1976-06-01

Great Lakes Water Quality Fourth Annual Report: Appendix A: Annual Report of the Water Quality Objectives Subcommittee to the Implementation Committee, Great Lakes Water Quality Board

Great Lakes Water Quality Board. Water Quality Objectives Subcommittee

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GREAT LAKES
WATER QUALITY BOARD

INTERNATIONAL JOINT COMMISSION

GREAT LAKES WATER QUALITY 1975
APPENDIX A
WATER QUALITY OBJECTIVES SUBCOMMITTEE REPORT

GREAT LAKES WATER QUALITY

FOURTH ANNUAL REPORT

APPENDIX A

ANNUAL REPORT OF THE WATER QUALITY OBJECTIVES SUBCOMMITTEE

TO THE
IMPLEMENTATION COMMITTEE
GREAT LAKES
WATER QUALITY BOARD
1976

GREAT LAKES WATER QUALITY

FOURTH ANNUAL REPORT

APPENDIX A

ANNUAL REPORT OF THE
WATER QUALITY OBJECTIVES
SUBCOMMITTEE

TO THE
IMPLEMENTATION COMMITTEE
GREAT LAKES
WATER QUALITY BOARD
1976

PREFACE

This is the second Annual Report of the Water Quality Objectives Subcommittee to be presented to the Implementation Committee and to the Great Lakes Water Quality Board since the signing of the Great Lakes Water Quality Agreement between the United States of America and Canada in 1972.

The report is a joint effort of the members of the Water Quality Objectives Subcommittee and the Research Advisory Board's Standing Committee on the Scientific Basis for Water Quality Criteria. It contains proposals for the revision of existing, and for new water quality objectives and supports these proposals with the latest information and data available.

The report is published as Appendix A to the Fourth Annual Report of the Great Lakes Water Quality Board of the International Joint Commission. This, however, does not imply that all of the proposals presented in this report will be approved and recommended in the Board's 1975 Annual Report to the Commission.

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SUMMARY

The specific water quality objectives recommended in this report are based on the best available scientific information on cause/effect relationships between pollutants and water use. Because they are set within the limits of presently available knowledge and data, provision has been made for revision as new knowledge becomes available.

In developing a specific water quality objective, the philosophy of protecting the most sensitive use was employed and, in addition, it was a condition of design that the objectives are to be met at the periphery of mixing zones.

The objectives presented in this report are:

Aluminum, total	No recommendation
Arsenic, total	50 µg/l
Cadmium, total	0.2 µg/l
Chromium, total	50 µg/l
Copper, total	5 µg/l
Cyanide, HCN	5 µg/l
Diazinon, total	0.08 µg/l
Fluoride, total	1.2 mg/l
Guthion, total	0.005 µg/l
Iron, total	300 µg/l
Lead, total	10 µg/l, Lake Superior
	20 µg/l, Lake Huron
	25 µg/l, Remaining Lakes
Mercury, filtered	0.2 µg/l and 0.5 µg/g in whole fish, (wet weight)
Nickel, total	25 µg/l
Parathion, total	0.008 µg/l
Selenium, total	10 µg/l
Total Dissolved Solids	No change from the Agreement.
Vanadium, total	No recommendation
Zinc, total	30 µg/l
Temperature	

INTRODUCTION

The Water Quality Objectives Subcommittee presents the results of its work in the year 1975-76 in this, the second annual report to the Water Quality Board. It has persevered in its charge to develop and recommend specific water quality objectives for the waters of the Great Lakes to ensure against the loss of beneficial uses. This concept was agreed to by the parties signatory to the 1972 Great Lakes Water Quality Agreement. This year's report follows the philosophical approach developed in the first annual report for the establishment and use of water quality objectives.

The report deals comprehensively with a number of metals and proposes an objective for each metal which, in the scientific judgment of the members of the Subcommittee, will adequately protect the most sensitive defined use of these Great Lakes waters. In addition to the metals, specific objectives are proposed for cyanide, fluoride, guthion, diazinon, parathion, and temperature.

The scientific information used by the Subcommittee in developing each of the specific objectives recommended is included as a rationale section with the objective.

Further, a status report is presented on the concept to limit effects on the biota from point source inputs to the lakes by the allocation of biological value loss in mixing zones.

1 RECOMMENDED REVISIONS AND NEW SPECIFIC WATER QUALITY OBJECTIVES

CHEMICAL CHARACTERISTICS

PERSISTENT TOXIC SUBSTANCES - INORGANIC

METALS

INTRODUCTION

It can be generally stated that all natural elements, (metals, metalloids and non-metallic elements) are present in all natural waters, in sediments, and in most living matter. For the majority of these elements, their occurrence is in minute concentrations, much below analytical detection limits of sophisticated monitoring (less than µg/l concentrations). Obviously, for most of these elements, no environmental concern is valid unless they are discharged at concentrations greater than present in the receiving waters.

Metals, such as sodium, potassium, calcium and magnesium are found in mg/l concentrations in most waters. They are essential to all forms of life as basic components of skeletal systems and for many biological processes in general. Ions of such metals are not lethal by themselves unless at very high concentrations (> 1,000 mg/l). On the other hand, metals, in particular aluminum, cadmium, copper, iron, mercury, nickel, silver and zinc, as well as the metalloids selenium and arsenic, are common to all natural aquatic systems in the µg/l range. In trace quantities, some of these elements are essential for certain biological processes while others have no known functions. Whether essential or not, these elements are lethal to biota at high concentrations (< 100 mg/l) and, in a few cases, their natural background levels are approaching toxic concentrations. To protect the aquatic ecosystem, these elements should not be allowed to increase in their concentrations, through man's activities.

Sources for all elements in surface waters are the result of weathering of rocks and soils (Table 1), industrial and municipal effluents and precipitation of airborne matter. In fact, it has been calculated that some large lakes with comparatively little human activity in the drainage basin may derive the major part of their metals from precipitation. Lake sediments, especially in shallow lakes, may also be an important source of trace metals. Such a recycling has been observed for mercury and is well known for the element phosphorus.

Chemistry

Dissolved Metals

In distilled water, dissolved metals largely exist in "free" ionic form, that is, as very weakly complexed hydroxy or aquo complexes. Because of

Table 1. Average concentrations of metals in rocks in mg/kg (Bowen, 1966)

METAL	IGNEOUS ROCK	SHALES	SANDSTONES	LIMESTONES	SOILS	COAL
Al	82,300	80,000	25,000	4,200	71,000	---
As	1.8	13	1	1	6	25
Cd	.2	.3	.05	.035	.06	.25
Cr	100	90	35	11	100	60
Cu	55	45	5	4	20	300
Fe	56,300	47,200	9,800	3,800	38,000	---
Hg	.08	.4	.03	.04	.03	---
Pb	13	20	7	9	10	5
Ni	75	68	2	20	40	35
Se	.05	.6	.05	.08	.2	$\leq$ 7
Ag	.07	.07	.05	.05	.1	.1
Zn	70	95	16	20	50	40

their low complex formation constants, these elements are readily available for any chemical reaction and for biological uptake. Consequently, any uptake by organisms of such ionic metals from water will be rapid and proportionate to their concentration.

However, natural waters always contain a significant amount of dissolved organic material including humic acids, lignin derivatives, fatty acids, amino acids, many other compounds from plant and animal origin, as well as increasing amounts of synthetic chemicals. Most of these compounds have one or more functional groups, such as hydroxy-, carboxy, sulfo- and amino- groups, which may combine with "free" metal ions to form metal-ligand complexes. Depending on the detailed structures of such ligands and the chemical characteristics of the metal ions, complexation can completely mask the availability of the metal ions for common reactions. Of course, any two ligands will act differently on a given set of metal ions and, as a result, the biological effects of a mixture of metal ions and organic compounds is extremely difficult to predict. If, as in the case of certain synthetic chemicals, the complex formation is very strong and no other physical, chemical or microbial degradation of the complex took place, quite high concentrations of toxic metal ions could be present without immediate harmful effects to aquatic life.

Organo-metallic Compounds

Chemical compounds of organo-metallic nature, that is with direct carbon-metal bonds, have been known to chemists for a long time. Recently it has been found that certain metals (for example mercury,) can be methylated by microbial action in sediments and these compounds can enter the aquatic food chain. Because of their partially organic nature, such compounds are likely to be associated with fatty tissues, where they may be stored and accumulated. At the same time, these compounds may produce strong toxic effects on the accumulator organism.

There is still comparatively little understanding of the biological and environmental behaviour and effects of organo-metallic compounds. Studies to determine which elements can be methylated or transformed to organo-metallic forms in aquatic ecosystems are presently under way. So far, in addition to mercury and arsenic, the elements lead, tin, cadmium, and selenium may be able to undergo such reactions.

Particulate and Colloid Metals

Trace metals may also be found in water in forms such as hydroxides, oxides, silicates, phosphates or carbonates which are commonly part of the particulate matter from either biological or mineral origin. Metals which become adsorbed or chemically bound by particulate organic matter are sedimented with the organic matter thereby providing a major route for their removal from aquatic systems.

Additionally, trace metals may be found as hydroxides and their dehydrated forms in very finely dispersed particulate matter of a few hundred to a few thousand molecular units. These aggregates or colloids are usually formed by precipitation of dissolved metals as a result of pH

changes, oxidation or through biological action. Processes of that nature occur primarily in effluents entering water of different quality. Because of the very small size of colloids and their inherently large surface area and high chemical and biological activity, they may be toxic to biota to a much higher degree than large size particulate matter of a similar chemical composition.

Analysis and Great Lakes Concentrations

Present analytical methods for the quantitative determination of metals in water, sediments and biota include the following: atomic absorption spectrometry, neutron activation analysis, polarography, anodic stripping, voltammetry, specific ion electrodes, titration with specific reagents and spectrophotometry.

As previously discussed, metals are found in dissolved, complexed and particulate forms in water. Consequently, analyses are performed for dissolved, suspended, extractable and total metals. At present, most analyses are for total metal, which may include dissolved and adsorbed or suspended metals irrespective of their oxidation state or form of complexation.

Many metals occur in natural waters at concentrations below direct routine analytical detectability. In such cases concentration procedures, usually by solvent extraction, have to be applied in order to obtain reliable quantitative results at these low levels.

Water samples for metal analyses are generally preserved by adding acid to pH 2 or less. At this pH all metals become available for solvent extraction except those very strongly bound by ligands.

Difficulty was experienced in obtaining accurate data on concentrations of metals in Great Lakes waters. Unpublished raw data from monitoring often contained incorrect values due to sample contamination at some stage between collection and analysis. Since analyses are generally quite accurate, the problem is one of sample collection and storage. Consequently, metal concentrations in offshore Great Lakes waters in Table 2 include only summary statistics derived from well-screened raw data on specific metals from the upper lakes. Similar statistics are not available for Lakes Michigan, St. Clair, Erie and Ontario. Other data on concentrations of metals in waters is from published sources and the accuracy of the data has not been assessed.

Biological Effects and Monitoring Problems

Sprague (1970) in his review of the utility of bioassay results indicated that for fish at different times and places, "precipitated" zinc was less toxic, equally as toxic or more toxic than "ionic" zinc. This ambiguity was probably the result of the inability of various authors to measure the various forms

Table 2.

Concentrations (µg/l) of metals in filtered water samples from the epilimnion of the Upper Great Lakes. These statistics represent values from many stations within a lake sampled several times within a year. The statistics are derived from an unpublished draft of the 1975 Report of the Upper Lakes Reference Group, IJC. Data on Lake Huron, Georgian Bay and the North Channel are from Vol. II, Chapter 5.3 and the data for Lake Superior, are from Vol. III, Chapter 5.3

	LAKE SUPERIOR, - 1973				NORTH CHANNEL, LAKE HURON - 1974			
	Detection Limit (D.L.)	Percent of Samples below D.L.	Modal Conc'n.	95 percentile Concentration	Detection Limit	Percent of Samples below D.L.	Modal Conc'n.	95 percentile Concentration
Cadmium	0.2	72	⩽0.2	0.6	0.2	100	⩽0.2	0.2
Chromium	0.2	63	⩽0.2	0.4	0.2	95	⩽0.2	0.2
Copper	0.5	5	2.0 - 2.5	5.0	0.5	5	1.0	4.0
Iron	0.5	3	1.0 - 1.5	7.0	0.5	3	1.5 - 2.5	4.5
Lead	1.0	63	⩽1.0	3.0	1.0	98	⩽1.0	1.0
Mercury	0.05	7	0.1 - 0.15	0.25	-	-	-	-
Nickel	1.0	46	⩽1.0	5.0	1.0	10	2.0 - 5.0	6.0
Zinc	1.0	72	7 - 10	40	1.0	2	3.0	6.0

	GEORGIAN BAY, LAKE HURON - 1974				LAKE HURON, - 1971			
	Detection Limit (D.L.)	Percent of Samples Below D.L.	Modal Conc'n.	95 percentile Concentration	Detection Limit	Percent of Samples Below D.L.	Modal Conc'n.	95 percentile Concentration
Cadmium	0.2	96	⩽0.2	0.2	0.2	98	⩽0.2	0.2
Chromium	0.2	94	⩽0.2	0.4	0.1	70	⩽0.1	0.6
Copper	0.5	25	1.0	4.5	0.25	28	⩽0.25	*
Iron	0.5	5	1.5	3.5	0.25	12	1.0	2.0
Lead	1.0	90	⩽1.0	1.0 - 2.0	0.5	38	⩽0.5	1.5
Mercury	-	-	-	-	-	-	-	-
Nickel	1.0	10	2.0	5.0	0.5	87	⩽0.5	5.0
Zinc	1.0	20	2.0	9.0	0.5	54	⩽0.5	*

*Could not be determined from the data available.

of zinc. On the other hand, toxicity of copper has been related, with reasonable success, to measurement of specific forms.

As a working method for some metals, fairly good correlations with biological availability and hence toxicity, have been obtained by assuming that soluble toxic forms pass a 0.45 μ filter while insoluble non-toxic forms do not. It is recognized however, that the actual separation of these forms is not that simple. Forms which were retained by the filter could be a reservoir of potentially toxic forms which may readily redissolve under changing conditions. Pulse polarography has been used to measure "labile" and "non-labile" forms of copper, but lability has not been directly related to toxicity to algae (Gächter et al., 1973). Specific ion electrodes were used to measure ion activity of copper (Zitko et al., 1973). While the measured ion activity was roughly related to copper toxicity to salmon, an ion activity below 200 μg/l could only be determined by extrapolation. Shaw and Brown (1974) also correlated copper toxicity to trout with ion activity as well as with estimated concentrations of carbonate-complexed and NTA-complexed copper. They concluded that toxicity was best characterized by the total of copper (II) (≃ ion activity) and copper carbonate and not by a single form alone.

The standard chemical procedure of acidifying samples to pH 2 solubilizes many loosely-bound forms of copper (= "acid extractable"). While this may be undesirable when carrying out toxicity tests, it is an essential procedure for assessing loadings and for assessing the potential harm of toxic forms and reservoirs of copper, as well as temporarily inactive forms of copper.

Removal of phyto- and zooplankton from a sample is probably unnecessary because their metal concentrations are low and their contribution to total metal concentrations in water samples is minor. For example, copper concentrations in Lake Michigan phyto- and zooplankton were 6 and 5 mg/kg wet weight respectively (Copeland and Ayers, 1972). Assuming a Lake Erie seasonal maximum density of phytoplankton of 14 mg/l (Vollenweider et al., 1974) and of zooplankton of 1 mg/l (Watson, 1974), the total copper in plankton would be equivalent to 0.089 μg/l of copper in the water. Copper concentrations in Lake Michigan water average 5 μg/l (Copeland and Ayers, 1972). Thus in whole-water the maximum error in the metal concentration of the sample during plankton blooms would be about 2%. This value may be too high since plankton in a bloom might deplete the metal ions in the water being sampled rather than adding metal ions. There is also a possibility of zooplankton "swarms" with densities approaching one gram per litre. Such "swarms" might contribute significantly to metal concentrations but the problem could be avoided by not sampling under such extreme conditions. In addition, filtration to remove micro-organisms could be another problem -- the filter may add or remove ionic copper (Marvin et al., 1970). A further problem may be anomalously high concentrations of metals in samples obtained from turbid inshore waters affected by shoreline erosion. These concentrations should be interpreted with caution. The measurement of metals in a sample that has been allowed to settle or that has been filtered could also give erroneous results if metals which are easily dissolved

from particulate matter were removed.

Stiff (1971) has assembled a variety of methods and has outlined an analytical routine for differentiating various forms of copper. However, results of this approach have yet to be correlated to toxicity tests in a variety of waters and are not suitable for application to routine monitoring. However, it is hoped that future developments in the methodology for identifying the various forms of metals will allow for refinements of objectives. Obviously any such refinement in the determination of the chemical and physical specification of an element will also require more elaborate sampling and storage procedures.

Therefore, until the relationship between metal forms and their toxicity is firmly established, and until there are reliable methods for monitoring such forms, water quality objectives for metals will refer to total concentrations of each metal in an unfiltered, (whole water), digested sample.

Setting Objectives for Metals for Aquatic Biota

Concentrations of metals that are above the level required for the nutrition of aquatic organisms but which are below their lethal level may produce subtle detrimental effects to their organisms. These effects may range from the inhibition of a single enzyme to failure in reproduction. The inhibition of a single enzyme may be of minor consequence or it may contribute to reproductive failure. If an aquatic organism is affected in some way by a metal so that it fails to reproduce, the population of that organism may disappear without evident direct mortality. Reductions in growth or in the efficiency of various physiological functions, changes in behaviour, or occurrence of physical abnormalities may all reduce the probability of successful reproduction of an organism. In particular, avoidance of sublethal concentrations of pollutants may be harmful to populations of fish by preventing migration to spawning areas or favourable feeding areas.

Thus, the objectives for metals are set at safe concentrations for aquatic species. Safe concentrations are determined as the maximum concentrations shown to have no harmful effect on any or all aspects of an aquatic organism's reproduction, physiology, behaviour, growth or any other function or activity essential for the maintenance of its population. In addition there should be no detrimental effect on a fishery based directly or indirectly on that organism. An unsafe concentration is any concentration having a harmful effect.

Safe concentrations are usually developed by laboratory measurements of sublethal toxicity. A measurement of concentrations inhibiting reproduction or producing mortality of a sensitive life stage provides a direct measurement of the unsafe concentration. Measurements of concentrations inhibiting physiological processes are most useful when the relevance to maintaining a population of the test organism is defined.

Safe concentrations may be derived from three measurements:

(a) The Maximum Acceptable Toxicant Concentration (MATC) as defined by Mount and Stephan (1967) consists of two numbers: (1) the lowest concentration of a toxicant having a harmful effect on an organism (unsafe) and (2) the highest concentration not producing that effect (safe). The threshold of response occurs somewhere between these two concentrations.

(b) A direct measurement of the threshold concentration causing the harmful effect. These data may be less useful if there are no limits given to the range of threshold concentrations.

(c) The application factor concept provides the third source of data for objectives since it is the ratio of MATC's to 96-hour LC_{50}'s. Consequently, an application factor can estimate the MATC for a particular species after a simple 96-hour LC_{50} measurement. Since there are error limits to both the application factor and the 96-hour LC_{50}, a direct estimation of the MATC by experimentation is preferable.

It is the intent of the Water Quality Board to provide a quality of water in the Great Lakes that will protect all water uses. Therefore, the proposed objectives for the metals that follow are based on the most sensitive of the defined uses of these Great Lakes waters.

Table 3

Concentrations (µg/l) of metals in filtered Great Lakes water sampled from municipal water intakes between 1962 and 1967 (Kopp and Kroner, 1970).

Metal	Detection Limits[8] (µg/l)	Lake Superior at Duluth Mean[1]	Lake Superior at Duluth Range	Lake Superior at St. Mary's R. Mean	Lake Superior at St. Mary's R. Range	Lake Michigan at Milwaukee Mean	Lake Michigan at Milwaukee Range	Lake Michigan at Gary Mean	Lake Michigan at Gary Range	Lake Huron at Port Huron Mean	Lake Huron at Port Huron Range	Lake St.Clair at Detroit Mean	Lake St.Clair at Detroit Range	Lake Erie at Buffalo Mean	Lake Erie at Buffalo Range	St. Lawrence R. at Massena Mean	St. Lawrence R. at Massena Range
Aluminum	40	11	ND[5]-26	6	ND-10	Not measured		21	ND-58	24	ND-65	29	ND-68	31	ND-66	39	ND-148
Arsenic	100	Not measured		—	—	—	—	—	—	—	—	—	—	—	—	38	ND-58
Cadmium	20	Not measured		—	—	—	—	—	—	—	—	—	—	7	ND-12	Not measured	
Chromium	10	9	ND-20	3	ND-7	—	—[4]	10	ND-19	5	ND-8	8	ND-13	7	ND-10	26	ND-112
Copper	10	3	3-36	5	2-28	13	ND-34	4	ND-7	10	4-20	8	6-13	24	10-56	7	ND-23
Iron	10	23	2-83	19	ND-168	20	ND-37	49	ND-114	16	ND-53	23	ND-62	19	4-84	22	ND-171
Lead	40	—	—[2]	6	ND-12	13	ND-20	34	ND-55	14	ND-28	21	ND-53	Not measured		22	ND-48
Nickel	20	—	—[3]	11	ND-28	ND	ND	ND	ND	ND	ND	—	—[6]	—	—[7]	7	ND-10
Silver	2	Not measured		—	—	—	—	—	—	—	—	—	—	—	—	2.6	ND-6.0
Zinc	20	9	ND-17	41	2-406	13	ND-23	25	10-55	12	ND-20	24	ND-69	178	64-423	41	ND-210

1. Mean of concentrations above limits of detection in extracted samples.
2. Only two detections: 7 and 20 µg/l.
3. Only one detection: 2 µg/l.
4. Only two detections: 2 and 4 µg/l.
5. ND = not detected at limits of analytical method.
6. Only two detections: 5 and 20 µg/l.
7. Only two detections: 13 and 21 µg/l.
8. Extraction methods allow the measurement of concentration below normal detection limits.

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ALUMINUM

RECOMMENDATION

No objective for aluminum is recommended at this time due to an inadequate data base on the effects of aluminum on aquatic biota.

RATIONALE

Aluminum is an abundant element in nature, especially in rocks and ores (Table 1). The metal itself is insoluble, but many of the salts are quite soluble. The occurrence of the metal in surface waters is limited because it is precipitated and adsorbed. The behaviour of aluminum in aqueous solutions is extremely intricate, forming a variety of sensitive complexes with water. The solubility of aluminum in natural waters is a direct function of pH, being more soluble at values of pH above and below 5.5. Concentrations of total aluminum in offshore Great Lakes waters are usually less than 10 µg/l (CCIW, unpublished data). In water intakes, concentrations may be as high as 148 µg/l but the mean values are 40 µg/l or less (Table 3). The high concentrations in offshore or intake waters appear principally in the Lower Lakes.

The usefulness of water for steam generation can be restricted by the presence of aluminum. Boiler feedwater to be used in high pressure situations may have to be treated to achieve aluminum concentrations less than 10 µg/l. Recirculation makeup water may be required to have an aluminum content of no more than 100 µg/l. Once-through cooling systems can utilize water as received without its use being restricted by the aluminum concentrations (NAS/NAE, 1973). Other industrial processes do not generally have restrictions on the aluminum concentration in process water.

There is no evidence for a nutritional requirement for aluminum of plants or animals (Bowen, 1966; Underwood, 1971). Water for livestock can be used with concentrations of up to 5,000 µg/l without harm (NAS/NAE, 1973). Aluminum is not considered a public health problem in public water supplies. In fact, "alum" is widely used as a coagulant in the treatment of water for public water supplies. A report by Capen (1939) states that alum at a concentration of 100 µg/l as aluminum in swimming pools may cause eye irritation. There are no standards for aluminum in the drinking waters of Canada (DNHW, 1969) or the U.S. (NAS/NAE, 1973).

Phytotoxicity from irrigation waters containing aluminum depends largely on soil pH. Alkaline pH values cause the aluminum salts to precipitate in the soil. Low soil pH (less than about 5.5) does not allow this precipitation to occur, thereby creating a toxicity condition thought to be due to the aluminum ion. A maximum aluminum concentration of 5,000 µg/l is recommended for irrigation waters with much higher values allowable for the neutral or alkaline soils (NAS/NAE, 1973).

The toxicity of aluminum to aquatic organisms varies with hardness, turbidity and pH. Symptoms of aluminum poisoning are different for dissolved and suspended aluminum. Acute toxicity is most pronounced when aluminum is dissolved, while chronic effects appear when aluminum is in suspended form. Freeman and Everhart (1971) calculated that at pH 7.0, the saturation level of aluminum in water was 50 µg/1. At this concentration there were no observed effects on rainbow trout. When the pH was increased to 9.0, at least 5,000 µg/1 of aluminum dissolved and fingerling rainbow trout were killed in 48 hours. These same investigators indicated that an aluminum concentration of about 500 µg/1 produced mortality, reduced feeding activity and caused dark coloration after a few weeks exposure at pH's of 7.0 and 8.0. Higher mortality rates were predicted for continued exposure. At pH 7.0, 90 percent of the aluminum would be in suspension whereas at pH 8.0, 100 percent would be dissolved. Freeman and Everhart (1971) encountered some difficulties in maintaining constant conditions at pH 7.0.

Since this experiment involved relatively short exposure times (45 days) and since reproduction of exposed fish was not measured, actual harmful concentrations may be much lower than 500 µg/1. Subsequent studies indicated that fish exposed to 500 µg/1 aluminum and transferred to fresh water, attained a growth rate equal to controls but did not make up the loss in growth suffered during the exposure (Freeman, 1973).

Doudoroff and Katz (1953), reporting on the work of Jones (1939), indicated a lethal threshold concentration of 70 µg/1 for stickleback using aluminum nitrate. The sticklebacks survived 300 µg/1 for one day and 100 µg/1 for one week. These tests were carried out with extremely soft water and at low pH. Other studies reported by Doudoroff and Katz (1953) revealed varying toxicity values with one study reporting no effect on rainbow trout in 48 hours at 1,000 µg/1. Biesinger and Christensen (1972) reported that 16 percent impairment of reproduction of Daphnia magna occurred at aluminum concentrations of 320 µg/1 and the three week LC_{50} for the same organism was 1,400 µg/1. Freeman and Everhart (1971) concluded with the statement that "If aluminum is present only in anionic and neutral or near neutral precipitated forms, a condition that should hold for most natural waters with pH greater than 5.50 tolerable concentrations of either form probably should not exceed 100 µg/1, if trout are to survive and grow normally".

Therefore an objective for aluminum of 100 µg/1 could be based on the protection of freshwater aquatic life, provision of good boiler feed water an provision of water not irritating to swimmers. However, none of the above studies have been confirmed by further experimentation and it is difficult, at this time, to justify an objective.

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ARSENIC

RECOMMENDATION

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

> *Concentrations of total arsenic in an unfiltered water sample should not exceed 50 micrograms per litre to protect raw waters for public water supplies.*

RATIONALE

There are several forms of arsenic found in fresh water; the most common are the arsenic and arsenious acids, the oxides of arsenic (As_2O_3), and some sulphur compounds (realgar and orpiment). The form in which one finds arsenic in fresh water is largely dependent upon the eH and pH values of the water (Ferguson and Gavis, 1972). Arsenic is also found in water in a variety of salt forms, such as sodium arsenite and sodium arsenate.

Physical forces such as weathering represent pathways by which arsenic may enter the aquatic ecosystem. It has been found that some igneous rocks have an arsenic content of about 2 µg/g; shale can yield arsenic concentrations as high as 13 µg/g while sandstone and limestone contain approximately 1 µg/g of arsenic (Table 1).

Other important sources of arsenic contamination are the burning of fossil fuels such as coal and oil, and various pesticides such as herbicides applied directly to water (Wiebe, 1930; Gilderhus, 1966). Arsenic also comes from various cleansing compounds in which levels as high as 35 µg/g have been measured (Zwick and Benstock, 1971). About 91,000 kg of arsenic were used in the Great Lakes basin in 1968, primarily as As_2O_3, for metallurgy (Fenwick, 1972).

Arsenic levels in surface waters, from natural or man made contamination vary considerably. Ferguson and Gavis (1972) report levels between 0 and 10 µg/l in freshwater; in Germany levels of 2 to 3 µg/l are normally found (Hutchinson, 1957, p. 563). Concentrations of arsenic in the Great Lakes are uniformly 1 µg/l or less in offshore waters (CCIW, unpublished data) but were found to be as high as 58 µg/l in a water intake at Massena, New York (Table 3). The Moira River, flowing into the Bay of Quinte, contains high levels of arsenic due to mining activity in its watershed. Concentrations of arsenic in the water of this river are normally greater than 10 µg/l but values as high as 300 µg/l have been recorded (OME, 1971).

Arsenic has no known nutritive value for plants (Bowen, 1966) and its essentiality for animals has not been proven. However, arsenic in the form of arsanilic acid, 4-nitrophenylarsonic acid, 3-nitro-4-hydroxy-phenylarsonic acid and phenyl-arsenoxide are proven growth stimulants for

pigs and poultry (Underwood, 1971).

Arsenic is classified by Bowen (1966) as moderately toxic to plants (toxic effects appear at concentrations between 1 and 100 mg/l in the nutrient solution). Arsenic is highly toxic to animals and it is a cumulative poison. Acute poisoning produces intestinal pain, vomiting and can lead to death. Chronic symptoms include cramps, nausea and liver damage (Fenwick, 1972).

In accordance with the "Safe Drinking Water Act", (PL-93-523), the U.S. Environmental Protection Agency proposed interim drinking water standards in the Federal Register on March 14, 1975. The maximum contaminant level for arsenic is proposed to be 50 µg/l, the same value as in the existing standards. Recently, the National Academy of Sciences (NAS/NAE, 1973) has recommended a maximum level of 100 µg/l total arsenic "because of adverse physiological effects on humans and because there is inadequate information on the effectiveness of defined (water) treatment procedures in removing arsenic." The existing guidelines for raw water in Canada (1968 Canadian Drinking Water Standards and Objectives - under review) specify an acceptable arsenic level of 10 µg/l and a maximum permissible level of 50 µg/l. For livestock an upper limit of 200 µg/l of arsenic in water is recommended (NAS/NAE, 1973)

The presence of arsenic in the aquatic environment has been shown in some cases to have deleterious effects on organisms. Some workers have used sodium arsenite to determine the lethality of arsenic on test organisms (Gilderhus, 1966), while others have used arsenite as arsenic trioxide (Holland, 1960). The lethal concentrations of both arsenate and arsenite for some algae fall between 2,000 and 10,000 µg/l (Wong, 1975).

The three week LC_{50} of sodium arsenate to Daphnia magna was 2,850 µg/l while the concentrations causing 50 and 16% impairment of reproduction were 1,400 and 520 µg/l, respectively (Biesinger and Christensen, 1971). Little is known about the effects of sodium arsenite on invertebrate and fish physiology. It is mainly used as a herbicide, but it may also be used as a deterrent to Toredo infestation of wooden structures in salt water. The 48-hour LC_{50} of sodium arsenite to chum salmon (Oncorhynchus keta) is about 11,000 µg/l (Alderdice and Brett, 1957). Holland (1960) noted 22% initial mortality of young pink salmon exposed to 5,300 µg/l arsenic, but mortality in the survivors continued for an additional 20 days. Recently, Speyer (1974) found 6,000 µg/l arsenic to be the lowest level affecting growth of rainbow trout although the response was increased by the presence of 200 µg/l HCN. Lawrence (1958) investigated the effect of arsenic trioxide on fish production using ponds stocked with bluegills. At 4,000 µg/l and 8,000 µg/l, reduction of bottom organisms as compared to the controls was 34% and 45%, respectively. The weight of fish harvested was also substantially reduced in the treated ponds. Conditioned avoidance behaviour

of goldfish was significantly impared by 100 µg/l arsenic as sodium arsenate but not by 50 µg/l (Weir and Hine, 1970).

Gilderhus (1966) studied the uptake of sodium arsenite by bluegills in outdoor pools containing invertebrates, vegetation and sediments. He noted that much of the arsenic applied ended up in the sediment. At 4,000 µg/l arsenic (a single treatment) maximum tissue residues in fish were 1,300 µg/kg for muscle, 2,400 µg/kg for skin and scales, 17,600 µg/kg for gills and digestive tract, 11,600 µg/kg for liver, 5,900 µg/kg for kidneys and 8,400 µg/kg for ovary. Average residues in Great Lakes fish vary from 3-43 µg/kg on a whole weight basis (Lucas et al., 1970), < 50-700 µg/kg on a dressed fish basis (Uthe and Bligh, 1971) and 6-80 µg/kg on a liver basis (Lucas et al., 1970). These values are considerably below those observed on an experimental basis.

Concentrations of arsenic considered safe for public drinking water supplies are substantially lower than those required to protect aquatic life. Consequently, the objective for arsenic should be 50 µg/l in keeping with the approved concentration for the protection of human health. However, to protect aquatic life, the Province of Ontario, specifies that "an environmental level of 10 µg/l should not be exceeded under any circumstances" (OWRC, 1970). This guideline is not well supported by scientific evidence.

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PERSISTENT CONTAMINANTS - METALS

CADMIUM

RECOMMENDATION

Concentrations of total cadmium in an unfiltered water sample should not exceed 0.2 micrograms per litre to protect aquatic life.

RATIONALE

Cadmium is a divalent metal that occurs mostly as a sulphide, usually in association with other metal sulphides, especially of lead and zinc. There is no mining activity specifically for cadmium; it is obtained principally as a by-product of zinc mining (Lymburner, 1974).

The properties of cadmium make it important in electroplating, in solders, as a pigment, as a catalyst, in photography, lithography and the electronics industry, and in the manufacturing of glass, alloys, biocides, lubricants and storage batteries (Lymburner, 1974, Cheremisinoff and Habib, 1972). In the Great Lakes Basin, cadmium is a by-product of zinc refining in Port Maitland, Ontario and cadmium-containing ores are mined in the Lake Superior region (Lymburner, 1974). There is considerable use associated with the automotive and metallurgical industries of the lower Great Lakes region. Therefore, cadmium may enter Great Lakes waters as a result of these processes. Additional inputs are derived from the weathering of rocks and the fallout from airborne cadmium originating in fossil fuels.

In water, cadmium may be complexed with soluble inorganic or organic materials as well as adsorbed to particulate matter. Hem (1972) derived theoretical limiting equilibrium solubilities for the carbonate and hydroxide complexes of cadmium in specific waters. He found that cadmium concentrations in surface waters of the United States, as reported by various authors, were much lower than the maximum permitted by the solubility product of the carbonate, the least soluble salt. He attributed the difference to the action of other complexing and adsorbing materials. Hahne and Kroontje (1973) also showed theoretically that, at high pH's or chloride concentrations, a high proportion of cadmium was mobilized as hydroxy or chloride complexes. However, their data show that at pH 7-8 and at chloride concentrations of 35 mg/l, the bulk of cadmium would occur as Cd^{++}. Using a cadmium specific ion electrode, Gardiner (1974a) measured the degree of complexation of cadmium in synthetic solutions and natural river waters containing varying amounts of carbonate, sewage effluent and humic acids. He found that a large proportion of cadmium occurred as Cd^{++} although the amount decreased with increasing pH, sewage effluent concentration or humic acid concentration. Humic substances accounted for most of the complexation. In natural waters, Gardiner (1974a) found that, of 1,000 µg/l added cadmium, 29-89% occurred as Cd^{++}, and the proportion was generally in excess of 50%. Suspended

solids originating from bottom muds will also adsorb cadmium (Gardiner, 1974b). The degree of adsorption depended on the type of solid, state of subdivision, concentration of metal ion, time of contact and concentration of other complexing ligands. Humic materials again appeared to be the major component of mud that is important. Gardiner (1974b) however, after these laboratory studies, was unable to satisfactorily explain the high proportion of measured free cadmium after adding cadmium to the effluent from a percolating filter. In a study of two streams in Tennessee, Perhac (1972) measured the metal content of coarse particulate suspended solids (Svedberg coefficient* (S) > 20000), in colloidal particulate suspended solids (100 < S <20000) and in dissolved solids. The mean cadmium concentrations in these fractions were 18, 519 and 12 µg/gm respectively. While the greatest concentration of cadmium was in colloidal solids, this represented the smallest proportion of heavy metal in water because colloids occurred only in trace amounts. The highest proportion of cadmium (≃98%) occurred in the dissolved solids. Presumably these materials would include humic acids, carbonates, chlorides, etc. Total cadmium in these waters ranged from 2-3 µg/l. Therefore, assessment of the impact of cadmium in water will probably be most concerned with free cadmium and soluble complexes.

Cadmium concentrations in the Upper Great Lakes are almost always less than 0.2 µg/l offshore (Table 2). In Lake Superior a small proportion of concentrations are between 0.2 and 0.6 µg/l. In Lake Erie, concentrations of cadmium in offshore filtered water never exceeded 1 µg/l, the detection limit at that time (Chawla and Chau, 1969; MWRC, 1972) but in a water intake at Buffalo, concentrations ranged as high as 12 µg/l and the mean was 7 µg/l (Table 3). In Lake Michigan, concentrations never exceeded 1 µg/l in 1970 although some tributaries were slightly higher (MWRC, 1972). In a 1974 survey of American nearshore waters, cadmium was always less than 2 µg/l (detection limit) in Lake Superior and Lake Huron (MWRC, 1975).

Cadmium is extremely toxic to mammals. Acute toxicity to humans includes severe nausea, salivation, vomiting, diarrhea, abdominal pains and myalgia. Liver and/or kidney damage may follow acute poisoning and respiratory distress may also occur (Flick et al., 1971). Chronic toxicity includes damage to liver, kidney, hematopoietic tissue and the respiratory tract (Flick et al., 1971). Cadmium has been implicated in bone degeneration in Japan although these findings are controversial (Dr. E. Sandi, personal communication). Epidemiological and experimental evidence suggests that cadmium may also cause hypertension. In experimental animals cadmium causes testicular damage, kidney damage, increased incidence of tumours and reduced growth (Flick et al., 1971). The biochemical bases for these effects may be the interaction of cadmium with thiol groups of enzymes or with phosphatidylethanolamine and phosphatidylserine monolayers (Vallee and Ulmer, 1972). As a result, many enzymatic reactions are inhibited by cadmium, and toxic effects occur in mitochondria, kidney tubules and nerve membranes (Vallee and Ulmer, 1972). The daily uptake of cadmium by an adult human from drinking water has been estimated as 15 µg, as compared to 200 µg in food and 1 µg in air (Nilsson, 1970). Of the total cadmium taken in,

* Svedberg coefficient is a numerical value related to the settling velocity of a spherical particle.

only 1-2% is retained and the rest is excreted in faeces and urine. To limit intake from water to 200 µg/day, a drinking water limit of 10 µg/l cadmium has been recommended (NAS/NAE, 1974). In Canada, the maximum permissible concentration of cadmium in drinking water is 10 µg/l while the acceptable concentration is less than 10 µg/l (DNHW, 1969). A recommendation of 50 µg/l is given to protect livestock (NAS/NAE, 1973).

Cadmium is not a nutrient for plants and is classified as highly toxic by Bowen (1966), (toxic at concentrations less than 1,000 µg/l in the nutrient solution). Since crop growths may be reduced at concentrations as low as 10 µg/l, recommendations for irrigation water are 10 µg/l for continuous use on all soils and 50 µg/l on neutral and alkaline fine textured soils for a 20-year period (NAS/NAE, 1973).

Low concentrations of cadmium are harmful to algae. Growth of Scenedesmus quadricauda in the laboratory was significantly inhibited at concentrations as low as 6 µg/l (Klass et al., 1974). Selenastrum capricornutum is somewhat less sensitive since 80 µg/l caused complete growth inhibition while 50 µg/l caused a slight inhibition (Bartlett et al., 1974). In a comparative study, Burnison et al. (1975) found that the concentrations of cadmium in Lake Ontario water causing 70% inhibition of primary productivity of Scenedesmus quadricauda, Chlorella pyrenoidosa, Ankistrodesmus falcatus and Chlorella vulgaris were 20, 100, 1,000 and 1,000 µg/l respectively. A macrophyte, Najas quadulepensis, was also affected by cadmium. Severe effects were observed at 90 µg/l while 7 µg/l caused reduced chlorophyll, turgor and stolon development (Cearley and Coleman, 1973).

The acute toxicity of cadmium to zooplankton varies considerably with the species tested. In water from Lake Monate, the 48-hour LC_{50}'s for Cyclops abyssorum prealpinus, Eudiaptomus padanus padanus and Daphnia hyalina were 3,800, 550 and 55 µg/l respectively (Baudouin and Scoppa, 1974). The 48-hour LC_{50} for Daphnia magna in Lake Superior water was 65 µg/l (Biesinger and Christensen, 1972), a value close to that of Daphnia hyalina. The 3-week LC_{50} for Daphnia magna was 5 µg/l while 0.17 µg/l caused 16% impairment of reproduction (Biesinger and Christensen, 1972). The 96-hour LC_{50} of the freshwater shrimp Paratya tasmaniensis at 10 mg/l hardness was 60 µg/l (Thorp and Lake, 1974). A 96-hour exposure of these shrimp to 30 µg/l cadmium caused a change in the ultrastructure of the gills (Lake and Thorp, 1974).

Aquatic insects are less sensitive than zooplankton. At a hardness of 44 mg/l, the 96-hour LC_{50}'s of cadmium for Acroneuria lycorias (stonefly), Ephemerella subvaria (mayfly) and Hydropsyche betteni (caddisfly) were >32,000, 2,000 and >32,000 µg/l respectively (Warnick and Bell, 1969). At 50 mg/l hardness, the 96-hour LC_{50}'s of a caddisfly, a damsel fly, and a midge (Chironomus sp.) were 3,400, 8,100, and 1,200 µg/l respectively (Rehwoldt et al., 1973). The species of caddisfly was unidentified and appeared 10 times more sensitive than that tested by Warnick and Bell (1969). The 96-

hour LC_{50}'s of a caddisfly, a damsel fly and a mayfly of Tasmania in water of 10 mg/l hardness was 2,000, 250,000 and 840 µg/l respectively (Thorp and Lake, 1974). Amphipods are much more sensitive since the 96-hour LC_{50} of Australochiltonia subtennis was 40 µg/l (Thorp and Lake, 1974), while that of a scud (Gammarus sp.) was 70 µg/l (Rehwoldt et al., 1973).

The 96-hour LC_{50}'s for a gastropod snail were 3,800 µg/l for eggs and 8,400 µg/l for adults (Rehwoldt et al., 1973). In contrast, the snail Helisoma sp. had a 14-day LC_{50} of 50 µg/l, and 20 µg/l reduced rates of survival and hatching of eggs (Heidel and McLaughlin, 1973). No effect was observed at 10 µg/l cadmium. Another benthic organism, the bristle worm (Nais sp.) had a 96-hour LC_{50} of 1,700 µg/l (Rehwoldt et al., 1973), while that for the rotifer Philodina sp. was about 100 µg/l (Sullivan et al., 1973). Tetrahymena pyriformis, a protozoan, showed a growth depression at 15,000 µg/l cadmium and slower swimming at 1,000 µg/l (Bergquist and Bovee, 1973).

The acute toxicity of cadmium to fish varies with species and the time of exposure. The 96-hour LC_{50} for fathead minnows (Pimephales promelas) at 200 mg/l hardness was 4,500 µg/l while the 8-day LC_{50} was 450 µg/l (Pickering and Gast, 1972). Similarily, the 96-hour LC_{50} for rainbow trout in hard water (290 mg/l) was about 2,000 µg/l while the 7-day LC_{50} was 8-10 µg/l (Ball, 1967). Kumada et al. (1972) observed a similar 10-day LC_{50} for rainbow trout of 5-7 µg/l cadmium. The 96-hour LC_{50}'s for bluegills (Lepomis macrochirus), Florida flagfish (Jordanella floridae, dace (Triborodon hakonensis) and striped bass (Morone saxatilis) were 17,200-24,200, 2,500, 56-100, and 2 µg/l respecitvely (Eaton, 1974; Spehar, unpubl. man.; Kumada et al., 1972; and Hughes, 1973).

The sublethal effects of cadmium on fish include lingering mortality and inhibition of reproduction. In hard water (200 mg/l) 57 µg/l of cadmium decreased the survival of fathead minnow larvae, the most sensitive stage. No effect was observed at 37 µg/l (Pickering and Gast, 1972). At a hardness of 120 mg/l, a mixture of cadmium, zinc and copper reduced the spawning of fathead minnows when the concentrations were 7.1, 42.3 and 6.7 µg/l respectively (Eaton, 1973). No effect was seen when the concentrations of cadmium, zinc and copper were 3.9, 27.3 and 5.3 µg/l respectively. It is not known whether the apparent increase in toxicity of cadmium is due to a change of water hardness or to the presence of the other metals. Since the toxic effects (larval mortality and reduced spawning) differed, it was probably the effect of the other metals.

Eaton (1974) showed that, at a hardness of 200 mg/l, bluegill survived and spawned successfully at 31 µg/l cadmium. Lingering mortality of adults occurred at 80 µg/l and bluegill appear as sensitive as fathead minnows at this hardness. In water of 180 mg/l hardness, Cearley and Coleman (1974) found that bluegill survival was not affected at 80 µg/l cadmium but 100%

mortality occurred at 850 µg/1 after 5 months. The principal difference between Eaton's (1974) study and that of Cearley and Coleman (1974) is that the latter used water of low alkalinity (49 mg/1) compared to the former (152 mg/1). In addition the chloride content of the water used by Cearley and Coleman (1974) was 193 mg/1. Largemouth bass (Micropterus salmoides) were more sensitive than bluegills. Significant mortality occurred at concentrations of 80 µg/1 cadmium and behaviour was affected at 8 µg/1 (Cearley and Coleman, 1974).

Survival of flagfish larvae in water of 44 mg/1 hardness was affected at 8 µg/1 cadmium and was normal at 4 µg/1. When the embryos were exposed to cadmium before hatching, the hatched larvae were less sensitive to cadmium (Spehar, Unpub. man.).

The reproductive physiology of brook trout (Salvelinus fontinalis) is also affected by cadmium. Exposures to 25 µg/1 for 24 hours or 10 µg/1 for 21 days at 20 mg/1 hardness caused extensive hemorhagic necrosis of the testes of male trout (Sangalang and O'Halloran, 1972, 1973). After about 4 months exposure, 1 µg/1 cadmium caused changes in testosterone and 11-ketotestosterone metabolism of male fish. There was no effect on secondary sexual characteristics and spermatogenesis, but testes regressed at least 2 weeks earlier than controls (Sangalang and Freeman, 1974). Brook trout alevins showed a decreased wet weight, increased protein content and increased acetylcholinesterase activity at 0.70 µg/1 cadmium in water of 45 mg/1 hardness (Christensen, 1975). These results correspond fairly well with the effects of cadmium on reproduction and survival of brook trout measured by Benoit et al. (1975). Survival of adult males during spawning and growth of juveniles were reduced at 3.4 µg/1 while no adverse effects were noted at 1.7 µg/1 cadmium.

Cadmium up to 100,000 µg/kg in the food of fish was not toxic to rainbow trout or dace after 18 weeks exposure (Kumada et al., 1972).

Cadmium residues in fish are fairly uniform. Lovett et al. (1972) measured cadmium concentrations in dressed fish from Lake Erie, Lake Ontario and the St. Lawrence River. Concentrations were generally between 10 and 30 µg/kg although a few had less than 10 µg/kg (the detection limit) and Gizzard shad from Lake Erie had 72 µg/kg. In another survey of dressed fish, from Lakes Erie and Ontario, cadmium concentrations were uniformly less than 50 µg/kg, the detection limit, with one exception - 60 µg/kg in rainbow smelt from Lake Erie (Uthe and Bligh, 1971). Using neutron activation, Lucas et al. (1970) measured cadmium concentrations of 62-140 µg/kg in whole fish from Lake Erie, Michigan and Superior. In fish livers, concentrations ranged from 60 to 1,400 µg/kg with most values around 400 µg/kg. This suggests that the liver concentrates cadmium. In Lake Michigan, fish (presumably whole) contained 100-300 µg/kg cadmium and there was no variation with feeding habits of the fish (MWRC 1972).

In experimental systems, bass and bluegills had total body accumulations of 8-15 and 6-20 times the concentration in water, depending on that concentration (Cearley and Coleman, 1974). Uptake and concentration in tissues levelled off within 2 months and the greatest accumulation occurred in internal organs. Kumada et al. (1972) found that cadmium concentrations in rainbow trout exposed to cadmium in water reached a plateau in 10-20 weeks and maximum concentrations were found in the kidneys. Concentrations in whole fish were about 10-80 µg/kg in control fish and increased only at cadmium concentrations above 1 µg/l. Concentrations in whole fish reached a maximum of 960 µg/kg after 30 weeks in 4.8 µg/l and declined to 440 µg/kg after 10 weeks in clean water. Similar increases in cadmium content were seen in rainbow trout and dace fed food containing up to 100,000 µg/kg of cadmium. Concentrations in whole trout fed this concentration reached 1,600 µg/kg after 12 weeks and declined dramatically to 70 µg/kg after 6 weeks on a clean diet (Kumada et al., 1972). The dramatic decrease was seen at all concentrations and indicates that cadmium taken in with the food is cleared faster than cadmium taken in from water. This could be illusory if the gills of fish exposed to cadmium in water contain high concentrations that are slowly released to the rest of the body after transferral to clean water.

White catfish (Ictalurus catus) given an intragastric dose of radioactive cadmium regurgitated 39-56% of the dose (Rowe and Massaro, 1974). Within one hour, 75% of the cadmium in the body was contained within the GI tract and 23% was in the gills. The fact that 2% was in the skin suggests that the gill load may have been picked up from the water after regurgitation. Over a period of 21 days, cadmium gradually moved down the intestine and concentrations gradually increased in both the liver and kidneys. By day 21, 34% of the cadmium was in the kidneys, 5% in the liver, about 56% still remained in the intestine and the rest was spread among other organs at low concentrations. Therefore, the total transfer from cadmium in the gut to other organs appears rather low.

Despite accumulation of cadmium, there is little evidence for bioconcentration up food chains. Mathis and Cummings (1973) found that mean concentrations of cadmium in Illinois River bottom sediments, worms, clams, omnivorous fish, carnivorous fish and water were about 2,000 µg/kg, 1,100 µg/kg, 600 µg/kg, 30 µg/kg and 0.6 µg/l respecitvely. Similarily, in eutrophic Wintergreen Lake, the concentrations of cadmium in bottom sediments, zooplankton, aquatic macrophytes, fish and water were 1,100 µg/kg, 500 µg/kg, 200 µg/kg, 40 µg/kg, and 0.9 µg/l respectively (Mathis and Kevern, 1975). Surprisingly, faeces from large flocks of migrating Canada geese contained up to 600 µg/kg cadmium.

A food chain model has been developed that predicts cadmium will bioconcentrate in Western Lake Erie food chains (Thomann et al., 1974). The model may not be useful since data on all trophic levels below fish are inadequate. However, future use of such models, based on adequate data, may give a clearer indication of the potential for bioconcentration.

Therefore, because of the extreme sensitivity to cadmium of trout and zooplankton reproduction, an objective for cadmium in the Great Lakes of 0.2 µg/l is recommended.

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CHROMIUM

RECOMMENDATION

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

> *Concentrations of total chromium in an unfiltered water sample should not exceed 50 micrograms per litre to protect raw waters for public water supplies.*

RATIONALE

Chromium as Cr(VI) can enter aquatic ecosystems from the production and use of explosives, paper, dyes, paints, plated materials and tanning. As Cr(III), chromium is present in glass, ceramics, photography processes and textile dying mordants (Cheremisinoff and Habib, 1972). Up to 1,700 mg/l of chromium as dichromate, are also added to cooling tower waters to prevent corrosion and this amount is discharged directly to water courses (Shepherd and Jones, 1971). Chromium occurs at very low concentrations in Great Lakes waters. Offshore, the average recorded concentrations are less than 0.2 µg/l, the detection limit, and 95% of samples contain less than 0.6 µg/l (Table 2). At water intakes, average concentrations are shown to be less than 10 µg/l and maxima less than 20 µg/l (Table 3). However, concentrations of chromium in water intakes in the St. Lawrence River appear much higher, (Table 3). Since Cr (III) is probably complexed as an insoluble hydrated oxide above pH 5 (NAS/NAE, 1973), most dissolved chromium in Great Lakes waters is probably in the Cr(VI) valence state. However, Schroeder and Lee (1975) have clearly demonstrated that Cr(III) added to natural lake waters is converted very slowly to Cr(VI) and that the conversion is slower at low temperatures. Consequently, significant concentrations of Cr(III) could exist in lake water for many days. Cr(VI) can potentially be reduced by H_2S at the interface of aerobic and anaerobic waters (Schroder and Lee, 1975). However, in aerobic lake waters Cr(VI) is not reduced and is removed principally by physical processes. For example, Cr(VI) is sorbed effectively by $Fe(OH)_3$. The result is a significant positive, linear correlation of chromium with iron in lake sediments (Schroder and Lee, 1975).

Chromium at low concentrations may be a nutrient for plants and animals. Although not proven to be essentialy for plants, low concentrations in soil and water appear to stimulate growth of terrestrial and aquatic species (NRCUS, 1974). In mammals, chromium interacts with insulin to increase glucose tolerance, and some diabetic conditions are alleviated by chromium treatment (Bowen, 1966; Underwood, 1971). A United States National Research Council panel on chromium concluded that: "Chromium deficiency can be produced in experimental animals but it can be prevented and cured by appropriate chromium supplementation. Its symptoms are reproducible and consist of a general decrease in the tissue response to insulin. On this basis, chromium must be considered an essentialy element" (NRCUS, 1974).

At high concentrations, chromium in air causes respiratory damage and cancer to mammals and contact with the skin can cause ulcers, scars and allergic effects (NRCUS, 1974). The effects on humans of chromium in drinking water are unknown but a standard of 50 µg/l total chromium has been set in the U.S. to limit total daily intake (NAS/NAE, 1973). In Canada, the maximum permissible concentration is 50 µg/l as Cr(VI) and the acceptable limit is less than 50 µg/l (DNHW, 1969).

The toxicity of chromium to aquatic biota is quite variable and depends on the species tested. Hervey (1949) used a subjective measurement of unicellular algal growth inhibition to demonstrate that some diatoms were sensitive to 320 µg/l but not to 32 µg/l of chromium. WiumAnderson (1974), using ^{14}C fixation to estimate growth, estimated that 650 µg/l of Cr(VI) caused 50% inhibition of photosynthesis by the diatom, Nitzschia palea. Patrick et al. (1968) indicated that 208 µg/l of Cr(III) also caused 50% reduction of photosynthesis of N. palea. Based on cell counts, 150 µg/l allowed very little growth after 4 days exposure at low cell densities (Wium-Anderson 1974). Daphnia magna reproduction and activity were inhibited by 330 and 320 µg/l chromium, respectively (Biesinger and Christensen, 1972; Anderson, 1946). Another invertebrate, Philodina roseola was shown to be 10 times less sensitive than Daphnia magna since its life cycle was affected between 3,400 and 4,600 µg/l (Schaeffer and Pipes, 1973).

A series of unpublished studies by Benoit and Pickering, reported in Water Quality Criteria, 1972 (NAS/NAE, 1973), demonstrated "safe" concentrations, based on reproduction, of 300, 600 and 1,000 µg/l of hexavalent chromium for rainbow trout (Salmo gairdneri), brook trout (Salvelinus fontinalis), and fathead minnow (Pimephales promelas), respectively. The "safe" concentration of trivalent chromium for fathead minnows was 1,000 µg/l. Therefore, both valence states of chromium appear equally toxic on a sublethal basis. However, Olson (1958) observed that chinook salmon fingerlings (Oncorhynchus tshawytscha), after 12 weeks exposure, had higher mortality rates (>50%) and lower growth rates in 200 µg/l Cr(VI) than in 200 µg/l Cr(III) or in control tanks. The fish in Cr(III) had mortality and growth rates identical to those of the control fish. Therefore, on an acute basis, Cr(III) appears less toxic than Cr(VI).

Chromium concentrations in fish tissue are low. Lucas and Edgington (1970) measured chromium by neutron activation and found that average whole body concentrations in alewife, spottail shiner and trout perch ranged from 0.9-1.6 µg/g. Chromium was also measured by neutron activation in dressed samples of whitefish, northern pike, smelt and perch. The concentrations ranged from <0.017 µg/g to 0.034 µg/g wet weight (Uthe and Bligh, 1971). These results are quite low compared to those in whole fish, suggesting that chromium is not retained by muscle. In addition, there was no variation in the chromium concentration in the fish within species from Lake Erie and from Moose Lake, Manitoba. Moose

Lake is a lake free of industrial activity. Experimental exposures indicate that Cr(VI) was taken up from water at concentrations as low as 1 µg/l (Fromm and Stokes, 1962). At 2,500 µg/l, uptake was via the gills and the metal occurred in the spleen, posterior gut, pyloric caeca, stomach and kidney (Knoll and Fromm, 1960). Little occurred in muscle and uptake across the stomach was minimal. It does not appear that chromium contamination of fish represents a problem since oral toxicity to mammals is low (NRCUS, 1974). Also, the residues reported in the uptake experiments were not associated with any damage to the fish. Therefore, no objective for chromium concentrations in fish tissues is recommended at this time.

The data presented on toxicity suggest an objective for chromium in water somewhat greater than the guideline for drinking water, to protect aquatic life. Since the U.S. and Canadian guidelines for drinking water are 50 µg/l, the objective for total chromium is 50 µg/l.

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COPPER

RECOMMENDATION

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

> *Concentrations of total copper in an unfiltered water sample should not exceed 5 micrograms per litre to protect aquatic life.*

RATIONALE

Copper in water originates from mining activities, application of copper-containing pesticides and algicides, burning of fossil fuels, industrial use in electronics, metallurgy, chemicals production, etc. and corrosion of copper pipes. Total copper in water is usually in the form of insoluble particulates, soluble complexes and soluble divalent ions. The proportion in each form depends on the amount of particulate and soluble complexing agents. These agents may be materials such as humates or carbonates (Stiff, 1971). Copper available to aquatic organisms for acute toxic action appears to be soluble Cu^{2+} and, less importantly, $CuCO_3$ (Andrew, 1975 and Shaw and Brown, 1974). On an experimental basis "toxic" or "labile" copper has been estimated by (a) calculation on the basis of cupric ion concentration, pH and alkalinity (Pagenkopf, 1974 and Shaw and Brown, 1974); (b) cupric ion electrode (Zitko et al., 1973 and Andrew, 1975) and (c) anodic stripping voltammetry (Gächter et al., 1973). These measures of toxic copper have yet to be adapted to field studies of copper toxicity and therefore most laboratory estimates of sublethal toxicity to aquatic biota are based on measurements of total copper. There is also a possibility that complexed, precipitated copper may ultimately have a toxic effect on benthos.

The concentrations of copper in the Great Lakes are dependent on local inputs. In parts of Georgian Bay and Lake Superior, for example, copper concentrations are shown to rise in the spring (Star File data summaries, Canada Centre for Inland Waters, Burlington and M.W.R.C., 1975). Possibly, the copper accumulated during the winter from aerosol fallout is transported to the water during the runoff period of the first snow melt. The modal copper concentrations in the upper lakes offshore are less than 2.5 µg/l, and 95% of the measurements are less than 5.0 µg/l. Chau et al. (1971) in their review of the variation of filtered copper in Lake Ontario showed that concentrations were highest in the western and eastern portions of the lake and that the high copper concentrations were associated with high iron concentrations, possibly because of human activity. The average copper concentrations in water intakes are shown to be as high as 24 µg/l, with maxima of less than 60 µg/l and minima of 2 µg/l (Table 3). Water intakes are more likely to be influenced by effluents from local industrial or municipal sources than are offshore areas.

There is evidence that copper loadings from man's activities contribute significantly to these observed concentrations. For example, in soils from bluffs eroded into Lake Erie, copper concentrations are about 27 µg/g. In deep cores representative of pre-colonial times, Lake Erie sediments contain about 29 µg/g. However, in recent sediments, the concentration is doubled to about 57 µg/g (Kemp et al., 1976). This suggests that man's activities account for 50% of the total animal input of copper to Lake Erie.

Copper is a constituent of many metalloenzymes and respiratory pigments and is an essential element for bacteria, fungi, blue green algae, green algae, angiosperms, invertebrates and vertebrates (Bowen, 1966). In public water supplies, copper presents an aesthetic rather than a toxicity problem. Since oral toxicity to adults is low, the U.S. drinking water standard is set at 1,000 µg/l to control taste (U.S.P.H.S., 1962). In Canada, the acceptable concentration is 1,000 µg/l while the objective is less than 10 µg/l (DNHW, 1969).

Concentrations of copper resulting in acute toxicity to organisms varies considerably with the hardness or alkalinity of water because the concentrations used during short term LC_{50} tests are close to the limits of solubility of copper. On the other hand, concentrations of copper that are safe for aquatic organisms may vary somewhat with the species being tested and the response being measured, but vary only slightly with water chemistry because copper concentrations are below the limits of solubility of copper.

Freshwater algae are quite sensitive to copper. Concentrations inhibiting respiration, photosynthesis, and growth are generally between 200 and 800 µg/l (Wong, 1975). However, photosynthesis of Chlorella pyrenoidosa was reduced 60% by only 20 µg/l copper (Nielsen et al., 1969). Nielsen et al. (1969) also showed that toxicity varied with exposure time, light intensity, initial cell concentration and composition of the medium. Obviously data for copper toxicity to algae of the Great Lakes should be derived from bioassays modelling Great Lakes conditions. In addition, synergistic effects on algae of copper and nickel have been noted, as well as heavy metal tolerance (Stokes and Hutchinson, 1975). These factors should be considered in future studies.

Growth of snails (Campeloma decisum, Physa integra) was reduced significantly between 8.0 and 14.8 µg/l of copper (Arthur and Leonard, 1970) while growth of crayfish, Orconectes rusticus was reduced at 15 µg/l (Hubschman, 1967). Mortality of young Gammarus pseudolimnaeus prevented completion of life cycles between 4.6 and 8.0 µg/l (Arthur and Leonard, 1970). Reproduction of Daphnia magna was reduced by 16% at 22 µg/l copper and by 50% at 35 µg/l copper after 3 weeks exposure in Lake Superior water of 45 mg/l hardness. The 3-week LC_{50} was 44 µg/l copper (Biesinger and Christensen, 1972). Activity and feeding of Daphnia magna were reduced at 27 µg/l copper in Lake Erie water of about 120 mg/l hardness (Anderson, 1948).

Atlantic salmon (Salmo salar) avoided 2.4 µg/l of copper in the laboratory. In a tributary of the Miramichi River, New Brunswick, a spawning run of adult salmon was reversed when copper and zinc concentrations were 19 and 240 µg/l, respectively (Sprague et al., 1965). It is impossible to tell if zinc increased or decreased the avoidance of copper by the salmon. The net result was that reproduction was prevented by a failure to reach spawning beds.

Cough frequency of brook trout increased between 6 and 15 µg/l of copper (Drummond et al., 1973). This provided an early indication of detrimental effects due to long-term exposures since alevins of brook trout died between 9.5 and 17.4 µg/l (McKim and Benoit, 1971). Further exposures of second generation brook trout confirmed that 9.5 µg/l was safe for alevins (McKim and Benoit, 1974). Larvae of bluegill also appeared quite sensitive to copper since mortality occurred between 21 and 40 µg/l of copper (Benoit, 1975). This was about twice the concentration required to kill brook trout larvae.

Fathead minnow (Pimephales promelas) reproduction in the presence of copper has been studied using several different dilution waters. In soft water of 30 mg/l hardness, a concentration between 10.6 and 18.4 µg/l of copper was effective in blocking spawning (Mount and Stephen, 1969). At 200 mg/l hardness, spawning was prevented between 14.5 and 33 µg/l copper (Mount, 1968). During long- and short-term exposures of fathead minnows to copper at 200 mg/l hardness, egg production was severely reduced between 24 and 37 µg/l, regardless of the length of exposure (Pickering et al. submitted for publication). These results suggest that the safe concentration of copper for fathead minnows does not vary to a great degree with water hardness under constant conditions. In addition, the study by Pickering et al. (submitted for publication) indicates that only short-term elevations of copper concentrations above safe levels during the spawning period are required to block fathead minnow reproduction.

The above were all laboratory studies under ideal conditions. In a field study, using water of variable quality enriched by a sewage treatment plant, Brungs et al. (1976) found that egg production and spawning success of fathead minnows were reduced between 66 and 118 µg/l copper. Hardness, pH, dissolved oxygen, total dissolved solids and many other parameters varied continuously. Therefore, it is difficult to compare these results with other studies since each parameter could affect both the solubility of copper, the physiology of the organism, and the biological availability of copper. It is impossible, from the field study, to identify which chemical characteristic of water, or combination of characteristics, influenced the response of fathead minnows to copper. The chemical characteristics of water of the Great Lakes, however, are relatively constant due to the lakes' large volumes. In addition, alkalinity, hardness, dissolved solids, etc. in the lakes are much less than in the field study. For example, the maximum hardness in Lake Ontario is 135 mg/l while

that in the field study was 352 mg/1. Therefore, the laboratory data probably provide the best data for consideration of the effects of copper in the Great Lakes.

Copper concentrations in aquatic biota are generally low. Copeland and Ayers (1972) measured 6 and 5 µg/g in phyto- and zooplankton, respectively, in Lake Michigan. In animals from the Illinois River, Mathis and Cummings (1973) measured the following mean concentrations:

Tubificids:	23 µg/g
Clams:	1.2-1.7 µg/g
Fish fillets: (Carnivorous)	0.07 - 0.19 µg/g
Fish fillets: (Omnivorous)	0.10 - 0.24 µg/g

These results suggest no food chain bioaccumulation. However, since only muscle of fish was measured, bioaccumulation cannot be adequately assessed. In Great Lakes fish, average copper in whole fish ranged from 0.8 - 2.7 µg/g (Lucas and Edgington, 1970) in fish livers from 1.5 - 28 µg/g (Lucas and Edgington, 1970), and in fish fillets from 0.5 - 1.28 µg/g (Uthe and Bligh, 1971). The concentrations varied little from lake to lake. However, in a more recent study, Brown and Chow (1975) showed that copper concentrations in fish muscle from Baie du Dore, Lake Huron averaged 0.45 µg/g while those in fish muscle from Toronto Harbour averaged 1.93 µg/g. This suggests that local copper contamination may be reflected in fish muscle concentrations. Experimental exposures of brown bullheads to copper did not produce significant accumulations of copper in the opercle, red blood cells or blood plasma (Brungs et al., 1973). However, there was a significant accumulation in the gills, kidney and liver at water concentrations of 27 - 53 µg/1. These residues have not been associated with any harmful effects on bullheads.

Although copper concentrations in fish tissues may indicate exposure of fish to copper, there have been no harmful effects to fish or to consumers of fish reported at present measured copper concentrations in the Great Lakes. Therefore, no objective for copper in fish tissues is recommended.

The data from laboratory studies of fish reproduction suggest an objective for copper alone in water of 10 µg/1. Eaton (1973) found that fathead minnow spawning success and egg production were reduced between 5.3 and 6.7 µg/1 copper when low concentrations of added cadmium and zinc were present. The cadmium and zinc concentrations were 3.9 and 27.3 µg/1, respectively, when no effect was observed and 7.1 and 42.3 µg/1, respectively, when reproduction was affected. Since all three metals could occur simultaneously at these concentrations in the Great

Lakes and since the young of Gammarus pseudolimnaeus are very sensitive to copper, an objective for copper of 5 µg/l is recommended. This objective is very close to copper concentrations measured offshore in the Great Lakes (Table 2). Since only total copper concentrations were measured during monitoring and sublethal toxicity studies in the laboratory, the relationship between toxicity and the amount of copper available for toxic action is unknown. In other words, measured total copper concentrations in the lakes exceeding the objective may be harmless if the copper is complexed and unavailable to aquatic organisms. Until adequate methods for assessing this situation are available, the objective will refer to total copper.

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IRON

RECOMMENDATION

It is recommended that the following revised objective for iron be adopted:

> *Concentrations of total iron in an unfiltered water sample should not exceed 300 micrograms per litre to protect aquatic life.*

RATIONALE

Iron is present in high concentrations in most rocks and soils, as well as in ore deposits. Input to the Great Lakes originates with weathering of rocks and soils; mining and processing of iron ores; steel making and metal fabricating; burning of fossil fuels; corrosion of iron or steel products in use; and corrosion of iron or steel products in junkyards, dumps and stream beds.

Total iron concentrations in water may easily exceed 1,000 µg/l. However, much of this iron may be in the form of suspended insoluble hydroxides or as a complex of an organic molecule such as a humic acid. The amount of iron in an insoluble nonionic form will approach 100 percent in waters of high dissolved oxygen because the ferric (Fe(III)) form predominates and it is relatively insoluble. In waters of low dissolved oxygen, iron may be reduced to the ferrous form (Fe(II)) and some iron will be in solution as an ion. This is accentuated by the fact that ferrous iron may be released from lake sediments if the water above the sediments becomes totally anaerobic; the release of iron being the result of a shift in the redox potential below a critical level at the sediment surface (OME, 1974).

Total concentrations of iron in the Great Lakes average less than 120 µg/l, (CCIW, unpublished data). Concentrations of iron passing a 0.45 µ filter are less than 7 µg/l in 95% of all samples (Table 2). Inshore, iron in filtered samples average less than 50 µg/l in water intakes and concentrations never exceeded 200 µg/l (Table 3). In waters adjacent to known sources of iron in the Great Lakes, concentrations may be much higher. For example, in Hamilton Bay in 1972, total iron concentrations were always greater than 200 µg/l, and most samples were between 300 and 700 µg/l. Some samples were recorded as high as 3,700 µg/l (OME, 1974).

Iron is an essential element for all organisms. It is a catalyst that activates a number of oxidases, and it is a constituent of many oxidizing metalloenzymes, respiratory pigments and proteins of unknown function (Bowen, 1966). Because iron can exist in oxidation states ranging from Fe (II) to Fe (VI), it is useful in a wide variety of biological processes. However, its low solubility in the ferric (Fe(III))

form means it is absorbed very poorly from drinking water (Jacobs and Worwood, 1974). This explains why iron deficiency anemia may occur where iron is found in drinking water. To adequately absorb iron, the iron must be reduced to the ferrous state or complexed with an organic or inorganic ligand that can be taken up by the organism across the gut or through the gills. Some components of food (e.g. meat proteins) may enhance iron absorption while other (e.g. oxalates) may inhibit it (Jacobs and Worwood, 1974).

Iron is moderately toxic to plants (toxic effects appear at concentrations between 1 and 100 mg/l in the nutrient solution--Bowen, 1966). The mode of toxic action appears to be binding of adenosine triphosphate (ATP). Iron is only slightly toxic to animals when taken orally (LC_{50} between 100 and 1,000 µg/g body weight--Bowen, 1966). In mammals, toxicity of iron is rarely encountered. One current problem is the consumption by children of large numbers of adult iron pills that can result in overdose and death. Very high levels of iron in the diet of livestock can cause phosphorus deficiencies and high concentrations in water cause palatability problems (NAS/NAE, 1973). Historically the most sensitive use to be protected by an iron limitation has been water supply. Concentrations above 300 µg/l may result in accumulations in distribution systems, staining of basins and toilet bowls and spotting of laundry. The Subcommittee reviewed the recommendation in Water Quality Criteria 1972 (NAS/NAE, 1973) and concluded that on the basis of user preference and because the defined treatment process can remove insoluble iron but may not remove soluble iron, 300 µg/l soluble iron should not be exceeded in public water supply sources. In Canada, the acceptable limit for dissolved iron in drinking water is 300 µg/l while the objective is less than 50 µg/l (DNHW, 1972).

The harmful effects of iron in aquatic systems are also related to its aqueous chemistry. Iron can cause alterations of the physical characteristics of streams and lakes by precipitation. Loose flocs of iron hydroxides can cause turbidity, reduced light penetration and reduced primary productivity. Presumably, reproduction of aquatic organisms that rely on visual behaviour cues would also be affected. High concentrations of precipitated iron flocs can also smother bottom fauna and, with time, consolidate to form pavement-like areas on the bottom of streams or lakes (U.S. E.P.A., draft, 1975). Obviously, organisms that burrow eggs or that require loose gravel through which oxygenated water can flow will not survive.

The direct detrimental effects of iron on aquatic organisms result from two separate mechanisms: (1) reduced pH due to hydrolysis of iron salts; and (2) effects of ferric hydroxide precipitate at neutral and basic pH's. The effects of low pH were discussed in Appendix A of the Water Quality Board Report to the IJC in 1974, and an objective was recommended.

Tolerance of iron by algae varies with the species. McLean (1974) only associated Cladophora glomerata with waters containing less than 450 µg/l of iron. Stigeoclonium tenue, however, was found in waters with iron concentrations up to 10,000 µg/l.

Invertebrates are fairly sensitive to iron. The 96-hour LC_{50}'s of iron for a stonefly (Acroneuria lycorius), a mayfly (Ephemerella subvaria), and a caddisfly (Hydropsyche bettoni) were > 16,000, ≃ 320 and > 16,000 µg/l, respectively (Warnick and Bell, 1969). Mayflies were more sensitive than other species to iron and this was also true for eight other metals (Warnick and Bell, 1969). Reproduction of Daphnia magna was reduced to 50 percent and 16 percent of control values by 5,200 and 4,380 µg/l, respectively and the 3-week LC_{50} was 5,900 µg/l (Biesinger and Christensen, 1972). The safe concentration for reproduction and growth of Gammarus minus was less than 3,000 µg/l (Sykora et al., 1975).

Fish are also affected by iron. Smith, et al. (1973) observed reduced survival of fry, and a 50 percent reduction in egg hatchability of fathead minnows (Pimephales promelas) at 1,500 µg/l. However, brook trout (Salvelinus fontinalis) egg hatchability was affected only at concentrations above 12,000 µg/l (Sykora et al., 1975). The safe concentration for brook trout, based on mortality of juveniles, was between 7,500 and 12,520 µg/l.

While there is considerable variation in acceptable concentrations, there is general agreement that the hydroxide precipitate interferes with respiration through the chorion in fish eggs and impairs gill function of gill-breathing organisms by occlusion of the lamellae. Warnick and Bell (1969) and Smith et al. (1973) have identified these effects at or near 1,000 µg/l. Sykora et al. (1972) found that the fine floc formed at low iron concentrations (1,500 µg/l) caused more damage to fathead minnow eggs than the large floc formed at high iron concentrations. Therefore, in order to protect all forms of aquatic organisms, formation of ferric hydroxide floc should be limited. A ferric hydroxide floc should not form at total iron concentrations less than 300 µg/l (NAS/NAE, 1973).

The water quality objective for iron, as specified in Annex 1, paragraph 1(f) of the Canada-U.S. Agreement on the Great Lakes is, "levels should not exceed 0.3 milligrams per litre". Since this was based on filtration of raw water to protect public water supplies, it did not provide an objective for the iron hydroxide floc. Therefore, to protect raw water for public water supplies, and to prevent harm to aquatic biota, it is recommended that the objective for total iron in unfiltered water be 300 µg/l.

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LEAD

RECOMMENDATION

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

> *Concentrations of total lead in an unfiltered water sample should not exceed 10 micrograms per litre in Lake Superior, 20 micrograms per litre in Lake Huron and 25 micrograms per litre in all remaining Great Lakes to protect aquatic life.*

RATIONALE

Lead is released to aquatic ecosystems from the production and use of lead in gasolines, paints, glazes, pipes, roofing materials and ammunition (especially shotgun pellets). Lead is also released during metal mining and refining processes, recycling of used lead products, burning of fuels and recycling or disposal of used motor oils (NRCC, 1973).

Lead generally occurs in very low concentrations in water due to its low solubility. Since carbonate, hydroxide, phosphate, chloride, etc. form insoluble salts with lead, any dissolved lead can be converted to an insoluble form and precipitated to the sediments. In Lake Ontario water, for example, it has been found that at concentrations above 100 mg/l lead, more than 98% is precipitated after 24 hours. Above 10 mg/l, 70% is precipitated and above 1 mg/l, 10% is precipitated. The precipitate does not appear to redissolve upon agitation (Hodson, unpublished data). Below 1 mg/l, lead may be in an insoluble form but not precipitated, perhaps due to particle size. The proportion in an undissolved form varies with water hardness (Davies and Everhart, 1973). At a hardness of 24.0 mg/l, alkalinity of 22.8 mg/l and pH of 6.91, about 100% of lead below 100 µg/l is in a dissolved form. In water with a hardness of 353 mg/l, alkalinity of 243 mg/l and pH of about 7.9, dissolved lead was only 2% of a total of 3,240 µg/l. As the total concentration decreased, dissolved lead increased to 27% of a total of 40 µg/l lead (Davies and Everhart, 1973). Lead solubility is strongly influenced by pH, and above pH 8.0 the solubility is less than 10 µg/l, regardless of alkalinity (Hem and Durum, 1973).

Modal lead concentrations in the Upper Great Lakes waters are less than 1.0 µg/l offshore, and 95% of all samples contain less than 3.0 µg/l (Table 2). A t water intakes, mean lead concentrations are as high as 34 µg/l with maxima at 55 µg/l or less (Table 3). The higher inshore concentrations probably reflect local inputs to the lakes.

Lead is not essential for plant and animal growth and is, in fact, quite toxic. Bowen (1966) has rated lead as being very toxic to plants, i.e. toxic effects may be seen below 1 mg/l in the nutrient solution.

Lead shot is also toxic to wildlife. Poisoning of diving and dabbling ducks, as well as swans and geese is a major problem of wetlands management (NRCC, 1973). Birds may die by feeding off bottom material heavily contaminated with lead shot from hunting. One lead pellet ingested by a mallard can cause elevated blood lead levels for up to three months (Dieter and Finley, 1975). The same exposure also caused marked changes in enzyme activity of brain and liver tissue (Dieter and Finley, 1975). The lethal dose of lead pellets is estimated as 5-6 for a mallard and 15-25 for a Canada Goose (NRCC, 1973) and toxicity varies with diet.

Lead toxicity to mammalian wildlife has not been reported but some domestic animals and humans are quite susceptible to lead. Domestic animals are exposed through ingestion of solid waste (e.g. lead-acid batteries) or contaminated drinking water. Chronic toxic effects include digestive problems, renal damage, neural damage and eventually death. Embryotoxicity due to transplacental lead transfer has been observed but teratogenicity has not been proven conclusively (NRCC, 1973). Many of these results are from experimental poisonings. The recommendation for lead in water for livestock in the U.S. is 100 µg/l (NAS/NAE, 1973).

Man is exposed to lead through food, water and air. Sources of lead include burning of fossil fuels, smoking, drinking water, non-food items such as paint chips, illicit liquor, containers improperly glazed with lead silicates and industrial operations (NRCC, 1973). Lead poisoning or plumbism, has three aspects: (1) mild or severe dysfunction of the alimentary tract; (2) neuromuscular atrophy; and (3) encephalopathy. Therefore, it has been recommended that total lead intake be limited to 0.6 mg/day by adults (NRCC, 1973; NAS/NAE, 1973) and 0.3 mg/day by children (NRCC, 1973). The recommendation for lead in drinking water in the U.S. is 50 µg/l (NAS/NAE, 1973) while in Canada, the maximum permissible limit is 50 µg/l, less than 50 µg/l is acceptable, and the objective is "not detectable" (DNHW, 1969).

Lead appears to be relatively non-toxic to algae. Concentrations reducing growth as determined by cell numbers, CO_2 fixation, chlorophyll production, etc. are generally between 1 and 100 mg/l and occasionally as high as 1,000 mg/l (Wong et al., in preparation). Toxicity varies considerably between species and between growth media. The growth media factor is of considerable importance since toxicity of lead in natural waters is much greater than in artificial media. Growth of Ankistrodesmus falcatus, a green alga of the Great Lakes, was reduced 50% by about 10,000 µg/l lead in Chu 10 medium. In Lake Ontario water, a similar effect was seen between 10 and 100 µg/l (Wong et al., in preparation). Temperature must also be considered, since toxicity increases with temperature (Wong et al., in preparation) and most laboratory studies are conducted at 20 C.

Daphnia magna reproduction was inhibited by 30 µg/l lead (Biesinger and Christensen, 1972). Conditioned behaviour of goldfish (Carassius auratus) was affected by 70 µg/l lead (Weir and Hine, 1970) but the importance of this change is unknown. Growth of brook trout (Salvelinus fontinalis) was reduced by periodic high concentrations of lead between 15,000 and 25,000 µg/l (Dorfman and Whitworth, 1969) while growth of guppies (Lebistes reticulatus) was reduced by continuous exposure to 1,250 µg/l (Crandall and Goodnight, 1962; 1963).

Prolonged lead exposure of rainbow trout (Salmo gairdneri), starting as fingerlings, caused black tails and lordosis (dorso-ventral spinal curvature) plus scoliosis (bilateral spinal curvature) (Davies and Everhart, 1973). These effects are probably due to neural damage and they occurred between 13.3 and 20 µg/l total lead at 27 mg/l hardness and 23 mg/l alkalinity. At 354 mg/l hardness and 243 mg/l alkalinity, the effects occurred between 120 and 360 µg/l total lead. When the results from hard water were expressed as "free" lead as measured by pulse polarography, the effects occurred between 18 and 32 µg/l. Therefore, a safe concentration based on total lead varies considerably with hardness while that based on "free" lead varies only slightly. In soft water, for trout exposed from the egg stage onwards and from parents exposed to lead for one year, the safe-unsafe range was 6-12 µg/l.

Interpolating from Davies and Everhart's (1973) results, safe-unsafe concentration ranges for total lead in the Great Lakes are as follows:

	Hardness (mg/l)	Alkalinity (mg/l)	Safe-unsafe range based on hardness (µg/l of lead)	Safe-unsafe range based on alkalinity (µg/l of lead)
Lake Superior	44	41	15 - 24	16 - 25
Lake Huron	94	75	21 - 37	22 - 38
Lake Michigan	119	--	25 - 46	--
Lake Erie	123	91	25 - 46	26 - 48
Lake Ontario	135	90	27 - 52	26 - 48

These results have been confirmed by Goettl et al. (1973) using the same dilution water. They found that lordosis plus scoliosis developed in young rainbow trout at lead concentrations between 8.0 and 14.0 µg/l. A third study of brook trout in water of 44 mg/l hardness gave similar results between 58 and 119 µg/l total lead (Holcombe et al., unpub.man.) On a dissolved basis, this represented 39 and 84 µg/l. It would appear that brook trout are not as sensitive as rainbow trout.

Some lead accumulation occurs in aquatic biota. Phytoplankton accumulate large quantities, perhaps due to adsorption by the relatively large surface areas of algal cells, or to ion exchange (Shukla and Leland, 1973). Leland and McNurney (1973) showed that concentrations of lead were always highest in periphyton of streams and decreased with increasing trophic level. Herbivorous fish had higher concentrations of lead than did carnivorous fish. All concentrations of lead in fish were less than 5.0 µg/g.

Lead concentrations in fillets of Great Lakes fish were found to be uniformly less than 0.5 µg/g, the detection limit, regardless of species or sample location (Uthe and Bligh, 1971). However, in a more recent survey, Brown and Chow (1975) reported that fish from Baie du Dore, Lake Huron, contained 0.19 µg/g lead in muscle while those from Toronto harbour contained 1.78 µg/g. Since only the values from Toronto Harbour appear elevated, muscle lead concentrations may reflect local contamination. Higher concentrations of lead occur in other organs of fish. In trout from a stream, concentrations of lead were higher in bone than in liver or gills (Pagenkopf and Neuman, 1974). In addition, there was a significant difference in lead content of bone between fish from a hatchery and fish from a river containing 2.65-2.93 µg/l lead, twice as much as in hatchery water. Lead may also occur in blood and accumulate in kidney tissue (Hodson, unpublished data). The significance of these residues to fish health has not yet been determined.

The criteria for lead for aquatic biota require a more stringent objective than for drinking water. Therefore, to account for the variation with water hardness of the response of rainbow trout to total lead in water, the objective for total lead is recommended as 10 µg/l in Lake Superior, 20 µg/l in Lake Huron and 25 µg/l in all other lakes.

Since lead may be methylated to tetramethyl lead by lake sediments (Wong et al., 1975), these objectives should be re-evaluated when the significance of methylation is defined.

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MERCURY

RECOMMENDATION

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

> *The concentration of total mercury in filtered water should not exceed 0.2 micrograms per litre nor should the concentration of total mercury in whole fish exceed 0.5 micrograms per gram (wet weight basis) for the protection of aquatic life and fish-consuming birds.*

RATIONALE

The biologically significant form of mercury is methylmercury. The bulk of the mercury found in fresh water fish occurs in the form of methylmercury (Johnels et al. 1967; Kamps et al. 1972).

Various forms of mercury may be methylated by at least two mechanisms (Wood et al., 1968; Ladner, 1971). The extent and rates of methylation are affected by many factors, among them are: concentration of mercury ions, availability of mercury ions, growth rate or metabolic activity of the methylating organisms, temperature, and pH (Bisogni and Lawrence, 1975). Methylmercury may also be demethylated by bacteria in sediments (Spangler et al., 1973). Thus the amount of methylmercury found in the environment at any one time is dependent on the combined reaction kinetics of the methylating and the demethylating processes. As a consequence, the combination of the available mercury concentrations and the operations of both transformation processes are significant. Since fish concentrate methylmercury preferentially over other forms of mercury, and since they excrete methylmercury very slowly, they provide a good indicator of long-term trends of the net methylation rate in an environment. Crayfish also accumulate significant amounts of methylmercury (Armstrong and Hamilton, 1973). Because of their shorter life cycles, they may be suitable to measure intermediate term trends in the net methylation rate in an aquatic environment.

The present administrative guideline for fish for human consumption promulgated by the U.S. Food and Drug Administration as well as the Canadian Food and Drug Directorate is 0.5 µg/g mercury in edible portions of fish. Natural background concentrations of mercury in fish are generally below this level, but may locally exceed it in some species. There is no evidence that concentrations of 0.5 µg/g in fish have any effect on them. Concentrations of mercury in fish that have been killed by chronic exposure to methylmercury ranged from 9.5 to 23.5 µg/g (McKim et al., 1975).

Table 4

MERCURY TOXICITY STUDIES

Organism	Compound	Effect	Lowest Conc. Producing Effect	Highest No. Effect Conc.	Remarks	Reference
Gammarus sp.	Hg^{++}	24hr LC_{50}	90 µg/l			Rehwoldt *et al.*
		96hr LC_{50}	10 µg/l			
Nais sp.	Hg^{++}	24hr LC_{50}	1900 µg/l			
		96hr LC_{50}	1000 µg/l			
Caddis fly	Hg^{++}	24hr LC_{50}	5600 µg/l			
		96hr LC_{50}	1200 µg/l			
Damsel fly	Hg^{++}	24hr LC_{50}	3200 µg/l			
		96hr LC_{50}	1200 µg/l			
Chironomus sp.	Hg^{++}	24hr LC_{50}	60 µg/l			
		96hr LC_{50}	10 µg/l			
Amnicola sp.	Hg^{++}	24hr LC_{50}	1100 µg/l			
		96hr LC_{50}	80 µg/l			
Brook trout embryos	CH_3Hg^+	GOT(decreased)	1.03 µg/l	0.08 µg/l	adults exposed 7 mo. before spawning; offspring maintained at same conc.	Christensen
alevins	CH_3Hg^+	GOT(enhanced)	0.93 µg/l	0.08 µg/l		
Rainbow trout	CH_3Hg^+	Decreased Hematocrit	10 µg/l		12 weeks exposure	O'Connor & Fromm
		Plasma electolytes		10 µg/l	"	
		in vitro O_2 metabol.		10 µg/l	"	
Brook trout	CH_3Hg^+	Cough response	3 µg/l		5 day exposure	Drummond *et al.*
Zebrafish	Phenyl mercuric acetate	No. eggs spawned	1 µg/l	0.2 µg/l	19-25 day exposures	Kihlstrom *et al.*
		% hatching	0.2 µg/l			
Rainbow trout	Hg^{++}	decreased activity	50 µg/l		4-6 day exposure	Alexander
Brook trout	CH_3Hg^+	deformities, deaths in	0.93 µg/l	0.29 µg/l	3 generation exposure	McKim *et al.*

Table 4 cont'd.

Organism	Compound	Effect	Lowest Conc. Producing Effect	Highest No. Effect Conc.	Remarks	Reference
Cat	CH_3Hg^+	C.N.S. deaths	0.25 mg/kg/ day		55-96 feeding of synthetic or "natural" CH_3Hg^+	Charbonneau et al.
Japanese quail	$HgCl_2$	Egg shell thinning	1 µg/g (diet)	2 µg/g (diet)		Stoewsand et al.
Mallard	N-(ethyl mercury) -p-toluene-sulfonanilide	Egg shell thinning		200 µg/g	85 day exposure (contains 3.1% Hg)	Haegele et al.
American kestrel	CH_3Hg^+	Egg shell thinning		10 µg/g (diet)	3 months exposure	Peakall & Lincer
Ring dove	CH_3Hg^+	Egg shell thinning		10 µg/g	intramuscular	"
		decreased egg laying	10 µg/g		"	"
Mallard	CH_3Hg^+	Decreased hatchling survival	3 µg/g (diet)	0.5 µg/g(diet)	21 week exposure	Heinz
Mallard duckling	CH_3Hg^+	enhanced avoidance response	0.5 µg/g (diet)		hens fed prior to and during reproductive phase	Heinz

It is nearly impossible to correlate environmental concentrations of total mercury in unfiltered water with concentrations of methylmercury which accumulate in fish. There appear to be several reasons for this: in aquatic ecosystems the vast majority of the total mercury is located in the sediments, where the highest concentration is associated with the smallest particles (Armstrong and Hamilton, 1973 and Walter and Wolery, 1974). The mercury associated with these small particles in the water sample would be included in unfiltered samples so that the turbidity of a sample significantly affects the mercury determination. The biological availability of mercury associated with these samples is probably significantly lower than that of any methylmercury in solution. In addition to mercury compounds adsorbed onto or incorporated into particles, an unfiltered water sample will contain mercury compounds chelated by dissolved organic substances such as fulvic acids (Andren and Harriss, 1975), and dissolved mercury compounds. The proportion of methylmercury in this complex mixture is probably variable, and is not readily determined by presently available techniques. Indirect evidence indicates that the amount of methylmercury in water constitutes a minor proportion of the total mercury content in unfiltered samples. Experimental exposure of brook trout to 0.03 µg/l of methylmercury has resulted in an accumulation of 0.96 µg/g after 239 days of exposure (McKim et al., 1975). Equilibrium concentrations were not reached during this exposure and were estimated to be significantly higher (>3 µg/g) by Hartung (1975). However, background levels of total mercury in water have been reported to range from 0.05 to 0.1 µg/l (N.A.S. Water Qual. Criteria 1972), and these have been associated with concentrations of 0.01 to 0.2 µg/g mercury in fish. Thus there is a significant discrepancy between bioaccumulation data derived from experimental exposures to methylmercury when compared with those derived from experimental data. As a consequence it must be concluded that measurements of total mercury in unfiltered water have only marginal usefulness in deriving environmental quality criteria, and therefore the measurement of mercury accumulated in biological organisms represents a significantly more persuasive criterion.

A series of toxicity studies is summarized in Table 4. It demonstrates that most organic mercury compounds are more toxic than inorganic mercury salts. No effects were noted in a three generation exposure of brook trout to 0.29 µg/l methylmercury. A slight reduction in the hatchability of eggs of zebrafish was noted at 0.2 µg/l. However, while this level should protect aquatic life, it will result in accumulations of methylmercury in aquatic life in excess of 0.5 µg/g. For the purpose of setting an objective to protect aquatic life, the total amount of mercury in filtered water samples is arbitrarily considered to be methylmercury. Concentrations of 0.2 µg/l of total mercury in filtered water should therefore protect aquatic life with a more than adequate safety margin.

Protection of organisms which consume aquatic life cannot be based on water concentrations, but must be based on an evaluation of the amounts of mercury accumulated in aquatic organisms.

On Lake St. Clair in 1970, great blue herons were found with mercury levels up to 23 µg/g in their flesh, and terns up to 7.5 µg/g in their flesh. Fish recovered from their stomachs contained up to 3.8 µg/g mercury (Dustman et al., 1972). No mortalities or population effects were noted in these species. Keith and Gruchy (1971) also reported finding gulls with elevated mercury residues in their eggs without finding effects on reproduction. The levels found in these instances are close to or identical to levels associated with mercury poisoning in some species of seed eating birds. It is therefore evident that species differences exist, and at least some fish-eating birds appear to be more resistant than some seed eating species.

Table 4 also lists the effects of feeding methylmercury to birds. Eggshell thinning was reported to occur in one study in Japanese quail at 1 µg/g of mercuric chloride in the diet. However, studies with organic mercury including methylmercury have not confirmed this in other species, even at higher dose levels. The most sensitive effects found, have been effects of hatchling survival in mallards at 3 µg/g, but not at 0.5 µg/g. The avoidance response of ducklings was enhanced slightly at 0.5 µg/g methylmercury fed to ducks prior to and during the reproductive phase. Since this effect was slight and may not be harmful, it is likely that the safe level for methylmercury in the diet of birds is close to 0.5 µg/g.

Therefore, fish-eating birds should be protected if the concentration of total mercury in whole fish does not exceed 0.5 µg/g. Since not all species of fish accumulate mercury equally, this provides an additional margin of safety. Also, since concentrations of 0.5 µg/g in fish produce no deleterious effects to fish, this limitation assures long-term protection of fish. Therefore, the simultaneous application of the proposed objectives for water and for bioaccumulated mercury in fish should protect aquatic life as well as the consumers of aquatic life.

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NICKEL

RECOMMENDATION

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

Concentrations of total nickel in an unfiltered water sample should not exceed 25 micrograms per litre to protect aquatic life.

RATIONALE

Nickel is both produced and used on a large scale in the Great Lakes basin. Production is centered in the Sudbury area and use is centred in the lower Great Lakes. Nickel is used primarily in metallurgy, metal fabricating and production of nickel pigments. The total used in the Great Lakes Basin was about 4.5 million pounds in 1968. Nickel enters the waters of the Great Lakes directly or indirectly from atmospheric inputs due to burning of fossil fuels, processing of nickel ores, waste incineration and possibly from gasoline to which nickel is added (Fenwick, 1972). Direct inputs to water may occur from manufacture of nickel pigments, nickel containing alloys, or nickel-plated metal products. Nickel salts are quite soluble but occur naturally only at very low concentrations (Fenwick, 1972).

Modal nickel concentrations offshore in the upper Great Lakes are less than 5.0 µg/l. Nickel is often not detectable in lake water and 95% of samples are less than 6.0 µg/l (Table 2). In water intakes, nickel concentrations are higher, with means as high as 11 µg/l and individual samples as high as 28 µg/l. However, nickel is often not detectable in water intakes (Table 3). There is some evidence for nickel contamination of Georgian Bay, perhaps due to fallout in the Sudbury Watershed from nickel smelting operations (CCIW, Star File).

Nickel has not been identified as a nutrient or essential element for plants and animals (Bowen, 1966; Underwood, 1971). Therefore, the principal biological activity of nickel is as a toxicant. Nickel toxicity to wildlife has not been reported but toxicity to man and experimental mammals has been demonstrated. The principal toxic actions are dermatitis following exposure to nickel in plating solutions and induction of lung cancer after chronic inhalation (Smith, 1972). Oral toxicity of nickel is extremely low, since concentrations greater than 1,000 µg/g in food are required to reduce growth of rats and mice (Underwood, 1971). There are no drinking water or livestock water criteria for nickel (NAS/NAE, 1973; DNHW, 1969).

Nickel is classified as very toxic to plants by Bowen (1966) (toxic effects observed at concentrations below 1 mg/l in a nutrient solution). However, some fungi and plants are adapted to grow in high concentrations (Bowen, 1966). This tolerance has also been observed in algal populations. Growth of "normal" Scenedesmus was inhibited 100 per cent by 500 µg/l nickel while 1,500 µg/l were required to produce the same effect in "tolerant" Scenedesmus (Stokes et al., 1973). These algae had originated from lakes in the Sudbury region contaminated by nickel. Other species of algae appear less sensitive, with toxic effects being observed at 1,500-10,000 µg/l (Wong, 1975). It would appear that algae are less sensitive than terrestrial plants according to Bowen's (1966) definition. While nickel does not appear to be overly toxic to algae by itself, recognition must be given to the synergistic effect of nickel on copper toxicity to algae (Stokes and Hutchinson, 1975).

Acute nickel toxicity to invertebrates varies with the species. The 96-hr. LC_{50}'s for a stonefly (Acroneuria sp.), a mayfly (Ephemerella subvaria) and a caddisfly (Hydropsyche bettoni) were 33,500, 4,000 and >14,000 µg/l, respectively, (Warnick and Bell, 1969). The 48-hour LC_{50} for Daphnia magna in Lake Superior water was 1,120 µg/l in the presence of food and 510 µg/l in the absence of food (Biesinger and Christensen, 1972). The 64-hour EC_{50} for immobilization of Daphnia magna in Lake Erie water was >700 µg/l (Anderson, 1948). Chronic three-week nickel exposures to Daphnia magna caused 50% mortality at 130 µg/l and 50% and 16% reproductive impairment at 95 and 30 µg/l, respectively (Biesinger and Christensen, 1972).

Acute nickel toxicity to fish is less than that of copper or zinc. The 48-hour LC_{50} for rainbow trout is 32,000 µg/l while those for copper and zinc were 750 and 4,000 µg/l, respectively (Brown and Dalton, 1970). The 96-hour LC_{50} for fathead minnows is 2,500-2,800 µg/l at a hardness of 210 mg/l (Pickering, 1974). In static bioassays, the 96-hour LC_{50} for fathead minnows was 5,000 µg/l at 20 mg/l hardness and 43,000 µg/l at 360 mg/l hardness (Pickering and Henderson, 1966).

In chronic exposures, reproduction of fathead minnows was unaffected by 380 µg/l nickel but 730 µg/l reduced egg production and hatchability of eggs (Pickering, 1974).

The nickel content of fish is normally quite low. Uthe and Bligh (1971) found nickel concentrations uniformly below 0.2 µg/g in fillets of Great Lakes fish. They noted no variation with location or species of fish. In a survey of an Illinois River ecosystem, nickel concentrations decreased from the sediments (27 µg/g) to tubificid worms (11 µg/g) to clams (1.5 µg/g) to omnivorous fishes (0.17 µg/g) to carnivorous fishes (0.12 µg/g) to water (2 µg/l) (Mathis and Cummings, 1973). Therefore, the concentrations decreased with increasing trophic level. Kariya et al. (1965) showed that, at lethal concentrations of nickel in water, nickel levels in fish increased from not detectable to as high as 70 µg/g.

However, at low, but still lethal concentrations, nickel was not detectable. Therefore, there are no concentrations of nickel that have been associated with sublethal toxicity. Since a nickel oral toxicity to fish consumers is not defined, no objective for nickel concentrations in fish is recommended.

For the protection of aquatic life, an objective of 25 µg/l is recommended for nickel in water.

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SELENIUM

RECOMMENDATION

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

> *Concentrations of total selenium in an unfiltered water sample should not exceed 10 micrograms per litre to protect raw water for public water supplies.*

NOTE: The effect of high dietary selenium concentrations on fish-eating birds and wildlife is unknown. Based on the response of laboratory mammals, concentrations of selenium approaching 3 µg/g, wet weight, in whole fish should be regarded with concern.

RATIONALE

Selenium is a common element appearing in the earth's crust at approximately 7×10^{-5}%. It is present largely as heavy metal selenides (together with sulphide minerals) but also occurs as selenates and selenites. In soils, excluding seleniferous soils not normally found in the Great Lakes region, it has been variously reported to be present at levels ranging from 0.1 µg/g to less than 2 µg/g (Cooper et al., 1974). Elevated levels of selenium are found in some sedimentary rock formations and their derived soils in central areas of Canada and the United States. There are no known mining activities for selenium and its production comes mostly as a by-product of copper and lead refining.

Commercial use of selenium was about 500 metric tons per year in 1968, mostly in the elemental form as red crystals or grey powder. It is used in electronics for rectifiers, photocells, and xerography. It is also used in steel and in pigments for paints, glass, and ceramics (Cooper, 1967; Lymburner and Knoll, 1973).

Selenium is usually present in water as selenate and selenite; the elemental form is insoluble but may be carried in suspension. Weathering of rocks and soil erosion is a major source of selenium in water. On a world basis, approximately 10,000 metric tons yearly are weathered and carried downstream to the sea. Of this, 140 tons is in solution but only 16 tons remains dissolved in the sea. The rest of it goes into sediments (Schroeder, 1974). The burning of fossil fuels is another source of soluble selenium. Analysis of coal and bottom and fly ash from a single burner has turned up levels of 2 µg/g, 3.4 µg/g and 41.3 µg/g, respectively (Lymburner and Knoll, 1973). Man's burning of fossil fuel puts about 450 tons per year of selenium (SeO_2) into the atmosphere, about 4.5% of the amount eroded naturally (Schroeder, 1974).

Disposal of waste containing selenium could be another source, although levels in effluents seem to be low. Sewage in California (both raw and treated) was found to have only 10 to 60 µg/l of selenium, except for a high value of 280 µg/l in an industrial area (Feldman, 1974).

Concentrations in water are usually low. The literature has been reviewed in several places (e.g. NAS/NAE, 1973), but many of the older estimates are probably too high because of the limitations of chemical methods. Most uncontaminated surface waters have less than 50 µg/l of selenium, and most drinking waters contain less than 10 µg/l (APHA et al., 1971). Surface waters in a province of Germany averaged 4 µg/l (Heide and Schubert, 1960). The normal concentration in sea water is only 0.4 µg/l (Chau and Riley, 1965). Even seepages from seleniferous areas do not contain more than 500 µg/l and this content is lost when the seepages empty into ponds or lakes, apparently by coprecipitation with ferric hydroxide (APHA et al., 1971). Selenium concentrations in the Great Lakes are below 1 µg/l offshore and mean concentrations are 0.2 µg/l or less (Table 2).

Lake sediments seem to act as reservoirs or sinks; in the northern United States they contained from 1.0 to 3.5 µg/g dry weight of selenium, considerably more than the usual concentration in soils (Wiersma and Lee, 1971). Small experimental ecosystem experiments showed that of the total amount of selenium in rain which fell on soil, 75% stayed in soil and 25% ran off into an aquatic system. Thirty-six percent of the amount of selenium that entered the aquatic system ended in the sediments and most of the rest was in the biota (Huckabee and Blaylock, 1974).

Deficiency of selenium in the soil and in grass eaten by livestock, leads to "white muscle disease". Dietary needs of livestock are in the vicinity of 0.1 to 0.2 mg/day (NAS/NAE, 1973) whereas the daily selenium requirement of humans has not been accurately determined. It would appear to be in the range of 0.1 to 0.2 mg/day (Levander, 1975), an amount normally found in an adequate diet (NAS/NAE, 1973).

Selenium poisoning of livestock has been divided into two classes: the acute type called blind staggers and the chronic type called alkali disease. The acute type is associated with ingestion of highly seleniferous plants containing 1,000 µg/g or more of selenium whereas the chronic type is associated with grains and plants which contain 5 to 20 µg/g of selenium (Moxon, 1958). The extensive literature on natural poisoning of livestock from selenium in their food plants agrees, in general, that 5 µg/g or more can lead to death in the herbivore, and that such levels in plants result from soil concentrations in the range 0.5 to 6 µg/g (National Technical Advisory Committee, 1968; NAS/NAE, 1973; McKee and Wolf, 1963). Also, a diet containing 3 µg/g of selenium in selenite form, in a lifetime study killed rates (Schroeder, 1967). The usual chronic effects in mammals may include weakness, visual impairment, paralysis, damage to heart, liver and viscera, stiff joints, and loss of hair and hooves. Additional symptoms in humans are marked pallour, red tainting of fingers, teeth and hair, dental caries, debility, depression

and irritation of nose and throat. In humans, overdoses resulting in acute toxicity may be characterized by nervousness, vomiting, cough, dyspnea, convulsions, abdominal pain, diarrhea, hypotension and respiratory failure (Schroeder, 1974; NAS/NAE, 1973; Rodier, 1971). No recognized cases of non-industrial chronic selenium poisoning in man have been reported (Sakurai and Tsuchiya, 1975).

The carcinogenic potential of selenium has been widely investigated (Schroeder, 1974). Recent critical evaluations made of these early studies leads to the conclusion that there are insufficient high quality data to allow evaluation of the carcinogenicity of selenium compounds (WHO, 1975; Palmer and Olsen, 1974). No suggestion that selenium is carcinogenic in man can be found in the available data (WHO, 1975).

Antagonism between toxicity of selenium and other metals has been pointed out; Levander (1973) reviewed the action of arsenic in counteracting selenium toxicity. Several cases in which cadmium poisoning is decreased by selenium are listed by Pakkala et al. (1972) and Anonymous (1972). The action against mercury toxicity has been mentioned by Koeman et al. (1973). There are other aspects such as the interrelationship with vitamin E and possible teratogenic effects (Anonymous, 1972).

Toxicity due to selenium in drinking water is not common, probably because concentrations in water are generally low, and cases of toxicity to livestock are usually related to intake with food. However, a level of 9,000 µg/l in well water resulted in human poisoning in 3 months (Beath, 1962).

Water Quality Criteria 1972 (NAS/NAE, 1973) suggests a limit of 10 µg/l of total selenium in drinking water assuming that two litres of water are ingested per person per day. This recommendation is also accepted by WHO, U.S.A., Canada and U.S.S.R. whereas some European countries such as France use a 50 µg/l limit on selenium in potable water.

The U.S. National Academy of Sciences (NAS/NAE, 1973) recommends that the upper limit for selenium in water given to livestock be 50 µg/l. This figure is also used by the Ontario Ministry of the Environment (1974).

Bowen (1966) has described selenium as moderately toxic to plants (toxic effects at concentrations between 1 and 100 mg/l in the nutrient solution). This appears to apply to freshwater algae as well. The concentrations of selenite causing 95% growth inhibition of Anabaena variabilis and Anacystis nidulans were 20 and 70 mg/l, respectively (Kumar and Prakash, 1971). Selenate produced the same results with these species at 30 and 50 mg/l, respectively. Kumar (1964) showed that growth of Anacystis nidulans, a bluegreen alga was also completely inhibited by 20 mg/l of selenate. However, a culture of this alga at increasing concentrations of selenate, over several generations, produced a tolerant strain that could grow in 250 mg/l of selenate. Scenedesmus sp. however,

was more sensitive since 2.5 mg/l was lethal (Bringman and Kuhn, 1959).

Little information is available on the toxicity of selenium to invertebrates, but Daphnia sp. has been found to be as sensitive as Scenedesmus sp. with a lethal threshold of 2.5 mg/l. (Bringman and Kuhn, 1959).

Niimi and LaHam (1975, 1976) have published the most comprehensive studies to date on toxicity of selenium to fish. Acute studies (Niimi and LaHam, 1976) indicated that lethality of selenium to zebrafish larvae (Brachydanio rerio) varied with the selenium salt used. The 96-hour and 10-day LC_{50}'s (Table 5) indicate that selenate salts are less toxic than selenite salts.

Table 5

Acute toxicity of selenium salts to zebrafish larvae (from Niimi and LaHam, 1976).

	96-hr. LC_{50} (mg/l)	10-day LC_{50} (mg/l)
selenium dioxide	20	5
sodium selenite	23	4
potassium selenite	15	≃2
sodium selenate	82	40
potassium selenate	81	50

These salts are the most common forms normally occurring in freshwaters. The selenides, selenomethionine and selenocystine, were also shown to be toxic. Selenocystine was about as toxic as the selenates and selenomethionine was more toxic. Reliable LC_{50}'s for selenides could not be calculated, however, due to a loss of compounds from the solution perhaps due to biological action. Biological action was also a problem in early experiments with inorganic compounds. It was noticed that bacterial slimes in test containers could produce a highly toxic, unidentified organic selenium compound. Daily cleaning alleviated the problem but it suggested that hazardous transformations of inorganic to organic selenium compounds might occur in aquatic systems.

Niimi and LaHam (1975) have also studied the toxicity of selenium dioxide to zebrafish embryos. Embryos were quite resistant and concentrations up to 10 mg/l had no effect on hatching. This was probably due to the extremely low permeability of the egg membrane. Larvae, by

comparison, were quite sensitive and high mortality was observed at concentrations as low as 3 mg/l after 10 days. No effect was observed at 1 mg/l.

The acute toxicity of selenium to goldfish is similar to that of zebrafish. In very soft water, the 5day LC_{50} of sodium selenite for goldfish was 10 mg/l (Ellis et al., 1937). Other work by Ellis et al. (1937) showed that 2 mg/l of the same salt killed goldfish in 1846 days. Weir and Hine (1970) found a 7day LC_{50} for goldfish of 12 mg/l in water of 50 mg/l $CaCO_3$. Using a conditioned avoidance response as an index, Weir and Hine (1970) also found that 0.25 mg/l could significantly affect learning behaviour as compared to controls. A concentration of 0.15 mg/l had no significant effect.

Selenium dioxide was also lethal to six species of fish in 4 days to 2 weeks, at concentrations between 2 and 20 mg/l, (Cardwell et al., no date).

Concentrations of selenium in the tissues of fish range from 0.16 to about 0.6 µg/g, wet weight, in a wide range of locations in fresh and ocean water. This range holds for Canadian dressed fish from industrial and isolated locations (0.17 to 0.38 µg/g, Uthe and Bligh, 1971); for a large series of freshwater fish from New York (0.2 to 0.5 µg/g, Pakkala et al., 1973); for ocean and freshwater fish in Finland (0.2 to 0.58 µg/g, Sandholm et al., 1973); seafoods (about 0.32 to 0.56 µg/g, Morris and Levander, 1970); the edible portion of trout (about 0.28 to 0.68 µg/g, Arthur, 1972); and for samples of marine food fish obtained in Ontario markets (0.16 to 0.4 µg/g, Dr. D. Arthur, Dept. Nutrition, University of Guelph). In a very large series of fish from central Canada, concentrations in muscle sample averaged about 0.26 µg/g, and most of the fish fell in the range mentioned above (Beal, 1974). However, the total range was wider. In the Great Lakes, concentrations of selenium in fish from the North Channel of Lake Huron, Georgian Bay, Lake Erie and Lake Ontario ranged from 0.56-2.00, 0.42-1.15, 0.10-0.75 and 0.06-0.96 µg/g, respectively.

Fish mortality in a Colorado reservoir was reported by Barnhart (1958) as being caused by selenium from bottom deposits which had passed through the food chain to accumulated levels of 300 µg/g. This is the single known case. In a less contaminated aquatic ecosystem, the animals were shown to have higher residues than the plants, but there was no pattern of continuing accumulation. Also, fish from pond culture where the artificial food was low in selenium, contained less selenium than those from a natural system (Sandholm et al., 1973). In an experimental system, Sandholm et al. (1973) also found that Scenedesmus dimorphus could actively concentrate selenomethionine but showed no active or passive uptake of inorganic selenium. Daphnia pulex, however, could absorb selenium from selenite. Fish (Puntius arulius) absorbed selenium principally from food and showed little uptake from inorganic and organic forms in water. Copeland (1970) reported that concentrations of selenium

from Lake Michigan zooplankton were highest downwind of industrialized areas, although this was not reflected in the sediments. Concentrations in the sediments were uniformly less than 0.5 µg/g, whereas, concentrations in zooplankton increased from 1 µg/g in uncontaminated areas to 7 µg/g in contaminated waters. Elimination of selenium by fish has not been studied but there appears to be no correlation of selenium with size, sex or age of fish (Pakkala et al., 1974). Therefore, selenium may be excreted in a similar fashion as determined in humans. A normal human intake of 0.06 to 0.15 mg/day is balanced by an output of 0.03 mg in faeces, 0.05 mg in urine, and 0.08 mg in sweat, air and hair (Schroeder et al., 1970).

A serious cause for concern may exist in the discovery that livers of some seals contain from 46 to 134 µg/g selenium (Koeman et al., 1973). These are much higher than the values of 0.5 to 1.3 µg/g found in the livers of land animals. Also, the single sample of tissue from a northern Canadian beluga whale showed a high level of 14.3 µg/g selenium. The topic is not well understood yet, Koeman et al. (1973) considered that the high selenium might be protective against high mercury residues.

Nevertheless, the possibility exists that fish-eating birds and mammals may be subject to a dangerous accumulation of selenium. The difference between optimal and toxic intake levels in the food is comparatively narrow (25 to 40 times, Hoffman et al., 1973). The fish mortality in Colorado indicates that accumulation can take place.

Since 3 µg selenium per gram of diet is toxic to rats over their lifetime and since the toxicity of selenium to fish-eating birds or wildlife is unknown, any accumulation of selenium in whole fish approaching 3 µg/g wet weight should be regarded with concern.

In summary, the recommendations for selenium in drinking water are more stringent than those for aquatic biota. Therefore, the recommended objective for selenium is 10 µg/l to protect raw drinking water supplies.

Selenium is known to be methylated biologically and Chau et al. (1976, in press) have recently demonstrated methylation of sodium selenite, sodium selenate, selenocystine, selenourea and seleno-DL-methionine by microbial action in lake sediments. All sediments that demonstrated microbial action were capable of methylating selenite and/or selenate. Three compounds, mono, and dimethyl selenide, and an unknown were produced. Since the bacterial action may have produced an unknown selenium compound of high toxicity to fish (Niimi and LaHam, 1976), the selenium objective should be reviewed when the environmental significance of selenium methylation is more completely understood.

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VANADIUM

RECOMMENDATION

Inadequate information on the possible degree of bioaccumulation or chronic effects to aquatic organisms exists to formulate a recommendation at this time.

RATIONALE

Vanadium is a common element, occuring in the earth's crust at approximately 100-200 µg/g (Schroeder, 1970; Vinogradov, 1959) and is present largely as V(III) and V(V) oxide salts (NAS, 1974) with some sulphides. Vanadium also occurs in fossil fuels (10-300 mg/l in oil, 15-35 µg/g in U.S. coal) and it is from these sources that most of the material introduced to the environment by man arises. In crude oil, the element largely remains with the undistilled residues and on burning it ends up as either air-born particulates or as a component of slag. A similar situation would exist for coal. Both modes permit the entry of vanadium to the aquatic ecosystem but, at least for the Great Lakes, the concentrations do not build up to any appreciable extent. In the Great Lakes, the concentrations observed in filtered water were generally less than the quantification limit of 0.5 µg/l and never exceeded 1.2 µg/l (C.C.I.W. 1970-71; Chau et al., 1970). Some minor accumulation in sediments over levels observed in soils has been observed for western Lake Erie (Zubkoff and Carey, 1970).

There is a considerable body of data on vanadium toxicity derived largely from inhalation studies with both humans and mammals. Deleterious effects to humans occur at 0.2 mg/m^3 in air (Zenz and Berg, 1967) but were reversible. The form of the toxicant is important with pentavalent vanadium being most toxic (Roshchin, 1967). Animal studies, reviewed by Faulkner Hudson (1964), indicate a lethal dose of some 20 µg/g body weight for mice and 1-2 µg/g for rabbits. At lower levels, vanadium appears to be readily excreted (Dimond et al., 1963; Jaraczewska, 1963) mainly via the urine. There is no reported indication of mutagenicity (NAS, 1974). The recommended limit for vanadium in drinking water for livestock is 100 µg/l, based on toxicity to chicks and accumulation by mice (NAS/NAE, 1973).

Vanadium toxicity to plants was also reviewed by NAS/NAE (1973). They recommended that, on soils irrigated every year, the maximum concentration of vanadium should be 100 µg/l or less. On soils irrigated for 20 years or less the concentration should be 1,000 µg/l or less.

In summary, there appears to be little data pertaining to aquatic organisms and this deficiency should be rectified. Sampling and analysis for fish and sediments in the Great Lakes might help to answer the question of accumulation and chronic toxicity testing should be undertaken after establishing the presence, if any, of this substance in biological tissue. At present, there is insufficient data to formulate objectives to protect aquatic biota.

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ZINC

RECOMMENDATION

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

Concentrations of total zinc in an unfiltered water sample should not exceed 30 micrograms per litre to protect aquatic life.

RATIONALE

Zinc, in various forms is used in metallurgy, metal fabricating, metal coatings, batteries, paint and varnish, industrial chemicals, rubber, soaps, medicines and pulp and paper production. In 1968, over 1,356 million pounds were used for these purposes in the Great Lakes basin (Fenwick, 1972). Zinc may enter the Great Lakes as a result of these uses in addition to inputs from mining and smelting of zinc ore, corrosion of metallic zinc and fallout from atmospheric contamination resulting from the burning of zinc-containing fossil fuels.

Zinc is quite soluble in water and weathering of rocks containing zinc contribute soluble forms to water (Fenwick, 1972). Offshore in the Great Lakes, modal concentrations of zinc are less than 10 µg/1, and 95% of samples contain less than 40 µg/1 (Table 2). However, the mean zinc concentrations range from 1.8 to 28.2 µg/1. At water intakes, the mean zinc concentrations are generally less than 45 µg/1 except in Lake Erie at Buffalo, where the mean is 178 µg/1. High concentrations have been observed at the St. Mary's River, the outlet of Lake Superior, at Buffalo (Lake Erie) and at Massena (outlet of Lake Ontario) (Table 3). At Buffalo, consistently high values suggest local zinc outputs near the water intake. Because zinc use is so widespread, sample contamination may be a problem.

Zinc is an essential element for both plants and animals. It is a constituent of many metalloenzymes and of several proteins of unknown function (Bowen, 1966). Zinc is necessary for reproduction, growth, formation of DNA and RNA, formation of the eye, and prevention of a fatal skin disease of pigs. It also promotes wound healing and prevents symptoms of poor blood supply in the legs that results from hardening of the arteries (Schroeder, 1974).

Zinc toxicity to land plants is rare and is usually observed on soils enriched with zinc as a result of mining operations (Bowen, 1966). Zinc is relatively non-toxic to man. However, when zinc metal is heated, zinc oxide fumes may be evolved that can cause "brass chills" or "brass founders ague". Direct doses of soluble zinc salts can cause nausea and vomiting (Fenwick, 1972). However, prolonged consumption of water containing up to 40,000 µg/1 zinc has been reported with no harmful effects on

humans (NAS/NAE, 1973). Consequently, the U.S. drinking water recommendation is based on taste and has been set at 5,000 µg/1 (NAS/NAE, 1973). The maximum permissible limit in drinking water in Canada is also 5,000 µg/1 but the objective is less than 1,000 µg/1 (DNHW, 1969).

Concentrations of zinc inhibiting growth of freshwater algae generally range between 1,000 and 10,000 µg/1 (Wong, 1975). However, growth inhibition of more sensitive species such as Oedogonium sp., Cladophora glomerata and Selenastrum capricornutum has occurred at 220, 240 and 700 µg/1, respectively (Whitton, 1970;Barlett et al., 1974).

Aquatic invertebrates are more sensitive to zinc than algae. Daphnia magna exposed to zinc for three weeks exhibited 50% mortality at 158 µg/1 and 50% and 16% inhibition of reproduction at 102 µg/1 and 70 µg/1, respectively (Biesinger and Christensen, 1972). Water hardness and alkalinity were 45.3 and 43.3 mg/1, respectively. In Lake Erie water, with a hardness and alkalinity of 123 and 91 mg/1, respectively, the 64-hour EC_{50} for immobilization of Daphnia magna was less than 150 µg/1 (Anderson, 1948).

Fish are more sensitive to zinc than other aquatic organisms. Sublethal exposures of zinc for fathead minnows in Lake Superior water (hardness 45 mg/1, alkalinity, 42 mg/1) caused reduced egg production during spawning at 180 µg/1. No effect was observed at 30 µg/1 (Brungs, 1969). In similar water, flagfish (Jordanella floridae) were more sensitive than fathead minnows. Eighty percent mortality of larvae of flagfish occurred at 85 µg/1 zinc and only 10% at 51 µg/1. However, if the larvae had been pre-exposed as embryos to the test concentrations of zinc, they were more tolerant of the zinc. Complete mortality occurred at 267 µg/1, 20-30% occurred at 139 µg/1 and 0-20% occurred at 75 µg/1 or less (Spehar, unpublished manuscript). Rainbow trout fry also die at low concentrations. In water of 26 mg/1 hardness and 25 mg/1 alkalinity, unacclimated trout had a 120-hr LC_{50} of 135 µg/1 while those pre-exposed as eggs had an LC_{50} greater than 526 µg/1. Based on lingering mortality of pre-exposed trout, the safe-unsafe concentrations were 135-251 µg/1 (Goettl, et al., 1973). Reproduction of bluegills was affected by zinc. Decreased spawning and complete mortality of fry occurred at 235 µg/1, while no effect was seen at 76 µg/1. Hardness and alkalinity were 51 and 41 mg/1, respectively (Sparks et al., 1972).

Avoidance of zinc may prevent reproduction of Atlantic salmon. In the laboratory, juvenile salmon avoided 54 µg/1 zinc, while in the field; migration of adults was prevented by about 240 µg/1 (Sprague et al., 1965) In the field, there were also 19 µg/1 copper in the water. The higher effective concentration of zinc could be due to the age of the fish or to the interaction between zinc and copper or some other constituent of natural waters. Growth of Phoxinus phoxinus in water with 63 mg/1 alkalinity was reduced at 130 µg/1 zinc but not at 50 µg/1 (Bengtsson, 1974).

Sublethal toxicity to zinc may be enhanced when in combination with copper and cadmium. At a hardness of 207 mg/1, alkalinity of 154 mg/1, copper of 6.7 µg/1, and cadmium of 7.1 µg/1, 42.3 µg/1 of zinc was associated with reduced spawning of fathead minnows. When copper, cadmium and zinc were 5.3, 3.9 and 27.3 µg/1, respectively, reproduction was unaffected (Eaton, 1973). Therefore, a safe concentration of zinc for fathead minnows was 30 µg/1 in soft water (Brungs, 1969) and 27.3 µg/1 in hard water in the presence of added copper and cadmium (Eaton, 1973). However, in Eaton's (1973) study, it cannot be stated that the effects observed were solely due to zinc. Nevertheless, concentrations of zinc causing sublethal harm to aquatic biota do not appear to vary significantly with hardness or alkalinity.

The average zinc content of Great Lakes fish ranged from 11-20 µg/g in fish fillets (Uthe and Bligh, 1971) and from 11-48 µg/g in fish livers (Lucas et al., 1970). From these data there appeared to be little variation in zinc content in fish with location within species. In contrast, Brown and Chow (1975) showed that the average concentration of zinc in fish muscle across 7 species of fish from Baie du Dore, Lake Huron, was 4.69 µg/g while the average across 11 species from Toronto Harbour was 36.02 µg/g. This suggests that levels may be influenced by local contamination. Experimental exposures of fish to ^{65}Zn in water indicated maximum accumulation in the gills and kidney. Following injection, maximum accumulation occurred in body tissues, such as kidney, hepato-pancreas, heart, intestine, gill and scales (Saiki and Mori, 1955). Therefore, the route of uptake will affect distribution. Saiki and Mori (1955) did not follow concentration or location beyond 48 hours of exposure, nor after transferral to clean water. Mount (1968) found that the ratio of zinc in gills to zinc in bones was relatively constant in fish exposed to low levels of zinc. This indicated equal rates of deposition in these tissues. In fish exposed to lethal zinc concentrations, the ratio increased dramatically as the gills took up zinc quickly. In fish killed by zinc, the ratio exceeded a definite threshold. For fish subject to sublethal zinc intoxication, there is, as yet, no data relating tissue concentrations to particular toxic effects.

Therefore, in view of the great sensitivity of fish to low concentrations of zinc, an objective of 30 µg/1 zinc is recommended for the Great Lakes.

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TOTAL DISSOLVED SOLIDS - EFFECT ON SPECIES COMPOSITION

RECOMMENDATION

It is recommended that the present objective for TDS in the Agreement (Annex 1) be retained. It is also suggested that concentrations of the major ions (Na^+, K^+, Mg^{+2}, Ca^{+2}, SO_4^{-2}, Cl^-, CO_3^{-2}, HCO_3^-) in each lake be monitored on a regular basis in order to identify any major shifts in relative ionic composition.

RATIONALE

Last year's report of the Water Quality Objective Subcommittee (1974) discussed the levels and trends of concentration of total dissolved solids (TDS) and its major constituents in the Great Lakes. The effects of TDS on aquatic life were considered, and it was recommended that the objective included in the Water Quality Agreement be left unchanged. This year the Subcommittee investigated the influence of TDS and its major constituents on the species composition of the Great Lakes ecosystems, especially the phytoplankton communities.

This subject was of concern because it is well established that the relative and absolute concentrations of the major ions in a body of water help determine the species that occur (Prescott, 1968). For example, the occurrence of large numbers of species of desmids is usually correlated with low concentrations of calcium or of calcium plus magnesium (Hutchinson, 1967); on the other hand, species of blue-green algae usually have a competitive advantage over most other species at high concentrations of sodium, potassium, chloride, and sulfate (Provasoli, 1969). Such observations raise the possibility that the numerous changes that have been noted in the relative abundance of the species of phytoplankton in the Great Lakes may have been partially caused by the changes in TDS and its constituents that have also been observed.

A rather thorough search of the literature has yielded very little evidence to support or refute this possibility. The effect of ionic composition on species composition has received relatively little study, and the few investigations that have been conducted have tended to deal with very large differences in concentration, usually by a factor of 10 or more. As shown in the TDS section of last year's report, the changes in concentration of major ions in the Great Lakes, while substantial in a number of local situations, have not been that large. Thus, there is no direct evidence from which to derive specific limits for these ions in order to protect against changes in species composition, and no limits are proposed.

However, the available evidence does nothing to alleviate the concern that the relative and absolute concentrations of the TDS constituents could strongly influence phytoplankton species composition. For this reason the

subcommittee feels even more strongly than previously that the present objective for TDS in the Agreement should be retained. Further, it recommends that the concentrations of the major ions (i.e., Na^{+}, K^{+}, Mg^{+2}, SO_4^{-2}, Cl^{-}, CO_3^{-2}, HCO_3^{-}) in each lake and each major within-lake subarea should be monitored on a regular basis in order to identify any major shifts in relative composition that occur.

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PERSISTENT COMPOUNDS

FLUORIDE

RECOMMENDATION

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

> *Concentrations of total fluoride in an unfiltered water sample should not exceed 1.2 milligrams per litre to protect raw waters for public water supplies.*

RATIONALE

Fluorine, chemically bound as fluoride, is the 17th most abundant element in the earth's crust. It occurs in both igneous and sedimentary rocks and enters surface waters mainly through the weathering process of these rocks (Kilham and Hecky, 1973). The main fluorine-containing minerals are fluorspar (CaF_2), cryolite (Na_3AlF_6) and fluorapatite [$Ca_5F(PO_4)_3$].

The fluoride cycle involves passage to and from the atmosphere, hydrosphere, lithosphere and biosphere. It has been estimated that 6,000 tons of fluoride are contained in the 30 million tons of soil distributed in the atmosphere each year in the United States (NAS/NRC 1971). Industrial sources to the atmosphere and surface waters have been steadily increasing over the past hundred years with the processing of new materials from the earth's crust. These industrial sources include manufacture of aluminum; steel; brick and tile products; phosphorus fertilizer and coal fired electric power generation. Fluoride is also used as a pesticide and for many other commerical purposes. It has been estimated that approximately 120,000 tons of fluoride were emitted to the atmosphere in the United States in 1968 from industrial operations. A large portion of these particulates and gases are removed from modern plants practising good emission control by the use of filters, electrostatic precipitators and various wet-scrubbing systems (NAS/NRC, 1971).

Groth (1975) estimated that the phosphate and aluminum industries discharge between 10,000 to 35,000 tons of fluoride into United States surface waters annually. He also estimates that fluoridation practices in municipal water supplies add another 20,000 tons each year.

Fluoride in Water

In general, most fluoride salts formed with mono-valent cations are water soluble (e.g., NaF, AgF and KF) but those formed with di-valent cations are usually quite insoluble (e.g., CaF_2 and PbF_2), (Sienko, 1957).

Natural or "background" fluoride levels in most freshwater streams have less than 0.2 mg/l (Neuhold and Sigler, 1960; Groth, 1975). Concentrations of 13 mg/l are present in the Firehole and Madison Rivers in Yellowstone National Park and in Pyramid and Walker Lakes in Nevada (Sigler and Neuhold, 1972). Many East African lakes contain more than 1,000 mg/l, the highest natural concentrations found anywhere (Kilham and Hecky, 1973). A 1970 "background" water quality survey of 23 streams in the urbanized S.E. portion of Michigan's lower peninsula compared to 32 streams in the upper peninsula showed mean fluoride concentrations of 0.40 and 0.18 mg/l, respectively (Michigan DNR, 1970).

Fluoride concentrations in the upper Great Lakes are below those predicted by the equilibrium constants of Kramer (1964). This was based on the calcium-carbonate-phosphate-fluoride system which is believed to regulate the concentrations of fluoride in inland lakes. This regulating system was postulated as the explanation for the observation of uniform concentrations of fluoride (0.46 mg/l) at different depths and even in the interstitial water of 14 foot deep core samples from a meromictic lake (Brunskill and Harriss, 1969). Comparisons of actual concentrations in the Great Lakes to Kramer's model (1964) are as follows:

Table 6

Comparison of Kramer's predicted fluoride levels for the Great Lakes with concentrations actually observed (in mg/l).

Lake	Predicted Levels	Average fluoride concentrations 1961 to 1963*	1968**	1971***
Lake Superior	0.23	0.15	0.032	0.05
Lake Michigan	0.18	0.1	0.1	--
Lake Huron	0.43	--	0.074	0.08
Lake Erie	0.4	0.1	0.110	0.12
Lake Ontario	0.35	0.2	0.116	0.14

* Kramer 1964

** Weiler and Chawla 1969

*** CCIW 1975

Fluoride concentrations have been observed to increase with high river flows (Baker and Kramer, 1971), below municipal wastewater discharges (Bahls, 1973), and in the vicinity of phosphate-mining operations (Moore, 1971).

It is also interesting to note that fluoride is considered one of the main ligands responsible for keeping beryllium, aluminum, scandium, niobium, tantalum, iron and tin in solution in natural waters (Pitwell, 1974).

Drinking Water Supplies

Fluoride in drinking water for domestic animals generally has the same effect as in man. Concentrations less than 2 mg/l generally have no effect. Levels higher than this can cause mottling of teeth and extremely high intakes can cause skeletal fluorosis. Since food is the major source of fluoride intake by domestic animals, it has been suggested that concentrations in forage averaging 40 µg/g or less will not cause significant fluorosis (NAS/NRC, 1971). It has been reported that 4 to 5 mg/l of fluoride in drinking water resulted in observable effects in cattle in the form of dental lesions, mottling, staining and abnormal wearing of the teeth. Thorough examination, however, established that these effects were insignificant in the health, vitality, reproduction or milk production of the animals (Neeley and Harbaugh, 1954). Even when cattle receive large amounts of fluoride neither their flesh nor their milk pass it along the food chain to man. The body burden they do accumulate is almost entirely in their bones (NAS/NRC, 1974). NAS/NAE (1973) recommends an upper limit for fluoride in livestock drinking water of 2 mg/l for prevention of excessive teeth mottling.

Fluoride is often added to domestic water supplies to a level in the distribution system of 1.0 mg/l to prevent dental caries. Water containing less than about 1 mg/l will seldom cause mottling of teeth even in the most susceptible children. Levels sufficient to cause other health problems will not be encountered in a water supply fit to drink, but could only be accumulated through a large intake of drinking water.

The World Health Organization European Drinking Water Standards recommend an upper fluoride drinking water limit of 1.5 mg/l.

The United States Public Health Service Drinking Water Standards (1962) specify the same standard for drinking water as does Ontario, Canada (1974). These standards, for the Great Lakes Basin, are 1.3 mg/l in each case. The Canadian Drinking Water Standards (DNHW, 1969) specify a fluoride concentration in drinking water of 1.2 mg/l.

Effects on Vegetation

All vegetation contains some fluoride due to uptake from soil and water. The normal range for terrestrial plants is 2 to 20 µg/g (dry weight). Plants can also absorb soluble fluoride salts through their leaves. Information on the amount of fluoride in plant tissue derived from irrigation water is limited. One investigation showed that irrigation water containing 6.2 mg/l of fluoride increased the fluoride content of forage crops from 11 µg/g to 15 to 25 µg/g (Rand and Schmidt, 1952). A U.S. group of experts recently recommended a maximum of 1.0 mg/l for continuous use in irrigation water for all general soil applications and 15 mg/l for use over a 20-year period on neutral and alkaline fine textured soils (NAS/NAE, 1973).

Apparently no plant injury occurs by irrigation water containing 10-15 mg/l fluoride (Bower and Hatcher, 1967; McKee and Wolf, 1963). No reduction in carbon dioxide uptake occurred in terrestrial mosses incubated in aqueous solutions containing 820 mg/l fluoride for 24 hours. Uptake was effectively stopped by 8,200 mg/l after 24 hours (Inglis and Hill, 1974). They concluded that fluoride was relatively non-toxic to mosses.

Aquatic plants have been found to contain higher concentrations of fluoride than terrestrial plants. The terrestrial plants contained 33.8 µg/g compared to 40.5 µg/g in the aquatics (Danilova, 1944). However, no bioaccumulation was observed in either Cladophora or diatoms experimentally exposed for 72 days to fluoride concentrations of 52 mg/l (Hemens and Warwick, 1972). Suppression of growth was observed by Smith and Woodson (1964) in a bioassay using the alga Chlorella pyrenoidosa at all levels between 4.2 to 4,200 mg/l of fluoride. They concluded that this antimetabolite has its greatest effect between 420 and 4,200 mg/l where 86 and 98% inhibition occurred after 72 hours. Fluoride concentrations of 4.2 and 42 mg/l had equal inhibitory effects of 19% after 72 hours. Using the same algal species, but measuring respiration instead of growth, Sargent and Taylor (1972), however, did not detect inhibition at high levels of fluoride (1,680 mg/l). They did find that copper sulfate and fluoride acted more than additively in inhibiting respiration.

Kilman and Hecky (1973) observed that the sedge Cyperus papyrus was absent in African lakes containing 5.4 and 6.6 mg/l of fluoride but was abundant in lakes with 0.95 mg/l of fluoride.

Effect on Aquatic Animals

Recently Groth (1975) reviewed the available literature and concluded that there was a fairly compelling case for dealing with fluoride as a pollutant with a great capacity to do ecological harm. As part of the evidence supporting this concern he cited the fact that downstream

concentrations of 0.5 to 3 mg/l fluoride can result from both industrial sources and municipal sewage. Concentrations are highest during summer months when biological activity is also at its peak. No ecological effects were correlated with these fluoride levels (Bahls, 1973). Groth (1975) states that much additional research is needed on the effects of fluoride and indicates that adverse effects on aquatic life may have been masked in the past by far more severe effects of untreated sewage, industrial effluents and other major pollutants.

Bacterial species commonly associated with municipal wastewaters were unaffected by concentrations up to 800 mg/l fluoride during a 48-hour bioassay. No changes in growth or morphology were observed in Escherichia coli, Pseudomonas fluorescens and Enterococcus species grown in nutrient broth and mineral media with the above-mentioned concentration of fluoride. No changes in viability were observed in these species after 4 months storage in the fluoride solution (Vajdic, 1966). Paramecia, Euglena and rotifers continued to live, reproduce and were active in fluoride concentrations of 2 to 1,000 mg/l (Wantland, 1956).

Using Lake Erie water as the diluent and fluoride as the toxicant, Anderson (1946) found a 48-hour EC_{50} of 504 mg/l for Daphnia magna. The measure of acute toxicity used was the 48-hour median effective concentration (48-hour EC_{50}) based on immobilization.

Indigenous populations of copepods were found in East African lakes containing 437 mg/l fluoride but not in lakes with 1,064 mg/l (Kilham and Hecky, 1973).

Studies with marine invertebrates indicate that only high fluoride concentrations were toxic to the bluecrab, Callinectes sapidus, (greater than 20 mg/l, (Moore, 1971)) and to oysters, (greater than 128 mg/l, (Moore, 1969)). However, Hemens and Warwick (1972) found a 30% mortality in brown mussels Prena perna after 5 days exposure to approximately 7.2 mg/l, and 60% mortality at 41.6 mg/l. No mortality was observed in 3 species of estuarine fish after 96 hours in 100 mg/l fluoride test solution. Stewart and Cornick (1964) found that exposure to 5 mg/l in sea water did not harm the lobster Homarus americanus at 2C, for 10 days.

Reliable bioassay data for freshwater fish are very limited and some researchers have used soft water as a diluent. Since calcium is antagonistic to fluoride toxicity it may not be valid to apply bioassay data from low calcium water, (less than 3 mg/l) to the Great Lakes which contain from 13 to 46 mg/l calcium (Vallin, 1968; Weismann, 1974; Sigler and Neuhold, 1972).

Neuhold and Sigler (1960) determined a 20-day LC_{50} for rainbow trout Salmo gairdneri of 2.7 to 4.7 mg/l fluoride (95% confidence level) using softened dilution water (calcium less than 3 mg/l). They concluded that this is much lower than would occur in high calcium water. They also

subjected rainbow trout to 30 different combinations of fluoride and calcium concentrations ranging from 0 to 25 mg/l fluoride and 0 to 25 mg/l calcium. From these bioassays they determined the antagonistic relationship between fluoride and calcium and expressed it in an equation. Applying their equation for calcium/fluoride antagonism to Lake Superior water with a calcium concentration of 13 mg/l the LC_{50} for rainbow trout is 26 mg/l fluoride (Appendix I). The LC_{50} they determined for rainbow trout eggs (237 to 381 mg/l) was very high compared with earlier observations by Ellis et al. (1948) indicating that 1.5 mg/l delayed hatching and caused a poorer hatch. Neuhold and Sigler (1960) also found that rainbow trout embryos and fry are more sensitive than eggs to fluoride. The 34-day LC_{50} was between 61 and 85 mg/l. In bioassays of the more tolerant carp they found an LC_{50} between 71 and 91 mg/l (95% confidence level) at temperatures ranging between 18 and 24C. The carp ranged from 10 to 33 cm in size.

Bioassays by Herbert and Shurben (1964) using rainbow trout showed a 96-hour LC_{50} of about 18 mg/l in very soft water (hardness 12 mg/l). However, the authors concluded that waters with a greater hardness significantly reduced the toxicity of fluoride. They further stated that 1.0 mg/l fluoride would have only a negligible toxic effect on a trout population.

Wallen et al. (1957) found the mosquitofish Gambusia affinis survived fluoride concentrations of 560 mg/l and lower in turbid water with an alkalinity of less than 100 mg/l. The 96-hour LC_{50} was 925 mg/l. Ellis (1937) reported that goldfish Carassius auratus survived in a concentration of 100 mg/l in hard water for four days (termination of experiment).

In a review, Sigler and Neuhold (1972) indicate that the response of fish to moderate fluoride concentrations (1.5 to 5 mg/l) is related to acclimation, environmental variables such as calcium concentrations and temperature, and is species dependent. They state that fish populations vary with respect to their resistance to fluoride toxicity giving as examples the healthy growing populations of trout in the Firehole River in Yellowstone National Park, Pyramid and Walker Lakes in Nevada where fluoride concentrations reach 13 mg/l. Yet their earlier tests showed that trout raised in low fluoride concentrations displayed LC_{50}'s of approximately 3 mg/l.

Bioaccumulation in Aquatic Animals

Fluoride concentrations in fish range from less than 0.1 to 24 µg/g (NAS/NRC, 1974). Most of the data available deal with marine fish and potential problems with high fluoride concentrations in fish flour (Farkas, 1974). Fish-protein concentrate made in the United States was found to contain 169 µg/g fluoride (Hadjimarkos, 1964). Hoskins and Loustaunau (1974) analyzed fish-protein concentrate made from two marine species and one freshwater species and found that all were less than the F.D.A.'s 100 µg/g limitation and many were less than 25 µg/g.

Bioassays using fluoride concentrations ranging from 0.5 to 128 mg/l showed accumulations occurred at 2 mg/l and above in oyster tissues. Maximum levels in tissue, which were obtained after the first five days of exposure, were 100 µg/g exposed to solutions of 32 mg/l fluoride while 18 µg/g was found after the 2 mg/l exposure (Moore, 1969). The blue crab similarly reached a concentration of 50 µg/g in muscle after 90 days exposure to 20 mg/l fluoride while the control (0.1 - 1.5 mg/l fluoride) contained 10 µg/g (Moore, 1971).

Generally potential problems occur only at high exposures of fluorides or when the total fish is consumed (including bone) as in fish-protein concentrate.

In summary, since most of the fluoride toxicity studies on aquatic life have involved either the use of low calcium dilution waters or marine organisms, it is not practical to set an objective based on the protection of aquatic life. Therefore, it is recommended that the objective for fluoride be: 1.2 mg/l total fluoride in an unfiltered water sample to protect raw waters for public water supplies.

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NON-PERSISTENT TOXIC SUBSTANCES

INORGANIC

CYANIDE

RECOMMENDATION

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

> *Concentrations of free cyanide in unfiltered water samples should not exceed 5 micrograms per litre for the protection of aquatic life.*

RATIONALE

Cyanide is one of the simplest and most readily formed organic compounds. Cyanide and derivatives are almost universally present where life and industry are found. Besides being very important in a number of manufacturing processes, they are found in many plants and animals as metabolic intermediates which generally are not stored for long periods of time.

Common forms of cyanide in effluents are metal cyanide complexes, hydrocyanic acid, and the free cyanide ion formed primarily from dissociated simple cyanide salts. The cyanide form present in the aquatic environment is largely pH dependent. Most of the free cyanide exists as HCN at Ph values of natural waters, with the fraction increasing rapidly as the pH of the solution decreases. When simple cyanide salts dissociate in aqueous solution, the cyanide ion combines with the hydrogen ion to form hydrocyanic acid, which is highly toxic to aquatic life. Chemically, the cyanide ion behaves similarly to the halide ions -- chloride, fluoride, bromide and iodide.

The cyanide ion combines with numerous heavy metal ions to form metallocyanide complexes. The stability of these anions is highly variable. Those formed with zinc and cadmium are not stable; dissociation and production of hydrocyanic acid in near neutral or acidic environments is rapid. In turn, some of the metallocyanide anions are extremely stable. Cobaltocyanide is difficult to destroy with highly destructive acid distillation in a laboratory. The iron cyanides are also very stable but exhibit the phenomenon of photodecomposition, and in the presence of sunlight the material dissociates to release the cyanide ion, thus affecting toxicity; at night the reaction may reverse to produce a less toxic environment.

A wide variety of organic compounds may contain cyanide functional groups. These compounds belong to a class of organic chemicals called

nitriles, none of which dissociates to liberate cyanide ions or molecular HCN. In addition, there are also complex organic acids, alcohols, esters, and amides that contain the cyanide radicals. These organic compounds are used for numerous products or may be a waste by-product. Their toxicity, persistence, and chemistry in the aquatic environment are not well known except for a few specific compounds.

Cyanide toxicity is essentially an inhibition of oxygen metabolism, i.e., rendering the tissues incapable of exchanging oxygen. The cyanogen compounds are true non-cumulative protoplasmic poisons (can be readily detoxified) since they arrest the activity of all forms of animal life. Cyanide shows a very specific type of toxic action. It inhibits the cytochrome oxidase system which facilitates electron transfer from reduced metabolites to molecular oxygen. The ferric iron-porphyrin molecule responsible for the catalytic action of cytochrome oxidase is the reactive site where cyanide combines with ferric [Fe(III)] iron atoms to form a reversible complex. Other enzymes containing a metal porphyrin molecule, e.g., peroxidases and xanthine oxidases, are also strongly inhibited by cyanide. Only undissociated HCN inhibits the consumption of oxygen in the tissues, causing cellular asphyxia (historic anoxia) by attaching itself to the iron of the prosthetic group of the enzyme cytochrome oxidase.

Hydrocyanic acid can be rapidly absorbed and carried in the plasma but does not combine with hemoglobin because its iron atom is divalent (ferrous). Instead, cyanide combines with methemoglobin, a mildly oxidized form of hemoglobin in which the iron atom is trivalent (ferric). Methemoglobin, which cannot carry oxygen, normally represents only a small fraction of the total hemoglobin. Since it forms an irreversible and innocuous complex with cyanide, it is an active cyanide detoxifying agent. Amyl nitrite and other agents can be used to increase the level of methemoglobin to counteract cyanide toxicity. A few of the ways in which cyanide can be metabolized within a pattern of normal physiology are by the production of thiocyanate, with amino acids, oxidation to carbon dioxide and formate, etc. The conversion of only free cyanide and not organically bound cyano groups to thiocyanate (SCN-) by action of the enzyme rhodanase is considered to be the primary method of detoxification of cyanide. Rhodanase is absent from blood and skeletal muscle, but is abundant in the liver. Thiocyanate is eliminated irregularly and slowly in the urine.

The action of cyanide on the respiration of the cell and the primary methods of detoxification of cyanide have been noted above. However, it should be pointed out that cyanide does not completely abolish cellular respiration. It is possible that a small amount of residual respiratory activity is made possibly by cytochrome-b activity, since this substance does not require the cyanide-susceptible cytochrome oxidases. An alternative explanation of residual respiratory activity of the cyanide-poisoned system is found in the action of the flavin aerobic dehydrogenases, which can transfer hydrogen to molecular oxygen without the cytochrome system.

The persistence of cyanide in water is highly variable. This variability is dependent upon the chemical form of cyanide in the water, the concentration of cyanide, and the nature of other constituents. Cyanide may be destroyed by strong oxidizing agents such as permanganates and chlorine. Chlorine is commonly used to oxidize strong cyanide solutions to produce carbon dioxide and ammonia; if the reaction is not carried through to completion, cyanogen chlorine may remain as a residual and this material is also toxic. If the pH of the receiving waterway is acid and the stream is well aerated, gaseous hydrogen cyanide may evolve from the waterway to the atmosphere. At low concentrations or toxicity and with acclimated microflora, cyanide may be decomposed by microorganisms in both anearobic and aerobic environments or waste treatment systems.

A review of the available pertinent data on the acute toxicity of simple cyanides to fish reveals that the minimum lethal (threshold) concentrations of free cyanide from data obtained from experiments ranging from 12 minutes to 10 days with brook trout, Salvelinus fontinalis (Karsten, 1934); rainbow trout, Salmo gairdneri (Herbert and Merkens, 1952); brown trout, Salmo trutta (Burdick et al., 1958); bluegills, Lepomis macrochirus (Doudoroff et al., 1966); and fathead minnows, Pimephales promelas (Doudoroff, 1956), are reported to be 50, 70, (60 determined concentration) 70, 104, 150 and 180 µg/1 as cyanide, respectively. The minimum lethal threshold concentration is the concentration nearly or barely tolerable for individuals of average resistance when the exposure thereto is indefinitely prolonged.

Research at the University of Minnesota has revealed that the minimum lethal threshold concentrations, as determined from continuous flow bioassays in which routine analyses for cyanide were performed, are generally lower than the above reported values. In addition, the acute data with fish indicate that, in general, juveniles are more sensitive to HCN than younger life history stages, and that their sensitivity is increased with reduction in dissolved oxygen concentration and with lowering of temperature. Over the temperature range of 25C to 8C the lethal threshold concentration (LTC) for juvenile bluegills (Lepomis machrochirus) was determined to decrease linearly from 130 to 58 µg/1 HCN. Should the above linearity persist to lower temperature - not as yet experimentally determined - the calculated LTC values at 5, 3, and 1C would be 41, 32 and 23 µg/1 HCN respectively.

In review it can be concluded that free cyanide concentrations in the range from 50 to 100 µg/1 as cyanide have proven eventually fatal to many sensitive fishes and levels much above 200 µg/1 probably are rapidly fatal to most species.

Downing (1954), Cairns and Scheier (1958), and Burdick et al. (1958) have shown that the toxicity of free cyanide increases with any reductions in dissolved oxygen below the 100 percent saturation levels. Cairns and Scheier (1958) observed that even periodic lowering of dissolved oxygen decreased the tolerance of bluegills to cyanide.

Contradictory information from the literature indicates that uncertainty exists between the relationship of toxicity of simple cyanides to fish and the pH of the test solution. However, since undissociated hydrogen cyanide has been demonstrated to be the toxic cyanide species in simple cyanide solutions, changes in the pH of natural waters below a value of about 8.3 should have no measurable effect on the acute toxicity of simple cyanides to fish. There is no apparent relationship between toxicity to fish and the alkalinity and hardness of the dilution water.

Cyanide is acutely toxic to most fishes at concentrations ranging from 50 to 200 µg/l (Herbert and Merkens, 1952; Burdick et al., 1958; Cairns and Scheier, 1958; Doudoroff, 1956; Turnbull et al., 1954; Lipschuetz and Cooper, 1955; Washburn, 1948).

Some information on chronic or sublethal effects of cyanide is also available. Leduc (1966) found increased intestinal secretions in the fish, Cichlasoma bimaculatum, at concentrations as low as 20 µg/l and reduced swimming capability at concentrations of 40 µg/l. Costa (1965) reported that three common species of fish detected and avoided cyanide concentrations of 26 µg/l in approximately one hour or less. Exposure to a cyanide concentration as low as 10 µg/l reduced the swimming ability or endurance of brook trout, Salvelinus fontinalis (Neil, 1957). Growth, or food conversion efficiency of coho salmon, Oncorhynchus kisutch, was reduced at hydrogen cyanide concentrations of 20 µg/l. Small freshwater fish of the family Cichlidae exposed to a cyanide concentration of 15 µg/l lost weight more rapidly than the control fish in water free from cyanide (Leduc, 1966).

Survival and growth tests of 56 days duration on young of the yellow perch (Perca falvescens) and newly hatched bluegill fry (Lepomis machrochirus) performed at the University of Minnesota indicate that with the bluegill fry significant mortality occurred after about one month at all levels above 26 µg/l HCN. With the yellow perch, a marked effect on growth and survival was observed at about 45 µg/l, and at 64 µg/l survival was approximately 50 percent (Broderius, 1975). Chronic tests were also performed by Broderius in 1975 from egg through reproduction and into the second generation of fathead minnows (Pimephales promelas) and through reproduction and hatching with brook trout (Salvelinus fontinalis). For the fathead minnow it was observed that HCN levels above 40 µg/l decreased growth rate through 84 days.

A significant reduction in fecundity was noted at HCN levels greater than 18 µg/1 liter. Mean percentage hatch was significantly decreased at levels of 40 µg/liter HCN and higher. When the combined effect of egg production and fertility (percentage of egg hatch) were calculated at about 50, 120, 180, 250, and 330 µg/liter HCN, effective reproduction was approximately 65, 59, 35, 24, and 21 percent of the controls respectively. From the brook trout chronic data, it can be calculated that at about 50, 100 and 300 µg/1 HCN effective reproduction was about 80, 50, and 30% of the controls respectively.

Based upon chronic effects on fish growth and reproduction, an objective of 5 µg/1 free cyanide as HCN is recommended.

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NON-PERSISTENT TOXIC SUBSTANCES

ORGANIC

NON-PERSISTENT PESTICIDES

RECOMMENDATION

Concentrations of unspecified, non-persistent pesticides should not exceed 0.05 of the median lethal concentration in a 96-hour test for any sensitive local species.

A persistent compound has been defined (Great Lakes Water Quality Board, 1974) as one which either a) by itself or as its transformation product, has a half-life for degradation under natural environmental conditions of more than eight weeks, or b) by itself or as its transformation products, on entering surface waters may bioconcentrate in the biota of the receiving waters. Most of the toxic substances dealt with under the category of persistent organic contaminants were organochlorine pesticides but there is a substantial number of biocides, particularly the organo-phosphates and carbamates which do not meet this definition but are of concern because of their actual or potential effects on biota in the Great Lakes region.

Where established standards for raw water supplies are limiting, the objective for any substance (persistent or not) will be based upon such standards, but these are generally not the most restrictive use. Rather, it will more likely be aquatic life which represents the most stringent use and objectives should be set, therefore, to protect all life stages of the most sensitive species identified.

In establishing objectives to protect aquatic life from any toxic substance, the preferred approach is to use data derived from chronic, long-term tests on at least one generation of a sensitive test organism. Accordingly, the approach adopted here is to establish objectives for those specific pesticides for which low level, long-term chronic testing has been conducted. Where scientifically determined "no-effect" levels are available, these levels shall be recommended as the specific numerical objective; but where such levels have not been determined, objectives will be established by applying an arbitrary safety factor of 0.2 to the lowest concentration which produced a subtle effect (e.g., reduction in reproductive success) on an appropriate test organism. This latter approach should provide a realistic estimate of "safe" levels, and is consistent with the philosophy established earlier for the establishment of objectives for persistent substances. Where neither the "no-effect" nor the estimated "safe" levels have been determined and where there are indications of potential and significant inputs to the Great Lakes basin, it is recommended that protection be afforded aquatic life through the use of a 0.05 safety factor applied to the 96-hour LC_{50} for the pesticide for sensitive local species.

It should be recognized that the preceding approach will significantly restrict the number of specific pesticides regulated within this category of substances, as an inadequate scientific data base exists for most of them to permit the establishment of defensible numerical objectives. For this reason, the use of the arbitrary safety factor of 0.05 times the 96 hour LC_{50} is employed. Objectives based on this latter procedure may be inadequate to protect aquatic life from a variety of deleterious sublethal effects or conversely, it may be unduly restrictive. Such a procedural objective is intended only as a temporary measure and not as a substitute for the requisite testing necessary to establish scientifically defensible objectives.

The presence of some of the organophosphorus pesticides has been investigated in the upper Great Lakes (Glooschenko et al., 1976) but none have been observed. While many of the compounds are not "persistent", they may survive long enough in localized areas to cause deleterious effects - either at acute levels or through accumulation of biological effects. The usage/discharge patterns are unknown for most of these substances and, to date, no pressing problems have been noted, at least on a basin basis. It is possible, however, to conceive of changing patterns such that localized exposure to these compounds might lead to undesirable levels. Such exposure could come about through direct application in spraying programmes and accidental spillage, via surface runoff or leaching, and with discharges in manufacturing operations. It is to protect against these eventualities that objectives are being formulated here.

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DIAZINON

RECOMMENDATION

The concentration of Diazinon in an unfiltered water sample should not exceed 0.08 micrograms per litre.

RATIONALE

Diazinon is the common name for the organo-phosphate pesticide diethyl-2-isopropyl-6-methyl-4-pyrimidyl phosphorothionate. It is commonly used to protect fruit trees, corn, tobacco and potatoes from sucking and leaf-eating insects. Diazinon is only slightly soluble in water (40 milligrams/litre at room temperature), and is stable in alkaline media, but is readily hydrolyzed in water (Martin, 1971).

Available data indicate that the persistence of diazinon in aquatic ecosystems is greatly influenced by pH. Cowart et al. (1971) demonstrated that the half-life of diazinon in water at a pH of 6.0 was 14 days. Miller et al. (1966) reported that 320 µg/l applied to a cranberry bog disappeared completely within 6 days. Gomaa et al. (1969) have indicated that the half-life of diazinon at pH values of 7.4, 9.0 and 10.4 was 184, 136 and 24 days, respectively. As pH values of 7.4-9.0 are normally encountered in waters of the Great Lakes system, it is possible that diazinon has the capability of persisting for up to several months in aquatic ecosystems. Because of the apparently conflicting data on its persistence, and as organophosphate compounds are generally non-persistent (i.e. half-life less than 8 weeks), diazinon is considered under the category of non-persistent pest control products.

Investigations of the accumulation rate of diazinon indicate that this compound does not appreciably accumulate in biological tissue. The Mummichog (Fundulus heteroclitus) concentrated diazinon to a level of approximately ten times the concentration in the surrounding water, but that 50% of tissue residue was lost in less than one week (Miller et al., 1966). Allison and Hermanutz (manuscript) reported that the accumulation factor for diazinon in fish is low (compared to that observed for most organochlorine pesticides), and that the tissue concentration is directly proportional to water concentrations.

There is currently no standard in use in either Canada or the United States which specifies maximum permissible concentrations of diazinon in raw public water supplies.

Exposure of the green alga Scenedesmus quadricaudata to diazinon concentrations of 100 and 1,000 µg/1 produced no effect on cell number, photosynthesis, or biomass over a ten-day study (Stadnyk and Campbell, 1971).

Studies of the toxicity of diazinon to fish are limited, and generally report the results of acute exposures. The 24-hour LC_{50} for rainbow trout (Salmo gairdneri) to diazinon was determined to be 380 µg/1 at 13°C (Cope, 1965). Cope (1966) reported that the 48-hr. LC_{50} for rainbow trout at 13°C and bluegills (Lepomis macrochirus) at 24°C was 170 µg/1 and 96 µg/1, respectively. Mean 96-hr LC_{50} values for diazinon were reported to be 7,800, 460, 770 and 1,600 µg/1 for fathead minnows (Pimephales promelas), bluegills, brook trout (Salvelinus fontinalis), and flagfish (Jordanella floridae) respectively (Allison and Hermanutz, manuscript).

The chronic effects of diazinon on fathead minnows and brook trout were studies by Allison and Hermanutz (manuscript). Statistically significant reductions in production rate for fathead minnows and brook trout were observed at 3.2 and 0.55 µg/1 (lowest concentrations tested). Exposure of brook trout for 6-8 months to concentrations of diazinon varying from 0.55 - 9.6 µg/1 resulted in equally reduced growth rates for progeny as well as adults. For fathead minnow, the hatch of progeny was reduced by 30% at a concentration of 3.2 µg/1. There is evidence that these effects on the progeny were the result of parental exposure alone, and not diazinon levels to which progeny were exposed following fertilization.

Available data indicate that the aquatic invertebrates are much more acutely sensitive to diazinon than are fish. The 48-hour EC_{50} (immobilization value at 15°C) for water fleas (Simocephalus serrulatus and Daphnia pulex) exposed to diazinon was 1.8 µg/1 and 0.90 µg/1, respectively (Sanders and Cope, 1966). Sanders (1969) has reported that the 96-hr LC_{50} for Gammarus lacustris was 200 µg/1. The 48-hour LC_{50} for the stonefly (Pteryonarcys californica) has been demonstrated to range from 6 µg/1 (FWPCA, 1968) to 7.5 µg/1 (Cope, 1966). The 96-hr. LC_{50} of diazinon for Acroneuria lycorias has been reported to be 1.7 µg/1 (NAS/NAE, 1973).

A number of studies have been conducted to determine the long-term acute toxicity of diazinon to aquatic invertebrates. These data are summarized in table 7 (NAS/NAE, 1973).

Table 7 - Toxicity of Diazinon to Aquatic Invertebrates (NAS/NAE, 1973)

Organism	30-day LC_{50} (µg/1)	30-day no effect (µg/1)
Gammarus pseudo-limnaeus	0.27	0.20
Daphnia magna	-	0.26
Pteronarcys dorsata	4.6	3.29
Acroneuria lycorias	1.25	0.83
Ophiogomphus rupinsulensis	2.2	1.29
Hydropsyche bettoni	3.54	1.79
Ephemerella subvaria	1.05	0.42

No studies have been conducted to evaluate the chronic effects of diazinon on reproduction and behaviour of invertebrates. Similarly, there have been no complete life cycle studies to establish a "no-effect", or safe concentration of diazinon for aquatic invertebrates.

Results from studies of the long-term acute toxicity of diazinon to aquatic invertebrates indicate that an objective less than 0.20 µg/1 would protect invertebrates from exposure to concentrations which are directly lethal. The unpublished work of Allison and Hermanutz would indicate that 0.55 µg/1 of diazinon is sufficiently high to exert a negative effect on brook trout productivity. In the absence of "no effect" concentrations established through the conduct of complete life-cycle studies, and information on the chronic toxicity of diazinon to invertebrates, it is recommended that the objective for diazinon be derived by application of a safety factor of 0.05 to the 96-hour LC_{50} for the most sensitive species. A review of the data presented here indicates that Acroneuria lycorius (96-hr LC_{50} of 1.7 µg/1) is the most sensitive organism. Accordingly, it is recommended that concentrations of diazinon in water not exceed 0.08 µg/1 to ensure protection of aquatic life. Available data on the long-term acute toxicity, and studies of the chronic effect of diazinon on brook trout, would indicate that this objective should protect sensitive species of fish and aquatic invertebrates.

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GUTHION

RECOMMENDATION

Concentrations of Guthion in an unfiltered water sample should not exceed 0.005 micrograms per litre for the protection of aquatic life.

RATIONALE

Guthion is a broad spectrum agricultural pesticide, also called azinphosmethyl and properly, O,O-dimethyl-S-[4-oxo-1,2,3-benzotriazin-3(4H)-ylmethyl] phosphorodithioate. It is used to protect fruit, grain and vegetable products in the agricultural industry as well as shrubs and trees and is soluble in water at approximately 30 mg/l (Chemagro Corp., 1957).

Hydrolysis of Guthion occurs in aqueous media (Heuer et al., 1974) at environmental pH's and temperatures with half-lives ($T_{\frac{1}{2}}$) of some 3-4 weeks. In a natural soil (Yaron et al., 1974), the half-lives varied between two weeks and a year, depending on the moisture content and temperature. Guthion has also been reported to degrade in pondwaters (Meyer, 1965; Flint, 1970) and also in a variety of fish ($T_{\frac{1}{2}}$'s less than one week) (Meyer, 1965). The degradation products have been shown to be non-toxic, at least to the insect target species (Liang and Lichtenstein, 1972).

The acute toxicity of Guthion to sensitive fish (96-hour LC_{50}) ranges from 3-14 µg/l. In static tests, LC_{50}'s for brown trout were 4 µg/l (Macek and McAllister, 1970); for rainbow trout, 3.2 µg/l (Katz, 1961) and 14 µg/l (Macek and McAllister, 1970); for bluegills, 5.2 µg/l (Katz, 1961) and for yellow perch, 13 µg/l (Macek and McAllister, 1970). Organophosphorus pesticides exert their lethal action toward fish by inhibition of acetylcholinesterase (AChE) (Weiss, 1961) and recovery from this condition is slow (Weiss, 1959, 1961; Darsie and Corrideu, 1959). Decrease in the activity of this nervous system enzyme, even when not lethal, will decrease the organism's activity and hence its potential for survival (Katz, 1961). Levels of Guthion as low as 1 µg/l have been observed to suppress AChE activity in bluegills (Weiss and Gakstatter, 1964). In other studies with fathead minnows, decreased spawning was observed during long-term exposures at concentrations as low as 0.7 µg/l (Adelman and Smith, unpublished 1976). These authors estimate a "safe" level at between 0.3 and 0.5 µg/l.

The most sensitive aquatic organisms for which observations are reported in the literature are the crustaceans and insects. Acute toxicities (96-hour LC_{50}) for these organisms have been observed as low as 0.1 - 0.2 µg/l for Gammarus lacustris and Gammarus fasciatus (Saunders, 1969; Saunders, 1972) and 1.5 µg/l for Pteronarcys californica (Sanders and Cope, 1968). The lowest long-term effects (20-30-day LC_{50}'s) have been noted in studies with grass shrimp (0.16 µg/l; Sanders, 1972) and stonefly naiads (0.24 µg/l; Jensen and Gaufin, 1966).

These above responses can hardly be considered as indicating "safe" concentration levels for all aquatic life and, consequently, the recommended safety factor of 0.05 is applied to the lowest of the 96-hour LC_{50}'s (Gammarus fasciatus at 0.1 µg/l) and should afford reasonable protection. The recommended objective is, therefore, 0.005 µg/l.

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PESTICIDES

PARATHION

RECOMMENDATION

Concentrations of parathion in an unfiltered water sample should not exceed 0.008 micrograms per litre for the protection of aquatic life.

RATIONALE

Parathion, O,O-diethyl-O-p-nitrophenylphosphorothionate, is a non-systemic contact and stomach insecticide and acaricide used extensively in the agricultural industry. It is slightly soluble in water at 24 mg/l (Martin and Worthing, 1974) and hydrolyses in distilled water with a half-life of 25-120 days (Peck, 1948; Cowart et al., 1971). Persistence of parathion in a natural environment has been studied by Eichelberger and Lichtenberg (1971) in which it was observed to have a half-life of one week in river water. In other studies, half-lives of three and five weeks were noted for "natural" waters of pH 8.4 and 7.0 respectively (Weiss and Gakstatter, 1964) and of 30-40 hours (Leland, 1968) in rainbow trout.

The effects of parathion (and other organophosphate pesticides) are reportedly via suppression of acetylocholinesterase (AChE) activity and this persists long after the actual exposure. Subjection of bluegills to 100 µg/l of parathion over 24 hours resulted in a 75% reduction of AChE activity which did not return to normal for a further sixty days (Weiss, 1961). The compound is metabolised to a number of products including its oxygenated analogue, para-oxon, and its amino form (Graetz et al., 1970). While some of these may be more toxic than the parent material and may even be responsible for parathion's AChE inhibition (Aldridge and Davison, 1952) they are generally more readily degraded as well.

Acute toxicity effects of parathion with fishes have been determined in flow-through systems with 96-hour LC_{50} values of 500 µg/l for bluegills, 1,600 µg/l for fathead minnows and 1,700 µg/l for brook trout (Spacie, 1975). In a similar test system, a 96-hour LC_{50} of 18 µg/l was noted for juvenile freshwater and estuarine striped bass (Korn and Earnest, 1974). Sub-acute effects (tremors) for brown bullheads are reported (Mount and Boyle, 1969) at 30 µg/l over a 30-day exposure. The lowest observed effect for fishes is with bluegills in which deformities were recorded over a 23-month exposure at 0.34 µg/l (Spacie, 1975).

Fishes are not however, the most sensitive organisms towards parathion - the insect target organisms, are much more susceptible. Acutely toxic levels for several more sensitive aquatic insects are recorded in table 8, along with sub-acute levels for the same species.

TABLE 8

Toxic Effects of Parathion to Insects and Crustaceans

Concentrations in µg/l in flow-through systems

Species	Acute LC_{50}	Sub-acute LC_{50}	Reference
Daphnia magna	0.62 (4 days)	0.14 (21 days)	Spacie (1975)
Acroneuria pacifica	0.93 (5 days)	0.44 (30 days)	Jensen & Gaufin (1964)
Gammarus fasciatus	0.40 (4 days)	0.07 (43 days)	Spacie (1975)

In the study by Spacie (1975) on Gammarus fasciatus, significant mortality was observed at 0.04 µg/l over 43 days and this is the lowest effect level reported for a freshwater organism. Also reported in the same studies were significant reproductive failure with Daphnia magna at parathion concentrations greater than 0.08 µg/l.

There do not appear to be any published data on actual "safe" concentrations of parathion for these sensitive organisms and in view of the fact that its physiological action is by suppression of acetylchlorinesterase activity, a condition from which recovery is slow, the safety factor of 0.2 is applied to the lowest of these levels (0.04 µg/l for Gammarus fasciatus) to arrive at the recommended level of 0.008 µg/l for the protection of aquatic life.

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PHYSICAL CHARACTERISTICS

TEMPERATURE

RECOMMENDATION

1. *Thermal additions to receiving waters or a designated segment thereof should be such that thermal stratification and subsequent turnover dates are not altered from those existing prior to addition of heat from artificial origin.*

2. *Maximum Weekly Average Temperature*

This is the mathematical mean of multiple, equally spaced daily temperatures.

A. *For Growth*

The maximum weekly average temperature (MWAT) in the zone inhabited by the species at that time should not exceed one-third of the range between the optimum temperature (To) and the ultimate upper incipient lethal temperature (Tu) of the species, in order to maintain growth of aquatic organisms at levels necessary for sustaining actively growing and reproducing populations (Table 1, Appendix II). Thus,

$$MWAT = To + \frac{Tu - To}{3}$$

The optimum temperature is assumed to be for growth but other physiological optima may be used in the absence of growth data. The MWAT must be applied with adequate understanding of the normal seasonal distribution of the important species.

B. *For Reproduction*

The MWAT for reproduction should not exceed those limits for normal spawning (Table 2 - Appendix II); in addition these objectives must protect gonad growth and gamete maturation, spawning migrations, spawning itself timing and synchrony with cyclic food sources, and normal patterns of gradual temperature changes throughout the year. The protection of reproductive activity must take into account normal months during which these processes occur in specific water bodies for which objectives are being developed.

C. *For Winter Survival (applicable at any place inhabitable by fish)*

The MWAT for fish survival during winter should not exceed the acclimation, or plume, temperature (minus a 2.0 C safety factor) that raises the lower lethal threshold temperature above the normal ambient water temperature for that season. This temperature limit will apply in any area to which the fish have access and would include areas such as unscreened discharge channels.

This objective is necessary to eliminate fish kills caused by rapid changes in temperature due to plant shutdown or movement of fish from a heated plume to ambient temperature.

3. *Short-term Exposure to Extreme Temperature*

A. *For the Season of Growth*

The temperature objective for (1) short-term exposure during the growth season is the 24-hr. median tolerance limit, minus 2°C, at an acclimation temperature approximating the MWAT for that month; and (2) short-term exposure during the spawning season is the upper temperature for successful incubation and hatching. These exposures should not be too lengthy or frequent or the species could be adversely affected. The length of time in minutes (t) that 50 percent of a population will survive temperatures above the incipient lethal temperature (T in °C) can be calculated from the following regression equation:

$$\log (t) = a + b (T)$$

where a and b are intercept and slope, respectively, which are characteristics of each acclimation temperature for each species (National Academy of Sciences, 1973).

B. *For the Season of Reproduction*

The short-term maximum temperature for the season of reproduction should be based on the maximum incubation temperature for successful embryo survival. The maximum temperature for spawning is probably an acceptable alternative.

RATIONALE

A detailed discussion of the development of these objectives and the rationale in support of them has been presented (NAS/NAE, 1973). The objectives are not designed to be unique for each thermal discharge. They are only different when the composition of sensitive important fish species is different. The following is a summarization of that rationale.

Living organisms do not respond to the quantity of heat but instead, to degrees of temperature or to temperature changes caused by transfer of heat. Organisms have upper and lower lethal tolerance limits, optimum temperatures for growth, preferred temperatures in thermal gradients, and temperature limitations for migration, spawning and egg incubation. Temperature also affects the physical environment of the aquatic medium (e.g., viscosity, degree of ice cover, and oxygen capacity). Therefore, the composition of aquatic communities depends largely on temperature characteristics of the environment, for example, warmwater fish such as bass and sunfish will not be able to compete with trout and salmon at temperatures that are optimal for the latter.

Because temperature changes may affect aquatic communities, an induced change in the thermal characteristics of an ecosystem may be detrimental. On the other hand, altered thermal characteristics may be beneficial, as evidenced in some of the newer fish hatchery practices and at other aquacultural facilities. The general difficulty in developing suitable objectives for temperature (which would limit the addition of heat) is to determine the deviation from natural temperature a particular body of water can experience without adversely affecting its desired biota. Whatever requirements are suggested, natural diurnal and seasonal cycles must be retained, annual spring and fall changes in temperature must be gradual, and large unnatural day-to-day fluctuations should be avoided. In view of the many variables, it seems obvious that no single temperature rise limitation can be applied uniformly to continental or large regional areas; the requirements must be closely related to each body of water and to its particular community of organisms, especially the important species found in it. These should include invertebrates, plankton, or other plant and animal life that may be of importance to food chains or otherwise interact with species of direct interest to man. Since thermal requirements of various species differ, the social choice of the species to be protected allows for different levels of protection among water bodies. Although such decisions clearly transcend the scientific judgments needed in establishing thermal criteria for protecting selected species, biologists can aid in making these decisions. Some measures useful in assigning levels of importance to species are: (1) high yield or desirability to commercial or sport fisheries, (2) large biomass in the existing ecosystem (if desirable), (3) important links in food chains of other species judged important for other reasons, and (4) endangered or unique status.

Criteria for making recommendations for water temperature to protect desirable aquatic life cannot be simply a maximum allowed change from natural temperatures. This is principally because a change of even one degree from an ambient temperature has varying significance for an organism, depending upon where the ambient level lies within the tolerance range. In addition, historic temperature records or, alternatively, the existing ambient temperature prior to any thermal alterations by man are not always reliable indicators of desirable conditions for aquatic populations. Multiple developments of water resources also change water temperatures both upward (e.g., upstream power plants or shallow reservoirs) and downward (e.g., deepwater releases for large reservoirs) so that ambient and natural temperatures at a given point can best be defined only on a statistical basis. Objectives for temperature should consider both the multiple thermal requirements of aquatic species and requirements for balanced communities. The number of distinct requirements and the necessary values for each require periodic re-examination as knowledge of thermal effects on aquatic species and communities increases. Currently definable requirements include:

o Maximum sustained temperatures that are consistent with maintaining desirable levels of productivity (growth minus mortality);

o Maximum levels of thermal acclimation that will permit safe return to ambient winter temperatures should artificial sources of heat cease;

o Temperature limitations for survival of brief exposures to temperature extremes, both upper and lower;

o Restricted temperature ranges for various stages of reproduction, including (for fish) gonad growth and gamete maturation, spawning migration, release of gametes, development of the embryo and larva, commencement of independent feeding (and other activities) by juveniles; and temperatures required for metamorphosis, emergence, and other activities of lower forms;

o Thermal limits for diverse compositions of species of aquatic communities, particularly where reduction in diversity created nuisance growths of certain organisms, or where important food sources or chains are altered.

Thermal objectives must also be formulated with knowledge of how man alters temperatures, the hydrodynamics of the changes, and how the biota can reasonably be expected to interact with the thermal regimes produced. It is not sufficient, for example, to define only the thermal objectives for sustained production of a species in open waters, because the large numbers of organisms may also be exposed to thermal changes by being pumped through the condensers and mixing zone of a power plant. Design engineers need particularly to know the biological limitations to their design options in such instances. Considerations such as impingement of fish upon intake screens, mechanical or chemical damage to zooplankton in condensers, or effects of altered current patterns on bottom fauna in a discharge area may reveal non-thermal impacts of cooling processes that may outweigh temperature effects. The environmental situations of aquatic organism (e.g., where they are, when they are there, in what numbers) must also be understood. Thermal objectives for migratory species should be applied to a certain area only when the species is actually there.

Available data for temperature requirements for growth and reproduction, lethal limits for various acclimation temperature levels, and various temperature-related characteristics of many of the more important or desirable freshwater fish species are included in Appendix II. General temperature objectives for these species are summarized in Tables 1 and 2 (Appendix II). The MWAT for winter survival are graphically presented in Figure 1 (Appendix II). In addition, examples of the development of numerial temperature objectives for a warmwater and a coldwater fish population are included in Appendix II. The derivation of the objectives in Tables 1 and 2, Figure 1, and the examples in Appendix II are reproduced from Brungs and Jones (1976).

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2 A PROPOSED MECHANISM TO PROTECT BIOLOGICAL INTEGRITY & LIMIT LOSS OF BENEFICIAL USES BY ALLOCATION OF VALUE

A portion of the narrative preceding the recommended water quality objectives in last year's report emphasized the necessity for development of a mechanism to limit the biological value loss and other beneficial use losses associated with mixing zones and other areas of non-compliance in such a manner that the integrity of the waterbody or portion thereof is assured.

A limiting mechanism incorporating management objectives and levels of protection was proposed which included the following basic steps:

1) Agreement on the biological and other uses to be protected;

2) Identification of the important species;

3) Biological mapping of the waterbody to establish biotic zones of the important species;

4) Assignment of a numerical biological value to the zones on the basis of importance to ecosystem function;

5) Selection of a level of protection for the waterbody;

6) Calculation of biological value available for allocation;

7) Allocation to present dischargers and reservation for future discharges.

The Water Quality Board acknowledged the future potential of the biological value allocation mechanism and authorized further development by referral back to the Implementation Committee and to the Research Advisory Board. The Water Quality Board was particularly interested in the availability of the biological data base and costs associated with gathering the missing information, the potential impact on existing dischargers, and the institutional arrangements for implementation.

The Research Advisory Board's Standing Committee on Scientific Basis for Water Quality Criteria and the Subcommittee concluded that the first obstacle to overcome enroute to acceptance and implementation of this mechanism is development of waterbody maps.

Last year almost total emphasis was on biological (zoogeographical) mapping, supported by chemical and physical mapping, to establish biotic zones of important species. Such a map, or series of maps on a seasonal basis,

would immediately identify areas important to ecosystem maintenance and provide insight into availability of habitat which by its scarcity is limiting to management objectives. This approach to waterbody mapping was too narrow in that beneficial uses other than biological were not included.

A Task Force working with biological, chemical, physical and social scientists familiar with Lake Ontario concluded that environmental value mapping to provide a holistic view of the system on an ecologically-meaningful scale was a prerequisite to development of any mechanism or plan for waterbody management.

Candidate information for environmental value maps includes: physical----- seasonal temperatures, depths, prevailing currents, bottom types; chemical----- areas of non-compliance, including mixing zones; biological-----fish (spawning, nursery, migration, living), marshes important to aquatic mammals and waterfowl, waterfowl feeding and wintering areas, gull nesting islands, benthos distribution and quality, zooplankton, phytoplankton, *Cladophora*, macrophytes; cultural uses-----dams and fishways, public recreation areas and parks, harbors of refuge and launching sites, fishing areas (sport and commercial), water intakes, discharge sites and areas of aesthetic and historical value.

The uses which could be made of waterbody environmental value maps include: identification and quantification of biotic zones; assignment of value; provision of a basis for limitations to configuration and conditions within mixing zones and other areas of non-compliance; site selection; guidance in dredging, filling and spoil disposal; background for aerial photography overlays; display of surveillance information; an extension of coastal zone mapping to permit holistic planning; identification of candidate areas of habitat rehabilitation; research planning; pinpointing priority areas to protect in the event of contaminant spills; and as a general education tool and popular guide.

The Research Advisory Board approved a workshop on Environmental Value Mapping of the Great Lakes for November 1976. Biological/environmental value maps as management aids have been produced for the Thames in England, the Danube in Germany, and Puget Sound, Chesapeake Bay, Galveston Bay, and San Francisco Bay in the United States. CCIW, Environment Canada, NOAA and New York Sea Grant are involved with special purpose mapping. The workshop will provide IJC cooperator organizations with an awareness of environmental mapping efforts ongoing in the Great Lakes and elsewhere in the world. What is the purpose of these maps? Their cost to construct? Why were certain characteristics mapped? Why were others left out? How have these maps been used? How can they be used in Great Lakes management? Why were certain display techniques chosen? Alternative displays?

The workshop will also provide an assessment of what information is essential or non-essential to support biological value allocation. Can we map biological entities in such a way that value becomes apparent? Is the necessary information available? Is our knowledge of ecosystem structure and function adequate?

The Subcommittee and the Standing Committee on Water Quality Criteria have refined theoretical allocation formulae and a computer program. Using the workshop product as a resource, further progress on the total mechanism is anticipated.

3 FUTURE DIRECTION OF SUBCOMMITTEE EFFORTS

The specific water quality objectives recommended by the Subcommittee are based on best available information. The Subcommittee will continue to review available information, and will recommend revisions as warranted from new knowledge. The Subcommittee will also consider, but will not necessarily develop objectives for the following parameters:

Chemical Characteristics

Inorganic

Barium - this element is considered to be toxic to humans, and drinking water standards for it exist in both the United States and Canada. This element was also listed in the 1972 Great Lakes Water Quality Agreement as requiring consideration for the development of a numerical objective.

Boron - this element is considered toxic to plants and animals, and drinking water standards for it exist in both countries.

Supersaturation of Dissolved Gases - modified dissolved gas pressures as a result of eutrophication, thermal or pressurized discharges present a widespread potential for adversely affecting fish and aquatic invertebrates.

Manganese - this element is considered to be toxic to humans and drinking water standards limit its concentration in finished public water supplies to 50 µg/l.

Nutrients and Chlorophyll-a - NO_3, PO_4 and SiO_2 - The Water Quality Objectives Subcommittee will take the lead in developing objectives for these three nutrients and consequently an objective for chlorophyll-a. The subcommittee has also requested the Standing Committee on Scientific Basis for Water Quality Criteria to obtain experts to assist the subcommittee in formulating these nutrient objectives.

Silver and Thallium - these elements are both known toxicants to aquatic life. They will be examined to determine if scientifically defensible objectives can be established from the present data base.

Phosphorus, elemental - elemental phosphorus is particularly toxic to fish and it is subject to bioaccumulation.

Pulp Mill Effluent Components - pulp mill effluents are known to contain toxic substances to aquatic life.

Organic

Detergents - these compounds will be examined by the Subcommittee as to their effects on water uses as surfactants and as builders. They are widely used in the Great Lakes basin, therefore their presence in the lakes is a certainty. A number of these compounds have known toxic effects to animals and to aquatic life.

Nitrilotriacetic Acid (NTA) - this compound is used widely in the Great Lakes drainage basin. Its presence, therefore, in the Great Lakes waters is a certainty.

Organophosphates and Carbamates - a large number of these compounds are in use throughout the Great Lakes basin for the control of insects and undesirable plant life. They are toxic to all living organisms but because their half-life in water is from a few days to a few weeks, they are not generally found in Great Lakes waters. The greatest danger of these compounds is to aquatic life through accidental spills and immediately following indiscriminate spraying programs. The Subcommittee will be examining the data to either set limits on individual brand named products or criteria that can be used to protect the water uses until the data base is adequate to set scientifically defensible objectives.

Polynuclear Aromatic Hydrocarbons (PAH) - many of these compounds are known to be carcinogenic to animals. These compounds occur in soils and there is evidence for their endogenous formation in plants. Materials associated with high temperature pyrolysis, such as coal-tar, coal-tar pitch, shale oil, carbon black have been shown to contain PAH. PAH has been isolated from cracked mineral oils, crude oil, and shipping and harbour oils. The Great Lakes waters are known to contain these compounds.

Rotenone - this compound is under registry as an insecticide. It is extremely toxic to fish but is not considered to be too toxic to mammals and birds.

Organo-tin Compounds - these compounds are insect feeding inhibitors. They are under registry as fungicides. The extent of their use in the Great Lakes basin is unknown. The Subcommittee is interested in these compounds because of their extreme toxicity to fish.

TFM - 3-trifluoromethyl-4-nitrophenol - this compound is applied directly to the waters of the Great Lakes for the control of the sea lamprey, Petromyzon marinus and could result in the possible loss of valuable fish species through the indiscriminate use of TFM.

Phenols - phenolic compounds contribute to the tainting of Great Lakes waters, and are also known to be toxic to aquatic life. The subcommittee will reconsider its objective of 0.001 mg/l proposed in the 1974 Appendix A to the Water Quality Board report.

Physical Characteristics

Asbestos - the data base is still insufficient to recommend a scientifically defensible objective for asbestiform fibres. The Subcommittee will continue to closely examine the scientific literature for new information on this material.

Biological Characteristics

Biological Effects of Intakes - concern over mortality of organisms, especially fish larvae, by intake entrainment continues. Many reports on studies to satisfy USEPA regulatory requirements at high volume intakes are either incomplete or not yet in the public domain. Research on putting intake mortality into perspective with natural mortality has high priority within the power industry and the Energy Research and Development Agency (ERDA). As data become available, WQOS will pursue development of a desired limit.

Microorganisms - the Standing Committee on Health Aspects of the Research Advisory Board is presently considering microorganisms to determine if it is scientifically justifiable to supplement total and fecal coliform objectives to provide a more accurate measure of conditions to protect Great Lake water users.

Toxicity Units - the concept of evaluating the damage potential of a wastewater effluent on the basis of toxicity units determined by volume of discharge and toxicity testing will be pursued.

APPENDIX I

EXTRAPOLATION OF LC_{50} USING CALCIUM CONTENT OF LAKE SUPERIOR WATER AND NEUHOLD'S EQUATION FOR CALCIUM/FLUORIDE ANTAGONISM

$y = 2.33 + 2.03\ X$

where y = probits - use 5, which is LC_{50}

$x = (\text{Log } F - \text{Log } Ca + 1)$

$5 = 2.33 + 2.03\ (\text{Log } F - \text{Log } Ca + 1)$ Ca (ppm) = 13

$(\text{Log } F = 1.11 + 1)$ Log 13 = 1.11

$5 = 2.33 + 2.03\ (\text{Log } F - .11)$

$5 - 2.33 = 2.03\ (\text{Log } F) - .11 \times 2.03$

$2.67 = 2.03\ (\text{Log } F) - .22$

$2.89 = 2.03\ (\text{Log } F)$

$2.89 \div 2.03 = \text{Log } F$

$1.42 = \text{Log } F$

$26.0 = F$ conc. in mg/l

The relationship between the concentrations of calcium and fluoride ions and the LC_{50} of rainbow trout subjected to varying combinations of calcium and fluoride was determined by plotting the log of the ratio of fluoride to calcium against the probit of responses to the varying combination of calcium and fluoride. A straight line relationship from which the LC_{50} can be determined was found (Figure 1). The LC_{50} was determined between 1.01 and 4.22 [fluoride] / [calcium] at the 95 percent confidence

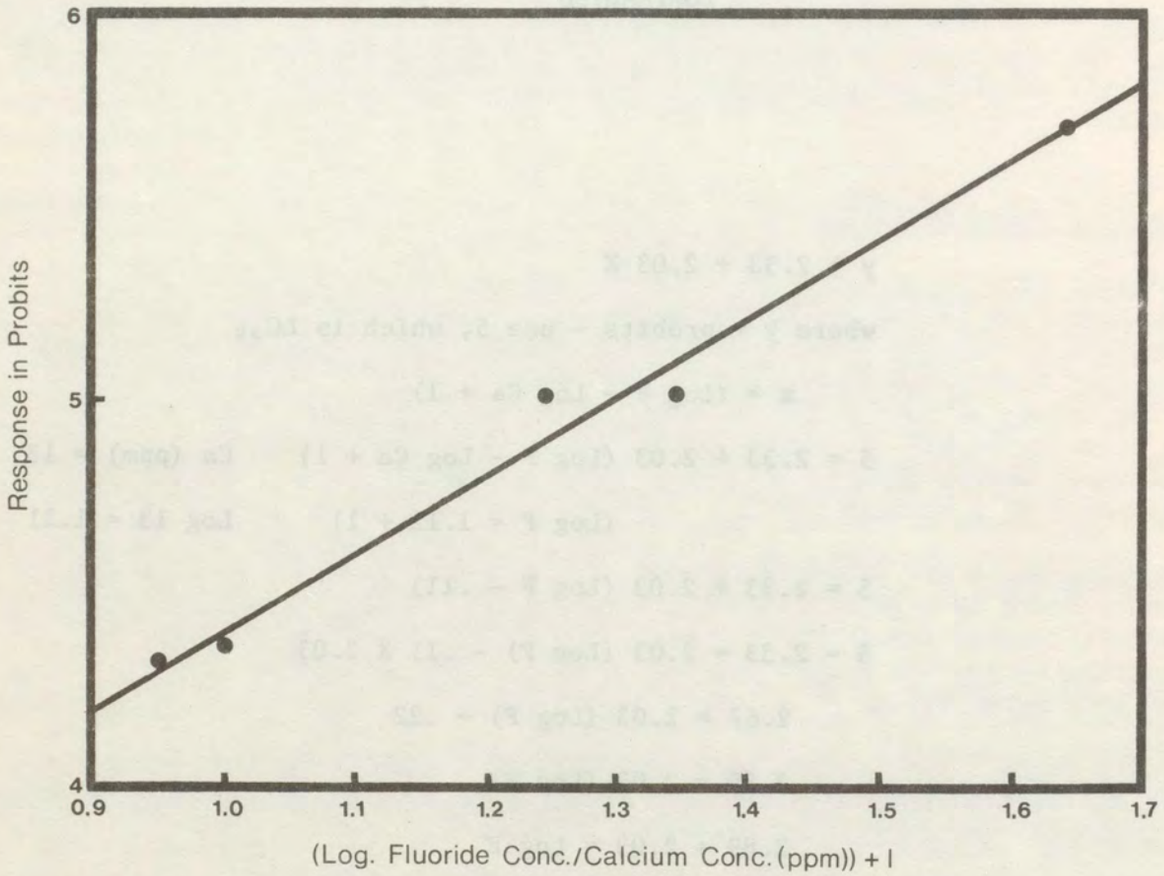

Figure 1. The response of rainbow trout to combinations of fluoride and calcium in the medium (expressed as the ratio between fluoride and calcium).

level. The sensitivity of the rainbow trout to the ratio of fluoride to calcium was between 1.71 and 2.35 probits of response per unit change in the log of the ratio. The relationship between the response and the log of the fluoride/calcium ratio (Figure 1) is expressed by the formula,

$$Y = 2.33 + 2.03X$$

where Y is the response in probits and X is the logarithm of the ratio between the fluoride concentration and the calcium ion concentration plus one unit characteristic.

A P P E N D I X II

THE PROCEDURES FOR CALCULATING NUMERICAL TEMPERATURE OBJECTIVES FOR FRESHWATER FISH

MAXIMUM WEEKLY AVERAGE TEMPERATURE

1. For Growth

The necessary minimum data for the determination of this objective are the physiological optimum temperature and the ultimate upper incipient lethal temperature. This latter temperature represents the breaking point between the highest temperatures to which an animal can be acclimated and the lowest of the extreme temperatures that will kill the warm-acclimated organism. Physiological optima can involve performance, metabolic rate, temperature preference, growth, natural distribution, or tolerance. However, the most sensitive function seems to be growth rate which appears to be an integrator of all factors affecting an organism. In the absence of data on optimum growth, the use of an optimum for a more specific function may be more desirable than not developing a growth objective at all.

MWAT for growth were calculated (Table 1) for fish species for which appropriate data were available. These data were obtained from the Fish Temperature Data Sheets in this Appendix. These data sheets contain the majority of thermal effects data for 34 species of freshwater fish and the sources of the data. In many instances no magic numbers jump off the page into the formula for the MWAT for growth. Some subjectivity is inevitable and necessary. For example, the data sheet for channel catfish includes four temperature ranges for optimum growth based on three published papers. It would be more appropriate to use data for growth of juveniles and adults rather than larvae. The middle of each range for juvenile channel catfish growth is 29 and 30 C. In this instance 29 C is judged the best estimate of the optimum. The highest upper incipient lethal temperature (that would approximate the ultimate upper incipient lethal temperature) is 38 C. Using the previous formula for the MWAT for growth:

$$29\ C + \frac{(38-29\ C)}{3} = 32\ C$$

The temperature objectives for the MWAT for growth of channel catfish would be 32 C (as appears in Table 1).

SHORT-TERM MAXIMUM DURING GROWTH SEASON

In addition to the MWAT, a short-term maximum temperature is necessary to protect against potential lethal effects. We have to assume that the incipient lethal data reflecting 50 percent survival necessary for this calculation would be based on an acclimation temperature near the MWAT for growth. Therefore,

Table 1. Objectives for Growth and Survival of Short-Exposures (24 hrs) of Juvenile and Adult Fish During the Summer, °C.

Species	Maximum Weekly Average[a] Temperature for Growth	Maximum Temperature for[b] Survival of Short Exposure
Alewife	--	--
Atlantic Salmon	20	23
Bigmouth Buffalo	--	--
Black Crappie	27	--
Bluegill	32	35
Brook Trout	19	24
Brown Bullhead	--	--
Brown Trout	17	24
Carp	--	--
Channel Catfish	32	35
Coho Salmon	18	24
Emerald Shiner	30	--
Fathead Minnow	--	--
Freshwater Drum	--	--
Lake Herring (Cisco)	17[c]	25
Lake Whitefish	--	--
Lake Trout	--	--
Largemouth Bass	32	34
Northern Pike	28	30
Pumpkinseed	--	--
Rainbow Smelt	--	--
Rainbow Trout	19	24
Sauger	25	--
Smallmouth Bass	29	--
Smallmouth Buffalo	--	--
Sockeye Salmon	18	22
Striped Bass	--	--
Threadfin Shad	--	--
Walleye	25	--
White Bass	--	--
White Crappie	28	--
White Perch	--	--
White Sucker	28[c]	--
Yellow Perch	29	--

a - Calculated according to the equation in the Recommendation (page 113).

b - Based on the equation in the Recommendation (page 114).

c - Based on data for larvae.

using the lethal threshold data for the channel catfish, we find four possible data choices near the MWAT of 32 C (again it is preferable to use data on juveniles or adults):

Acclimation temperature (°C)	a	b
30	32.1736	-0.7811
34	26.4204	-0.6149
30	17.7125	-0.4058
35	28.3031	-0.6554

The formula for calculating the short-term maximum is

$$\log (t) = a + b (T)$$

where t is the time in minutes and T is the temperature in °C.

Since this temperature objective is a mean weekly value, we will assume that an appropriate length of time one might expect a short-term maximum temperature to persist would be 24 hr. Any longer time would probably result in a violation of the mean weekly temperature.

Since the time is fixed at 24 hrs (1440 min), we need to solve for temperature:

$$\text{Temperature in °C} = \frac{\log 1440 - a}{b}$$

Upon solving for each of the four data points we obtain 37.1, 37.8, 35.9 and 38.4 C. The average would be 37.3 C and after subtracting the 2 C safety factor to provide 100 percent survival, the short-term maximum for channel catfish would be 35 C as appears in Table 1.

MAXIMUM WEEKLY AVERAGE TEMPERATURE FOR SPAWNING

These objectives are the easiest to determine. Using the data sheets in this Appendix, one would use either the optimum temperature for spawning or, if that is not available, the middle of the range of temperatures for spawning. Again, using the channel catfish data, the MWAT for spawning by the channel catfish would be 27 C (Table 2). Since spawning may occur over a period of a few months in a particular water body and only a MWAT for optimum spawning is estimated, it would be logical to use that optimum for the middle month of the spawning season. For a spring-spawning species, the MWAT for the next earlier month would approximate the lower temperature of the range in spawning temperature and the MWAT for the last month of a usual 3-month spawning season would approximate the upper temperature for the range. For example, if the channel catfish spawned from April

Table 2. Objectives for Spawning and Embryo Survival of Short-Exposures During the Spawning Season, °C.

Species	Maximum Weekly Average[a] Temperature for Spawning	Maximum Temperature for[b] Embryo Survival
Alewife	22	28[c]
Atlantic Salmon	5	11
Bigmouth Buffalo	17	27[c]
Black Crappie	17	20[c]
Bluegill	25	34
Brook Trout	9	13
Brown Bullhead	24	27
Brown Trout	8	15
Carp	21	33
Channel Catfish	27	29[c]
Coho Salmon	10	13[c]
Emerald Shiner	24	28[c]
Fathead Minnow	24	30
Freshwater Drum	21	26
Lake Herring (Cisco)	3	8
Lake Whitefish	5	10[c]
Lake Trout	9	14
Largemouth Bass	21	27[c]
Northern Pike	11	19
Pumpkinseed	25	29[c]
Rainbow Smelt	8	15
Rainbow Trout	9	13
Sauger	12	18
Smallmouth Bass	17	23[c]
Smallmouth Buffalo	21	28[c]
Sockeye Salmon	10	13
Striped Bass	18	24
Threadfin Shad	19	34
Walleye	8	17[c]
White Bass	17	26
White Crappie	18	23
White Perch	15	20[c]
White Sucker	10	20
Yellow Perch	12	20

a - The optimum or mean of the range of spawning temperatures reported for the species.

b - The upper temperature for successful incubation and hatching reported for the species.

c - Upper temperature for spawning.

to June the MWAT for the 3 months would be approximately 21, 27 and 29 C. For fall-spawning fish species the pattern or sequence of temperatures would be reversed due to natural declining temperatures during their spawning season.

SHORT-TERM MAXIMUM DURING SPAWNING SEASON

If these maxima were determined in the same manner as for the growing season, we would be using the time-temperature equation as before. However, these data are based usually on survival of juvenile and adult individuals. Egg incubation temperature requirements are more restrictive (lower) and this biological process would not be protected by maxima designed using data on juvenile and adult fish. Also, spawning itself could be prematurely stopped if those maxima were achieved. It is also likely that the maximum spawning temperature approximates the maximum successful incubation temperature.

Consequently, the short-term maximum temperature should preferably be based on maximum incubation temperature for successful embryo survival but the maximum temperature for spawning is probably an acceptable alternative. In fact, the higher of the two is probably the preferred choice as variability in available data may indicate discrepancies in this relationship.

For the channel catfish (see Fish Temperature Data Sheet) the maximum reported incubation temperature is 28 C and the maximum reported spawning temperature is 29 C. Therefore, the best estimate of the short-term survival of embryos would be 29 C (Table 2).

MAXIMUM WEEKLY AVERAGE TEMPERATURE FOR WINTER

As discussed earlier the MWAT for winter is necessary usually to prevent fish mortality in the event the water temperature drops rapidly to an ambient condition. This could occur due to power plant shutdown or a movement of the fish itself. These MWAT are meant to apply whatever fish can congregate, even if that is within the mixing zone.

Some stocks of yellow perch appear to require a long chill period during the winter for optimum egg maturation and spawning. However, protection of this species would be outside the mixing zone. In addition, the embryos of fall spawning fish such as trout, salmon and other related species such as cisco require low incubation temperature. For these species the MWAT during winter would have to consider embryo survival, but again, this would be outside the mixing zone.

With these exceptions in mind, it is unlikely that any significant effects on fish populations would occur as long as mortality was prevented. In many instances growth could be enhanced by winter heat addition.

There are fewer data for lower incipient lethal temperatures than for the previously discussed upper incipient lethal temperatures (NAS/NAE, 1973). Consequently, the data were combined to develop a generalized MWAT for winter survival rather than use the species specific approach as in the other types of objectives.

All the lower lethal threshold data for freshwater fish species were used to calculate a regression line. This line had a slope of 0.50 and a correlation coefficient of 0.75. This regression line was then displaced by approximately 2.5 C since it passed through the middle of the data points and did not represent the more sensitive species. This new line on the edge of the data array was then displaced by a 2 C safety factor, that same factor discussed earlier, to account for the fact that the original data points were for 50 percent survival and the 2 C safety factor should result in 100 percent survival. These two adjustments in the original regression line, therefore, result in a line (Figure 1) that should ensure no more than negligible mortality of any fish species. At lower temperatures the coldwater species appeared to be different than the warmwater species and the resultant objective (Figure 1) takes this into account.

If fish can congregate at a point close to the discharge, this objective could be a limit on the degree rise permissible at a particular site. Obviously, if there is a screened discharge channel in which some cooling occurs, the permissible plant heat rise could be greater.

An example of the use of this objective would be if the ambient water temperature were 10 C, the maximum temperature where fish could congregate would be 25 C, a difference of 15 C. At a lower ambient of about 2.5 C, the MWAT would be 10 C, a 7.5 C difference.

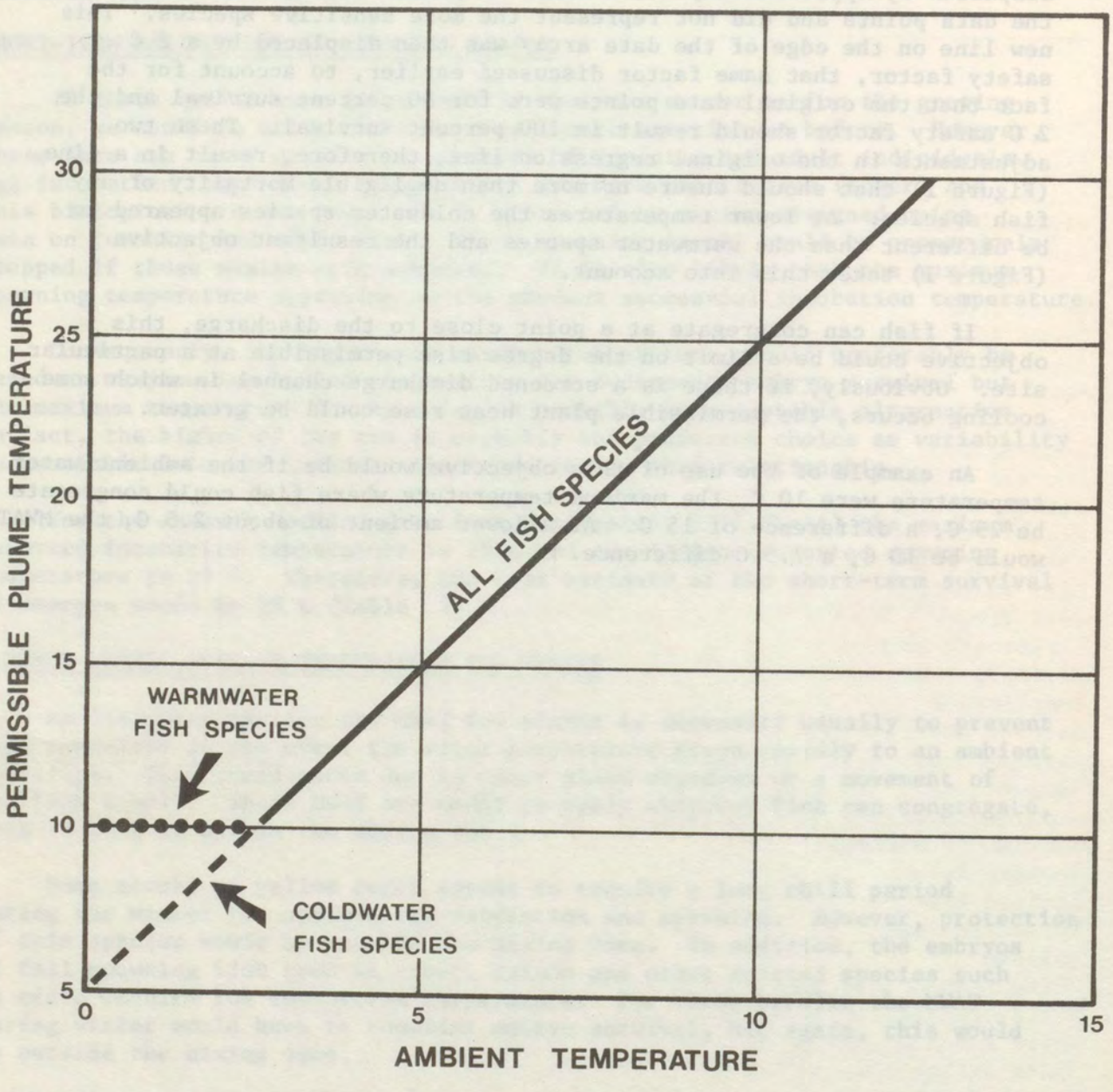

Figure 1. Nomograph to determine the maximum weekly average temperature of plumes for various ambient temperatures °C.

FISH TEMPERATURE DATA

Species: Alewife, *Alosa pseudoharengus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	10			20	5
	20			23	5
				32*	2
Lower					

*ultimate incipient

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration	13*(3)	7(1)**-? <10(1)-?		1 1,3
Spawning		16-28(1)	Apr-Aug(5)	1,5
Incubation		13-21**(1)		1
and hatch	17	11-27		1

*peak run

**marine stocks

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	24			23*	2
	31			23*	2
	18		20		4
	21		22		4

*age unknown

[1] References on following page.

Alewife

References

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FISH TEMPERATURE DATA

Species: Atlantic salmon, *Salmo salar*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	5		22*		1
	6	22			1
	10		23*		1
	20		23*		1
	27.5		27.8**		8
Lower					

*30 days ~~after~~ hatch
**ultimate upper incipient temp.

II. Growth:	larvae	juvenile	adult	
Optimum and [range]	10(9)	16-18(4)		4,9

III. Reproduction:	optimum	range	month(s)	
Migration	adults 23 or less,	smolt 10 or less		3
Spawning	4-6(3)	2-10(11)	Oct-Dec(7)	3,7,11
Incubation and hatch		3(3)-11(12)		3,12

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	4	14			2
	Summer		17(5)	14-16(6)	5,6
				14	10

[1] References on following page.

Atlantic salmon

References

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FISH TEMPERATURE DATA

Species: Bigmouth buffalo, *Ictiobus cyprinellus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	16-18(6)	14(1)-27(6)	Apr(4)-June(3)	1,3,4,6
Incubation and hatch		14(5)-17(2,5)		2,5

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				31-34*	7

**Ictiobus* sp. field

[1] References on following page.

Bigmouth buffalo

References

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FISH TEMPERATURE DATA

Species: Black crappie, *Pomoxis nigromaculatus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	29		33*		2
Lower					

*Ultimate incipient level

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		22-25		2
		(11-30)*		2

*Limits of zero growth

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		14(4)-20(3)	Mar(4)-July(3)	3,4
Incubation and hatch				

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Summer	18-20(5)		24-34(1)	1,5
			27-29*		6

*50% catch/effort

[1] References on following page.

Black crappie

References

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FISH TEMPERATURE DATA

Species: Bluegill, *Lepomis macrochirus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	15(2), 12(8)		27(8)	31(2)	2,8
	20			32	2
	25(2), 26(8)		36(8)	33(2)	2,8
	30		34		2
	33		37		8
Lower	15(2), 12(8)		3 (8)	3(2)	2,8
	20			5	2
	25(2), 26(8)		10(8)	7(2)	2,8
	30			11	2
	33		15		8

II. Growth:	larvae	juvenile	adult	
Optimum and		30(10)	24-27(3)	3,10
[range]		(22-34)(10)	[16(1)-30(4)]	1,4,10

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	25(5)	19(5)-32(6)	Feb(6)-Aug(1)	1,5,6
Incubation and hatch	22-24	22-34		8

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	26 Aug(11)		32(9,11)		9,11
	8 Nov		18		11
	3 Feb		16		11
	26 June		31		11
	30 June		32		7

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Bluegill

References

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FISH TEMPERATURE DATA

Species: Brook trout, *Salvelinus fontinalis*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	3		23		3
	11		25		3
	12	20*, 25**			2
	15		25		3
	20		25		3
Lower	25		25		3

*Newly hatched
**Swimup

II. Growth:	larvae	juvenile	adult	
Optimum and	12-15(2)		16(1)	1,2
[range]	(7-18)(2)		(10-19)(1)	1,2

III. Reproduction:	optimum	range	month(s)	
Migration			Sept(1)	
Spawning	<9(1)	2(6)-12(1)	Dec (5)	1,5,6
Incubation and hatch	6	?-13		1

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	6		12		4
	24		19		4

[1] References on following page.

Brook trout

References

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FISH TEMPERATURE DATA

Species: Brown bullhead, *Ictalurus nebulosus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	30		35		5
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		21(4)-?	Mar-Sept(3)	3,4
Incubation and hatch		21(4)-27(3)		3,4

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	18 May(2)		21(2)	29-31*(1)	1,2
	26 July		31		2
	23 Sept		27		2
	10 Mar		26		2

*final preferendum

[1] References on following page.

Brown bullhead

References

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FISH TEMPERATURE DATA

Species: Brown trout, *Salmo trutta*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	20(2)	23(2)		26*(5)	2,5
	23			25**	4
Lower					

*approx. ultimate upper incipient lethal
**age unknown

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		7-19*		4

*ages 0-IV

III. Reproduction:	optimum	range	month(s)	
Migration	6-7			1
Spawning	7-9(11)	1(7)-13(8)	Oct(9)-Jan(10)	7,8,9,10,11
Incubation and hatch	7-12(4)	5(4)-15(3)		3,4

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				12-18	6

[1] References on following page.

Brown trout

References

1. Stuart, T.A. 1953. Water currents through permeable gravels and their significance to spawning salmonids. Nature. 172:407-408.

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11. Scott, W.B. and E.J. Crossman. 1973. Freshwater fishes of Canada. Fish. Res. Bd. Can. Bull. 184. 966 p.

FISH TEMPERATURE DATA

Species: Carp, *Cyprinus carpio*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	20		31-34*		3
	26		36*		3
	25-27		40-41		10
Lower			*24 hr. TL_{50}		

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				
	(16-30)(9)			9

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	19-23(2)	14(4)-26(2)	Mar-Aug(5)	2,4,5
Incubation and hatch	17-22(7)	?-33(1)		1,7
	Limit for 10 min. exposure of early embryo is 35°			1

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	25-35		31-32		6
	Summer			33-35	8
	10		17		6

[1] References on following page.

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10. Horoszewicz, L. 1973. Lethal and "disturbing" temperatures in some fish species from lakes with normal and artifically elevated temperatures. J. Fish. Biol. 5:165-181.

FISH TEMPERATURE DATA

Species: Channel catfish, *Ictalurus punctatus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	15		30*		2
	25(2) 26(1)		37(1) 34(2)*		1,2
	29	31			3
	30		37		1
	34		38		1
Lower			*88-122 grams		
	15			0	2
	20			3	2
	25			6	2

II. Growth:	larvae	juvenile	adult	
Optimum and [range]	29-30(3) (27-31)(3)	28-30(8) (26-34)(4)		3,8
				3,4

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	27(5)	21-29(5)	Mar(10)-July(6)	5,6,10
Incubation and hatch		24-28(5)		5

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Summer			30-32*	7
	2 Jan(11)		11(11)	32**(9)	9,11
	22		35		11
	29		35		11

*field
**14-hr. photoperiod

[1] References on following page.

Channel catfish

References

1. Allen, K.O. and K. Strawn. 1968. Heat tolerance of channel catfish, *Ictalurus punctatus*. Proc. Conf. of S.E. Assoc. of Game and Fish Comm. 1967.

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9. Kilambi, R.V., J. Noble, and C.E. Hoffman. 1970. Influence of temperature and food conversion efficiency of the channel catfish. Proc. 24th Ann. S.E. Game and Fish Comm., p. 519-531.

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FISH TEMPERATURE DATA

Species: Coho salmon, *Oncorhynchus kisutch*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	5		23		1
	10		~~24(1)~~	~~21*(3)~~	~~1,3~~
	15		24		1
	20		25		1
	23		25		1
				*Accl. temp. unknown	
Lower	5		0.2		1
	10		2		1
	15		3		1
	20		5		1
	23		6		1

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		15*		2
		(5-17)**		6

*unlimited food
**depending upon season

III. Reproduction:	optimum	range	month(s)	
Migration		7-16		5
Spawning		7-13	Fall	3
Incubation and hatch	8(2)	?-11(7)		2,7

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Winter			13	4

[1] References on following page.

Coho salmon

References

1. Brett, J.R. 1952. Temperature tolerance in young Pacific salmon, genus *Oncorhynchus*. J. Fish. Res. Bd. Canada. 9:265-323.

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FISH TEMPERATURE DATA

Species: Emerald shiner, *Notropis atherinoides*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	5		23		1
	10		27		1
	15		29		1
	20		31		1
	25		31		1
Lower	15		2		1
	20		5		1

II. Growth:	larvae	juvenile	adult	
Optimum and		29		2
[range]		(24-31)		2

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		20(3)-28(5)	May-Aug(1,4)	1,3,4,5
Incubation and hatch				

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Summer		25*		3

*unknown age

[1] References on following page.

Emerald shiner

References

1. Carlander, R.D. 1969. Handbook of freshwater fishery biology. Vol. 1, Iowa State Univ. Press, Ames, Iowa.

2. McCormick, J.H. and C.F. Kleiner. 1976. Growth and survival of young-of-the-year emerald shiners (*Notropis atherinoides*) at different temperatures. Accepted for pub. J. Fish. Res. Bd. Canada.

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FISH TEMPERATURE DATA

Species: Fathead minnow, *Pimephales promelas*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]			23.5-30	1

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	23.5(1)	18(2)-30(1)	May-Aug(2)	1,2
Incubation and hatch	23-28	23.5-30		1

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	

[1] References on following page.

Fathead minnow

References

1. Brungs, W.A. 1971. Chronic effects of constant elevated temperature on the fathead minnow (*Pimephales promelas* Rafinesque). Trans. Am. Fish. Soc. 100:659-664.

2. Carlson, D.R. 1967. Fathead minnow, *Pimephales promelas* Rafinesque, in the Des Moines River, Boon County, Iowa, and the Skunk River Drainage, Hamilton and Story Counties, Iowa. Iowa State Jour. of Science. 41:363-374.

FISH TEMPERATURE DATA

Species: Freshwater drum, *Aplodinotus grunniens*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		18-24(4)	May(1)-Aug(3)	1,3,4
Incubation and hatch		22(2)-26(1)		1,2

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				29-31*	5

*Field

[1] References on following page.

Freshwater drum

References

1. Wrenn, B.B. 1969. Life history aspects of smallmouth buffalo and freshwater drum in Wheeler Reservoir, Alabama. Proc. 22nd Ann. Conf. S.E. Assoc. Game and Fish Comm., 1968. p. 479-495.

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FISH TEMPERATURE DATA

Species: Lake Herring (cisco), *Coregonus artedii*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	2(3), 3(2)	20(2)	20(3)	20(4)*	2,3,4
	5(3), <10(5)		22(3)	<24(5)	3,5
	>13		26		3
	20		26		3
	25		26		3
				*accl. temp. unknown	
Lower	2		0.3		3
	5		0.5		3
	10		3		3
	20		5		3
	25		10		3

II. Growth:	larvae	juvenile	adult	
Optimum and [range]	16 (13-18)			2
				2

III. Reproduction:	optimum	range	month(s)	
Migration	To spawning grounds at ≈ 5			7
Spawning	3(6,7)	1-5(8)	Nov-Dec(6)	6,7,8
Incubation and hatch	6(1)	2-8(1)	Nov(6)-May(8)	1,6,8

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				13	6

[1] References on following page.

Lake herring (cisco)

References

1. Colby, P.J. and L.T. Brooke. 1970. Survival and development of the herring (*Coregonus artedii*) eggs at various incubation temperatures. In: Biology of Coregonids, C.C. Lindsay and C.S. Woods, ed., Univ. Manitoba. pp. 417-428.

2. McCormick, J.H., B.R. Jones and R.F. Syrett. 1971. Temperature requirements for growth and survival of larval ciscos (*Coregonus arteddi*). J. Fish. Res. Bd. Canada. 28:924-927.

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5. Colby, P.J. and L.T. Brooke. 1969. Cisco (*Coregonus artedii*) mortalities in a Southern Michigan lake, July 1968. Limnology & Oceanog. 14:958-960.

6. Dryer, W.R. and J. Beil. 1964. Life history of lake herring in Lake Superior. U.S. Fish. Bull. 63:493-530.

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8. Carlander, K.D. 1969. Handbook of Freshwater Fishery Biology. Vol. 1, Iowa State Univ. Press, Ames, Iowa.

FISH TEMPERATURE DATA

Species: Lake trout, *Salvelinus namaycush*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		3-14(3)	Aug-Dec(2)	2,3
Incubation and hatch	8(1)	0.3-10(3)		1,3

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
			12*		4
			8-15**		5

*yearling
**age unknown

[1] References on following page.

Lake trout

References

1. Edsall, T.A. and W.E. Berlin. 1973. In: Progress in Fishery Research 1973. Great Lakes Fishery Laboratory, U.S. Fish and Wildlife Service. Eschmeyer, P.H. and J. Kutkuhn, eds.

2. Carlander, K.D. 1969. Handbook of freshwater fishery biology, Vol. 1, life history data on freshwater fishes of the United States and Canada, exclusive of the Perciformes. Iowa State Univ. Press, Ames, Iowa. 752 p.

3. Royce, W.F. 1951. Cited in: Brown, H.W. 1974. Handbook of the effects of temperature on some North American fishes. Amer. Elect. Power Service Corp., Box 487, Canton, Ohio.

4. McCauley, R.W. and J.S. Tait. 1970. Preferred temperature of yearling lake trout, *Salvelinus namaycush*. J. Fish. Res. Bd. Canada. 27:1729-1733.

5. Ferguson, R.G. 1958. The preferred temperature of fish and their midsummer distribution in temperate lakes and streams. J. Fish. Res. Bd. Canada. 15:607-624.

FISH TEMPERATURE DATA

Species: Lake whitefish, *Coregonus clupeaformis*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		0.5-10	Sept-Dec	2
Incubation and hatch	3-8			1

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				13*	3

*2 year old

[1] References on following page.

Lake whitefish

References

1. Brooke, L.T. 1975. Effect of different constant incubation temperatures on egg survival and embryonic development in lake whitefish (*Coregonus clupeaformis*). Trans. Amer. Fish. Soc. 3:555-559.

2. Carlander, K.D. 1969. Handbook of freshwater fishery biology, Vol. 1, life history data on freshwater fishes of the United States and Canada, exclusive of the Perciformes. Iowa State Univ. Press, Ames, Iowa. 752 p.

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FISH TEMPERATURE DATA

Species: Largemouth bass, *Micropterus salmoides*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	20		33		1
	25		35		1
	30		36		1
Lower	20		5		1
	25		7		1
	30		12		1

II. Growth:	larvae	juvenile	adult	
Optimum and	27(2)	30(8)		2,8
[range]	(20-30)(2)	(23-31)(8)		2,8
		29(10)	22(11)	10,11

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	21(4)	16-27(4)	Apr-June(3) Nov-May(4)	3,4
Incubation and hatch	20(5)	13(6)-26(9)		5,6,9

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
			30-32*		7
			27-28**		7

*Lab., small
**Field, larger

[1] References on following page.

Largemouth bass

References

1. Hart, J.S. 1952. Geographic variations of some physiological and morphological characters in certain freshwater fish. Univ. Toronto Biological Series No. 60.

2. Strawn, Kirk. 1961. Growth of largemouth bass fry at various temperatures. Trans. Amer. Fish. Soc. 90:334-335.

3. Kramer, R.H. and L.L. Smith, Jr. 1962. Formation of year class in largemouth bass. Trans. Amer. Fish Soc. 91:29-41.

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6. Kelley, J.W. 1968. Effeces of incubation temperature on survival of largemouth bass eggs. Prog. Fish. Cult. 30:159-163.

7. Ferguson, R.G. 1958. The preferred temperature of fish and their midsummer distribution in temperate lakes and streams. J. Fish. Res. Bd. Canada. 15:607-624.

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9. Carr, M.H. 1942. The breeding habits, embryology and larval development of the largemouth black bass in Florida. Proc. New Eng. Zool. Club. 20:43-77.

10. Johnson, M.G. and W.H. Charlton. 1960. Some effects of temperature on metabolism and activity of largemouth bass *Micropterus salmoides* Lacepede. Prog. Fish. Cult. 22:155-163.

11. Markus, H.C. 1932. Extent to which temperature changes influence food consumption in largemouth bass (*Huro floridans*). Trans. Am. Fish. Soc. 62:202-210.

FISH TEMPERATURE DATA

Species: Northern pike, *Esox lucius*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	18	25,28*			2
	25		32		1
	27		33		1
	30		33**		1
Lower					
	18	3*			2

*At hatch and free swimming, respectively
**Ultimate incipient level

*At hatch and free swimming

II. Growth:	larvae	juvenile	adult	
Optimum and	21	26		2
[range]	(18-26)			2

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		4(4)-18(3)	Feb-June(5)	3,4,5
Incubation and hatch	12	7-19		2

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
			24,26*		6

*Grass pickerel and musky, respectively

[1] References on following page.

Northern Pike

References

1. Scott, D.P. 1964. Thermal resistance of pike (*Esox lucius L.*) muskellunge (*E. masquinongy*, Mitchell) and their F_1 hybrid. J. Fish. Res. Bd. Canada. 21:1043-1049.

2. Hokanson, K.E.F., J.H. McCormick and B.R. Jones. 1973. Temperature requirements for embryos and larvae of the northern pike, *Esox lucius* (Linnaeus). Trans. Amer. Fish. Soc. 102:89-100.

3. Fabricus, E. and K.J. Gustafson. 1958. Some new observations on the spawning behavior of the pike, *Esox lucius* L. Rep. Inst. Freshwater Res., Drottningholm. 39:23-54.

4. Threinen, C.W., C. Wistrom, B. Apelgren and H. Snow. 1966. The northern pike, its life history, ecology, and management. Wis. Con. Dept. Publ. No. 235, Madison.

5. Toner, E.D. and G.H. Lawler. 1969. Synopsis of biological data on the pike *Esox lucius* (Linnaeus 1758). Food and Ag. Org. Fisheries synopsis No. 30, Rev. 1.

6. Ferguson, R.G. 1958. The preferred temperature of fish and their mid-summer distribution in temperate lakes and streams. J. Fish. Res. Bd. Canada. 15:607-624.

FISH TEMPERATURE DATA

Species: Pumpkinseed, *Lepomis gibbosus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]			30	1
			15-?	1

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		20-29	May-Aug	3
Incubation and hatch				

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	19 May		21		2
	24 June		31		2
	26 Sept		33		2
	8 Nov		10		2

[1] References on following page.

Pumpkinseed

References

1. Pessah, E. and P.M. Powles. 1974. Effect of constant temperature on growth rates of pumpkinseed sunfish (*Lepomis gibbosus*). J. Fish. Res. Bd. Canada. 31:1678-1682.

2. Peterson, S.E., R.M. Schtusky and S.E. Allison. 1974. Temperature preference, avoidance and shock experiments with freshwater fishes and crayfishes. Ichthyological Associates, Bulletin 10. Ichthyological Associates, Inc., Box 12, Drumore, Pa.

3. Breder, C.M., Jr. 1936. Cited in: Brown, H.W. 1974. Handbook of the effects of temperature on some North American fishes. Amer. Elect. Power Service Corp., Box 487, Canton, Ohio.

FISH TEMPERATURE DATA

Species: Rainbow smelt, *Osmerus mordax*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration	4-5			1
Spawning		0.6-15	April	2
Incubation and hatch		5-15		3

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				6-14	4

[1] References on following page.

Rainbow smelt

References

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FISH TEMPERATURE DATA

Species: Rainbow trout, *Salmo gairdneri*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	18		27		1
	19			21	2
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		17-19		5
	[3(8)-20(11)]			8,11

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	9(10)	5-13(6)	Nov-Feb(7) Feb-June(7)	6,7,10
Incubation and hatch	5-7(9)	5-13(4)		4,9

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Not given		14		3
		13-20	13-19		11
	18&24		18&22, resp.		12

[1] References on following page.

References

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FISH TEMPERATURE DATA

Species: Sauger, *Stizostedion canadense*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	10		27		4
	12		27		4
	18		29		4
	22		30		4
Lower	26		30		4

II. Growth:	larvae	juvenile	adult	
Optimum and		22		4
[range]		(16-26)		4

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	9-15(4)*	6(1)-15(4)	Apr(1)-June(3)	1,3,4
Incubation and hatch	12-15	9-18		4

*for fertilization

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				19*	2
				27-29	5

*field

[1] References on following page.

Sauger

References

1. Nelson, W.R. 1968. Reproduction and early life history of sauger, *Stizostedion canadense*, in Lewis and Clark Lake. Trans. Amer. Fish. Soc. 97:159-166.

2. Ferguson, R.G. 1958. The preferred temperature of fish and their midsummer distribution in temperate lakes and streams. J. Fish. Res. Bd. Canada. 15:607-624.

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FISH TEMPERATURE DATA

Species: Smallmouth bass, *Micropterus dolomieui*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper		38*(8)	35(3)		8,3
		*acclimation not given			
Lower	15(3)	4(8)*	2(3)		3,8
	18		4		3
	22		7		3
	26		10		3
		*acclimation temperature not given			

II. Growth:	larvae	juvenile	adult	
Optimum and [range]	28-29(2)	26(3)		2,3

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	17-18(5)	13-23(9)	May-June(7)	5,7,9
Incubation and hatch		13-22		10

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Summer			21-27	6
	Winter			>8*(1)-28(4)	1,4
	18&30		23&31 resp.		11
			*juvenile and adult		

[1] References on following page.

Smallmouth bass

References

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2. Peek, F.W. 1965. Growth studies of laboratory and wild population samples of smallmouth bass. Master's Thesis, Univ. Arkansas.

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11. Cherry, D.S., K.L. Dickson and J. Cairns, Jr. 1975. Temperatures selected and avoided by fish at various acclimation temperatures. Jour. Fish. Res. Bd. Canada. 32:485-491.

FISH TEMPERATURE DATA

Species: Smallmouth buffalo, *Ictiobus bubalus*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	17(1)-24(5)	14(1)-28(5)	Mar(3)-Sept(5)	1,3,5
Incubation and hatch		14(1)-21(2)		1,2

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				31-34*	4

**Ictiobus* sp. field

[1] References on following page.

Smallmouth buffalo

References

1. Wrenn, W.B. 1969. Life history aspects of smallmouth buffalo and freshwater drum in Wheeler Reservoir, Alabama. Proc. 22nd Ann. Conf. S.E. Assoc. Game & Fish Comm., 1968. pp. 479-495.

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5. Jester, D.B. 1973. Life history, ecology, and management of the smallmouth buffalo, *Ictiobus bubalus* (Rafinesque), with reference to Elephant Butte Lake. Ag. Exp. Sta. Res. Dept. 261, New Mexico State Univ., 111 p.

FISH TEMPERATURE DATA

Species: Sockeye salmon, *Oncorhynchus nerka*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	5		22		1
	10		23		1
	15		24		1
	20		25		1
Lower	5		0		1
	10		3		1
	15		4		1
	20		5		1
	23		7		1

II. Growth:	larvae	juvenile	adult	
Optimum and [range]	15(5)	15(2)*		2,5
		(10-15)		4
		(11-17)		7

*Max. with excess food

III. Reproduction:	optimum	range	month(s)	
Migration		7-16		4
Spawning		7-13	Fall	6
Incubation and hatch				

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Summer		15		3

[1] References on following page.

Sockeye salmon

References

1. Brett, J.R. 1952. Temperature tolerance in young Pacific salmon, genus, *Oncorhynchus*. J. Fish. Res. Bd. Can. 9:265-323.

2. Griffiths, J.S. and D.F. Alderdice. 1972. Effects of acclimation and acute temperature experience on the swimming speed of juvenile coho salmon. J. Fish. Res. Bd. Can. 29:251-264.

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FISH TEMPERATURE DATA

Species: Striped bass, *Morone saxatilis*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	not given		35*	28**	2
Lower					

*Laboratory
**Field observation

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration		6-8		2
Spawning	16-19(2)	12-22(1)	Apr-June(1)	1,2
Incubation and hatch		16-24		1

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	5 Dec		12		3
	14 Nov		22		3
	21 Oct		26		3
	28 July		28		3

[1] References on following page.

Striped bass

References

1. Shannon, E.H. 1970. Effect of temperature changes upon developing striped bass eggs and fry. Proc. 23rd Conf. S.E. Assoc. Game and Fish Comm. October 19-22, 1969, pp. 265-274.

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3. Meldrim, J.W., J.J. Gift, and B.R. Petrosky. 1974. Supplementary data on temperature preference and avoidance responses and shock experiments with estuarine fishes and macroinvertebrates. Ichthyological Associates, 56 p. mimeo. Ichthyological Associates, Inc., Middletown, Delaware.

FISH TEMPERATURE DATA

Species: Threadfin shad, *Dorosoma petenense*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower			9*		1

*lowest permitting some survival

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		14(3)-23(4)	Apr-Aug(4)	3,4
Incubation and hatch		23(4)-34(5)		4,5

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				>19	2

[1] References on following page.

Threadfin shad

References

1. Strawn, K. 1963. Resistance of threadfin shad to low temperatures. Proc. 17th Ann. Conf. Southeastern Assoc. of Game and Fish Comm. pp. 290-293.

2. Adair, W.D. and D.J. DeMont. 1970. Effects of thermal pollution upon Lake Norman fishes. N. Caroline Wildlife Res. Comm., Div. Inland Fisheries. Summary Report, Fed. Aid Fish Restoration Project F-19-2. 14 p.

3. Carlander, K.D. 1969. Handbook of freshwater fishery biology. The Iowa State Univ. Press, Ames, Iowa.

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FISH TEMPERATURE DATA

Species: Walleye, *Stizostedion vitreum*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	12		29		1
	16		31		1
	22		31		1
Lower	26		31		1

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		22(1)	20(6)	1,6
		(16-28)		1

III. Reproduction:	optimum	range	month(s)	
Migration		3-7		4
Spawning	6-9(1)*	4(7)-17(5)	Apr-May(4)	1,5,7,4
Incubation and hatch	9-15			1

*for fertilization

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				23*	2
			22-25(1)	25(3)*	1,3

*field

[1] References on following page.

Walleye

References

1. Smith, L.L. Jr., and W.M. Koenst. 1975. Temperature effects on eggs and fry of percoid fishes. USEPA Ecological Research Series Report 66013-75-017.

2. Ferguson, R.G. 1958. The preferred temperature of fish and their mid-summer distribution in temperate lakes and streams. J. Fish. Res. Bd. Canada. 15:607-624.

3. Dendy, J.S. 1948. Predicting depth distribution of fish in three TVA storage reservoirs. Trans. Am. Fish. Soc. 75(1945):65-71.

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FISH TEMPERATURE DATA

Species: White bass, *Morone chrysops*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower	17	14*			3

*% mortality not given

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		24-30		7

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		14-20 (north) 12-? (Tenn)	May-June (north) Mar-May (Tenn)	4 1
Incubation and hatch		16(2)-26(6)		2,6

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Summer			28-30*	5

*Field

[1] References on following page.

White bass

References

1. Webb, J.F. and D.D. Moss. 1967. Spawning behavior and age and growth of white bass in Center Hill reservoir, Tennessee. Master's Thesis, Tenn. Tech. Univ.

2. Yellayi, R.R. 1972. Ecological life history and population dynamics of white bass, *Morone chrysops* (Rafinesque) in Beaver Reservoir. Part 2, Report to Arkansas Game and Fish Commission

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4. Ruelle, R. 1971. Factors influencing growth of white bass in Lewis and Clark Lake. In: Reservoir Fisheries and Limnology, G. Hall, ed., pp. 411-423.

5. Gammon, J.R. 1973. The effect of thermal input on the populations of fish and macroinvertebrates in the Wabash River. Tech. Rept. 32, Purdue Univ. Water Resources Research Center.

6. McCormick, J.H. Temperature effects on white bass (*Morone chrysops*) embryo development, and survival of one-day-old larvae. Unpublished ms., Environmental Research Laboratory-Duluth, Duluth, Minn.

7. McCormick, J.H. Temperature effects on the growth of juvenile white bass. Unpublished data. Environmental Research Laboratory-Duluth, Duluth, Minn.

FISH TEMPERATURE DATA

Species: White crappie, *Pomoxis annularis*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	29		33		4
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		25		4

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	16-20(5)	14-23(5)	Mar-July(3)	3,5
Incubation and hatch	19	14-23		5
	Hatch in 24-27-1/2 hrs. at 21-23			2

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	27 July(6)		28(6)	28-29(1)	1,6
	3 Jan		8		6
	5 Mar		10		6
	24 June		26		6

[1] References on following page.

White crappie

References

1. Gammon, J.R. 1973. The effect of thermal input on the populations of fish and macroinvertebrates in the Wabash River. Tech. Rept. 32, Purdue Univ. Water Resources Research Center.

2. Breder, C.M. and D.E. Rosen. 1966. Modes of reproduction in fishes. Nat. History Press.

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4. Kleiner, C.F. and K.E.F. Hokanson. Effects of constant temperature on growth and mortality rates of juvenile white crappie, *Pomoxix annularis* Rafinesque. Unpublished data, Environmental Research Laboratory-Duluth, Duluth, Minnesota.

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6. Peterson, S.E., R.M. Schutsky, and S.E. Allison. 1974. Temperature preference, avoidance, and shock experiments with freshwater fishes and crayfishes. Ichthyological Associates, Bulletin 10. Ichthyological Associates, Inc., Box 12, Drumore, Pa.

FISH TEMPERATURE DATA

Species: White perch, *Morone americana*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper					
Lower					

II. Growth:	larvae	juvenile	adult	
Optimum and [range]				

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning		11(3)-20(1)	May-June(3)	1,3
Incubation and hatch				

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	6		10		2
	15		20		2
	20		25		2
	26-30		31-32		2

[1] References on following page.

White perch

References

1. Holsapple, J.G. and L.E. Foster. 1975. Reproduction of white perch in the lower Hudson River. New York Fish and Game Jour. 22:122-127.

2. Meldrim, J.W., J.J. Gift and B.R. Petrosky. 1974. Supplementary data on temperature preference and avoidance responses and shock experiments with estuarine fishes and macroinvertebrates. Ichthyological Associates Rept., 56 p. mimeo. Ichthyological Associates, Inc., Middletown, Delaware.

3. Sheri, A.N. and G. Power. 1968. Reproduction of white perch, *Roccus americana*, in the bay of Quinte, Lake Ontario. J. Fish. Res. Bd. Canada. 25:2225-2231.

FISH TEMPERATURE DATA

Species: White sucker, *Catostomus commersoni*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	5		26(2)		2
	10	28(1)*	28(2)		1,2
	15	31(1)	29(2)		1,2
	20(2), 21(1)	30(1)	29(2)		1,2
	25		29		2
	25-26		31		3
Lower		*7-day TL50 for swimup			
	20		2-3		2
	21	6*			1
	25		6		1

*7-day TL50 for swimup

II. Growth:	larvae	juvenile	adult	
Optimum and	27			1
[range]	(24-27)			1

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	~10(5)	~4-18(5,6)	Mar-June(2)	2,5,6
Incubation and hatch	15	9-20		1

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
				19-21	4

[1] References on following page.

White sucker

References

1. McCormick, J.H., B.R. Jones, and K.E.F. Hokanson. 1976. Temperature effects on embryo development, early growth, and survival of the white sucker, *Catostomus commersoni* (Lacepede). Accepted for publication. J. Fish. Res. Bd. Canada.

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6. Raney, E.C. 1943. Unusual spawning habitat for the common white sucker *Catostomus c. commersonii*. Copeia. 4:256.

FISH TEMPERATURE DATA

Species: Yellow perch, *Perca flavescens*

I. Lethal threshold:	acclimation temperature	larvae	juvenile	adult	reference[1]
Upper	5			21	1
	10(1), 10(4)	10(4)*		25(1)	1,4
	15(1), 20(4)	19(4)*		28(1)	1,4
	25	*swimup		32	10
Lower	25		9		10

II. Growth:	larvae	juvenile	adult	
Optimum and [range]		28		11
		(26-30)(11)	[13(6)-20(7)]	6,7,11

III. Reproduction:	optimum	range	month(s)	
Migration				
Spawning	12(3)	2(5)-15(3)	Mar-June(3)	3,5
Incubation and hatch	10 up 1°/day to 20	7-20		4

IV. Preferred:	acclimation temperature	larvae	juvenile	adult	
	Winter			21(2)	2
	Summer		24		2
	24		20-23	18-20	9
	25		22		8
	7		19		8
	2		20		8

[1] References on following page.

Yellow perch

References

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EXAMPLES

Again, it is necessary at times to make subjective decisions based on knowledge of existing aquatic systems and common sense. For some fish species for which there may be few or relatively poor data, this subjectivity becomes important. Even if several people were to calculate various temperature objectives for species with numerous, high quality data, it is likely that they would not agree in all instances.

The following examples for warmwater and coldwater species are presented only as examples and are not at all intended to be waterbody-specific recommendations. Local extenuating circumstances may warrant differences, or the basic conditions of the examples may be slightly unrealistic.

EXAMPLE I

Tables 1 and 2, Figure 1, and NAS/NAE (1973) are the principal data sources for the objectives for this example. The following waterbody-specific data are necessary and in this example are not precisely factual:

1. Species to be protected by the objectives: They will be the channel catfish, largemouth bass, bluegill, white crappie, freshwater drum and bigmouth buffalo.

2. Local spawning seasons for these species: They are April to June for the white crappie and the bigmouth buffalo. For the other species the spawning season is from May to July.

3. Normal ambient winter temperature: In this example it will be 5 C in December and January; and 10 C in November, February and March.

4. The principal growing season for these fish species: This would be July through September.

5. Any local extenuating circumstances should be incorporated into the objectives as appropriate. Some examples would be yellow perch gamete maturation in the winter, very temperature sensitive endangered species, or important fish food organisms that are very temperature sensitive. For the example we will have no extenuating circumstances.

In some instances there will be insufficient data to determine each necessary objective for each species. One must make estimates based on any available data or by extrapolation from data for species for which there are adequate data. For instance, this includes the bigmouth buffalo and freshwater

drum for which no growth or short-term summer maxima are available (Table 1). One would of necessity have to estimate that its summer objective would not be lower than that for the white crappie which has a spawning requirement as low as for the other two species.

The choice of important fish species is very critical. Since in this example the white crappie is as temperature sensitive as any of the species, the maximum weekly average temperature for summer growth is based on the white crappie. Consequently, this objective would result in lower than optimal conditions for the channel catfish, bluegill and largemouth bass. An alternate approach would be to develop objectives for the single most important species even if the most sensitive is not well protected. The choice is a socioeconomic one.

Before developing a set of objectives such as in Table 3, one should study the material in Tables 1 and 2 for the species of concern. In this example it is evident that the lowest objective for summer growth for the species for which data are available, would be the white crappie (28 C). However, there is no short-term maximum since the data are not available (see Fish Temperature Data Sheets). For the species for which there are data, the lowest short-term maximum is for the largemouth bass (34 C). In this example, we have all the necessary data for spawning and short-term maxima for embryo survival for all species of concern (Table 2).

During the winter, objectives may be necessary both for the heated plume or mixing zone as well as for the receiving water. Receiving water objectives would be necessary if an important fish species were known to have gamete maturation requirements like the yellow perch, or embryo incubation requirements like trout, salmon, cisco, etc. In this example there is no need for receiving water objectives in the winter.

At this point, we are ready to complete Table 3 for our first example.

TABLE 3. Objectives for Example 1

Month	Maximum Weekly Average Temperature (°C) Receiving Water	Heated Plume	Decision Basis
January	--[a]	15	Figure 1
February	--[a]	25	Figure 1
March	--[a]	25	Figure 1
April	18	---	White crappie spawning
May	21	--	Largemouth bass spawning
June	25	--	Bluegill spawning and white crappie growth
July	28	--	White crappie growth
August	28	--	White crappie growth
September	28	--	White crappie growth
October	21	--	Normal gradual seasonal decline
November	--[a]	25	Figure 1
December	--[a]	15	Figure 1

Month	Short-term Maximum	Decision Basis
January	None needed	--
February	None needed	--
March	None needed	--
April	26	Largemouth bass[b] survival (estimated)
May	29	Largemouth bass[b] survival (estimated)
June	34	Largemouth bass[b] survival
July	34	Largemouth bass[b] survival
August	34	Largemouth bass[b] survival
September	34	Largemouth bass[b] survival
October	29	Largemouth bass[b] survival
November	None needed	--
December	None needed	--

[a] If a species had required a winter chill period for gamete maturation of egg incubation, receiving water objectives would also be required.

[b] No data available for the slightly more sensitive white crappie.

EXAMPLE 2

All of the general concerns and data sources presented throughout the discussion and derivation of Example 1 will apply here.

1. Species to be protected by the objectives: They will be the rainbow and brown trout and the coho salmon.

2. Local spawning seasons for these species: They are November through January for rainbow trout; and November through December for the brown trout and coho salmon.

3. Normal ambient winter temperature: In this example it will be 2 C in November through February and 5 C in October, March and April.

4. The principal growing season for these fish species: This would be June through September.

5. Consider any local extenuating circumstances: There are none in this example.

TABLE 4. Objectives for Example 2

Month	Maximum Weekly Average Temperature (°C) Receiving Water	Heated Plume	Decision Basis
January	9	10	Rainbow trout spawning and Figure 1
February	13	10	Normal gradual seasonal rise and Figure 1
March	13	15	Normal gradual seasonal rise and Figure 1
April	14	15	Normal gradual seasonal rise and Figure 1
May	16	--	Normal gradual seasonal rise
June	17	--	Brown trout growth
July	17	--	Brown trout growth
August	17	--	Brown trout growth
September	17	--	Brown trout growth
October	12	15	Normal gradual seasonal decline
November	8	10	Brown trout spawning and Figure 1
December	8	10	Brown trout spawning and Figure 1

Month	Short-term Maximum	Decision Basis
January	13	Embryo survival for rainbow trout and coho salmon
February	13	Embryo survival for rainbow trout and coho salmon
March	13	Embryo survival for rainbow trout and coho salmon
April	--	--
May	--	--
June	23	Short-term maximum for brown trout survival
July	23	Short-term maximum for brown trout survival
August	23	Short-term maximum for brown trout survival
September	23	Short-term maximum for brown trout survival
October	--	--
November	13	Embryo survival for rainbow trout and coho salmon
December	13	Embryo survival for rainbow trout and coho salmon