FN-41
Olympia, WA 98504

Jeff Frost, Chief
Planning & Coordination Div.
Interagency Committee for
Outdoor Recreation
MS: KP-11
Olympia, WA 98504

David Heiser, Environmental
Coordinator
Parks & Recreation Comm.
MS: KY-11
Olympia, WA 98504

Environmental Coordinator
Office of Financial
Management
MS: AL-01
Olympia, WA 98504

Washington St. Library
Documents Section, Library
Bldg.
MS: AJ-11
Olympia, WA 98504

Environmental Protection
Agency
1200 - 6th Ave.
Seattle, WA 98101

Environmental Coordinator
U.S. National Park Service
83 S. King St. #212
Seattle, WA 98104-2848

Project Manager
U.S. Bureau of Reclamation
Ephrata, WA 98823

Donald Ricketts, Exec.
Secretary
WA St. Cattlemen's Assoc.
P.O. Box 96
Ellensburg, WA 98925

WA Fly Fishing Club
P. O. Box 639
Mercer Island, WA 98040

Washington Bass Assoc.
P.O. Box 574
Marysville, WA 98270

Ed Brezina, Chairman
Trout Committee
WA State Sports Council
15520 Grant Ave.
Tillicum, WA 98498

Frank Stricklin, Chairman
Bass & Panfish Committee
WA St. Sports Council
8300 Hideaway Lane NW
Silverdale, WA 98383-9314

Inland Empire Fly Fishing
Club
Box 2926, Terminal Annex
Spokane, WA 99220

Vim Crane Wright
Institute for Environmental
Studies
University of Washington
Seattle, WA 98155

Tom Deming
Puyallup Indian Tribe
6824 Pioneer Way W
Puyallup, WA 98371

Nels Hanson
WA Forest Protection Assoc.
711 Capitol Way, Suite 608
Olympia, WA 98501

Chairman
Sauk-Suiattle Indian Tribe
5318 Chief Brown Lane
Darrington, WA 98241

NWIFC
6730 Martin Way
Olympia, WA 98503

Jack Silvers, Master
WA State Grange
3104 Western Ave.
Seattle, WA 98121

Don Ahrenholtz, Exec. VP
WA Farm Bureau
P. O. Box 2009
Olympia, WA 98507-2009

Elliott Marks
Nature Conservancy
1601 2nd Ave., Suite 910
Seattle, WA 98101

Anne Mahnke
Audubon Society
619 Joshua Green Bldg.
Seattle, WA 98101

Green River Steelhead Trout
Club
P. O. Box 214
Auburn, WA 98071

WA Environmental Council
4516 University Way NE
Seattle, WA 98105

Bertha Simpson
Legislative Chairperson
WA St. Sports Council
437 N. Percival
Olympia, WA 98502

Bob Heirman, President
Snohomish Co. Sportsmen
1920 Lake St.
Snohomish, WA 98920

Okanogan Co. Planning Dept.
P. O. Box 1009
Okanogan, WA 98840

Linda Cattebay
Bonneville Power Admin.
905 NE 11th
Portland, OR 97208

Snohomish Co. Planning Dept.
Co. Admin. Bldg.
Everett, WA 98201

U.S. Dept. of Agriculture
Evergreen Plaza Bldg.
816 E. 5th
Olympia, WA 98502

Spokane Regional Planning
Conf.
N. 221 Wall St.
City Hall, Room 353
Spokane, WA 99201

Chairman, Tulalip Indian
Tribe
3901 Totem Beach Rd.
Marysville, WA 98270

Chairman, Muckleshoot Indian
Tribe
39015 172nd Ave. NE
Auburn, WA 98002

Chairman, Spokane Indian
Tribe
P. O. Box 385
Wellpinit, WA 99040

Jim Rochelle
Weyerhaeuser Technical Center
Tacoma, WA 98477

The Journal American
P. O. Box 310
Bellevue, WA 98009

The Columbia Basin Daily
Herald
P. O. Box 910
Moses Lake, WA 98837

Jim Torrence, Regional
Forester
U.S. Forest Service
P. O. Box 3623
Portland, OR 97208

Pierce Co. Planning Dept.
2401 South 35th St., Room 6
Tacoma, WA 98124

U.S. Army Corps of Engineers
Seattle Dist.
P. O. Box C-3755
Seattle, WA 98124

Chairman, Stillaguamish
Indian Tribe
2439 Stoluckaquamish
Arlington, WA 98223

Grant Co. Planning Dept.
P. O. Box 37
Ephrata, WA 98823

King Co. Planning Dept.
506 2nd Ave. #707
Seattle, WA 98104

Columbia Basin Fish &
Wildlife
Authority
Metro Center, Suite 170
200 SW First Ave.
Portland, OR 97201-5346

Joe Jaquet
1121 W. Jackson
Olympia, WA 98502

The Everett Herald
P. O. Box 930
Everett, WA 98206

The Seattle Times
P. O. Box 70
Seattle, WA 98111

The Tacoma News Tribune
P. O. Box 11000
Tacoma, WA 98411

The Omak Chronicle
P. O. Box 553
Omak, WA 98841

Pend Oreille Co. Planning
Dept.
P. O. Box 5000
Newport, WA 99156

Lincoln Co. Planning Dept.
P. O. Box 297
Davenport, WA 99122

Adams Co. Bldg. & Planning
Dept.
165 N. First St.
Othello, WA 99344

Puget Sound Water Quality
Authority
217 Pine St., Suite 1100
Seattle, WA 98101

Jefferson Co. Planning &
Bldg. Dept.
County Courthouse
Port Townsend, WA 98368

Grays Harbor Co. Planning &
Bldg. Dept.
P. O. Box 390
Montesano, WA 98563

Thurston Regional Planning
Council
Bldg. #1 Administration
2000 Lakeridge Dr. SW
Olympia, WA 98502

Whatcom Co. Planning Dept.
401 Grand Ave.
Bellingham, WA 98225

Skagit Co. Planning &
Community Development
County Administration Bldg.,
Room 204
Mt. Vernon, WA 98273

Lewis Co. Planning Dept.
P. O. Box 418
Chehalis, WA 98532

The Spokesman Review
P. O. Box 2160
Spokane, WA 99210

The Journal Times
P. O. Box 288
Ritzville, WA 99169

Stevens Co. Planning &
Community Development
P. O. Box 191
Colville, WA 99114

Yakima Co. Planning Dept.
County Courthouse, Room 417
Yakima, WA 98901

Clallam Co. Dept. of
Community Development
223 E. 4th St.
Pt. Angeles, WA 98362

Mason Regional Planning
Council
P. O. Box 186
Shelton, WA 98584

Pacific Co. Planning Dept.
P. O. Box 68
South Bend, WA 98586

San Juan Co. Planning Dept.
P. O. Box 947
Friday Harbor, WA 98250

Island Co. Planning Dept.
P. O. Box 698
Coupeville, WA 98239

Skamania Co. Planning Dept.
P. O. Box 790
Stevenson, WA 98648

Cowlitz/Wahkiakum
Governmental Conf.
Admin. Annex
207 4th Ave. N
Kelso, WA 98626

Klickitat Co. Planning Dept.
Courthouse Annex
228 W. Main St., Room 150
Goldendale, WA 98620

Kittitas Co. Planning Dept.
County Courthouse
Ellensburg, WA 98926

Douglas Co. Regional Planning
Comm.
110 Thrid St. NE
E. Wenatchee, WA 98801

Ferry Co. Planning Dept.
P. O. Box 305
Republic, WA 99166

Walla Walla Co. Regional
Planning Dept.
310 W. Poplar St., Suite 117
Walla Walla, WA 99362

Garfield Co. Board of
Commissioners
P. O. Box 278
Pomeroy, WA 99347

Spokane Co. Planning Dept.
N. 721 Jefferson St.
Spokane, WA 99260

Barry Jenkins
Mason Co. Trout Unlimited
P.O. Box 855
Shelton, WA 98584

Clark Co. Planning &
Code Admin.
P.O. Box 5000
Vancouver, WA 98668

Chelan Co. Planning Dept.
411 Washington St.
Wenatchee, WA 98801

Benton Co. Planning Dept.
P. O. Box 910
Prosser, WA 99350

Franklin Co. Planning Dept.
1016 N. Fourth
Pasco, WA 99301

Whitman Co. Regional Planning
Old National Bank Bldg., Room
8
Colfax, WA 99111

Columbia Co. Planning
Department
County Courthouse
341 E. Main St.
Dayton, WA 99328

Asotin Co. Planning Dept.
County Courthouse
Asotin, WA 99402

Jeff Dickinson
Squaxin Island Tribe
W. 81, Hwy. 108
Shelton, WA 98584

Kitsap Co. Planning Dept.
614 Division St.
Port Orchard, WA 98366

Dave Lamb
W. 1720 4th Ave.
Spokane, WA 99204

Liberty Lake Sewer Dist.
S. 1827 Liberty Lake Dr.
Liberty Lake, WA 99019

U.S. Fish & Wildlife Service
Fish & Wildlife Enhancement
3704 Griffin Ln SE, Suite 102
Olympia, WA 98501

Spokane Review/Chronicle
W. 999 Riverside
Spokane, WA 99204

Larry Minkler, Managing
Director
WA Wildlife Heritage
32610 Pacific Hwy S
Federal Way, WA 98003

SAVE
16524 104th Ave.
Bothell, WA 98011

NW Fund for Environment
93 Pike Place 313
Seattle, WA 98101

Friends of the Earth
4512 University Way NE
Seattle, WA 98105

Greenpeace
5018 17th Ave NW
Seattle, WA 98105

The Mountaineers
300 3rd W
Seattle, WA 98119

Sierra Club
1516 Melrose
Seattle, WA 98122

WA Wilderness Coalition
P.O. Box 45187
Seattle, WA 98145

Adopt-A-Stream Foundation
P.O. Box 5558
Everett, WA 98204

Coalition of Sports Fishermen
1719 N. Oakes
Tacoma, WA 98406

Izaak Walton League
3509 N. 8th
Tacoma, WA 98406

National Audubon Society
4011 Alameda Ave.
Tacoma, WA 98466

Bill Robinson, President
Trout Unlimited
2401 Bristol Ct. A-18
Olympia, WA 98502

Dennis Austin
Washington Department of
Fisheries
P.O. Box 43150
Olympia, WA 98504-3150

William E. Barnett
305 Chatham Hill Road
Wenatchee, WA 98801

Carol Biagi
1826 Delmont
Walla Walla, WA 99362

Gary Graves
Northwest Indian Fisheries
Commission
6730 Martin Way E
Olympia, WA 98506

Frank Haw
3811 15th Court NE
Olympia, WA 98502

Keith Herrell
Pacific Salmon Sportfishing
Council
1023 S. Adams, Suite G52
Olympia, WA 98501

Rome Hutchings
23204 25th Ave. S
Des Moines, WA 98198

John Kelly
1612 SE 166th
Seattle, WA 98166

Ray Lebsack
N. 3505 Vista Rd.
Spokane, WA 99212

Bill McMillan
MPO 25 R
Laurel Lane S
Washougal, WA 98671

Rod Meeseberg
800 O'Sullivan Dam Rd.
Othello, WA 99344

Pete Soverel
18430 72nd W
Edmonds, WA 98026

Al Sussee
1807 E. 72nd St.
Tacoma, WA 98404

Dick Thompson
10104 NW 4th Ave.
Vancouver, WA 98686

Ron Tingley
1024 Ridge Place
Sedro Woolley, WA

Patrick Trotter
4926 26th Ave. S
Seattle, WA 98108

Paul Schneider
1411 Hubbard Road
Yakima, WA 98903

Ron Barnes
Quilcene Tribe
P. O. Box 187
LaPush, WA 98350-0187

Ben Schroeter
923 Kaiser Rd NW, #5
Olympia, WA 98502

Lower Elwha Klallam Tribe
P. O. Box 1666
Pt. Angeles, WA 98362

Hoh Tribe
HC 80 Box 917
Forks, WA 98331

Jamestown Klallam Tribe
150 S. Fifth, Suite 2
Sequim, WA 98382

Lummi Tribe
2616 Kwina Rd.
Bellingham, WA 98226

Makah Tribe
P.O. Box 115
Neah Bay, WA 98357

Nisqually Tribe
4820 She-Nah-Num Dr. SE
Olympia, WA 98503

Nooksack Tribe
P.O. Box 157
Deming, WA 98244

Point No Point Treaty Council
7850 NE Little Boston Rd.
Kingston, WA 98346

Port Gamble Tribe
P.O. Box 280
Kingston, WA 98346

Quinault Tribe
P.O. Box 189
Taholah, WA 98587

Skokomish Tribe
N. 80 Tribal Center Rd.
Shelton, WA 98584

Squaxin Island Tribe
W. 81 Hwy. 108
Shelton, WA 98584

Suquamish Tribe
P. O. Box 498
Suquamish, WA 98392

Swinomish Tribe
P. O. Box 817
La Conner, WA 98257

Upper Skagit Tribe
2284 Community Plaza
Sedro Woolley, WA 98284

Colville Tribe
P. O. Box 150
Nespelem, WA 99115

Spokane Tribe
P. O. Box 385
Wellpinit, WA 99040

Yakima Indian Nation
P. O. Box 151
Toppenish, WA 98948

Columbia River Inter-Tribal
Fisheries Commission
729 NE Oregon St., Suite 200
Portland, OR 97232

Deneen M. Person
333 Ridge view Dr #150
Kent WA 98032

Steve Hotchkiss
333 Ridgeview Dr #150
Kent WA 98032

Dori H. Miller
2027 Eastlake Ave #303
Seattle WA 98102

Garrett W. Jackson
2027 Eastlake Ave East #303
Seattle WA 98102

Renee Reed
PO Box 45494
Seattle WA 98145

The Mountaineers
Conservation Division
300 Third Ave West
Seattle WA 98119

Women Climbers Northwest
PO Box 20573
Seattle WA 98102

Federation of Western Outdoor
Clubs
OUTDOORS WEST
512 Boylston Ave
Seattle WA 98102

Janine Blaeloch
7040 14th NW
Seattle WA 98117

Maria Zurowske
Thurston Co Health Dept
2000 Lake Ridge Dr
Olympia WA 98502

Bob Crowell
PO Box 171
Okanogan WA 98840

George Draffan
PO Box 95316
Seattle WA 98145

Lake Lawrence Improvement
Club
16646 Pleasant Beach Dr
Lake Lawrence WA 98597

Sue Koenig
PO Box 393
Indianola WA 98342

Barbara Fournier
6959 NE Buck Lake RD
Hansville WA 98340

Nike Eir Quester
PO Box 224
Indianola WA 98342

Marion Kling
8800 NE Ohman Road
Kingston WA 98346

Robert Gibson
PO Box 11
Tenino WA 98589

Robert T. Lindley
16646 Pleasant Beach Dr SE
Yelm WA 98597

Richard Kich
16648 Pleasant Beach Rd SE
Yelm WA 98597

Joe Zelazny
15737 Wildaire DR SE
Yelm WA 98597

Joe Depinto
15805 Wildaire DR SE
Yelm WA 98597

Norm Reitner
15705 Wildarie DR SE
Yelm WA 98597

Joe Sokolik
530 Sawyer SE
Olympia WA 98502

William Grone
5422 34th St LP NE
Tacoma WA 98422

Dennis Kellogg
14922 Cedarwood Dr
Tenino WA 98589

Patsy J. Clearman
14916 Cedarwood Dr
Tenino WA 98589

Bob Boyer
PO Box 212
Tenino WA 98589

Richard Goodwin
1021 H St
Centralia WA 98531

James A Jars
PO Box 762
Tenino WA 98589

Gerri Woolf
PO Box A
Tenino WA 98589

Del Hinzpeter
16040-A Tilley RD S
Tenino WA 98589

Pete Stewart
12909 Silver Cr Rd SE
Tenino WA 98589

Ron Knackstedt
611 N Columbia #14
Olympia WA 98501

Eric Stewart
12909 Silver Cr Dr
Tenino WA 98589

Ted Habermen
14846 Cederwood Dr
Tenino WA 98589

L.B.Prince
16811 17th Ave E
Spanaway WA 98387

Doug Vassar
PO Box 15
Tenino WA 98589

Chuck Flory
15908 Lawrence Pl Se
Yelm WA 98597

Ruby Lavon
14814 Cedarwood Dr SE
Tenino WA 98589

Robert E Frose
PO Box 251
Tenino WA 98589

Bill Cleland
15002 Military Rd
Tenino WA 98589

Lloyd & Mona Facklam
16035 Lawrence Pl SE
Yelm WA 98597

John Carpenter
PO Box 516
Yelm WA 98597

Dan Staro
1840 NE Serpentine Pl
seattle wa 98155

Ellen L. Wilson
15617 Lawrence Lk Rd SE
Yelm WA 98597

Dave & Karen Wilson
2513 395th St Ct S
Roy WA 98580

Pamela Lyons
3813 Biskey St NW #18
Olympia WA 98502

Burgess L. Meredith
15824 Lawrence Pl SE
Yelm WA 98597

John Hevener
9605 Tilley Rd
Olympia WA 98580
Robert Gibson
PO Box 11
Tenino WA 98589

Robert T. Lindley
16646 Pleasant Beach Dr SE
Yelm WA 98597

Richard Kich
16648 Pleasant Beach Rd SE
Yelm WA 98597

Joe Zelazny
15737 Wildaire DR SE
Yelm WA 98597

Joe Depinto
15805 Wildaire DR SE
Yelm WA 98597

Norm Reitner
15705 Wildarie DR SE
Yelm WA 98597

Joe Sokolik
530 Sawyer SE
Olympia WA 98502

William Grone
5422 34th St LP NE
Tacoma WA 98422

Dennis Kellogg
14922 Cedarwood Dr
Tenino WA 98589

Patsy J. Clearman
14916 Cedarwood Dr
Tenino WA 98589

Bob Boyer
PO Box 212
Tenino WA 98589

Richard Goodwin
1021 H St
Centralia WA 98531

James A Jars
PO Box 762
Tenino WA 98589

Gerri Woolf
PO Box A
Tenino WA 98589

Del Hinzpeter
16040-A Tilley RD S
Tenino WA 98589

Pete Stewart
12909 Silver Cr Rd SE
Tenino WA 98589

Ron Knackstedt
611 N Columbia #14
Olympia WA 98501

Eric Stewart
12909 Silver Cr Dr
Tenino WA 98589

Ted Habermen
14846 Cederwood Dr
Tenino WA 98589

L.B.Prince
16811 17th Ave E
Spanaway WA 98387

Doug Vassar
PO Box 15
Tenino WA 98589

Chuck Flory
15908 Lawrence Pl Se
Yelm WA 98597

Ruby Lavon
14814 Cedarwood Dr SE
Tenino WA 98589

Robert E Frose
PO Box 251
Tenino WA 98589

Bill Cleland
15002 Military Rd
Tenino WA 98589

Lloyd & Mona Facklam
16035 Lawrence Pl SE
Yelm WA 98597

John Carpenter
PO Box 516
Yelm WA 98597

Dan Staro
1840 NE Serpentine Pl
seattle wa 98155

Ellen L. Wilson
15617 Lawrence Lk Rd SE
Yelm WA 98597

Dave & Karen Wilson
2513 395th St Ct S
Roy WA 98580

Pamela Lyons
3813 Biskey St NW #18
Olympia WA 98502

Burgess L. Meredith
15824 Lawrence Pl SE
Yelm WA 98597

John Hevener
9605 Tilley Rd
Olympia WA 98580

Washington Department of Wildlife



Serving Washington's
wildlife and people—
now and in the
future

The Washington Department of Wildlife will provide equal opportunities to all potential and existing employees without regard to race, creed, color, sex, sexual orientation, religion, age, marital status, national origin, disability, or Vietnam Era Veteran's status. The department receives Federal Aid for fish and wildlife restoration. The department is subject to Title VI of the Civil Rights Act of 1964 and Section 504 of the Rehabilitation Act of 1973, which prohibits discrimination on the basis of race, color, national origin or handicap. If you believe you have been discriminated against in any department program, activity, or facility, or if you want further information about Title VI or Section 504, write to: Office of Equal Opportunity, U.S. Department of Interior, Washington, D.C. 20240, or Washington Department of Wildlife, 600 Capitol Way N, Olympia WA 98501-1091.