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# Great Lakes Water Quality Fifth Annual Report 1976: Appendix A Annual Report of the Water Quality Objectives Subcommittee and the Task Force on the Scientific Basis for Water Quality Criteria

Great Lakes Water Quality Board. Water Quality Objectives Subcommittee

Great Lakes Water Quality Board. Task Force on the Scientific Basis for Water Quality Criteria

# GREAT LAKES
## WATER QUALITY BOARD
## RESEARCH ADVISORY BOARD

INTERNATIONAL JOINT COMMISSION

# GREAT LAKES WATER QUALITY 1976
# APPENDIX A
# WATER QUALITY OBJECTIVES

Presented to the
Implementation Committee
of the
Great Lakes
Water Quality Board
and to the
Great Lakes
Research Advisory Board

# GREAT LAKES WATER QUALITY

## ANNUAL REPORT OF THE WATER QUALITY OBJECTIVES SUBCOMMITTEE AND THE TASK FORCE ON THE SCIENTIFIC BASIS FOR WATER QUALITY CRITERIA

JUNE 1977

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# PREFACE

The 1976 Annual Report on Water Quality Objectives was prepared jointly by the Water Quality Objectives Subcommittee of the Water Quality Board and the Scientific Basis for Water Quality Criteria Task Force of the Research Advisory Board. It is the third report on water quality objectives since the signing of the Great Lakes Water Quality Agreement between the United States of America and Canada in 1972.

The report outlines recommended new and revised water quality objectives with supporting rationale; presents rationale for substances for which objectives were researched but for which adequate scientific background data to derive objectives were not available; objectives under active review; and intended future activities. Research needs required to aid in the revision and derivation of certain objectives are also outlined.

The material presented in this report represents the best scientific judgement of the members of the Subcommittee and Task Force. This does not imply, however, that all of the proposals presented in this report will be approved and recommended to the Commission.

# TABLE OF CONTENTS

# LIST OF TABLES

# LIST OF FIGURES

# SUMMARY

This report presents new and revised water quality objectives, information on substances researched but for which scientifically defensible objectives could not be developed due to an inadequate data base and substances under active review for the formulation of objectives.

The report also presents a brief discussion of the Environmental Mapping Workshop, an outline of the future direction of the Subcommittee and the Task Force and the research needs identified by the Subcommittee and Task Force in developing water quality objectives.

The substances examined by the Subcommittee and Task Force during the period of this report are as follows:

New and Revised Water Quality Objectives
- Chlorine
- Dissolved Oxygen
- Silver
- Mirex

Substances Researched - Insufficient Data to Recommend Defensible Objectives
- Antimony
- Barium
- Boron
- Cobalt
- Manganese
- Molybdenum
- Vanadium
- Organotin Compounds
- Phosphorus, Elemental
- Polynuclear Aromatic Hydrocarbons

Substances and Materials Under Active Review
- Nutrients
- Bacteria
- Detergents
- Atrazine
- Malathion

# INTRODUCTION

The Water Quality Objectives Subcommittee and the Scientific Basis for Water Quality Criteria Task Force present the results of their activities for the year 1976-77 in this, their 1976 report to the Water Quality Board and to the Research Advisory Board.

In selecting priorities, consideration was given to the likelihood of harmful substances reaching a living organism, whether man or animal, in sufficient concentration and for a sufficient period for harm to occur.

Toxicological information on substances is rarely complete or precise enough to meet the requirements of those who use the information to establish justifiable water quality objectives. Major problems arise in the measurement of sub-acute effects and the results of exposure to low levels of toxic substances over the short and the long term, in allowing for possible interactions between different toxic substances and other water quality characteristics, and in monitoring the differences in response between individuals and the rates of bioaccumulation of persistent materials.

Although the Subcommittee and Task Force recognized these problems, they had to rely heavily on acute and chronic toxicity data of single substances for determining safe limits to protect the most sensitive use. The objectives therefore must be kept under review and revised when relevant new information becomes available. There is also a problem of applying toxicity data based on carefully controlled conditions to "real" situations.

The scientific information used to recommend an objective for each substance to protect the most sensitive use is presented as a rationale. Objectives are recommended for total residual chlorine, dissolved oxygen, silver and mirex. The report also presents information on substances for which the scientific data were inadequate to recommend objectives and on substances which are currently under active review.

In addition to the above, information is presented on environmental mapping, the future direction of the work of the Subcommittee and Task Force, and research needs which will assist the Subcommittee and Task Force in their charge to develop and recommend water quality objectives specific to the Great Lakes.

# 1 WATER QUALITY OBJECTIVES

## RECOMMENDED NEW AND REVISED WATER QUALITY OBJECTIVES

### CHLORINE

*RECOMMENDATION*

It is recommended that the following new objective for chlorine be adopted:

*Total residual chlorine, as measured by the amperometric (or equivalent) method, should not exceed:*

*0.002 milligrams per litre in order to protect aquatic life.*

*RATIONALE*

The extensive study of chlorine as a disinfectant has resulted in a thorough understanding of the chemistry of chlorine in water. Elemental chlorine hydrolyzes in water to form hypochlorous acid (equation 1). The hypochlorous acid is a weak acid and it dissociates to form the hypochlorite ion according to equation 2.

$$Cl_2 + H_2O \rightleftharpoons HOCl + H^+ + Cl^- \quad (1)$$

$$HOCl \rightleftharpoons H^+ + OCl^- \quad (2)$$

Thus, free available chlorine is present as hypochlorous acid (HOCl), hypochlorite ion ($OCl^-$), and elemental chlorine ($Cl_2$).

Ammonia is present to a significant degree in most wastewater and is of prime importance in wastewater treatment plants using halogenation for disinfection. At pH 4.5-8.5 and 20°C, chlorine reacts with ammonia in wastewater to produce monochloramine ($NH_2Cl$) and dichloramine ($NHCl_2$).

Total residual chlorine (TRC) is the sum of free available chlorine and combined available chlorine (chloramines and similar compounds). Free available chlorine is seldom found in treated wastewaters because chlorine is added in an amount less than the chlorine demand before discharge to a surface water.

## METHODOLOGY

Many wastewater treatment plants are required to maintain a residual chlorine concentration of 0.5 to 2.0 mg/1. Most plant operators use the orthotolidine method which has been shown to be biased on the low side resulting in much higher concentrations than necessary for adequate disinfection. This compounds toxicity problems in receiving waters. Total residual chlorine concentrations in 20 Illinois effluents ranged from 0.98 to 5.17 mg/1 (Snoeyink and Markus, 1974). A similar study at 22 plants in southern Wisconsin resulted in observed concentration of TRC between 0.18 and 10.3 mg/1 (McKersie, 1974). Both studies demonstrated that the orthotolidine methods provided the poorest results when compared against better methods such as the amperometric titration technique. Other studies (Martens and Servizi, 1974; Servizi and Martens, 1974) reached the same conclusion that the commonly used orthotolidine method is inadequate to determine TRC in wastewaters or receiving streams.

## TOXICITY OF CHLORINATED WASTEWATERS

There is an extensive data base on the toxicity of TRC to freshwater aquatic life and these data have been adequately summarized (Isom, 1971; McKee and Wolf, 1963; Doudoroff and Katz, 1950; Brungs, 1973 and 1976; and Michigan Department of Natural Resources, 1971). The following discussion is limited to those studies that have involved chlorinated wastewaters and does not include numerous studies of TRC in clean waters.

The Michigan Department of Natural Resources (1971) reported the effects on caged fish in several receiving streams below wastewater discharges. Fifty percent of the rainbow trout died within 96 hr (96-hr $LC_{50}$) at TRC concentrations of 0.014 to 0.029 mg/1; some fish died as far as 0.8 mile (1.3 km) below the outfall. These same discharges were studied when chlorination was temporarily interrupted, and no mortality was observed. In addition, dechlorination with sodium thiosulfate eliminated toxicity in 4-day tests with undiluted effluent.

Tsai (1973) studied the effects on fish of 156 wastewater treatment plants in Maryland, northern Virginia, and southeastern Pennsylvania. All the plants discharged chlorinated municipal wastes into small streams containing fish. In most of the plants in Maryland and Virginia, 0.5 to 2.0 mg/1 residual chlorine is maintained in the effluents, and Pennsylvania requires 0.5 mg/1 in effluents prior to discharge to natural surface water. Tsai (1973) studied principally fish, but observed typically a bottom devoid of living organisms in the area immediately below the chlorinated outfalls. Unchlorinated discharge areas were typically characterized by abundant growths of wastewater fungi. No fish were found in water with a TRC above 0.37 mg/1, and the species diversity index reached zero at 0.25 mg/1. A 50 percent reduction in species diversity index occurred at 0.10 mg/1. Of the 45 species of fish observed in the study areas, the brook trout and brown trout were the most sensitive and were not found at concentrations above approximately 0.02 mg/1. These and 8 other species were not found above 0.05 mg/1. In this sensitive group were 5 minnow species.

Arthur et al. (1975) have studied the effect of chlorinated secondary wastewater treatment plant effluent containing only domestic sewage effluent on reproduction of fathead minnows, Daphnia magna, and the scud (Gammarus pseudolimnaeus). Daphnia magna apparently was the most sensitive invertebrate species and died at a TRC concentration of 0.014 mg/1, and acceptable reproduction occurred at 0.003 mg/1 and below. Scud reproduction was reduced at concentrations above approximately 0.012 mg/1 (1.2 percent effluent). No effects on any life cycle stage, including reproduction, of the fathead minnow was observed at a concentration of 0.014 mg/1; adverse effects were observed at 0.042 mg/1. Acute toxicity studies with eight species of fish, crayfish (Orconectes virilis), scud (Gammarus pseudolimnaeus), snails (Physa integra and Campeloma decisum), and stoneflies (Acroneuria lycorias) indicated that the crayfish, snails, and stonefly larvae were least sensitive with 7-day $LC_{50}$ values greater than 0.55 mg/1. Seven-day $LC_{50}$ values for the other organisms were between 0.083 and 0.261 mg/1; coho salmon, brook trout, fathead minnow, white sucker, and walleye were the most sensitive (0.086 to 0.150 mg/1). Nearly 50 percent of the observed mortalities occurred in the first 12 hours of the acute tests indicating that the lethal effect of TRC occurs rapidly. Comparable acute and chronic tests with the effluent dechlorinated with sulfur dioxide indicated that most, if not all, of the toxicity of the chlorinated effluent was eliminated. Esvelt et al. (1971; 1973) and Krock and Mason (1971) completed an extensive study on the toxicity of chlorinated municipal wastewaters entering San Francisco Bay and surrounding areas. They observed a significant increase in toxicity following chlorination. Chlorine toxicity was still significant in aged (up to 3 days) chlorinated wastewater, in which TRC concentrations were as high as 25 percent of the initial level. Rainbow trout was the most sensitive of the species tested, followed by the golden shiner and three-spined stickleback. A calculated chlorine residual of 0.03 mg/1, based on dilution of a measured concentration of 2.0 mg/1, reduced phytoplankton photosynthesis by more than 20 percent of the value obtained with a dilution of effluent having no chlorine residual. Dechlorination with sodium bisulfite also eliminated chlorine-related toxicity. One of the conclusions of the California study was that chlorination may be the largest single source of toxicity in San Francisco Bay.

Martens and Servizi (1974) and Servizi and Martens (1974) observed mortality of salmon in receiving streams at TRC concentrations as low as 0.02 mg/1. Determinations of the effect of time on chlorine residuals were made by sample storage and lagoon retention. Lethal concentrations persisted in undiluted effluent for at least 50 hours. Twenty to one dilutions resulted in the chlorine residual declining to a non-detectable concentration after 22 hours. Studies with caged fish at points downstream of the effluent demonstrated acutely lethal conditions that did not persist during periods when the chlorinator was inoperable.

Ward et al. (1976) conducted acute and chronic tests of chlorinated wastewater at the Grandville, Michigan sewage treatment plant. The 96-hr $LC_{50}$ values for fish ranged from 0.04 to 0.278 mg/1. The $LC_{50}$ values for rainbow trout, coho salmon, lake trout, golden shiner, common shiner, pugnose shiner, and fathead minnow were from 0.04 to 0.095 mg/1. The most resistent species were largemouth bass and other sunfish. The highest tested concentration of residual chlorine that had no chronic effect on the fathead minnow was 0.01 mg/1. A second series of acute and chronic studies at the Wyoming, Michigan wastewater treatment plant has produced similar results (Ward et al., 1977).

Several reviewers of chlorine toxicity have recommended numerical objectives for concentrations of TRC that would not adversely affect aquatic populations when discharged continuously. Basch and Truchan (1974) recommended maximum concentrations of 0.02 and 0.005 mg/1 for warmwater and coldwater intolerant fish, respectively. European Inland Fisheries Advisory Commission (1973) has suggested objectives dependent upon pH and temperature with an acceptable upper limit of 0.004 mg HOCl/1 (TRC from 0.004 mg/1 at a pH of 6.0 and 5°C to 0.121 mg/1 at a pH of 9.0 and 25°C). A third review by Brungs (1973) has recommended objectives of 0.01 mg/1 for warmwater fish and 0.002 mg/1 for coldwater species and the most sensitive fish food organisms. Since these recommendations, additional data on warmwater fish species (Arthur et al., 1975; Tsai, 1973; Ward et al., 1976, 1977; Bogardus et al., 1976) do not support the distinction between coldwater and warmwater fish species. A more recent recommendation (Brungs, 1976) supports the objective for total residual chlorine of 0.002 mg/1.

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## DISSOLVED OXYGEN

*RECOMMENDATION*

It is recommended that the following revised objective for dissolved oxygen be adopted to replace the existing objective in Annex 1 of the Water Quality Agreement:

*Dissolved oxygen should not be less than the values specified below for the protection of aquatic life:*

| *Water Temperature °C* | *Oxygen Concentration* | |
|---|---|---|
| | *Percent Saturation* | *mg/litre* |
| *0* | *69* | *10.0* |
| *5* | *70* | *9.0* |
| *10* | *70* | *7.9* |
| *15* | *71* | *7.2* |
| *20* | *79* | *7.2* |
| *25* | *87* | *7.2* |

EXISTING OBJECTIVE

The above objective is recommended to replace the existing objective in Annex 1 paragraph 1 (c) of the Water Quality Agreement, which states:

> "In the Connecting Channels and in the upper waters of the lakes, the dissolved oxygen level should be not less than 6.0 milligrams per litre at any time; in hypolimnetic waters, it should be not less than necessary for support of fishlife, particularly cold water species."

*RATIONALE*

INTRODUCTION

Dissolved oxygen (DO) is a critical constituent which is essential for the continued healthy functioning of aquatic systems. Inadequate dissolved oxygen concentrations in surface waters may contribute to an unfavourable environment for fish and other aquatic life; the absence of dissolved oxygen may degrade the aesthetic quality of waters by giving rise to the malodorous products of anaerobic decomposition. Although supersaturated concentrations of DO which can arise from excessive algal production may affect one or more beneficial uses of water, the emphasis of the rationale is on the implications of a deficiency of dissolved oxgyen.

OXYGEN REGIMES IN THE GREAT LAKES

Table 1 summarizes summertime dissolved oxygen conditions in the hypolimnetic waters of the Great Lakes, and illustrates the variation in conditions within that system. In Lakes Superior and Huron, the hypolimnion remains essentially

# TABLE 1.

## Summary Of Dissolved Oxygen Conditions In The Great Lakes' Summertime Hypolimnia As Observed By The Canada Centre For Inland Waters (Compiled By Hugh F.H. Dobson, April 22, 1977)

| LAKE | NUMBER OF CRUISES | YEARS OF THE OBSERVATIONS CONSIDERED | APPROXIMATE TEMPERATURES (°C) | DISSOLVED OXYGEN CONDITIONS | | |
|---|---|---|---|---|---|---|
| | | | | MAXIMUM VALUES (MG/L, % SATN) | MINIMUM VALUES (MG/L, % SATN) | MEAN DEPLETION RATE (MG/L/MONTH) |
| Superior | 9 | 1968 - 73 | 3.8° | 13.8/107% | 12.0/93% | 0.14 |
| Huron | 17 | 1968 - 74 | 3.9° | 13.8/108% | 11.4/88% | 0.24 |
| Georgian Bay | 10 | 1969 - 74 | 4.1° | 14.5/114% | 11.6/90% | 0.27 |
| Central Erie | 5 | 1970 | 7 to 12° | ~11. /~100% | 0.0/0% | 3.3 |
| Eastern Erie | 6 | 1970 | 6° | ~12. /~100% | 5.7/48% | 1.2 |
| Ontario, main basin | 21 | 1972 - 75 | 3.8° | ~13. /~100% | 10./80% | 0.6 ? |
| Ontario, outlet basin | 33 | 1966 - 75 | 5 to 12° | 13.1/105% | 4.2/39% | 2.0 |

saturated with oxygen, though areas of oxygen depletion do occur. This depletion is generally observed during the August-September period; the reduction seldom exceeds 10%.

In contrast to the upper lakes, there is a marked seasonal variation in oxygen conditions in Lakes Erie and Ontario. The existing objective for dissolved oxygen is frequently violated in both lower lakes. Zero values of DO commonly occur during the summer months in the bottom waters of the central basin of Lake Erie; minimum values of 5.7 mg/l (48% saturation) have been observed in the hypolimnion of the eastern basin of Lake Erie. Dissolved oxygen values in the central and eastern basin are depicted in Figure 1.

In Lake Ontario, oxygen conditions are more stable, with a maximum depletion of 20% from complete saturation. However, significant reductions occur in the outlet basin of Lake Ontario. Minimum cycles of 39% saturation have been recorded. The seasonal cycle of DO in the bottom water of Prince Edward Bay is presented in Figure 2.

In contrast to the bottom waters, the eplimnetic waters throughout the Great Lakes are normally saturated with oxygen, and violations of water quality objectives, would not be expected to occur.

## AESTHETIC QUALITIES OF WATER

Maintenance of aesthetic qualities of water requires sufficient dissolved oxygen to avoid the onset of septic conditions. The absence of dissolved oxygen in the water column causes the anaerobic decomposition of any organic materials present. Such decomposition results in the formation of gases such as hydrogen sulfide, carbon dioxide and methane in the sediments.

## POTABLE WATER SUPPLIES

Dissolved oxygen in bodies of water used for municipal water supplies is desirable as an indicator of generally satisfactory water quality. In addition, dissolved oxygen in the water column prevents the chemical reduction and subsequent leaching of iron and manganese principally from the sediments (Environmental Protection Agency, 1973). These metals cause additional expense in the treatment of water or affect consumers' welfare by causing taste problems and staining plumbing fixtures and other surfaces which contact the water in the presence of oxygen (NAS/NAE, 1974).

Dissolved oxygen also is required for the biochemical oxidation of ammonia ultimately to nitrate in natural waters. This reduction of ammonia reduces the chlorine demand of waters and increases the disinfection efficiency of chlorination (NAS/NAE, 1974).

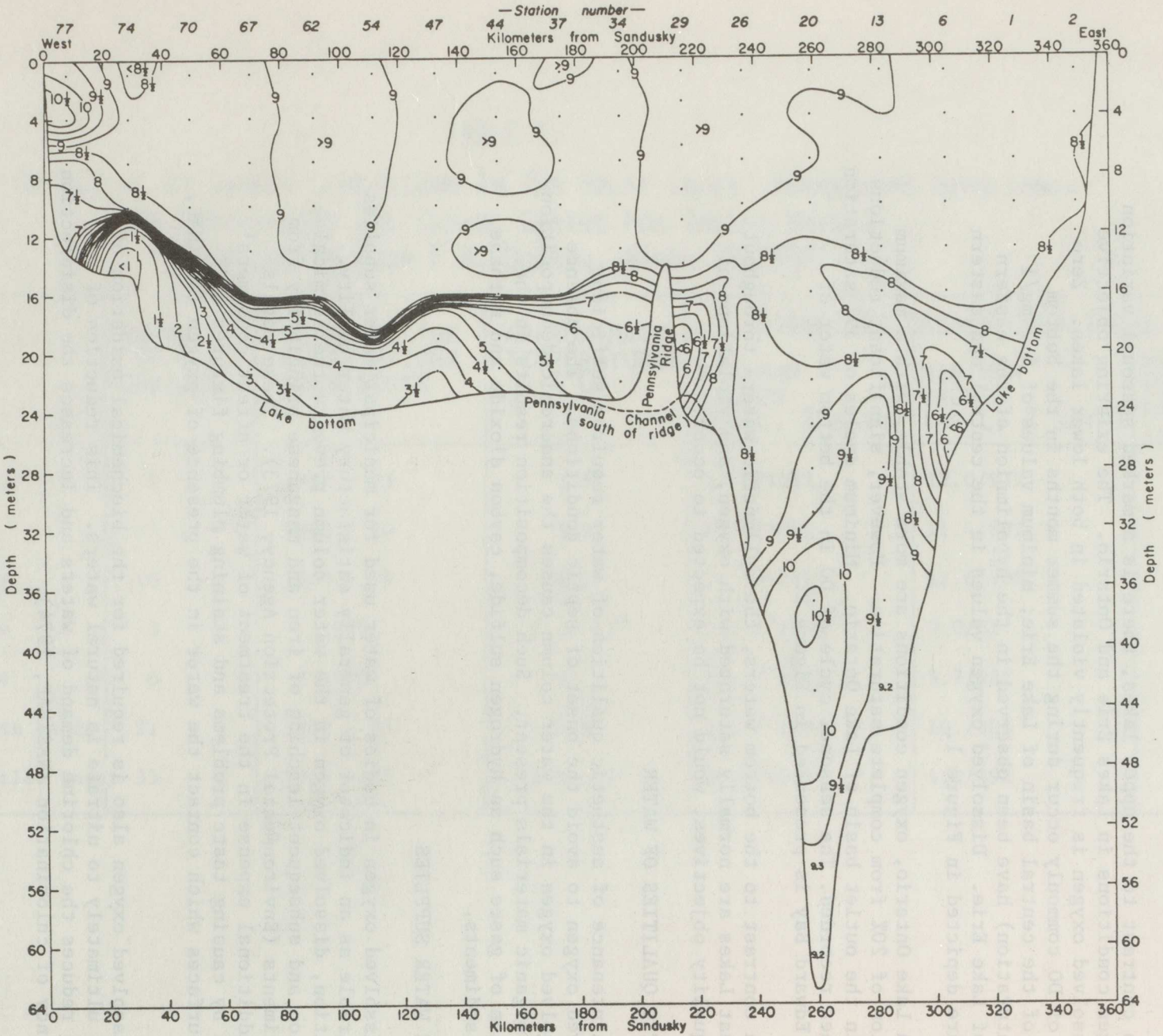
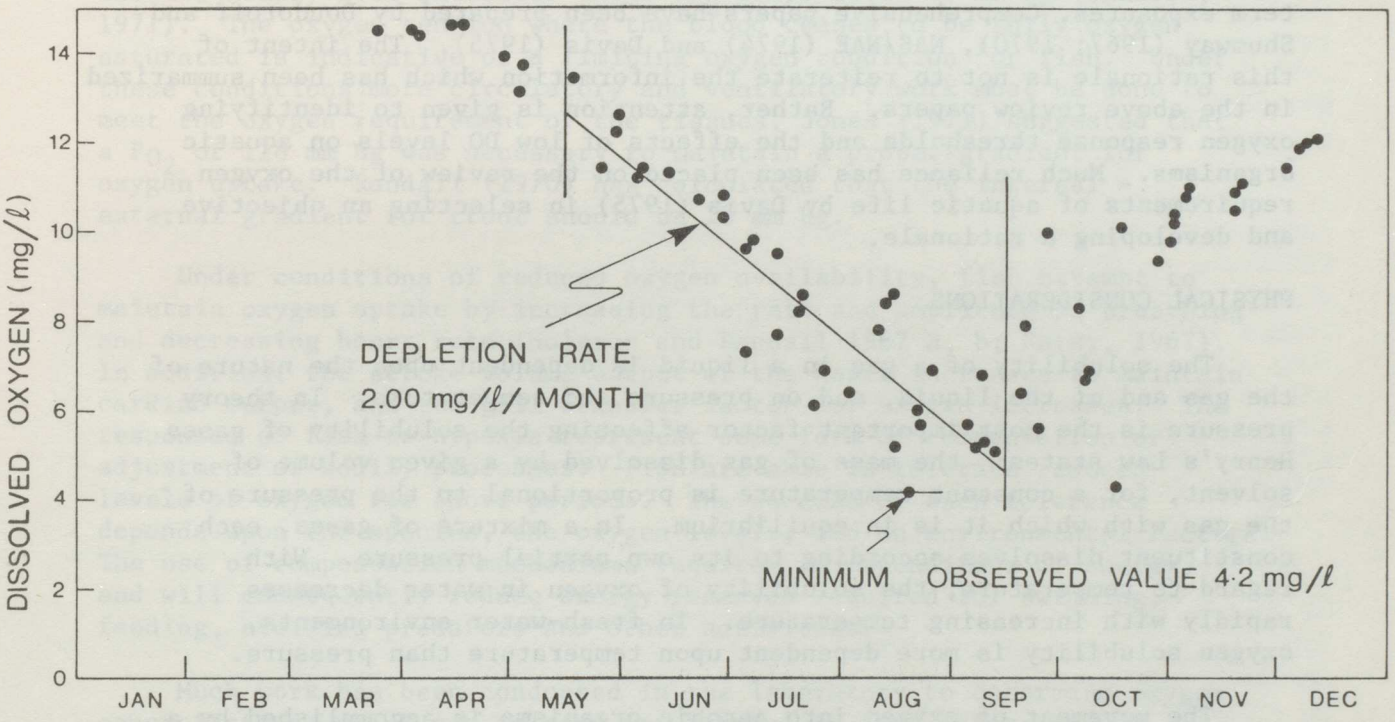



FIGURE 1: Lake Erie: Dissolved oxygen ( mg/l ) in a vertical section from Sandusky to Buffalo, July 29 to August 2, 1968. Vessel 'Theron' ( Canada Centre for Inland Waters ), Cruise 68-1-08. Vertical exaggeration: X 5,000.

FIGURE 2:




SEASONAL CYCLE OF DISSOLVED OXYGEN IN THE BOTTOM WATER OF PRINCE EDWARD BAY (LAKE ONTARIO), 1966 to 1975: DATA OF THE CANADA CENTRE FOR INLAND WATERS.

The disadvantage of substantial quantities of dissolved oxygen in water used as a source of municipal water supply is the increased rates of corrosion of metal surfaces in both the water treatment facilities and in the distribution system (NAS, 1974).

Such corrosion, in addition to the direct damage, can increase the concentration of iron (and other metals) which may cause taste in the water, as well as staining.

## AQUATIC LIFE REQUIREMENTS

There is a large volume of literature pertaining to the minimum dissolved oxygen concentration necessary to sustain aquatic life, especially fish, and the concentrations below which fish will be affected by short-term exposures. Comprehensive papers have been prepared by Doudoroff and Shumway (1967; 1970), NAS/NAE (1974) and Davis (1975). The intent of this rationale is not to reiterate the information which has been summarized in the above review papers. Rather, attention is given to identifying oxygen response thresholds and the effects of low DO levels on aquatic organisms. Much reliance has been placed on the review of the oxygen requirements of aquatic life by Davis (1975) in selecting an objective and developing a rationale.

## PHYSICAL CONSIDERATIONS

The solubility of a gas in a liquid is dependent upon the nature of the gas and of the liquid, and on pressure and temperature. In theory pressure is the most important factor affecting the solubility of gases. Henry's Law states: the mass of gas dissolved by a given volume of solvent, for a constant temperature is proportional to the pressure of the gas with which it is in equilibrium. In a mixture of gases, each constituent dissolves according to its own partial pressure. With regard to temperature, the solubility of oxygen in water decreases rapidly with increasing temperature. In fresh-water environments, oxygen solubility is more dependent upon temperature than pressure.

The movement of oxygen into aerobic organisms is accomplished by a process of diffusion which depends upon an internal - external oxygen pressure gradient. It is the oxygen tension gradient between tissues and the external medium that is critical to the gas exchange process.

Water which is equilibrated with the atmosphere at sea level will have an oxygen partial pressure of about 154 - 158 mm Hg. In fish, the internal oxygen tension usually ranges from 50 - 110 mm Hg, and oxygen therefore diffuses across the gills into the blood down an $O_2$ gradient of 40 - 100 mm Hg (Randall, 1970). A decrease of the external oxygen partial pressure ($P_{O_2}$) reduces the gradient or depresses arterial $P_{O_2}$.

As temperature increases, oxygen solubility decreases, but oxygen partial pressure drops only slightly. In warm water, a fish must pump more water over the gills to provide a given volume of oxygen per unit time, even though the oxygen partial pressure gradient between water and blood has changed but little. Metabolic demand for oxygen increases with elevated temperatures. Therefore, severe respiratory problems can be associated with a combination of high temperature and reduced oxygen tension (i.e. water not saturated) as both oxygen availability and the gradient for oxygen diffusion are reduced. To provide protection for aquatic life, oxygen objectives must aim to maintain a critical oxygen content and necessary oxygen tension as well as provide for the effects of temperature on metabolism.

## OXYGEN REQUIREMENTS OF FISH

The blood oxygen dissociation curve (relating percent saturation of the blood to the $P_{O_2}$ applied) for rainbow trout indicates that the blood remains nearly 100% saturated until $P_{O_2}$ drops below 80 mm Hg (Cameron, 1971). The oxygen tension where the blood ceases to be fully oxygen saturated is indicative of a limiting oxygen condition for fish. Under these conditions more circulatory and ventilatory work must be done to meet the oxygen requirement of the tissues. Jones (1971) suggested that a $P_{O_2}$ of 118 mm Hg was necessary to maintain a proper gradient for oxygen uptake. Randall (1970) has calculated that the internal - external gradient for trout should be 20 mm Hg.

Under conditions of reduced oxygen availability, fish attempt to maintain oxygen uptake by increasing the rate and amplitude of breathing and decreasing heart rate (Holeton and Randall 1967 a, b; Garey, 1967). In addition, the stroke volume output of the heart increases to maintain cardiac output, and the gill transfer factor for oxygen increases. The responses of fish to hypoxia represent some form of compensation or adjustment of bodily processes. Fish are able to tolerate reduced levels of oxygen for short periods. The success of such tolerance depends upon the species, the oxygen levels, and on environmental factors. The use of compensation mechanisms requires an expenditure of energy, and will consequently reduce energy reserves required for swimming, feeding, avoiding predators and other activities.

Much work has been conducted in the laboratory to determine oxygen concentrations which are in some way harmful to fish maintained in quiescent conditions. However, Fry (1957) recognized that identification of oxygen thresholds (where metabolic rate ceases to be dependent upon available oxygen) should be studied in relation to active metabolism of the test organism. He states:

> "Any reduction of the oxygen content below the level where the active metabolic rate begins to be restricted is probably unfavourable to the species concerned. From the ecological point of view this "incipient limiting level" (the critical level under conditions of activity) can be taken as the point where oxygen content becomes unsuitable".

Brett (1970) determined the oxygen requirements for fingerling sockeye salmon during various important activities. His data indicate that a reduction in oxygen to 50% saturation (at 20°C) would severely limit energy expenditure for migrating or maximum feeding. Similarly, 30 and 43% reductions of maximal swimming speed in rainbow trout resulted when environmental oxygen fell to 50% of saturation at 21-23°C and 8-10°C, respectively (Jones, 1971). Davis et al. (1963) reported that any reduction from ambient oxygen at 10-20°C usually reduced maximum sustained swimming speed in coho and chinook salmon. In contrast, Katz et al. (1959) has reported that the swimming ability in reduced oxygen may be affected by season and temperature, based upon experiments with large mouth bass.

The ability of fish to avoid hypoxic water has been reported by Randall (1970). Whitmore et al. (1960) have observed a seasonal variability in behavioral sensitivity of juvenile chinook salmon to low dissolved oxygen. Juvenile fish avoided DO concentrations of 1.5 - 4.5 mg/l in the summer, but showed little avoidance of 4.5 mg/l in the fall when temperatures were lower. Other behavioral responses to hypoxia include a negative phototaxis in walleye (Scherer, 1971), and violent bursts of activity and attempts to surface (Shepard, 1955).

Growth rate of fish is dependent upon dissolved oxygen. Growth rate and food consumption in juvenile largemouth bass and coho salmon increased as DO concentrations approached air-saturation levels (Stewart et al., 1967; Herrman, 1958). Herrman (1958) concludes that minimal oxygen concentrations to which juvenile coho can be exposed for relatively long periods without markedly affecting growth, feeding, food conversion and general activity, lie within the range of 4-6 mg/l. Exposure to fluctuating DO levels reduced growth of juvenile largemouth bass (Stewart, et al., 1967), and Whitworth (1968) observed loss of weight in brook trout to daily oxygen fluctuations.

The ability to acclimate to low DO would be a useful factor for populations regularly exposed to such conditions. However, the ability of fish to acclimate to low DO has not been clearly demonstrated, and there is a lack of field data to demonstrate that this ability would markedly improve the survival chances of fish populations. The ability to acclimate would be useful if the transition to a low DO regime were sufficiently slow to enable acclimation to occur without a severe physiological stress. Acclimation would be of no value if fish encounter a rapid downward shift of oxygen.

In addition to the direct impact of low oxygen levels in fish, there is evidence that low oxygen enhances the lethal effect of toxicants by producing a metabolic stress, thus lowering the resistance of the animal, and by increasing toxicant uptake as the result of elevated water flow across the gills. Alderdice and Brett (1957) observed an

apparent increase in the toxicity of kraft pulpmill waste to young sockeye salmon in the presence of low oxygen. Similarly, Lloyd (1961) reported an increase in the toxicity of a number of chemical species (ammonia, lead, copper, zinc, phenols) to rainbow trout when oxygen levels fell below 60% saturation at 17.5°C. In tests with rainbow trout, Downing (1954) demonstrated that at 17°C, any reduction in DO below 100% saturation (9.74 mg/1) led to a significant enhancement of the lethal effect of cyanide.

Developing fish eggs and larvae show a number of responses to low oxygen including respiratory dependence, retarded growth, reduced yolk sac adsorption, developmental deformities, and mortality. As development proceeds, the oxygen requirements of both eggs and larvae increase. Alderdice et al. (1957) observed that eggs at early stages of incubation required oxygen concentrations of approximately 1 mg/1, while those about to hatch required about 7 mg/1. It is apparent that hatching eggs and larval fish represent the most sensitive stage in the life history. Davis (1975) has calculated the mean threshold of incipient oxygen response for salmonid larvae to be 8.09 mg/1.

## OXYGEN REQUIREMENTS OF AQUATIC INVERTEBRATES

Available data on the responses of freshwater invertebrates to low dissolved oxygen have been summarized by Davis (1975). Davis indicated that a great range of tolerance responses and requirements for oxygen exist amongst aquatic invertebrates, and concluded that insufficient evidence exists at this time to allow meaningful dissolved oxygen objectives to be established for aquatic invertebrates. He suggests that any depression of natural oxygen conditions can result in a change in an aquatic invertebrate community. However, as many invertebrates are able to temporarily withstand periods of low oxygen, it is likely that establishment of objectives to protect fish will also ensure the protection of aquatic invertebrates.

## APPROACHES TO THE DEVELOPMENT OF A DISSOLVED OXYGEN OBJECTIVE FOR AQUATIC LIFE

There are currently two philosophies with regard to establishment of an objective for dissolved oxygen to protect aquatic life. One view is that <u>any</u> reduction of DO can reduce the efficiency of oxygen uptake by aquatic animals and hence reduce their ability to meet the demands of their environment. This approach espouses the view that there is no DO concentration or percentage saturation to which the oxygen content of natural waters can be reduced without causing or risking some adverse effects on the reproduction, growth and productivity of resident fish populations. This view has been endorsed by Doudordoff and Shumway (1970), NAS/NAE (1974) and Davis (1975). Objectives which are based upon this approach generally are expressed as percent saturation or concentration minima and are in effect a continuum of values dependent upon temperature.

The opposing view is that the response of organisms to DO concentrations below 100% saturation simply reflects an acclimation response which has no negative impact on the continued survival or productivity of the aquatic community. Based largely upon field observations of fish populations which have been found at oxygen concentrations considerably below that considered suitable for a thriving population, single minimum concentrations of DO are believed to adequately protect aquatic populations. This view has been adopted by the EPA "Quality Criteria for Water" (EPA, 1976) which recommends an objective of 5 mg/l for dissolved oxygen in freshwater.

There are merits to both of these philosophies. However, the approach which has been adopted for establishment of a DO objective for the Great Lakes is that any change in the DO regime is likely to have some effect on the ecosystem and the magnitude of that effect will depend on the severity and duration of the change. It is felt that this approach is justified for the following reasons.

1. The objective provides both the correct oxygen tension gradient to move oxygen into the blood and sufficient oxygen to fulfill the requirements of metabolism.
2. The objective recognizes the greater metabolic demand for DO at elevated temperatures.
3. A reduction of DO levels to below saturation limits active metabolism, reduces maximum swimming speed of fish and causes a physiological, behavioral or other stress induced response. While the consequences of the chronic occurrence of such stress are not well understood, there is a reasonable likelihood that it is an important factor in the long term survival of the organism.
4. There is a small safety factor included between the objective and the concentration causing measurable harm to aquatic biota.

## DEVELOPMENT OF OXYGEN CRITERIA FOR FISH POPULATIONS

In line with the above discussion, the approach proposed by Davis (1975) for deriving DO criteria to protect fish has been adopted here. His approach involved the establishment of mean thresholds of incipient oxygen response thresholds by averaging data on oxygen levels for various assemblages of fish where sublethal responses to hypoxia first become apparent. Using the calculated mean threshold level, three levels of protection (A, B, and C) were devised as follows:

1. Level A: This level is one standard deviation above the mean average oxygen response level for a fish community (e.g. freshwater fish, including salmonids). It represents a high degree of safety for important fish stocks and permits 13-20% depression of oxygen from full saturation.

2. Level B: This level is the caluclated mean oxygen response threshold, and represents the oxygen value where the average member of a species of a fish community starts to exhibit symptoms of oxygen distress. Oxygen concentrations are allowed to be depressed from 35 to 45% below saturation.
3. Level C: This level is one standard deviation below the mean oxygen response threshold, and permits a large portion of a fish population to be affected. Oxygen concentrations may be depressed to 60% below saturation.

The calculated oxygen response thresholds for freshwater fish populations is presented in Table 2 (abridged from Davis, 1975).

## TABLE 2.
## Incipient dissolved oxygen Response Thresholds For Freshwater Assemblages Of Fish

| GROUP | PROTECTION LEVEL | $P_{O_2}$ MMHG | MG $O_2$/LITER |
|---|---|---|---|
| Mixed freshwater fish population including salmonids (15 °C) | A | 113 | 7.27 |
| | B | 86 | 5.26 |
| | C | 58 | 3.25 |
| Mixed freshwater fish population with no salmonids (18 °C) | A | 92 | 5.63 |
| | B | 73 | 3.98 |
| | C | 54 | 2.33 |
| Freshwater salmonids (including steelheads) (15 °C) | A | 119 | 7.84 |
| | B | 90 | 6.00 |
| | C | 61 | 4.16 |
| Salmonid larvae and mature eggs (9 °C) | A | 115 | 9.74 |
| | B | 120 | 8.09 |
| | C | 85 | 6.44 |

On the basis of the calculated critical partial oxygen pressures presented in Table 2, extrapolated objectives over the range of 0-25 °C were calculated as percent of saturation required to provide a desirable oxygen partial pressure and content. These values are intended to be minimum oxygen levels of body of water. Oxygen criteria based upon percent saturation are provided in Table 3 (abridged from Davis, 1975).

These objectives expressed as percent saturation over a range of temperatures, are intended to protect fish by providing both the correct oxygen gradient to move oxygen into the blood and sufficient oxygen to

## TABLE 3.

## CRITICAL CONCENTRATIONS OF OXYGEN REQUIRED TO MAINTAIN DIFFERING LEVELS OF PROTECTION FOR VARIOUS ASSEMBLAGES OF FRESHWATER FISH

| GROUP | PROTECTION LEVEL | $P_{O_2}$ MMHG | ML $O_2$ /LITER | MG $O_2$ /LITER | % SATURATION AT °C REQUIRED TO MAINTAIN PROTECTION LEVEL | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | 0° | 5° | 10° | 15° | 20° | 25° |
| Freshwater mixed fish population including salmonids | A | 110 | 5.08 | 7.25 | 69 | 70 | 70 | 71 | 79 | 87 |
| | B | 85 | 3.68 | 5.25 | 54 | 54 | 54 | 54 | 57 | 63 |
| | C | 60 | 2.28 | 3.25 | 38 | 38 | 38 | 38 | 39 | 39 |
| Freshwater mixed fish population with no salmonids | A | 95 | 3.85 | 5.50 | 60 | 60 | 60 | 60 | 60 | 66 |
| | B | 75 | 2.80 | 4.00 | 47 | 47 | 47 | 47 | 47 | 48 |
| | C | 55 | 1.75 | 2.50 | 35 | 35 | 35 | 35 | 35 | 36 |
| Freshwater salmonid population (including steelhead) | A | 120 | 5.43 | 7.75 | 76 | 76 | 76 | 76 | 85 | 93 |
| | B | 90 | 4.20 | 6.00 | 57 | 57 | 57 | 59 | 65 | 72 |
| | C | 60 | 2.98 | 4.25 | 38 | 38 | 38 | 42 | 46 | 51 |
| Salmonid larvae and mature eggs of salmonids | A | 155 | 6.83 | 9.75 | 98 | 98 | 98 | 98 | 100 | 100 |
| | B | 120 | 5.60 | 8.00 | 76 | 76 | 76 | 79 | 87 | 95 |
| | C | 85 | 4.55 | 6.50 | 54 | 54 | 57 | 64 | 71 | 78 |

Figure 3: Oxygen objective expressed as
mg/litre at various temperatures (interpolated between points)

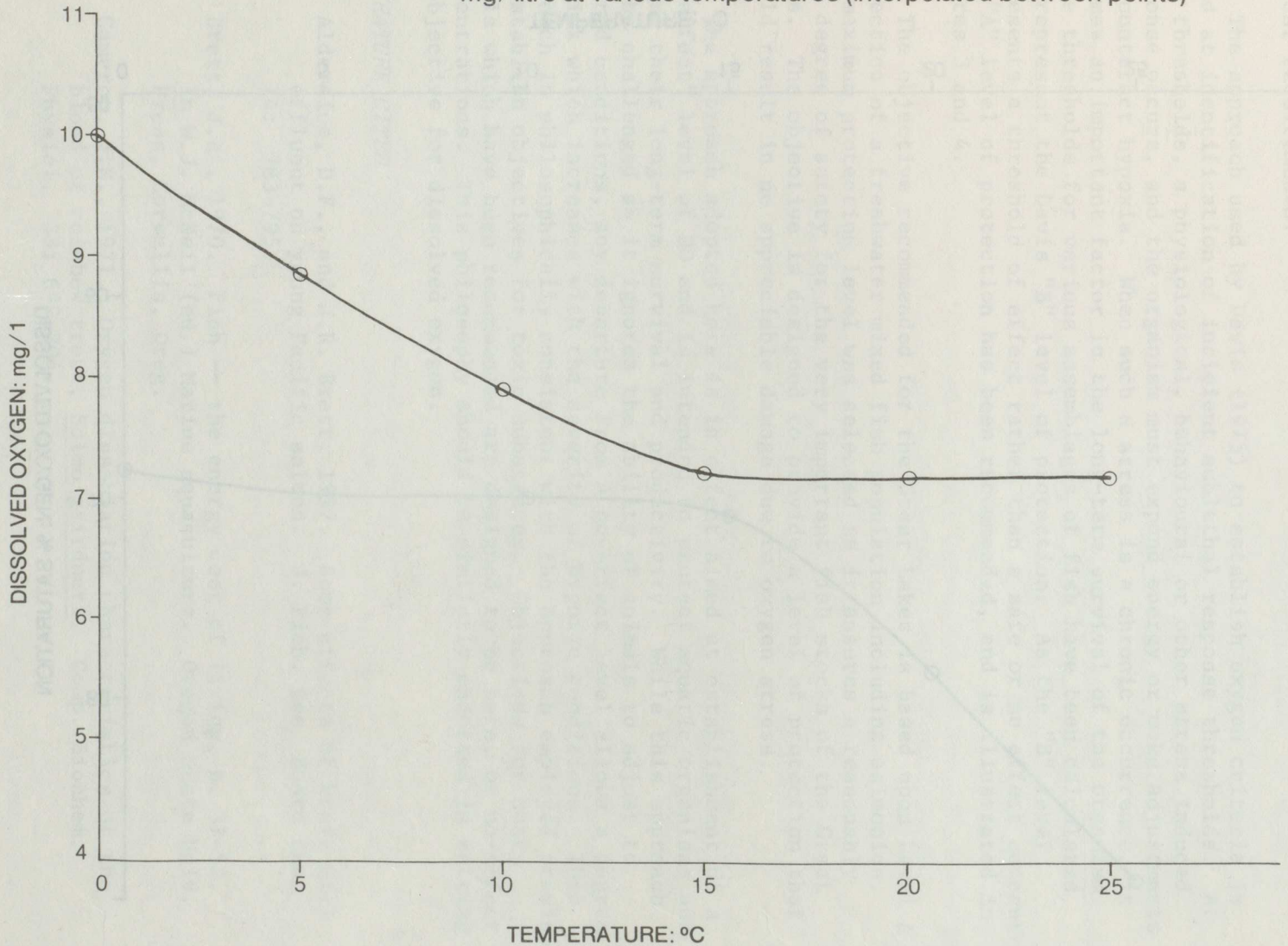
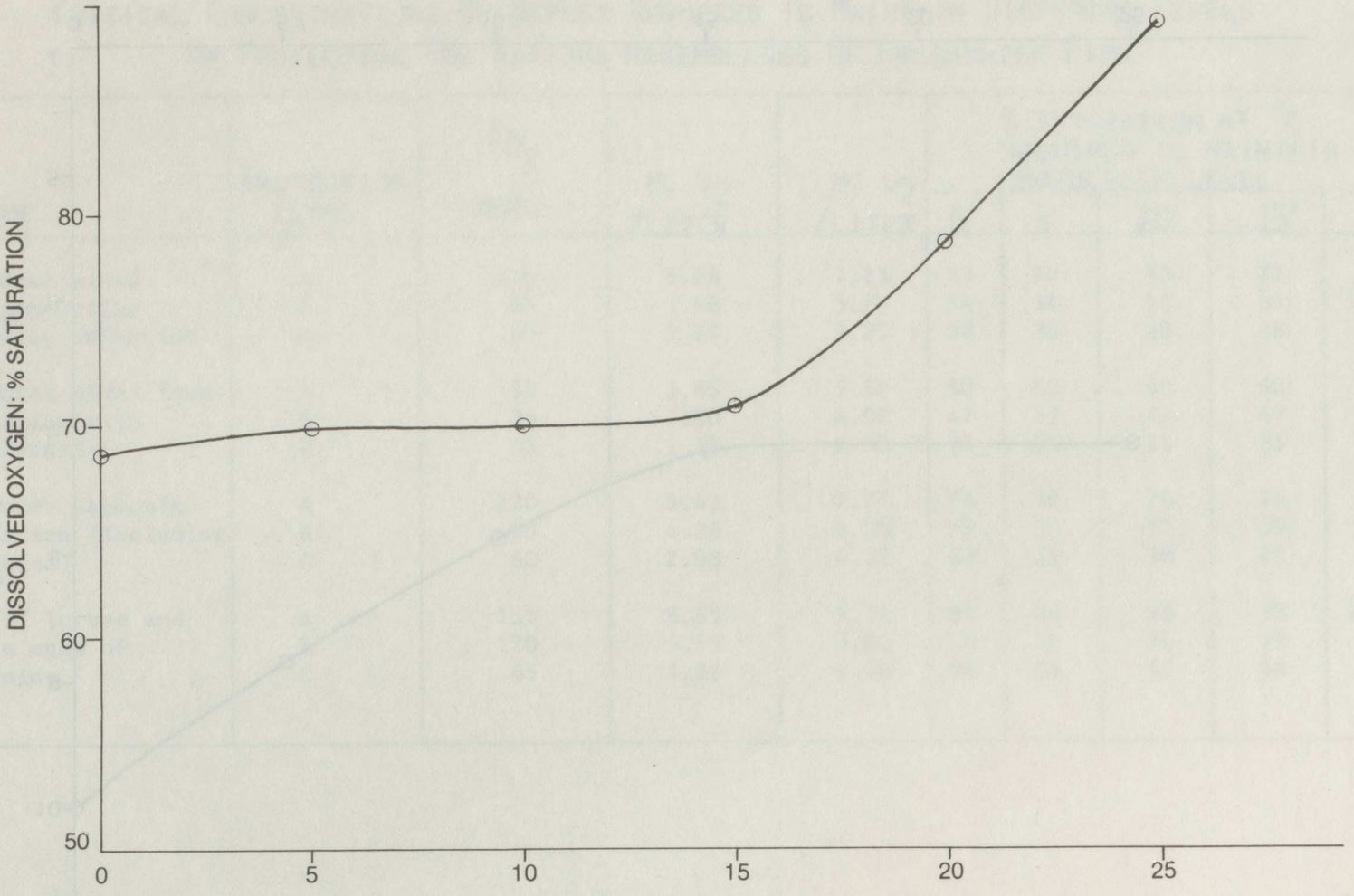


Figure 4: Oxygen objective expressed as percent saturation at various temperatures (interpolated between points)



fulfill the requirements of metabolism. At the lower temperatures where water solubility of oxygen is high, the criteria are based upon the $P_{O_2}$ values (Table 3) to ensure that a percent saturation value consistent with the required $P_{O_2}$ gradient was present. At the higher temperatures, higher percentage saturation values were necessary to provide the oxygen content requirements.

The approach used by Davis (1975) to establish oxygen criteria is aimed at identification of incipient sublethal response thresholds. At such thresholds, a physiological, behavioural or other stress induced response occurs, and the organism must expend energy or make adjustments to counteract hypoxia. When such a stress is a chronic occurrence, it becomes an important factor in the long-term survival of the organism. These thresholds for various assemblages of fish have been calculated and represent the Davis "B" level of protection. As the "B" level represents a threshold of effect rather than a safe or no effect concentration, the "A" level of protection has been recommended, and is illustrated in Figures 3 and 4.

The objective recommended for the Great Lakes is based upon Level A protection of a freshwater mixed fish population including salmonids. The maximum protection level was selected as it assures a reasonably high degree of safety for the very important fish stocks of the Great Lakes. The objective is designed to provide a level of protection that should result in no appreciable damage due to oxygen stress.

The approach adopted here is in effect aimed at establishment of a "no-effect" level of DO and is intended to protect aquatic organisms and ensure their long-term survival and productivity. While this approach can be challenged as it ignores the ability of animals to adjust to altered conditions, any departure from a no-effect level allows a degree of risk which increases with the severity of hypoxic conditions. This approach is philosophically consistent with the approach employed previously to establish objectives for toxic substances. Objectives for heavy metals which have been recommended are designed to be safe, or no-effect concentrations. This philosophy should be similarly endorsed in setting an objective for dissolved oxygen.

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## SILVER

*RECOMMENDATION*

It is recommended that the following new objective for silver be adopted:

*Concentrations of total silver in an unfiltered water sample should not exceed 0.1 micrograms per litre to protect aquatic life.*

*RATIONALE*

Silver occurs in the native state as a constituent of various natural alloys and in a great variety of minerals combined with sulfur, antimony, arsenic, tellurium and selenium (Boyle, 1968). The average silver content of soils is about 0.10 mg/kg and certain coals contain considerable amounts of silver (2-10 mg/kg in the ash). The silver halides, such as silver iodide, are relatively insoluble in water in contrast to silver nitrate, the most common salt. A very comprehensive review of the sources, use, distribution, losses to the environment and human health aspects of silver has been prepared by Carson and Smith (1975).

The results of calculated particulate emissions of trace elements from air pollution sources in Chicago, Milwaukee and northwest Indiana indicate that approximately 3,000 kg per year of silver, attributed totally to fuel oil, enters the atmosphere in the vicinity of Lake Michigan (USEPA, 1972). Photoprocessing is also a source of silver. The mean silver concentration in effluent from a photographic industry's activated sludge plant was 70 µg/1. In the Genessee River, downstream from another photoprocessing plant, silver concentrations were as high as 260 µg/1 although most samples were less than 20 µg/1. Water in a nearby Lake Ontario water intake had a concentration of 1 µg/1 silver, and sediments of the Genessee showed elevated silver concentrations (Bard et al., 1976).

Other sources of silver emissions to the environment are from mining and processing of silver ores, industrial electronics use, and cloud seeding with silver halides. Analyses of various effluents from municipal wastewater treatment plants indicated silver concentrations from 0.05 - 45 µg/1 (Carson and Smith, 1975).

Copeland and Ayers (1972), found an average of 0.3 µg/1 silver in Lake Michigan waters. In a survey of trace metals in the waters of the United States, Kopp and Kroner (1967) indicated that silver was detectable in less than 7 percent of all samples with a mean observed value of 2.6 µg/1. The greatest occurrence of silver was in the Colorado River basin where it was observed in 18% of the samples at a mean concentration of 5.8 µg/1. In the St. Lawrence River, concentrations in water from inshore intake pipes ranged from non-detectable to 6 µg/1. The mean concentration was 2.6 µg/1. These concentrations are suspiciously high in light of toxicity data and the much lower values observed by Copeland and

Ayers (1972) and Bard et al, (1976). They suggest either local inshore contamination or deficiencies in the analytical procedures. Silver has not been included in past monitoring of offshore water quality in the Great Lakes.

There are few data on mammalian toxicity of silver. In rats, the 24-48 hr. $LD_{50}$ for $AgNO_3$ was 25.2 mg/kg and the silver accumulated in the heart, lungs, spleen, kidney and liver (DeQuidt et al., 1974). Quality Criteria for Water (EPA, 1976) recommends a limit of 50 µg/l for domestic water supplies based on various human symptoms including argyria, a localized skin discoloration (greyness) following prolonged ingestion. Argyria in the eye can lead to blindness and in the lung to interference in function (Carson and Smith, 1975). In Canada, the maximum permissible concentration is also 50 µg/l (DNHW, 1969).

A survey of silver toxicity to bacteria and fungi showed that growth of Mycobacterium, the most sensitive species tested, was completely inhibited by 10 µg/l (Golubovitch, 1974). Growth of Aspergillus niger, the least sensitive species, was completely inhibited by 500 µg/l.

Silver is classified as very toxic to plants by Bowen (1966) who observed toxic effects at concentrations below 1 mg/l in the nutrient solution. Growth of Chlorella pyrenoidosa, Chaetoceros galvestonensis and Cyclotella nana was completely inhibited by 100 µg/l of silver (Fitzgerald, 1967; Hannan and Patouillet, 1972). A concentration of 420 µg/l was highly toxic to six species of algae (Palmer and Maloney, 1955). The most sensitive alga appears to be Scenedesmus sp. Scenedesmus demonstrated no increase in cell growth after five days exposure to a concentration of silver of 30 µg/l (Bringmann and Kuhn, 1959).

Invertebrates are also sensitive to silver. After 24 hours, 50 percent or more of Daphnia magna were dead or obviously incapacitated at a concentration of 19 µg/l silver (Bringmann and Kuhn, 1959). After 64 hours, 50 percent of Daphnia magna were immobilized at a concentration of silver of 3.2 µg/l (Anderson, 1948). Fifty percent of a planarian population (Polycelis nigra) were killed within 2 days at an exposure to silver of 150 µg/l (Jones, 1940). Nehring (1973) tested the response of several aquatic insects to silver. The 15-day $LC_{50}$ for silver for mature stonefly naiads (Pteronarcys californica) was 8.8 µg/l. Immature naiads of the same species were more sensitive with a 7-day $LC_{50}$ of less than 4 µg/l. Nearly all mayflies (Ephemerella grandis) were killed in concentrations of silver as low as 9 µg/l after 10 days. The largest bioconcentration factor was about 300 times with a tissue level of 20 mg/kg. The 96-hr. $LC_{50}$'s for Mercenaria mercenaria (hard clam) and Crassostrea virginica (oyster) were 21 and 5.8 µg/l respectively in seawater at a temperature of 26°C. (Calabrese and Nelson, 1974; Calabrese and Collier, 1973). "Effective control" of the nematode Pratylenchus penetrans, a plant parasite, was achieved with 10 µg/l silver, although other nematodes were less sensitive (Pitcher and McNamara, 1972). First stage larvae of a marine crustacean, Palaemon serratus, changed their orientation to light at 200-500 µg/l silver, concentrations similar to the 96-hr. $LC_{50}$. Larvae of Carcinus maenus were more sensitive with a 96-hr. $LC_{50}$ of 10-100 µg/l and a change in photokinesis at 1 µg/l (Amiard, 1976).

Jones (1939) exposed sticklebacks (Gasterosteus aculeatus) for 10 days and observed no mortality different from the controls at a silver concentration of 3.0 µg/1. At higher concentrations, mortality was increased. Bluegills (Lepomis macrochirus) and largemouth bass (Micropterus salmoides) were exposed to silver nitrate at concentrations of 77 and 0.9 µg/1 (Coleman and Cearley, 1974). At 77 µg/1, all of the largemouth bass died within 24 hours while the bluegill survived for six months. Tissue concentrations equilibrated within 2 months of exposure and resulted in a maximum bioconcentration factor of approximately 200 times. The 96-hr. $LC_{50}$ for killifish (Fundulus heteroclitus) was 40 µg/1 while 30 µg/1 inhibited hepatic alkaline phosphatase, xanthine oxidase, catalase and RNA-ase and 20 µg/1 increased activity of hepatic δ-amino levulinic acid dehydratase (Jackim, 1973).

A two-month exposure of rainbow trout starting with the egg stage resulted in a total mortality of the fish at all concentrations equal to or greater than 2.5 µg/1 (Goettl et al., 1973). Hatching of the eggs was greatly accelerated at those concentrations and this caused insufficiently developed larvae. At a concentration of 1.2 µg/1 there was a 40 percent mortality and the next lower concentration of 0.6 µg/1 resulted in reduced growth of the rainbow trout. In a subsequent study (Goettl et al., 1974) there was a significant reduction in growth after nine months at a concentration of silver of 0.69 µg/1. Premature hatching of eggs and retarded sac fry development occurred at silver concentrations of 0.69, 0.34 and 0.17 µg/1. After 18 months, percent mortality for the various concentrations of silver were: 0.69 µg/1, 95%; 0.34 µg/1, 49%; 0.17 µg/1, 36%; and at 0.09 µg/1, 17%. The mortality at 0.09 µg/1 was not different from that of the control. Goettl and Davies (1975) continued their studies on silver toxicity by using silver iodide (solubility = 0.3 µg/1) in contrast to earlier studies that used silver nitrate. The exposure began with rainbow trout eggs and after three months exposure there was significant mortality of fry after swim-up at concentrations of 0.19 µg/1 and above. A concentration of 0.11 µg/1 was not significantly different from the control. More recent data presented by Davies (1976) show that mortality of fry after swim-up decreased from 38% at 0.50 µg/1 silver to 18% at 0.13 µg/1. There was no control mortality, and 3% mortality at 0.07 µg/1. Davies (1976) recommended a concentration between 0.07 and 0.13 µg/1 as being "safe".

Freeman (1975) studied the annual atmospheric and hydrologic silver budget of an alpine lake system. In this area silver iodide is the most widely used precipitation nucleating agent for weather modification. Silver iodide is commonly injected into the atmosphere by surface generators or airborne pyrotechnic flares. Sub-micron silver iodide particles are thus introduced into the atmosphere. Routine burn rates range from 0.1 to 0.5 g of silver iodide per minute for periods ranging from 2-8 hours. The amount of silver falling in rain will depend on the success of seeding and the concentration will depend on the ratio of "silver seed" to water. Some typical values for rainfall are 1-216 ng/1 while rainfall affected by cloud seeding may contain 1-4,500 ng/1. Snow affected by cloud seeding contained 110 ng/1 during one study (Carson and Smith, 1975). Concentrations

of silver in muscle of cutthroat trout, determined by atomic absorption spectrophotometry, were from 0.11 to 0.37 mg/kg. The concentrations in the bone ranged from 2.6 to 4.4 and in the liver from 0.29 to 2.32 mg/kg. Concentrations in the water itself ranged from 0.24 to 0.76 µg/1. Concentration factors for liver and muscle appear to range from 140 - 10,000 with a potential concentration factor for bone of up to 18,000.

Copeland and Ayers (1972) determined the average trace element concentrations in Lake Michigan using neutron activation. The concentration in water was 0.3 µg/1; sediment, 0.6 mg/kg; zooplankton, 0.04 mg/kg; benthos 0.10 mg/kg; phytoplankton, 0.09 mg/kg; and fish, 0.01 mg/kg. The fish samples were the edible portions and all results are based on wet weight. The observed silver concentration factors in Lake Michigan biota were: phytoplankton, 300; zooplankton, 133; and benthos, 330.

Silver concentrations were also measured in various species of fish in Lake Michigan by Copeland, et al., (1973). Only muscle fillets were analyzed using neutron activation with results based on wet weight. The residues were: coho salmon, 0.034 mg/kg; yellow perch, 0.028 mg/kg; lake trout, 0.036 mg/kg; brown trout, 0.044 mg/kg; and whitefish, 0.032 mg/kg. Whole fish analyses were performed on spottail shiners, 0.036 mg/kg; rainbow smelt, 0.039 mg/kg; and alewife, 0.034 mg/kg.

Kibiya and Oguri (1961) determined the distribution of radioactive silver injected into goldfish. The silver was concentrated to a large extent in the liver of these fish with small amounts distributed throughout other tissues. Silver concentrations were determined by neutron activation of whole fish and fish livers from Lakes Michigan, Superior and Erie. In all samples, silver concentrations were equal to or less than 1 ng/kg (Lucas, et al., 1970). These values appear unrealistically low compared to those of the other authors.

The silver concentration in lake trout of known age from Lake Cayuga was analyzed by spark source mass spectrometry. The concentration of silver in these samples ranged from 0.48 to 0.68 µg/kg fresh weight (Tong, et al., 1974). There was no relationship between silver concentration and age of the lake trout and those values are about 10 times higher than seen by Copeland et al., (1973). In an earlier study various fish from 11 New York State waters were analyzed for silver by spark source mass spectrometry following dry ashing, Tong et al., (1972). Of 48 samples all but three fell within the range of 0.01 to 0.06 µg/kg. The other samples were 0.11, 0.25 and 0.65 µg/kg.

It would appear from the foregoing analyses that there is a wide discrepancy in the reported results for silver residues in freshwater fish. This may be due to a variety of analytical procedures that were not well established at the time of analysis.

## CONCLUSION

It is obvious that many natural silver compounds are relatively insoluble but concentrations below the solubility limit are toxic to fish. Because of its extreme toxicity to fish during long term exposures, an objective for total silver of 0.1 µg/1 is recommended.

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## MIREX

*RECOMMENDATION*

*For the protection of aquatic organisms and fish consuming birds and animals, mirex including its degradation products should be substantially absent from water and aquatic organisms. Substantially absent here means less than detection levels as determined by the best scientific methodology available.*

Note: The best detection levels of mirex (1977) as determined by a survey of laboratories in the Great Lakes region are 0.005 µg/l for water and 0.005 µg/g for biological tissues.

*RATIONALE*

Although mirex was never used as an insecticide in the Great Lakes' region, it was reported as a contaminant in fish tissues from Lake Ontario (Kaiser, 1974). Subsequent investigations showed the chemical's other use as a flame retardant under the tradename Dechlorane and its manufacture, processing, and use in the Great Lakes' Basin. Further studies also proved its widespread presence in Lake Ontario sediments (Thomas, 1976), and in herring gulls (Hallett et al., 1976). Other monitoring surveys showed its presence in fish of all trophic levels in Lake Ontario (OME, 1976; NYDEC, 1976; OMAF, 1975; FIB, 1974). This wide spread contamination of the Lake Ontario and St. Lawrence ecosystems is evident from the data presented in Tables 4 and 5.

### TABLE 4.

### MIREX IN LAKE ONTARIO FISHES, SEDIMENTS AND HERRING GULL EGGS
### DATA FROM OME, 1976; OMAF 1975; NYDEC, 1976; FIB, 1974; THOMAS ET AL., 1977; AND GILMAN ET AL., 1977.

| SAMPLE | (NO. OF SPECIMEN) | MIREX (µG/G) | |
|---|---|---|---|
| | | MEAN | RANGE |
| Sediments | (229) | - | ND-0.40 |
| Herring gull eggs | (39) | 5.06 | 1.95-18.6 |
| American eel | (17) | 0.46 | ND-1.39 |
| Lake trout | (47) | 0.22 | 0.02-0.97 |
| Chinook salmon | (16) | 0.21 | 0.08-0.36 |
| Brown trout | (50) | 0.18 | 0.01-0.35 |
| Coho salmon | (91) | 0.17 | ND-0.50 |
| Splake | (1) | 0.16 | - |
| Smallmouth bass | (76) | 0.13 | ND-0.39 |
| Smelt | (296) | 0.13 | 0.01-0.35 |
| White perch | (41) | 0.10 | ND-0.64 |
| Gizzard shad | (16) | 0.09 | ND-0.21 |
| Yellow perch | (148) | 0.08 | ND-0.18, 1.20* |

TABLE 4. (CONT'D)

| SAMPLE | NO. OF SPECIMEN | MIREX (µG/G) | |
|---|---|---|---|
| | | MEAN | RANGE |
| Brown bullhead | (175) | 0.07 | ND-0.38, 0.76* |
| Rock bass | (15) | 0.07 | ND-0.24 |
| White bass | (60) | 0.07 | ND-0.16 |
| Alewife | (86) | 0.05 | ND-0.23 |
| Rainbow trout | (77) | 0.04 | ND-0.31 |
| Northern pike | (31) | 0.03 | ND-0.10 |
| Carp | (12) | 0.02 | ND-0.14 |
| White sucker | (117) | 0.02 | ND-0.10 |
| Black Crappie | (5) | 0.01 | 0.01-0.02 |
| Largemouth bass | (7) | 0.01 | <0.01-0.02 |
| Walleye | (4) | 0.01 | ND-0.02 |
| Pumpkinseed | (51) | <0.01 | ND-0.03 |
| Freshwater Drum | (1) | ND | ND-0.03 |

* ND, None detected
* One analysis high, not considered for mean value

TABLE 5.

MIREX IN ST. LAWRENCE RIVER FISHES

| SAMPLE (NO. OF ANALYSES) | MIREX (µG/G) | | REFERENCE |
|---|---|---|---|
| | MEAN | RANGE | |
| Maskinonge (2) | 0.08 | 0.04-0.11 | NYDEC, 1976 |
| Northern pike (4) | - | 0.04-0.09 | NYDEC, 1976 |
| Smallmouth bass (34) | - | 0.01-0.27 | NYDEC, 1976 |
| Walleye (4) | - | 0.02-0.10 | NYDEC, 1976 |
| Yellow perch (12) | - | 0-0.11 | NYDEC, 1976 |

Mirex, the common name for dodecachloro-octahydro-1,3,4-metheno-2H -cyclobuta(c,d)-pentalene, ($C_{10}Cl_{12}$), is extremely resistant to biological and chemical degradation and has been reported as one of the most stable compounds when evaluated in aquatic model ecosystems (Mehendale et al., 1972; Metcalf et al., 1973). Only trace amounts of incompletely identified metabolites were observed in biota at 33 days exposure in an aquatic and terrestrial model system (Stein et al., 1976). Under ultraviolet or gamma-irradiation, mirex has been shown to undergo slow and structurally quite limited reactions with the formation of "photomirex" ($C_{10}HCl_{11}$), dihydro derivatives and Kepone ($C_{10}Cl_{10}O$) a related insecticide (Carlson et al., 1976; Lane, 1973; Alley et al., 1972). Some of these degradation products have been observed in herring gulls and fish (Hallet et al., 1976; Norstrom et al., 1976; Sabatino, 1976).

EFFECTS ON AQUATIC BIOTA

Effects of mirex on the growth and productivity of Chlorella pyrenoidosa were studied by Kricher et al. (1975). Mirex concentrations of 100 µg/1 had some negative effects on this alga at an exposure time of 164 hrs. Hollister et al. (1975) reported the accumulation of mirex from water in several species of algae. At 0.2 µg/1 mirex for seven days, the growth of these species was little affected. In separate experiments, the seven days exposure of the algae to mirex concentrations of 0.010, 0.025, and 0.050 µg/1, respectively, led through bioaccumulation to levels of approximately 10 to 300 µg/kg in Chlorococcum sp., indicating an accumulation factor greater than 10,000-fold.

Ludke et al. (1971) studied the response of juvenile crayfish (Procambarus blandingi and Procambarus hayi) to mirex. At an exposure of 5 µg/1 mirex in water for six hours, no dead crayfish were observed. However, the same animals, subsequently placed in clean water, showed 26% mortality 10 days after the exposure relative to the control. At an exposure concentration of 5 µg/1 for 58 hours, 5% mortality was recorded at the termination of the exposure and 98% mortality after an additional 10 days in clean water. Similarly, a concentration of 1 µg/1 mirex for 144 hours resulted in 0% mortality within that period and in 95% mortality 10 days after treatment. Procambarus hayi, exposed to 0.5 and 0.1 µg/1 mirex for 48 hours, resulted in initial mortalities of 12% and 19%, respectively, and in 71% and 65% mortalities within 4 days after the exposure. In other experiments by Ludke et al., (1971), juvenile crayfish were exposed to water leaching, mirex bait granules. At seven days exposure, 95% mortality was observed, corresponding to a mirex concentration of 0.86 µg/1 in the water. The dead crayfish were found to have mirex accumulated to a mean of 1.5 µg/g. At a mirex concentration as low as 0.073 µg/1, 12.5% mortality was observed after the first day of exposure. From this report, it appears obvious that mirex is extremely toxic to those species of crayfish. Mortality increases with the time exposure to the insecticide and, in most cases, significantly delayed toxic action was observed several days after exposure.

A study by Tagatz (1976) on the effect of mirex on predator-prey interaction resulted in significantly increased mortality of grass shrimp (Palaemonetes vulgaris) through predation by pinfish (Lagodon rhomboides). Effects of mirex exposures were observed at concentrations as low as 0.025 µg/l at times of 13 days. Mirex also proved to be a stomach poison more potent than DDT to juvenile blue crabs (Callinectes sapidus) as shown by Leffler (1975). Dietary concentrations of 1.5 and 3.0 µg/g when given to the blue crabs resulted in a strong reduction of autotomisation of damaged limbs. Brown et al., (1975) reported the effect of mirex on estuarine microorganisms. No inhibition of bacterial growth, was observed at exposure times up to 96 hrs and mirex concentrations up to 10 µg/l with several pure bacterial cultures in saline medium. At 100 µg/l mirex for four days, no significant effects were observed on the rates of ammonification of protein and on nitrate reduction, although the latter process was initially delayed.

In another study by Van Valin et al. (1968), bluegills (Lepomis macrochirus) and goldfish (Carassius auratus) were exposed to mirex by feeding with mirex treated diet or by treating holding ponds with mirex bait. At an exposure level of 5 mg/kg day bodyweight, the growth of bluegills was reduced. Gill and kidney breakdown occurred in goldfish when exposed to mirex in water at 1 µg/l for 56 days.

Collins et al. (1973) studied the uptake of mirex by caged and uncaged channel catfish (Ictalurus punctatus) in ponds. Uncaged fish had a mean of 0.65 µg/g mirex residue six months after the application, while caged adult fish and caged fingerlings had less than 0.01 µg/g and 0.03 µg/g residue respectively, indicating a strong bioaccumulation of mirex via the food chain. Residues in cricket frogs were found to be 2.88 µg/g mirex six months after the treatment. In similar work by Hyde et al. (1974), the effects of mirex on the channel catfish production in ponds were investigated. After three areal applications of mirex over six months, at the normal rate of 1.4 kg/ha bait (4.2 g/ha mirex), the fish had accumulated mirex to an average of 0.015 µg/g in fillets and 0.255 µg/g in fat. At the same time, survival of fish in the experimental ponds was reduced to 43.3% compared with 71.6% for the control. The body weight of the treated fish was approximately 17% that of control fish.

The acute effects of mirex on juvenile and adult striped mullet (Mugil cephalus L.) were studied by Lee et al. (1975). The survival of young juvenile mullet was significantly reduced in 96-hr flow-through tests at mirex concentrations of 0.1 and 1.0 mg/l. The movement of mirex from contaminated sediment was observed by Kobylinski and Livingston (1975). Under flow conditions, approximately 40% of the contaminant was lost from the sediment and accumulated in the tissues of hogchoker (Trinectes maculatus). The authors proposed a direct uptake route of mirex from the sediment, and also found a correlation of mirex concentrations in sediment and water.

Borthwick et al. (1973) reported the accumulation of mirex in South Carolina estuaries after the application of mirex bait (1.7 g/ha mirex) for the fire ant control program. They concluded that mirex was translocated from treated land and high marsh to the estuarine biota and that bio-accumulation occurred especially in predators, such as racoons and birds. In the test area water samples contained mirex only below the detection limit of 0.01 µg/1. Approximately one year after the bait application, sediments contained 0-0.07 µg/g, crabs 0-0.60 µg/g, shrimps 0-1.3 µg/g, mammals 0-4.4 µg/g, and birds 0-17.0 µg/g mirex at a detection limit of 0.01 µg/g for sediments and biota.

Wolfe and Norment (1973) sampled an area for one year following an application of mirex bait (1.7 g/ha mirex). Crayfish residues ranged from 0.04 to 0.16 µg/g. Fish residues were about 2 to 20 times greater than in controls and averaged from 0.01 µg/g for bluegills to 0.53 µg/g for mosquitofish. These values are, in general similar to those reported by Naqui and DeLaCruz (1973) and by Borthwick et al. (1973). Naqui and DeLaCruz (1973) observed mirex residues of 0.15 µg/g in molluscs, 0.25 µg/g in fish, 0.29 µg/g in insects, 0.44 µg/g in crustaceans and 0.63 µg/g in annelids.

An evaluation of mirex and other organochlorine pesticides by Metcalf et al. (1973) found mirex to be one of the most stable compounds studied in such a model ecosystem.

## EFFECTS ON BIRDS AND MAMMALS

Mirex fed to birds was observed to be slow-acting, causing delayed mortality up to 15 days after dosage ceased (Stickel et al., 1973). Reproduction among penned mallards and bobwhite was not affected when fed diets containing 1 or 10 µg/g mirex (Heath and Spann, 1973), although carcass residues of surviving birds were as high as 287 µg/g. In similar experiments by Medley et al. (1974), roosters were fed diets containing mirex at 0.007 µg/g, 0.06 µg/g, 0.710 µg/g and 7.2 µg/g for periods up to 32 weeks. The highest residue levels observed were 994 µg/g in fat of roosters on a 7.2 µg/g diet. Studies by Hyde et al. (1973) on the reproductive success of mallard ducks fed mirex at concentrations of 0, 1, and 100 µg/g in the diet for 25 weeks did not show significant differences on egg production, shell thickness, shell weight, embryonation, and hatachability, but at 100 µg/g duckling survival was reduced to a mean of 72.6% compared to 95.7% for the control for a 14-day post-hatch period on uncontaminated food, based on ducklings hatched. At the 100 µg/g diet, fat of adult ducks contained a mean of 2,964 µg/g, while the eggs contain a mean of 277 µg/g mirex. Further reports by Woodham et al. (1975), indicated the accumulation of mirex in tissues and eggs of hens exposed to dietary mirex levels of 0.01 and 1.0 µg/g for periods up to 40 weeks. The highest residues observed in the eggs were 2.03 µg/g. After 13 weeks of contaminant free diet, the mirex residues in the eggs had decreased to 0.66 µg/g.

Feeding experiments of Japanese quail, given a single oral dose (1.2 µg/g) of $^{14}C$ - mirex, were studied by Ivie et al. (1974). Partial excretion of mirex via feces was initially observed for both sexes, but male quails excreted approximately twice the amount as female birds. However, after the initial excretion, no further residues were found in the feces and male birds had body burdens of more than 50% of the single

dose, 84 days after dosing. Egg laying quail hens had eliminated 85% of the single dose via the egg yolks in 84 days. The recoveries of the $^{14}C$ - mirex from the whole body residues were 97% or greater and the analysis of the extracts revealed unmetabolized mirex only.

Innes et al. (1969) reported on bioassays for the tumorigenicity of 120 chemicals. At a dietary level of 26 µg/g mirex, a significantly elevated tumor incidence was observed after 18 months. Similar effects were observed for ten of the other 120 tested compounds.

## CONCLUSIONS

In general, little information exists on the effects of mirex on freshwater organisms as most of the results mentioned above are on estuarine and marine ecosystems and their respective biota. Several of such species, however, are also common to Great Lakes' waters, but the information is still insufficient to allow the formulation of safe levels of mirex in water or biota. There is a lack of studies on chronic exposure at low concentration. Mirex has been shown to be extremely persistent under most environmental conditions, is accumulating in food chains and is toxic to a wide variety of species. In addition, there is evidence for its cumulative and strongly delayed toxic effects in some species.

Recently the U.S. Food and Drug Administration and the New York State Department of Environmental Conservation (U.S.FDA, 1974; Berle, 1976) have introduced an action guideline of 0.1 µg/g mirex for edible portions of fish for the protection of human consumers. Recognizing that this limit for edible portion of fish may be inadequate for the protection of fish consuming birds and mammals, it is recommended, that the objective for "Persistent Organic Contaminants" (Great Lakes Water Quality, 1974, Appendix A, p. 32) be applied. Therefore, it is recommended that the concentrations of mirex in water and biota should not exceed the detection limits as determined by the best scientific methodology available at the time. The present detection levels, as determined by a survey of four laboratories in the Great Lakes region, are 0.005 micrograms per liter for water and 0.005 micrograms per gram for biological tissues.

In view of the occurrence of the structurally related mirex derivatives "photomirex" and Kepone together with mirex, and in the absence of the necessary information on their sub-lethal properties, it is recommended that such derivatives be included in further investigations of the toxic effects of mirex. Furthermore, such derivatives should be included in the residue analysis for mirex for the purpose of this objective, which is indicated by the expression "mirex including its degradation products". For surveillance purposes, it is further advised, that emphasis be placed on biological tissues rather than water analysis, as it appears likely that mirex concentrations in water below the detection limit may result in detectable residues in aquatic life.

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## SUBSTANCES RESEARCHED - INSUFFICIENT SCIENTIFIC DATA TO RECOMMEND DEFENSIBLE OBJECTIVES

The available scientific information on the substances presented below was thoroughly researched. Information was found to be inadequate to the degree that, in the judgement of the members of the Subcommittee and Task Force, scientifically defensible objectives could not be developed at this time. The toxicity data researched are recorded to eliminate duplication of effort when sufficient new data becomes available to justify the formulation of objectives.

### ANTIMONY, BARIUM, BORON, COBALT, MANGANESE, MOLYBDENUM, VANADIUM

*RECOMMENDATION*

No objectives for these metals are recommended at this time due to an inadequate data base on their effects on aquatic life.

*RATIONALE*

For the listed metals, there are some raw water standards for the protection of human health and some standards for agricultural purposes, either livestock watering or irrigation (NAS/NAE, 1974; Ontario Ministry of the Environment, 1974; Environment Canada, 1972). These are presented in Table 6.

### TABLE 6.

### MAXIMUM ALLOWABLE CONCENTRATIONS (MG/L)* OF VARIOUS METALS IN RAW WATER AND AGRICULTURAL WATER SUPPLIES

| | RAW WATER | | AGRICULTURE | |
|---|---|---|---|---|
| | CANADA | U.S. | CANADA | U.S. |
| Antimony (Sb)** | - | - | - | - |
| Barium (Ba) | 1 | 1 | - | - |
| Boron (B) | 5 | 1 | 0.5 | 0.75 |
| Cobalt (Co) | - | - | 10 | 5 |
| Manganese (Mn) | 0.05 | 0.05 | 20 | 10 |
| Molybdenum (Mo) | - | - | 0.05 | 0.01 |
| Vanadium (V) | - | - | 10 | 1.0 |

*Raw water quality objectives and standards for a number of these metals are currently under review in both the U.S. and Canada.

**No levels specified.

While there is an inadequate data base to assess the necessary levels of these metals for protection of aquatic life, there is enough information to demonstrate that the concentrations presented in Table 6 will not necessarily provide such protection. An indication of such data is presented in Table 7.

# TABLE 7.

## EFFECTS OF SB, BA, B, CO, MN, MO AND V ON AQUATIC ORGANISMS

| | | FISH (MG/L) | INVERTEBRATES (MG/L) |
|---|---|---|---|
| Antimony (Sb) | 9-20 | 96 hr $LC_{50}$, Pimephales promelas (fathead minnow) | 9.0 Retarded movement, D. magna |
| Barium (Ba) | 50 | C.N.S. effects[1], Oncorhynchus kisutch (Coho salmon) | 6-9 Reproductive impairments, D. magna |
| Boron (B) | 113 | 283 hr. $LC_{50}$, O. kisutch Coho salmon) | <.68 $NaBO_3$[3] - immobilization, D. magna |
| Cobalt (Co) | 10 | asymptotic[2] $LC_{50}$, (Gasterosteus aculeatus (Stickleback) | 0.01 reproductive impairment, - 0.012 D. magna |
| Manganese (Mn) | 40 | asymptotic[2] $LC_{50}$, Gasterosteus aculeatus (Stickleback) | 4-5 reproductive impairment, D. magna |
| Molybdenum (Mo) | 70-370 | 96 hr $LC_{50}$, P. promelas (Fathead minnow) | -- ----- |
| Vanadium (V) | 4-55 | 96 hr $LC_{50}$, P. promelas (Fathead minnow) | -- ----- |

1. Central Nervous System 2. Exposure Until Direct Mortality ceases
3. Other salts, less toxic

As a result, no objectives for these metals are being presented at this time. However, the data are being continuously examined and objectives will be recommended when adequate data become available.

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## ORGANOTIN COMPOUNDS

*RECOMMENDATION*

No objective can be recommendated for organotin compounds at this time due to an inadequate data base on the effects of the individual alkyltin compounds on aquatic life.

*RATIONALE*

There are three main areas in which organotin compounds have product and process utility: (1) heat stabilizers, (2) catalytic agents, and (3) biocidal compounds. Organotin derivatives account for the fourth largest production of organometallics amounting to about 3-4 million pounds per year.

The structures and use patterns of these compounds are shown in Table 8 and Figure 5 (Piver, 1973). The largest proportion of organotin stabilizer production is for the stabilization of polyvinyl chloride (PVC) polymers. The concentration range of organotin compounds required to heat stabilize PVC during processing is between 0.5 to 3.0 parts per hundred parts of resin. In the production of polyurethane foams, organotin catalysts allow the foam to be made directly from hexamethylene dioxocyanate and 1, 4-butanediol. The organotin catalysts more commonly used are dibutyltin salts such as the diacetate dilaurate, dichloride, dilauryl mercaptide, and dimethyltin dichloride.

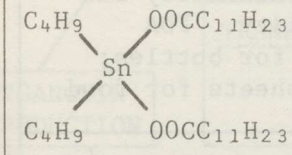
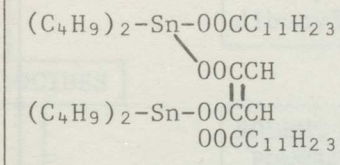
Organotins are employed in a host of other applications that include: (a) in rubber products and paints - as antioxidants and anticracking agent, to retard rubber deteriorations, and as stabilizers of chlorinated rubbers or chlorinated paints; (b) in transformers, capacitors and cables - to prevent corrosion by serving as scavengers for HC1 formed if a short circuit occurs in transformers, etc.: (c) in lubricants and textile oils - as anti oxidants and corrosion-reducing adjuvants for lubricants and as anti-oxidants for textile oils; (d) as activators and catalysts - in oxidation, polymerization; (e) tin-containing polymers: (f) treatment of fibreglass for adhesion to resins; and (g) curring catalysts for application of silicones to textiles and paper.

The biocidal applications of organotin include: (a) agricultural fungicides - triphenyltin acetate (Brestan, Fentin acetate), triphenyl hydroxide (Duter, Fentin hydroxide), and bis (tri-n-butyltin oxide (TBTO)): (b) general fungicidal action (triphenyltin chloride, TBTO) in paints, for preservation of manila and sisal ropes, leather, and textiles, to confer mildew resistance to fabrics, for protection of jute and jute bags, as wood preservative, slimicide, and for production process paper; (c) bactericides and biostats - disinfectant (triaryltin) and bactericides for seeds; (d) antihelmintics - against worms in poultry (dibutylin laurate, tin oleate, tetraisobutyltin; (e) nematocide- p-bromophenoxytriethyltin; (f) herbicides -vinyltin compounds (trivinyltin chloride); (g) rodent repellants - protecting food in treated bags (tributyltin chloride,

triphenyltin chloride and acetate); (h) molluscicides - triphenyltins; (i) ovicides - trialkyl - and triaryltin chlorides; and (j) as insecticides and ovicides in combination with DDT or pyrethrum.

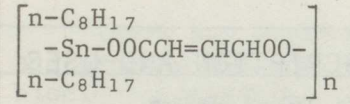
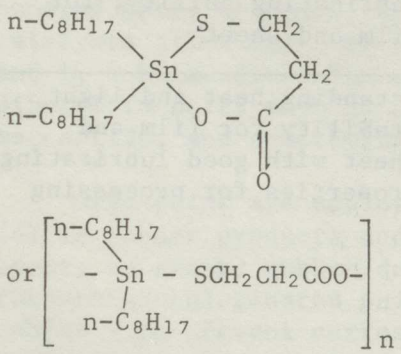
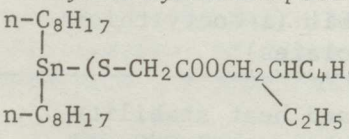
## TABLE 8.

## COMMERCIALLY IMPORTANT BUTYLTIN AND N-OCTYLTIN STABILIZERS (PIVER, 1973)

| NAME AND STRUCTURE | TRADE NAMES | DESCRIPTION AND USES |
|---|---|---|
| Dibutyltin dilaurate<br>  <br> | Thermolite 12<br>Advastab DBTL<br>Niax D-22<br>Clear 1 | Excellent lubricating properties for easy processing; usually combined with other stabilizers; for film and sheet |
| Dibutyltin maleate<br>$[(C_4H_9)_2SnOOCCH=CHCOO]_n$ | Thermolite 13<br>Advastab T-340 | Good stabilizer, poor lubricating ability; for film and sheet |
| Dibutyltin laurate-maleate<br>May be solution of dilaurate and maleate or<br>  <br> | Thermolite 17 | Outstanding heat and light stability for film and sheet with good lubricating properties for processing |
| Dibutyltin bis (lauryl mercaptide)<br>$(C_4H_9)_2$-Sn-$(SC_{12}H_{25})_2$ | Thermolite 20<br>Advastab TM-918<br>Mark A | Good lubricating properties; used with dibutyl tin S,S-bis (isooctylthio-glycolates) |
| Dibutyltin bis(monoalkyl maleate) (alkyl usually is $C_4$ or $C_8$)<br>$(C_4H_9)_2$-Sn-$(OOCCH=CHCOOR)_2$ | Thermolite 25, 26<br>Advastab T-52N, T-150<br>Mark 275 | Light and heat stability to tubing; and PVC and PVC copolymer sheet and film |
| Dibutyltin, S,S-bis (isooctyl thioglycolate)<br>$(C_4H_9)_2$-Sn-$(SCH_2COOC_8H_{17})_2$ | Thermolite 31<br>Advastab TM-180<br>Mark 292<br>Synpron 1001 | Stabilizer for rigid PVC pipe; control of melt viscosity; permanence; good for pigmented rigid applications |
| Dibutyltin β - mercapto-propionate<br>$[(C_4H_9)_2$-$SNSCH_2CH_2COO-]_n$ | Thermolite 35<br>Advastab T-360<br>Mark 488 | Used for bottles, film and sheet |

## TABLE 8. (CONT'D)

## COMMERCIALLY IMPORTANT BUTYLTIN AND N-OCTYLTIN STABILIZERS (PIVER, 1973)

| NAME AND STRUCTURE | TRADE NAMES | DESCRIPTION AND USES |
|---|---|---|
| Di-n-octyltin maleate<br>  | Thermolite 813<br>Mark OTS | Used in bottles, film and sheet for food contact according to CFR, FDA, Title 21, Part 121, Sub-part F, 121.2602 |
| Di-n-octyltin S,S bis(isooctyl thioglycolate)<br>$(n\text{-}C_8H_{17})_2\text{-Sn.}(C_8H_{17}\text{-}OOCCH_2S)_2$ | Thermolite 831<br>Advastab TM-188<br>Mark OTM | Maximum heat stability and processability. FDA sanctioned for bottles; films and sheets for food contact |
| Di-n-octyltin β-mercapto-propionate<br>  | Advastab T-270<br>Mark 492 | |
| Di-n-octyltin S,S - bis(2-ethyl hexyl mercaptoacetate)<br>  | Mark OTM | FDA sanctioned for bottles films and sheets for food contact |

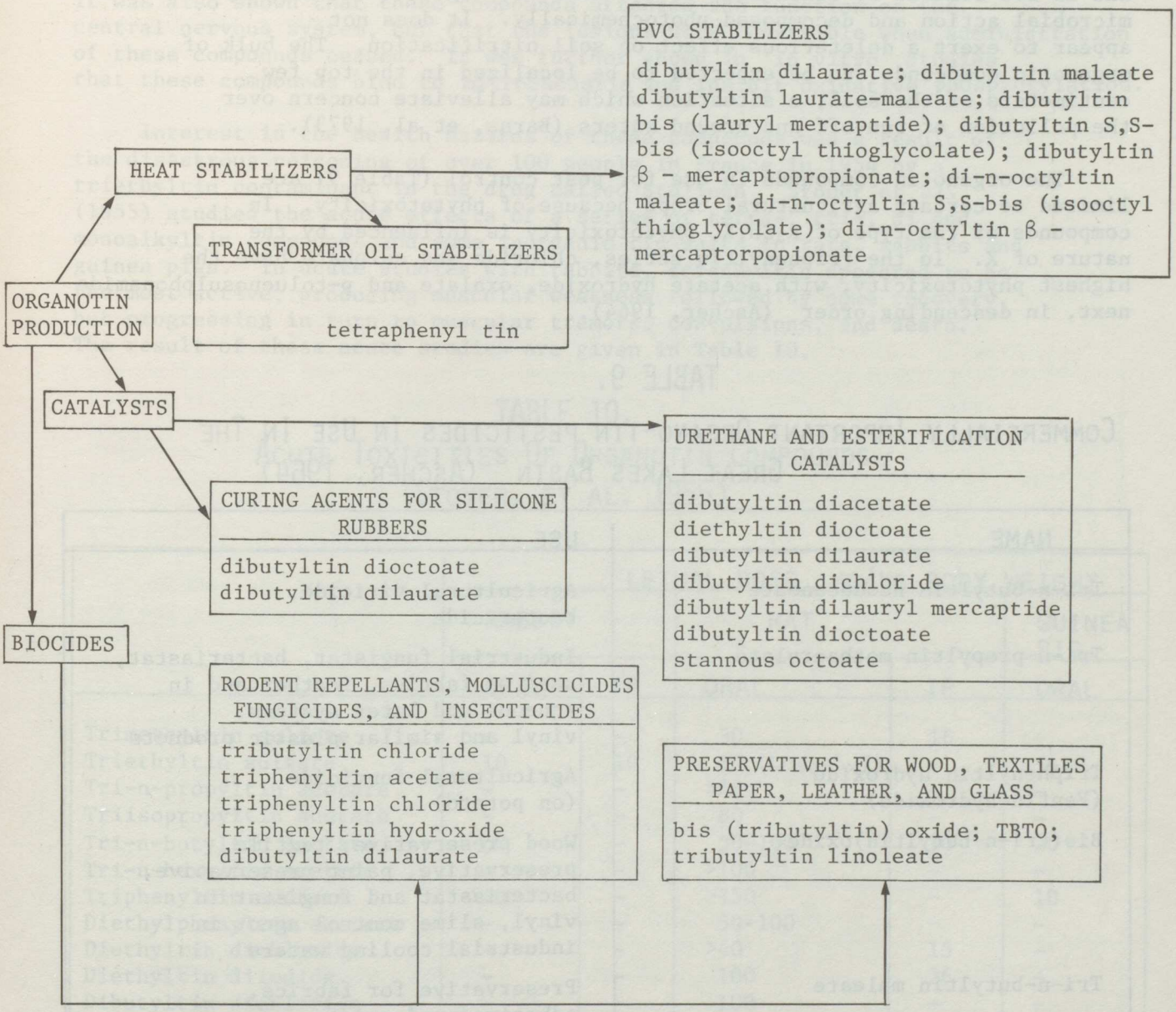



FIGURE 5: ORGANOTIN PRODUCT DISTRIBUTION CHART (PIVER, 1973)

In the presence of water, the acetate salts of organotin compounds hydrolyze completely into the hydroxide, which behave as weak bases. The chloride salts are very stable.

Triphenyltin acetate (Fentin acetate) in soil is negligibly volatile and is not removed by leaching. It is, however, degraded in soils by microbial action and decomposed photochemically. It does not appear to exert a deleterious effect on soil nitrification. The bulk of organotin compounds can be expected to be localized in the top few centimeters of the soil, a situation which may alleviate concern over the pollution of run-off and ground waters (Barns, et al. 1973).

The organotin compounds in use for pest control (Table 9) are limited to certain agricultural crops because of phytotoxicity. In compounds of the type of $R_3Sn.X$, phytotoxicity is influenced by the nature of X. In the triphenyltin series, chloride and sulphate have the highest phytotoxicity, with acetate hydroxide, oxalate and p-toluenosulphonamide next, in descending order (Ascher, 1964).

## TABLE 9.

## Commercially Important Organo-tin pesticides In Use In The Great Lakes Basin (Ascher, 1964)

| NAME | USE |
|---|---|
| Tri-n-butyltin neodecanoate | Agricultural viricide, bactericide |
| Tri-n-propyltin methacrylate | Industrial fungistat, bacteriastat, used on fabrics, leather and in aqueous and latex systems, vinyl and similar plastic products |
| Triphenyltin hydroxide (Fentin hydroxide) | Agricultural fungicide (on potato) |
| Bis(tri-n-butyltin)oxide | Wood preservative, textile preservative, paint preservative, bacteriastat and fungistat in vinyl, slime control agent in industrial cooling waters |
| Tri-n-butyltin maleate | Preservative for fabrics, adhesives and paints |
| Bis(tri-n-propyltin)oxide | Canvas shoe fabric laminate preservative, alkyd paint film fungistat |

Organotins compounds have the potential of being environmental health hazards. Acute and chronic toxicity studies in rats with these compounds showed them to be very toxic with the dialkyl derivatives being less toxic than the trialkyl and triaryl derivatives. The toxicity was shown to diminish as the size and stability of the organic ligand increased. It was also shown that these compounds affected the function of the central nervous system, but that the lesions were reversible when administration of these compounds ceased. It was further shown in 'in vitro' studies that these compounds bind to mitrochondria and inhibit oxidative phosphorylation.

Interest in the health hazards of these compounds was a result of the disastrous poisoning of over 100 people in France in 1954 by a triethyltin contaminant in the drug called Stalinon. Stoner et al. (1955) studied the acute effects of a series of tetra-, tri-, di and monoalkyltin compounds and some inorganic tin salts in rats, rabbits and guinea pigs. In acute studies with rabbits, triethyltin appeared to be the most active, producing muscular weakness followed by some recovery, but progressing in turn to muscular tremors, convulsions, and death. The result of these acute studies are given in Table 10.

## TABLE 10.
## ACUTE TOXICITIES OF ORGANOTIN COMPOUNDS
## (STONER, ET AL. 1955)

| | LETHAL DOSE, MG/KG BODY WEIGHT | | | | |
|---|---|---|---|---|---|
| | RABBIT | | RAT | | GUINEA PIG, |
| | ORAL | IP | ORAL | IP | ORAL |
| Trimethyltin sulfate | - | - | 30 | 16 | - |
| Triethyltin sulfate | 10 | 10 | 10 ($LD_{50}$-5.7) | - | 5-10 |
| Tri-n-propyltin acetate | - | - | >40 | - | - |
| Triisopropyltin acetate | - | - | 80 | - | - |
| Tri-n-butyltin acetate | 60 | - | 50-100 | 10 | 20 |
| Tri-n-hexyltin acetate | - | - | >100 | - | - |
| Triphenyltin acetate | >40 | - | >150 | - | 10 |
| Diethylphenyltin acetate | - | - | 50-100 | - | - |
| Diethyltin dichloride | - | - | >40 | 15 | - |
| Diethyltin diiodide | - | - | 100 | 26 | - |
| Dibutyltin dichloride | - | - | 100 | - | - |
| Dibutyltin dilaurate | - | - | - | 85 | - |
| Monoethyltin trichloride | - | - | - | 200 | - |

Work by Magee et al. (1957) made it clear that triethyltin derivatives exerted a powerful toxic action on the central nervous system of rats. The first reaction in the rat to poisoning was the accumulation of fluid in the white matter of the central nervous system. The accumulation of fluid persisted for as long as the organotin compounds were administered. When feeding of the triethyltin compound was stopped, the lesion was reversible.

Pelikan and Cerny (1968) studied the acute toxicity of tri-n-butyltin derivatives in white mice. The compounds were given per gavage (tube fed) in a single dose of each compound at 500 mg/kg body weight. After eight hours, autopsy findings showed signs of damage to the digestive tract, liver, and spleen. The $LD_{50}$ results of these tests are shown in Table 11.

## TABLE 11.

## $LD_{50}$ Results For Tributyltin Derivatives In White Mice (Pelikan and Cerny, 1968)

| | $LD_{50}$, MG/KG BODY WEIGHT | $LD_{50}$ RANGE, MG/KG |
|---|---|---|
| Tributyltin oleate | 230 | 175-301 |
| Tributyltin laurate | 180 | 136-237 |
| Tributyltin chloride | 117 | 80-170 |
| Tributyltin benzoate | 108 | 74-156 |
| Tributyltin acetate | 46 | 24.8-85.16 |

A large amount of acute toxicity data has been obtained for tri-substituted organotin compounds; the results of these and several other important studies are summarized in Table 12.

## TABLE 12.

## Acute Toxicity Data For Trisubstituted Organotin Compounds (Pelikan and Cerny, 1968)

| COMPOUND | $LD_{50}$, MG/KG | ANIMAL USED | REFERENCE |
|---|---|---|---|
| Triethyltin chloride | 5 (IP) | Female rats | Robinson, 1969 |
| Tributyltin oxide | 7 (IP) | Female rats | Robinson, 1969 |
| Trioctyltin dilaurate | >800 (IP) | Female rats | Robinson, 1969 |
| Tributyltin acetate | 133 (oral) | Male rats | Klimmer, 1969 |
| Tributyltin salicylate | 137 (oral) | Male rats | Klimmer, 1969 |
| Bis(tributyltin)oxide | 122 (oral) | Male rats | Klimmer, 1969 |
| Tri-n-octyltin chloride | >10000 (oral) | Male rats | Klimmer, 1969 |

Suzuki (1971) performed acute and chronic studies with triethyltin sulphate on newborn rats. In the acute studies, 5 mg/kg body weight of the triethyltin sulphate was injected daily intraperitoneally. The rats in this study died after 3 days. The brains of the test animals showed diffuse hemorrhagic encephalopathy. In the chronic studies, 5 mg/l in drinking water was taken each day. The test animals showed no clinical symptoms of cerebral involvement, but severe, diffuse status spongiosus was evident throughout the white matter of the central nervous system.

Pelikan (1969) studied the effects of bis(tri-n-butyltin)oxide, an important preservative for wood, textile paper, leather and glass, on the eyes of rabbits. The actual concentrations of the organotin compound administered were: 6.1 mg/kg; 4.6 mg/kg; 0.61 mg/kg; and 0.46 mg/kg. A single dose of 0.03 ml per rabbit was administered to the conjunctival sac of the left eye. The largest concentration approximates the highest accidentally administered dose. The lower two concentrations produced very profound changes in the rabbits' eyes. Within three hours, erythema and mild edema of the eyelids was observed. In addition, there were numerous large necrotic areas, patechial hemorrhages, early chemosis of the bulbar and polpebral conjunctivae, and distinct pericorneal injection and dullness of the shine of the cornea with decreased transparency. In the rabbits given the larger concentrations, these effects were more pronounced and produced in two of the animals a clinical situation which showed marked deterioration after three to four days. On day 11 and 12 the rabbits died. This commercially important biocidal agent, when administered in low concentration doses, has a very pronounced and irreversible effect on normal eye functions.

Barnes and Magee (1959) studied the toxicity of the salts of dibutyltin hydroxide because these stabilizers were used for plastics which were made into flexible tubing for blood-transfusion sets and the transport of liquids such as beer and milk. In acute studies with rats, a single oral dose of dibutyltin dichloride of 50 mg/kg body weight produced bile-duct lesion.

Chronic toxicity studies on di-n-butyltin dichloride in rats were conducted by Gaunt et al. (1968). The no-effect level in rats was established at 40 ppm in the diet for 90 days. The level corresponded to an intake of approximately 2 mg/kg day. Other important acute toxicity studies with di- and monosubstituted organotin derivatives are summarized in Table 13.

# TABLE 13.

## Acute Toxicity Data For disubstituted Organotin Compounds

| COMPOUND | $LD_{50}$, MG/KG | ANIMAL USED | REFERENCE |
|---|---|---|---|
| Dibutyltin oxide | 520 (oral, oil solution) | Male rats | Klimmer, 1969 |
| Dibutyltin oxide | 39.9 (IP) | Female rats | Robinson, 1969 |
| Dibutyltin dichloride | 35 (oral) | White mice | Gaunt, 1968 |
| Dibutyltin dichloride | 112 (oral) | White mice | Gaunt, 1968 |
| Dibutyltin dichloride | 100 (oral, oil solution) | Male rats | Klimmer, 1969 |
| Dibutyltin di-2-ethyl-hexyl thioglycolate | 510 (oral, oil solution) | Male rats | Klimmer, 1969 |
| Dibutyltin di(monobutyl) maleate | 120 (oral, oil solution) | Male rats | Klimmer, 1969 |
| Dibutyltin di(mononony1) maleate | 170 (oral oil, solution) | Male rats | Klimmer, 1969 |
| Dibutyltin dilaurate | 175 (oral, oil solution) | Male rats | Klimmer, 1969 |
| Di-n-octyltin thioglycolate | 945 (oral) | Male rats | Klimmer, 1969 |
| Di-n-octyltin β mercaptopropionate | 1,850/2,050 (oral) | Male rats | Klimmer, 1969 |
| Di-n-octyltin 1,4-butanediol bismercaptoacetate | 2,950 (oral) | Male rats | Klimmer, 1969 |
| Di-n-octyltin ethyleneglycol dithioglycolate | 880 (oral) | Male rats | Klimmer, 1969 |
| Di-n-octyltin maleate | 1,265 (oral) | Male rats | Klimmer, 1969 |
| Di-n-octyltin di-(1,2 propyleneglycol maleate) | 4,775 (oral)/30 (IP) | Male rats | Klimmer, 1969 |
| Di-n-octyltin di-monobutyl maleate) | 2,030/2,750 (oral) | Male rats | Klimmer, 1969 |
| Di-n-octyltin bis(2-ethylhexylmaleate) | 2,760/3,500 (oral) | Male rats | Klimmer, 1969 |
| Di-n-octyltin dilaurate | 6,450 (oral), 95 (IP) | Male rats | Klimmer, 1969 |
| Di-n-octyltin bis (lauryl thioglycolate) | 3,700 (IP) | Male rats | Klimmer, 1969 |
| Di-n-octyltin oxide | 2,500 (IP) | Male rats | Klimmer, 1969 |
| Di-n-octyltin acetate | >800 (IP) | Female rats | Robinson, 1969 |
| Di-n-octyltin bis(2-ethylhexyl mercaptoacetate) | 2,010 (stomach tube) | White mice | Pelikan, 1970a, 1970b |
| Di-n-octyltin bis (dodecyl mercaptide) | 4,000 (stomach tube) | White mice | Pelikan, 1970a 1970b |
| Di-n-octylin bis (butylmercaptoacetate) | 1,140 (stomach tube) | White mice | Pelikan, 1970a 1970b |
| Mono-n-octyltin tris(2-ethylhexyl mercaptoacetate) | 1,500 (stomach tube) | White mice | Pelikan, 1970a 1970b |
| Mono-n-octyltin trichloride | 10,000 (oral) | Male rats | Klimmer, 1969 |

In general, in the whole animal, pharmacological and toxicological effects of organotin compounds are confined to the central nervous system (Fishbein, 1974) and to the thymus (Seinen and Williams, 1976). For rats, the oral toxicities of trialkyltins are in the order triethyl > trimethyl > triisopropyl > tri-n-butyl (Fishbein, 1974).

The conversion of tetraalkyltins to trialkyltins, in vivo, as demonstrated for tetraethyltin, accounts for the latent toxicity of the tetraalkyltins, with the site of conversion being the liver, (Cremer, 1958). Once tetraethyltin has been converted to triethyltin, it appears to persist in the body in that form without apparent further reaction to the dialkyl derivative. The trialkyltin ion is a stable entity which is toxic per se and persists for some time in the tissues (Cremer, 1958).

Seiffer and Schoof (1967) reported values for 6 hr. $LC_{50}$ and 95% kill is 6 hours for the snail Australorbis glabratus of tri-n-butyltin as 0.29 and 0.57 mg/l and of bis- (tri-n-butyltin) oxide as 0.41 and 0.84 mg/l, respectively.

Boultin, et al. (1971) studied the effects of triethyltin sulfate (TETS) on the metabolism of the barnacle (Elminus modestus) by exposing them to concentrations of this anti-fouling compound which was lethal after 30-36 hours. The changes in metabolism observed suggested that pyruvate utilization was restricted via pyruvate dehydrogenase. Although metabolic phenomena were observed, this compound did not inhibit any of the enzymes tested sufficiently strongly to account for its toxicity. Neither does its action as an uncoupling agent appear to correlate with its antifouling properties since Elminius was shown to be highly resistent to intoxication by 2,4-dinitrophenol. However, Aldridge and Rose (1969) suggested that trialkyltins interact differentially to 2,4-DNP with the mammalian respiratory chain system, so that resistence to 2,4-DNP may not preclude a toxic action to TETS on respiration in Elminius. A further possibility would be that the metabolic effects which imply a restriction of pyruvate utilization by TETS, result from an action on the dehydrolipoyl transacetylase component of pyruvate dehydrogenase.

The Centre for Overseas Pest Research, in 1974, reported that the slow release formulation of tributyltin oxide which is claimed to be molluscicidal without harming fish, when tested in concentrations as little as 0.005 mg of active ingredient per litre can be harmful (though not lethal) to the tropical food fish Sarotherodon mossambicus. The most noticeable effect is lesion of the cornea which may interfere with vision. Exposure to 0.005 mg/l for 24 hours caused the cornea to become completely opaque and the surface to start to peel away. In addition, exposure for several months has been found to reduce the growth rate of fish, and fry of pre-exposed parents are generally smaller than normal. Although not directly lethal, these effects could lead to serious reductions in the productive capacity of natural fish populations.

Alabaster (1969) reported values of 24-hr. and 48-hr. $LC_{50}$ for rainbow trout as, respectively, 0.028 and 0.021 mg/1 of tributyl tin oxide. This indicates that aquatic life is more sensitive to organotin compounds.

It appears that trialkyltin compounds are, in general, more toxic than the di- and mono- alkyltin compounds. Until more information is available on the effects of the individual alkyltin compounds on aquatic life, an objective for these compounds cannot be scientifically supported.

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## ELEMENTAL PHOSPHORUS

*RECOMMENDATION*

No objective for elemental phosphorus can be recommended at this time due to an inadequate data base on the effects of elemental phosphorus on freshwater aquatic life.

*RATIONALE*

Phosphorus normally occurs in the environment in mineral compounds called apatites that form the basis for many soils. They are common in the Great Lakes Basin as typified by the Lake Erie bluffs. In water, phosphorus is usually in the form of phosphate anions, either free, complexed to inorganic cations, or complexed or incorporated into organic biological material. Phosphorus in this form is a nutrient. Elemental phosphorus (known as white or yellow phosphorus) is rarely observed in natural water due to its high reactivity and rapid oxidation to phosphates. However, industrial production of elemental phosphorus has resulted in significant losses to the environment with resulting extensive fish mortality (for a comprehensive review of the Placentia Bay, Newfoundland problem see Jangaard, 1972). In Newfoundland, elemental phosphorus is produced from fluorapatite ($Ca_3(PO_4)_2CaF_2$). In the Great Lakes Basin, phosphate fertilizers are manufactured and elemental phosphorus is used in the process.

Elemental phosphorus solubility in water is about 3 mg/l (Isom, 1960) but it may also exist suspended as a colloid. Most toxicity studies with aquatic organisms involve emulsification to produce a stock solution, followed by dilution to the desired test concentrations. Consequently, the ratio of dissolved to colloidal material is usually unknown. Because of its strong reducing nature, phosphorus should oxidize very rapidly to phosphate. However, observed concentrations of elemental phosphorus in a marine system were quite high and elemental phosphorus persisted in the sediments for months, perhaps due to low oxygen concentrations (Zitko et al., 1970). The behaviour of elemental phosphorus has not been investigated in a natural fresh water system. In a laboratory study at 3 mg/l phosphorus, Isom (1960) did not measure the rate of oxidation but noted a slight decrease in pH after 6 days indicating formation of phosphoric acid. If all phosphorus was oxidized, the pH drop might have been larger. Zitko et al. (1970) reported that oxidation of elemental phosphorus dispersions was kinetically a first order reaction with a half-life of 2-7.5 h. Adsorption on to a solid support such as suspended bottom mud decreased the rate of oxidation.

There are several analytical techniques for elemental phosphorus but the best for water, mud or tissue samples appears to be that of Addison and Ackman (1970). It involves extraction into a suitable solvent, isolation by gas-liquid chromatography and measurement by a specific flame photometric detector. The whole process takes 5-10 minutes /sample and the detection limit is $10^{-12}$g which gives a sensitivity in a concentrated sample of 2 parts/$10^{12}$.

Elemental phosphorus is extremely toxic. The $LD_{50}$ for rats was between 0.5 and 3.5 mg (Fletcher, 1973) and sublethal doses caused fatty changes throughout the hepatic lobules, and hyperplasia and changes in the parallel arrangement of the rough endoplasmic reticulum (Kim, 1971). Vitamin E did not alleviate these symptoms so that the toxic action of phosphorus may not be due to its reactivity.

The phosphorus discharge in Long Harbour, Placentia Bay, Newfoundland was associated with a deposit of sediment near the effluent pipe. In this region there were no benthos. In the region adjacent to the sediment, the only living organism was a nudibranch, the burrowing anemone Edwardsia sp. The numbers and diversity of benthos increased sharply with distance from the discharge but scallops and sand dollars (Echinarachinius parina) showed heavy mortality (Peer, 1972). The incipient lethal level of elemental phosphorus for lobster (Homarus americanus) and beach flea (Gammarus oceanicus) was 40 and 3000-4000 µg/l respectively (Zitko et al., 1970). The apparent cause of death in lobster was coagulation of the hemolymph followed by suffocation. Sublethal exposure caused damage to the antennal gland and hepatopancreas of lobster. Histological observations of the hepatopancreas included disorientation and cell membrane destruction in embryonic and fibrillar cells, an increase in the vacuole size and cell number, especially of secretory and absorptive cells, and an obliteration of the lumen of tubules (Aiken and Byard, 1972).

In salt water, very low concentrations of elemental phosphorus are lethal to fish, with no apparent threshold. A threshold of lethality in continuous-flow exposures was not apparent for Atlantic salmon (Salmo salar) or cod (Gadus morhua). The $LT_{50}$ (medial lethal time) for salmon at 0.79 µg/l was 195 hr., while for cod at 1.89 µg/l, it was 125 hr. (Fletcher and Hoyle, 1972). In static exposures, mortality correlated well with the product of concentration X exposure time with an estimated threshold of 700 µg-h/l for salmon and 400 µg-h/l for cod (Fletcher and Hoyle, 1972). Zitko et al. (1970) were able to establish a threshold of 18 µg/l for salmon but not for herring (Clupea harengus). A concentration of 2.5 µg/l gave an $LT_{50}$ for herring of about 130 hr. The $LT_{50}$'s for seawater adapted brook trout (Salvelinus fontinalis) and smelt (Osmerus mordax) were 121 and 180 hr. respectively at 0.5 µg/l. Again, no thresholds of lethality were apparent.

For all of the above studies, elemental phosphorus was emulsified in water and the proportions of dissolved and colloidal were unknown. Maddock and Taylor (1976) developed a system for eliminating colloidal phosphorus from the test chamber. In this system, the 48 hr. $LC_{50}$ for cod was 14.4 µg/l with a threshold or incipient lethal level of about 11 µg/l, a higher concentration than shown above. This suggests that both colloidal and soluble elemental phosphorus were toxic with the colloidal appearing more toxic.

Freshwater fish are also sensitive to elemental phosphorus with a 163 hr. $LC_{50}$ of bluegill (Lepomis macrochirus) of 25 µg/l total phosphorus (Isom, 1960). In solutions filtered through a 100 µm pore filter, there was no toxicity at the limit of phosphorus solubility (3 mg/l). This suggested to Isom (1960) that only colloidal phosphorus was toxic in contradition to the studies of Maddock and Taylor (1976). There is a possibility that the filtering of a colloidal suspension not only removed colloidal phosphorus but also provided the right conditions for oxidizing soluble phosphorus to non-toxic phosphate.

Phosphorus has a residual toxic effect since lobsters and fish died 24 hours post-exposure (Zitko et al., 1970; Fletcher and Hoyle, 1972). This probably explains why fish died with symptoms of phosphorus poisoning at a great distance from Long Harbour in Placentia Bay. Another cause of delayed mortality may have been the toxic effects of phosphorus in the diet. Fletcher (1973) exposed cod for 1 hour to 5000 µg/l P. He sacrificed these fish, removed the liver and muscle, and then fed the cod flesh to brook trout. The liver contained 194 µg/g of elemental phosphorus and, when fed to trout, caused symptoms of phosphorus poisoning within 2-5 days. The muscle contained 4-11 µg/g but caused the same effect in 21-31 days. The calculated lethal dose was about 1.23-2.73 mg which compares favourably to the lethal dose for rats of 0.5-3.5 mg.

Contaminated mud was also toxic to herring. A suspension of 0.1% contaminated mud from Long Harbour caused death in 28 hr. while a 1% suspension caused death in 7 hr. (Idler, 1969). The mud contained up to 5800 mg/kg elemental phosphorus (Ackman et al, 1971). Herring were observed to spawn in this mud in Long Harbour and shortly after many dead and dying herring were found.

The symptoms of phosphorus poisoning in fish included disintegration of leucocytes, hemolysis of red blood cells, low hematocrits, destruction of gill epithelium, tubular necrosis in kidneys, vacuolation and disruption of the liver cord structure and vacuolation and edema of the spleen (Odense et al, 1972). The hemolysis of red blood cells caused the characteristic "red herrings" due to subcutaneous hemorrhaging . Although this undoubtedly contributed to mortality in herring, salmon and trout, there may be other causes of death for cod and smelt since these species do not exhibit redness. (Fletcher et al., 1970; Fletcher and Hoyle, 1972; Zitko et al., 1970).

There is considerable concentration of phosphorus over water levels by various marine organisms. In cod, concentration of phosphorus by white muscle was 10-30 X the exposure level (Maddock and Taylor, 1976; Dyer et al, 1970), 50-100 X by red muscle, and 1000 X by liver (Dyer et al., 1970). In addition, the concentration in fish varied directly with the concentration in water with cod tissues accumulating phosphorus in the order: Liver > pyloric caeca > spleen > gonad > esophagus > muscle > intestine > skin, gills (Fletcher, 1974). In salmon, the order was: pyloric caeca > liver > esophagus > muscle > intestine > gills > skin > kidney. The biological half-time for various tissues of fish transferred to clean water was 4-6 hr. for cod and 1-1.5 hr. for salmon (Fletcher, 1974). While these depuration rates appear rapid, fish exposed once to a lethal concentration will still continue to die in clean water; i.e. the toxic action is irreversible (Zitko et al., 1970). Concentration factors for whole invertebrates and seaweed were: Mussels (Mytilus edulis): 10 X; quahog (Arctica islandica): 18 X; Fucus vesiculosus: 22 X; clam (Mya arenaria): 23 X; Fucus distichus: 23 X; starfish (Asterias vulgaris): 27 X; and periwinkle (Littorina littorea): 43 X (Fletcher, 1971). On an organ basis, the pyloric caecae of starfish accumulated 100 X the exposure level while the ovary and hepatopancreas of lobster accumulated 300 and 1000 X respectively. On a commercial basis, elemental phosphorus could be hazardous to consumers of exposed fish since normal processing involving iced storage, freezing and thawing, frozen storage, or cooking reduced elemental phosphorus by 60% at most. Salting was slightly more effective and reduced levels by up to 75%. However, some fish showed no reduction at all (Dyer et al., 1972).

In view of the extreme toxicity of elemental phosphorus, the absence of a lower lethal threshold for some species and its persistence in water, mud and biota, an objective of 0.5 µg/l of phosphorus in esa water would be appropriate. However, since the response of freshwater species has not been tested adequately, and since the relationship of salt water toxicity to freshwater toxicity is unknown, an objective for freshwater is not recommended.

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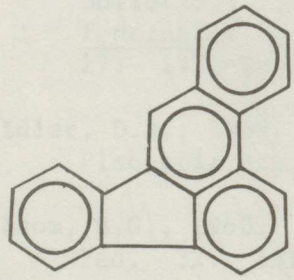
## POLYNUCLEAR AROMATIC HYDROCARBONS

*RECOMMENDATION*

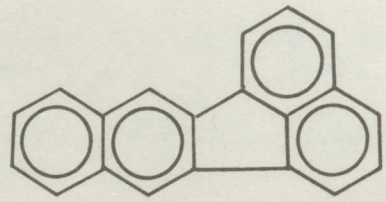
Inadequate information exists on natural environmental levels and on effects of these substances on aquatic life. Consequently, no objective is recommended at this time.

*RATIONALE*

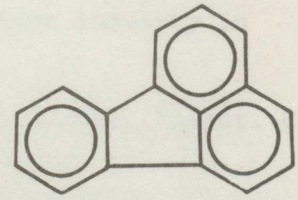
Polynuclear (or polycyclic) aromatic hydrocarbons, PAH's, are also referred to as fused ring or condensed nuclear compounds. They are loosely defined as aromatic compounds with two or more carbon atoms per ring shared between two or more rings. Such a definition does not exclude the nitrogen heterocyclic PAH's but, due to a lack of data on their environmental significance, the comments in this rationale largely pertain to the carbon and hydrogen PAH's. Examples of this class of compounds, those recommended by the World Health Organization (1971) for monitoring, are:



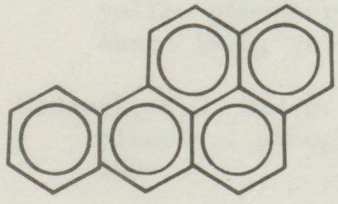
benzo(b)fluoranthene



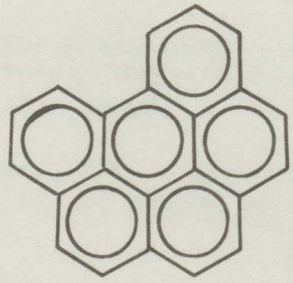
benzo(k)fluoranthene



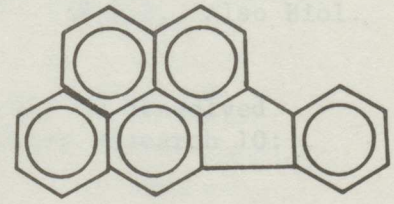
fluoranthene



benzo(a)pyrene



benzo(g,h,i)perylene



Indeno(1,2,3-cd)pyrene

Such compounds, and their various alkylated derivatives which should also be included in environmental considerations of PAH's (Badger, 1948; Sawicki et al., 1966; Youngblood and Blumer, 1976), are ubiquitous in the environment (Gunther and Buzzeti, 1965). They are also present in fossil fuels (McKay and Latham, 1973) and can be derived from the incomplete combustion of any plant or plant-derived material. Auto exhausts and all forms of combustion engines (N.A.S., 1972) are major sources of PAH's in the atmosphere. Additionally, for some of the human population, cigarette smoke is probably an even greater exposure than the urban air which has been frequently examined.

PAH's are susceptible to oxidation, a fact which has been observed photochemically in the atmosphere (N.A.S., 1972; Fatiadi, 1967; Pierce and Katz, 1976) in the aquatic environment (Andelman and Suess, 1970) and in mammals metabolically (Borgen et al., 1973; Maugh, 1974). Quinones and epoxides are the products which themselves are mutagenic (Huberman et al., 1971) and probably may be carcinogenic (Epstein et al., 1968; Ames, et al., 1972). PAH's are also readily chlorinated in water treatment processes (Harrison et al., 1976a) forming stable, chlorinated aromatic compounds (Reichert, 1968 a and b) which might be expected to have a similar environmental behaviour and effects to those of the polychlorinated biphenyls. All of these reactions will be strongly influenced by the structures of the particular PAH's but since, at this time, information is not available on either their occurrence or their chronic effects, they are not considered further in the context of water quality.

The solubility of PAH's in water, as reported for several of the series (Davis et al., 1942; Klevens, 1950), is in the range 0.5 - 12,500 µg/l. Compounds with four or more rings are at the bottom of this range and consequently, partitioning of such, favours their adsorption on particulate matter. It is likely that this is the manner in which PAH's occur in the aquatic environment. From a long list of PAH's that were observed at 0 - 20 µg/g in the surface sediments of Lakes Ontario and Erie (Strosher and Hodgson, 1973), only two were found in 0.45 µ-filtered water at levels barely above the detection limit of 0.004 µg/l. Because of such adsorption, PAH's can be substantially removed in water treatment processes (Borneff, 1969; Reichert et al., 1971) although not completely so, presumably due to their association with fine suspended material (Harrison et al., 1976b).

Analytical methods are available for PAH determination in water, in air particulates, as well as in sediments and biological tissue. Schaad (1970), Grimmer and Hildebrandt (1972), Fishbein (1973a), Giger and Blumer (1974) and Zitko (1975) in particular have detailed reviews of the various procedures. Generally, ultraviolet or fluorescent detection techniques after liquid chromatography have been employed and presently gas chromatography, particularly coupled with mass spectrometry, is becoming more common in order to provide unequivocal identifications.

Environmental levels of PAH's have long been reported for a wide variety of components of the environment. Blumer (1973) has reported levels as high as 1300 µg/kg of a single carcinogenic PAH, benzo(a)pyrene, in remote forest soils of America while Graef (1965) and Borneff and Kunte (1963) obtained similar results from Europe. These compounds have also been reported as constituents of terrestrial plants (Hancock et al., 1970; Graef and Diehl, 1966), marine zooplankton (Lee, et al., 1976; Niaussat and Auger, 1970), sediments (Aizenshat, 1973; Strosher and Hodgson, 1973; Niaussat and Auger, 1970) and groundwater (Borneff and Kunte, 1964). Much of these data have been reviewed recently by Harrison et al., (1975), Andelman and Suess (1970), Fishbein (1973b) and Suess (1976) and a summary of some relevant material is given in Table 14.

## TABLE 14.
## Environmental Levels Of Total*
## Polynuclear Aromatic Hydrocarbons

| SUBSTRATE | | |
|---|---|---|
| Forest soil | 36-180 µg/kg (T) | Borneff & Kunte (1963) |
| Plant leaves | ~200 µg/kg (5) | Hancock et al. (1970) |
| Urban air (U.S.) | 0.006-0.2 µg/m$^3$(T) | Fishbein (1973b) |
| Urban air (U.K.) | 1-37 µg/m$^3$ (4) | Commins (1958) |
| Urban air (Budapest) | 0.04-2.1 µg/m$^3$ (11) | Kertesz-Saringer & Morlin (1973) |
| Cigarettes | ~2 µg/cigarette (150) | Lee et al. (1976) |
| Groundwater | 0.001-0.08 µg/1 (T) | Borneff & Kunte (1964) |
| Rain | 0.2-4. µg/1 (T) | Hellman (1974) |
| Unpolluted lakes | 0.01-0.025 µg/1 (T) | Suess (1976) |
| Sediments (Great Lakes) | 0.02-54 µg/kg (T) | Strosher & Hodgson (1973) |
| Polluted river | 0.1-2.1 µg/1 (T) | Borneff & Kunte (1965) |
| Marine plankton | 6-60 µg/kg (T) | Mallet et al. (1963) |
| Freshwater algae | 10-50 µg/kg (T) | Suess (1976) |
| Sewage | 1-20 µg/1 (T) | Hellman (1974) |
| Sewage sludge | 20,000-40,000 µg/kg (T) | Hellman (1974) |

* total implies here some 4 or more compounds, numbers indicated in parentheses or T = "total".

The case for the natural synthesis of PAH's is apparently well established. Bacterially, E. coli (Knorr and Schenk, 1968; Mallet and Tissier, 1969) and other microorganisms (Niaussat and Auger, 1970) are capable of producing such compounds. In the plant world, the evidence is strong that PAH's are readily synthesized and even serve to stimulate growth (Graef and Nowak, 1966). Algae have been shown (Borneff et al., 1968) to be capable of forming PAH's. It would seem that animals are not capable of such synthesis (Boger, 1968) but generally can detoxify (Bogen et al, 1973; Maugh, 1974), or excrete them (Lee, 1975). Such background levels, though, are thought to be minor (Suess, 1976) as far as human exposure is concerned when compared with exposure from the urban atmosphere which is largely derived from the burning of fuel. It is also debatable whether such sources are the primary environmental sources (Youngblood and Blumer, 1976).

The persistence of PAH's in the aquatic environment has been examined somewhat. A study by Malaney et al. (1967) showed 2 - 20% microbial degradation of a variety of PAH's in 144 hours with activated sludge, corresponding to a mean half-life of approximately 12 weeks. Compounds with fewer rings have been found, in this study, to degrade more rapidly than their analogues with a higher number of rings.

PAH's can be metabolized in biological systems, generally through the processes of ring hydroxylation and subsequent cleavage (Gibson, 1971). Lee, in a variety of papers between 1972 and 1975, identified hydroxy PAH metabolites in marine organisms ranging from zooplankton (Lee, 1975) through unspecified microorganisms (Lee and Ryan, 1976) to mudsucker, sculpin and sand dabs (Lee et al., 1972). Others have also observed metabolic degradation (Gibson, 1971) in particular in rainbow trout (Pedersen et al., 1974) in which the liver was observed to be the most active degradative site.

Other than concentration of PAH's in shellfish (Cahnmann and Kuratsune, 1957; Ehrhardt, 1972; Zitko, 1975) and mussels (Koe and Zechmeister, 1952; Dunn and Stich, 1975) and fish, plus the carcinogenicity to mammals (I.A.R.C., 1973), few data have been reported on effects of these compounds. That these compounds are carcinogens has been established for many years, but the significance of environmental levels, with respect to human health (I.A.R.C., 1973) on the aquatic environment, is unknown. There are indications that some PAH's are co-carcinogens with other environmental materials (Wynder and Hoffmann, 1967; Bingham and Falk, 1969).

There are no raw or finished water standards for PAH's in any of the Great Lakes States, provinces, or federal jurisdictions. The World Health Organization, however, includes such compounds in the International Standards for Drinking Water (WHO, 1971) where they recommend a maximum allowable limit of 0.2 µg/l for the six PAH compounds cited earlier in this rationale. Of these six, three [benzo(b)fluoranthene, benzo(a)pyrene, and indeno(1,2,3-cd) pyrene] are documented carcinogens. It is apparent that this restriction is intended to limit PAH's in general from a health viewpoint only since levels of 0.1 µg/l have been identified by WHO as being associated with "heavily polluted waters". This, together with the unlikely fact that there is any safe level for carcinogenic substances, precludes the possibility of recommending any level as an objective. Since such substances are already naturally present to one degree or another in the aquatic environment, the general objective of "non-degradation" will have to be employed to limit concentrations of PAH's. What is required, however, is that a survey of the levels of these substances be undertaken for the waters covered by the Canada-U.S. Water Quality Agreement. It is also necessary, for interpreting transportation and behaviour, that the sediments, seston and fish of these waters be simultaneously examined for PAH burden.

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## SUBSTANCES AND MATERIAL UNDER ACTIVE REVIEW

The Subcommittee and Task Force have various activities underway which are considering new and revised objectives for the following parameters:

NUTRIENTS - nitrogen, phosphorus and silicon - To develop objectives for these nutrients, the Task Force on the Scientific Basis for Water Quality Criteria has a Work Group consisting of outside experts.

BACTERIA - Efforts are underway to form a Work Group under the Task Force on the Scientific Basis for Water Quality Criteria to review the current total coliform and fecal coliform levels specified in the Agreement and to consider other alternatives for objectives for bacteria in the Great Lakes system.

DETERGENTS - The Research Advisory Board currently has Task Forces which are reviewing the health implications of NTA usage and the ecological effects of non-phosphate detergent builders. Following a review of the Task Forces' reports which are expected in May 1977, the Task Force on Scientific Basis for Water Quality Criteria and the Water Quality Objectives Subcommittee will consider objectives for detergents and components of detergent formulations.

ATRAZINE AND MALATHION - With the recent receipt of new knowledge pertaining to the toxicity of atrazine and malathion to aquatic organisms, the Water Quality Objectives Subcommittee and the Task Force on Scientific Basis for Water Quality Criteria are preparing to review the information and if possible to propose objectives.

# 2 ENVIRONMENTAL MAPPING WORKSHOP

In last year's report it was pointed out that a mechanism is needed to limit the biological value loss and other beneficial use losses associated with mixing zones and other areas of non-compliance with the objectives. In this report it is concluded that the first requirement for the development, acceptance, and implementation of such a mechanism is development of maps of the environmental properties and cultural stresses of the water bodies involved. Thus, in May 1976, the Scientific Basis for Water Quality Criteria Task Force proposed to the Research Advisory Board that it sponsor a workshop on environmental mapping of the Great Lakes. This workshop was approved and a Steering Committee of six members was set up to direct and coordinate the effort.

The workshop was divided into three sessions: (1) concepts and applications of maps, (2) previous mapping efforts, and (3) availability of information. It was held November 8 - 10, 1976, in Windsor, Ontario. Thirty-four presentations were given and approximately 70 persons were in attendance. A report of the proceedings of this workshop is in preparation and will be published in 1977.

There was general but not complete agreement during the workshop that environmental mapping holds great promise in aiding the IJC in restoring and enhancing the water quality of the Great Lakes and specifically, in assisting in establishing where water quality objectives would apply, as required by the Water Quality Agreement. It was also agreed that such mapping is, in general, a very promising technique to aid in developing and agreeing on sound resource management decisions, in informing and involving the public concerning the conservation and protection of waters, and in advancing our scientific knowledge of status for those waters.

Recommendations based on the deliberations of the meeting will be forwarded to the Research Advisory Board in May 1977.

# 3 FUTURE DIRECTION

The Subcommittee and Task Force will continue to review and research available scientific information on substances known to be harmful or with the potential to be harmful to living organisms and will recommend new objectives or revisions to accepted objectives as warranted from the available and from new knowledge. The Subcommittee and Task Force will also research, but will not necessarily develop guidelines for the following:

MIXING ZONES - represent a loss of biological value to an ecosystem. There is a limit to this encroachment on ecosystems before an imbalance and possible collapse of aquatic communities occur. The Subcommittee and Task Force will present a proposal for reducing biological losses imposed by mixing zones on water bodies by the use of environmental maps.

PROCEDURES FOR EVALUATING COMPLEX EFFLUENTS - It is apparent that insufficient data exist to allow establishment of water quality objectives for the large number of organic and inorganic compounds discharged to the Great Lakes as compounds of complex effluents. A mechanism to establish receiving water objectives to protect aquatic life from the effects of such unregulated substances will be considered. The presently used methods for evaluating the toxicity of complex effluents are not well defined. The Subcommittee and Task Force will develop a set of procedures for evaluating these effluents to assure greater protection for the organisms using Great Lakes waters.

# 4 RESEARCH NEEDS

During the course of their deliberations the Subcommittee and the Task Force identified a number of areas where inadequate knowledge was available for development of objectives.

During the year's deliberations, a list was maintained of these areas of inadequate information. For example, in this report, inadequate data are indicated for acute and chronic toxicity to aquatic freshwater biota for aluminum, antimony, barium, boron, cobalt, molybdenum, vanadium, organotins, mirex, elemental phosphorus, phthalate esters other than dibutyl phthalate and di-(2-ethylhexyl) phthalate and polynuclear aromatic hydrocarbons.

On the basis of inadequacies noted in this and the previous reports of the Subcommittee and Task Force, the following few research needs were considered of higher priority:

- Strong efforts are urgently needed to address the problems of toxicity of mixtures of hazardous organic and inorganic chemicals, with emphasis on identifying the chemical properties which determine additive, synergistic, antagonistic and independent biological activity.

- Efforts should be made to acquire data on acute and chronic toxicity to aquatic freshwater biota of organotins, mirex, and phthalate esters other dibutyl phthalate and di-(2-ethylhexyl) phthalate.

- The levels of polynuclear aromatic hydrocarbons in the waters, sediments and biota of the Great Lakes should be assessed.

- Water quality objectives for metals currently refer to total concentrations of each metal in an unfiltered (whole water), digested sample. It may be better to base these objectives only on the toxic and potentially toxic forms of the metals. This can be done only if:

  - the kinetics of transformations (equilibria) of metal forms in Great Lakes waters are defined,

  - the relationship between metal forms and their toxicity is established,

  - reliable methods are developed for monitoring such metal forms.

- More sensitive analytical methods are required for persistent organic compounds in water and biota.

- The fate of toxic substances (or their partitioning) in the water environment must be determined, with particular attention to transformations, ultimate sink(s) and methods of disposal.

- Efforts to determine the effect of changing ionic composition of waters on the composition and abundance of the biota.

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