RATIONALE FOR THE ESTABLISHMENT OF ONTARIO'S PROVINCIAL WATER QUALITY OBJECTIVES

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INTRODUCTION

This is a companion publication to the Ministry of the Environment booklet entitled "Water Management - Goals, Policies, Objectives and Implementation Procedures of the Ministry of the Environment.

Table 1 in that booklet contains the Provincial Water Quality Objectives, a set of numerical and narrative criteria designed for the protection of aquatic life and recreation. These Objectives represent a desirable level of water quality that the Ministry strives to maintain in the Province. The scientific rationale for the Objectives have been compiled in this publication.

APPROACH TO ESTABLISHING THE PROVINCIAL WATER QUALITY OBJECTIVES

Staff of Ministry of the Environment have reviewed and compared water quality objectives, criteria and standards established by Canadian, American and other agencies. The majority of the Provincial Water Quality Objectives adopted in this publication were established by the Water Quality Objectives Subcommittee of the International Joint Commission, and by the U. S. Environmental Protection Agency. The use of the documentations prepared by these two agencies is gratefully acknowledged. The basic approach to establishing the Provincial Water Quality Objectives is given below.

The Provincial Water Quality Objectives for protection of recreational water uses are based on public health and aesthetic considerations. With respect to aquatic life, the objectives are set at such values as to protect all forms of aquatic life and all aspects of the aquatic life cycles. The clear intention is to protect all life stages during indefinite exposure to the water. With the exception of dissolved oxygen, temperature classification of organisms is not considered relevant to the application of criteria. Numerous studies have found that the biological variation of response within species to a toxicant is as great or greater than the biological variation among species. Consequently, criteria will be applied equally to "warm water" species and "cold water aquatic organisms". This approach will also incorporate the variation of sensitivity occurring when "warm water" species are exposed to contaminants under low temperature conditions and when "cold water" species are exposed under high temperature conditions.

Ideally, water quality objectives should be established based on "no negative effect" data derived from chronic long-term tests on sensitive organisms. However, current understanding of chemical dynamics and effects on aquatic life are limited to a few species and contaminant levels that are lethal in short-term tests.

Therefore, the numerical Objectives are generally derived from short-term toxicity data and "application factors". Data from short-term toxicity tests are usually expressed as median lethal concentrations (LC_{50}), indicating the concentration of *test* material that caused death to 50 percent of the test organisms within a given period of time. An application factor can be defined as the ratio of the maximum concentration having no negative effect on the test organism to the 96 hour LC_{50} concentration. This approach to establishing water quality objectives is not ideal, but is a practical method of making an estimate of a safe concentration from limited toxicity data. The application factors commonly used are:

> Concentration of materials that are nonpersistent or have noncumulative effects should not exceed 0.1 of the 96-hour LC_{50} at any time or place after mixing with the receiving waters. The 24-hour average of the concentration of these materials should not exceed 0.05 of the LC_{50} after mixing.
>
> For toxicants which are persistent or cumulative the concentrations should not exceed 0.05 of the 96-hour LC_{50} at any time or place, nor should the 24-hour average concentration exceed 0.01 of the 46-hour LC_{50}.

The Objectives represent minimum water quality conditions and are based on data produced by exposing healthy, stress-free organisms to one variable at a time. The Objectives do not account for additive effects of more than one chemical nor for additional environmental stress arising from temperature and predation factors.

Consequently, water with a quality at or near the Objectives for several parameters may not protect aquatic life because of synergistic effects.

The scientific data base used in setting these Objectives is constantly changing. The Objectives will be reviewed on a continuing basis and revised whenever warranted.

RATIONALE FOR INORGANICS, HEAVY METALS AND OTHER PARAMETERS

ALKALINITY AND pH

OBJECTIVE

Alkalinity should not be decreased by more than 25 percent of the natural concentrations. The pH should be maintained within the range of 6.5 - 8.5.

RATIONALE

NATURAL CONDITIONS AND SIGNIFICANCE

Acidity in natural waters is caused by carbon dioxide, mineral acids, weakly dissociated acids, and the salts of strong acids and weak bases. The alkalinity of a water is actually a measure of the capacity of the carbonate-bicarbonate system to buffer the water against change in pH. Technical information on alkalinity has recently been reviewed by Kemp (1971).

An index of the hydrogen ion activity is pH. Even though pH determinations are used as an indication of acidity or alkalinity or both, pH is not a measure of either. There is a relationship between pH, acidity, and alkalinity (Standard Methods 1971): water with a pH of 4.5 or lower has no measurable alkalinity, and water with a pH of 8.3 or higher has no measurable acidity. In natural water, where the pH may often be in the vicinity of 8.3, acidity is not a factor of concern. In most productive fresh waters, the pH falls in a range between 6.5 and 8.5 (except when increased by photosynthetic activity). Some regions have soft waters with poor buffering capacity and naturally low pH. They tend to be less productive. Such conditions are found especially in dark colored waters draining from coniferous forests or muskegs, and in swampy sections of the Southeast. For a variety of reasons, some waters may exhibit quite extreme pH values. Before these are considered natural conditions, it should be ascertained that they have not actually resulted from man-made changes, such as stripping of ground cover or old mining activities. This is important because the recommendations refer to estimated natural levels.

TOXICITY TO AQUATIC LIFE

Some aquatic organisms, especially algae, have been found to live at pH 2 and lower, and others at pH 10 and higher; however, such organisms are relatively few. Some natural waters with a pH of 4 support healthy populations of fish and other organisms. In these cases the acidity is due primarily to carbon dioxide and natural organic acids, and the water has little buffering capacity. Other natural waters with a pH of 9.5 also support fish but are not usually highly productive.

The effects of pH on aquatic life have been reviewed in detail in excellent reports by the European inland Fisheries Advisory Commission (1969) and Kata (1969). Interpretations and summaries of these reviews are given in Table 1.

ADVERSE INDIRECT EFFECTS OR SIDE EFFECTS

Addition of either acids or alkalies to water may be harmful not only by producing acid or alkaline conditions, but also by increasing the toxicity of various components in the waters. For example, acidification of water may release free carbon dioxide. This exerts a toxic action additional to that of the lower pH. Recommendations for pH are valid if carbon dioxide is less than 25 mg/L.

A reduction of about 1.5 pH units can cause of thousandfold increase in the acute toxicity of a metallocyanide complex (Deudoroff et al 1966). The addition of strong alkalies may cause the formation of undissociated NH_4OH or un-ionized NH_3 in quantities that may be toxic (Lloyd 1961, Burrows 1964). Many other pollutants may change their toxicity to a lesser extent. It is difficult to predict whether toxicity will increase or decrease for a given direction of change in pH.

Weakly dissociated acids and bases must be considered in terms of their toxicities, as well as their effects on pH and alkalinity.

The availability of many nutrient substances varies with the hydrogen ion concentration. Some trace metals become more soluble at low pH. At higher pH values, iron tends to become unavailable to some plants, and hence the production of the whole aquatic community may be affected.

The major buffering system in natural waters is the carbonate system that not only neutralizes acids and bases to reduce the fluctuations in pH, but also forms a reservoir of carbon for photosynthesis.

This process is indispensable, because there is a limit on the rate at which carbon dioxide can be obtained from the atmosphere to replace that in the water. Thus the productivity of waters is closely correlated to the carbonate buffering system. The addition of mineral acids preempts the carbonate buffering capacity, and the original biological productivity is reduced in proportion to the degree that such capacity is exhausted. Therefore, the minimum essential buffering capacity and tolerable pH limits are important water quality considerations.

Because of this importance, there should be no serious depletion of the carbonate buffering capacity, and it is recommended that reduction of alkalinity of natural waters should not exceed 25 per cent.

TABLE 1: A Summary of Some Effects of pH on Freshwater Fish and Other Aquatic Organisms

pH	Known Effects
11.5 - 12.0	Some caddis flies (Trichloptera) survive but emergence reduced.
11.0 - 11.5	Rapidly lethal to all species of fish.
10.5 -11.0	Rapidly lethal to salmonids. The upperlimit is lethal to carp *(Cyprinus carpio*),goldfish (*Carassius auratus*),one pike. Lethal to some stoneflies Plecoptera)and dragonflies (odonata). Caddis fly emergence reduce.
10.0 -10.5	Withstood by salmonids for short periods but eventually lethal. Exceeds tolerance of bluegills (*Lepomis machrochirus*) and probably goldfish. Some typical stoneflies and mayflies (Ephemera) survive with reduced emergence.
9.5 - 10.0	Lethal to salmonids over a prolonged period of time and no viable fishery for coldwater species. Reduces populations of warmwater fish and may be harmful to development stages. Causes reduced emergence of some stoneflies.
9.0 - 9.5	Likely to be harmful to salmonids and perch (Perca) if present for a considerable length of time and no viable fishery for coldwater species. Reduced populations of warmwater fish. Carp avoid these levels.
8.5 - 9.0	Approaches tolerance limit of some salmonids, whitefish (*Coregonus*), catfish (*Ictaluridae*),and perch. Avoided by goldfish. No apparent effects on invertebrates.
8.0 - 8.5	Mobility of carp sperm reduced. Partial mortality of burbot (*Lota Iota*) eggs.
7.0 - 8.0	Full fish production. No known harmful effects on adult or immature fish, but is a Gammarus reproduction and perhaps for some other crustaceans.
6.5 - 7.0	Not lethal to fish unless heavy metals or cyanides that are at low pH are present. Generally full fish production, but for fathead minnow *(Pimephales promelas*),frequency of spawning and number of eggs are somewhat reduced. Invertebrates except crustaceans relatively normal, including common occurrence of mollusks. Microorganisms, algae, and higher plants essentially normal.

6.0 - 6.5 Unlikely to be toxic to fish unless free carbon dioxide is present in excess of 100 ppm. Good aquatic populations with varied species can exist with some exceptions. Reproduction of Gammarus and Daphnia prevented, perhaps other crustaceans. Aquatic plants and microorganisms relatively normal except fungi frequent.

5.5 - 6.0 Eastern brook trout (*Salvelinus fontinalis*) survive at over pH 5.5. Rainbow trout (*Salmo gairdneri*) do not occur. In natural situations, small populations of relatively few species of fish can be found. Growth rate of carps reduced. Spawning of fathead minnow significantly reduced. Mollusks rare.

5.0 - 5.5 Very restricted fish populations but not lethal to any fish species unless CO_2 is high (over 25 ppm), or water contains iron salts. May be lethal to eggs and larvae of sensitive fish species. Prevents spawning of fathead minnow. Benthic invertebrates moderately diverse, with certain blackflies (Simuliidae), mayflies (Ephemerella), stoneflies, and midges (Chironomidae) present in numbers. Lethal to other invertebrates such as the mayfly. Bacterial species diversity decreased; yeasts and sulfur and iron bacteria (Thiobacillus-Ferrobacillus) common. Algae reasonably diverse and higher plants will grow.

4.5 - 5.0 No viable fishery can be maintained. Likely to be lethal to eggs and fry of salmonids. A salmonid population could not reproduce. Harmful, but not necessarily lethal to carp. Adult brown trout (*Salmo trutta*)can survive in peat waters. Benthic fauna restricted mayflies; reduced. Lethal to several typical stoneflies. Inhibits emergence of certain caddisfly, stonefly, and midge larvae. Diatoms are dominant algae.

4.0 - 4.5 Fish populations limited; only a few species survive. some coarse fish, and pike can acclimate to this pH, but only pike reproduce. Lethal to fathead minnow. Some caddisflies and dragonflies found in such habitats; certain midges dominant. Flora restricted.

3.5 - 4.0 Lethal to salmonids and bluegills. Limit of tolerance of pumkinseed (*Lepomis gibbosus*), perch, pike, and some coarse fish. All flora and fauna severely restricted in number of species. Cattail (Typha)is only common higher plant.

3.0 - 3.5 Unlikely that any fish can survive for more than a few hours. A few kinds of invertebrates such as certain midges and alderflies, and a few species of algae may be found at this pH range and lower.

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SOURCE

The above rationale information was taken from "Water Quality Criteria 1971", National. Academy of Sciences, National Academy of Engineering, Washington D. C., 1972, EPA. R3.033, March 1973, pages 140-141 and 203.

AMMONIA

OBJECTIVE

Concentrations of un-ionized aa (NH_3) should not exceed 0.02 milligrams per litre for the protection of aquatic life.

RATIONALE

Ammonia enters natural water systems from several sources, either directly as ammonia or indirectly by formation from other nitrogenous matter. Direct sources are precipitation of ammonia with rain and snow, gas exchange with the atmosphere and the influx of ammonia-containing effluents from urban, industrial and agricultural sources. Indirect sources re the chemical and biochemical transformation of nitrogenous organic and inorganic matter in soil and water, nitrogen fixation processes of dissolved nitrogen gas in water and excretion of ammonia by biota.

Ammonia is consumed by chemical and biochemical processes, some of them resulting its oxidation to nitrite and nitrate ions, other reactions resulting in its incorporation into organic matter, particularly with the formation of proteins.

Aqueous Ammonia Equilibrium System

At high concentrations, ammonia becomes a significant toxicant to the aquatic biota. The toxicity of ammonia to fish is primarily due to un-ionized ammonia (NH_3) (Chipman, 1973; Wuhrmann *et al.* 1947; Wuhrmann and Woker, 1948; Hemens, 1966). Ionized ammonia (NH_4^+) is considered non toxic or significantly less toxic than un-ionized ammonia (NH_3) (Tabata, 1962). The percent of un-ionized ammonia in an aqueous ammonia solution is strongly dependent on pH, according to the equilibrium equation:

$$NH_3 + H_2O \leftrightharpoons NH_3 \bullet H_2O \leftrightharpoons NH_4^+ + OH^-$$

The above equation is abbreviated and does not consider the hydrogen bonding of the molecules and ions to adjacent water molecules (Butler, 1964).

The equilibrium of ionized with unionized ammonia also influenced by the temperature and salinity of the water. As shown in Table 2, the fraction of unionized ammonia increases with rising temperature, particularly at low pH levels. The presence of low to moderate amounts of dissolved solids (200-1000 mg/L) will slightly lower the concentration of un-ionized ammonia. In Great Lakes waters, the magnitude of this effect will usually be less than the effect of lowering the temperature by 1°C under otherwise constant conditions. For practical purposes and for the definition of this objective the influence of the salinity will hence be neglected.

Table 2: Percent Un-ionized Ammonia In Aqueous Ammonia Solution

(Values at zero salinity) (After Thurston *et al.*, 1974)

Temperature	pH Value								
(°C)	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0
5	0.013	0.040	0.13	0.39	1.2	3.8	11	28	56
10	0.019	0.059	0.19	0.59	1.8	5.6	16	37	65
15	0.027	0.087	0.27	0.86	2.7	8.0	22	46	73
20	0.040	0.13	0.40	1.2	3.8	11	28	56	80
25	0.057	0.18	0.57	1.8	5.4	15	36	64	85
30	0.081	0.25	0.80	2.5	7.5	20	45	72	89

Conditions under which objective for total ammonia is limiting.

Conditions under which the objective for un-ionized ammonia is limiting.

TOXICITY TO FISH

At concentrations above 0.5 mg/L un-ionized ammonia (NH_3) is strongly toxic to many fish. Comprehensive reviews on the toxicity of ammonia to aquatic biota are published by EIFAC (1973), NAS/NAE (1974), and by McKee and Wolf (1963). A compilation of major studies on fish is given in Table 3. Most of these data on rainbow trout, Atlantic salmon, striped bass, three-spined stickleback, carp and other species are in the form of one to four day LC_{50} concentrations and under pH, temperature and dissolved oxygen conditions as they prevail in the Great Lakes. Two to four day LC_{50} concentrations for rainbow trout range from 0.25 to 0.75 mg/L NH_3 with coarser fishes being slightly less sensitive.

The equilibrium of un-ionized and ionized ammonia, and thus the toxicity of ammonia, is strongly dependent on the pH and temperature, and, to a lesser degree on the salinity of the water. The toxicity of un-ionized ammonia is to a minor degree further dependent on the alkalinity and free carbon dioxide of water, and, to an unknown degree, on other synergistic and antagonistic factors, such as dissolved oxygen levels, biota acclimation, etc.

There are few published data available on chronic sub-lethal effect of ammonia to fish of any species. A three-month test on 200 rainbow trout (Water Pollution Research 1967; 1968) showed a 15 percent mortality at 0.27 mg/L NH_3 and a 5 percent mortality at un-ionized ammonia concentrations of 0.13 and 0.07 mg/L NH_3, respectively.

Although no mortality data have been reported at concentrations less than 0.2 mg/L NH_3, deleterious effects of ammonia at comparable concentrations and lower have been observed by a number of researchers. Reichenback-Klinke (1967), in a series of one-week tests on 240 fish of 9 species at concentrations of 0.1 to 0.4 mg/L NH_3 observed, as well as inflammations and hyperplasia, swelling of and diminishing of the number of blood cells. Irreversible blood damage occurred in trout fry at 0.27 mg/L NH_3. He also noted that these low NH_3 doses inhibited the growth of young trout and lessened their resistance to diseases. Flis (1968) reported that a 35-day exposure of carp to a concentration of approximately 0.1 mg/L NH_3 resulted in extensive necrobiotic and necrotic changes and tissue disintegration in various organs.

Reduction in growth rates for rudd has also been observed after 95 days at concentrations greater than 0.1 mg/L NH_3 Water Foliation Research 1971; 1972) and for rainbow trout at 0.02 mg/L NH_3 after 6 months (Smith and Piper, 1974). Smith and Piper (1974) also reported severe pathological changes in gills and livers of rainbow trout after 12 months exposure at 0.02 mg/L NH_3. On a test of rainbow trout for the 21-day period between egg hatching and swim-up stage, a reduction in development (length and sac absorption) was observed at concentrations of 0.07 mg/L NH_3 and higher (Thurston, 1974). Concentrations as low as 0.002 mg/L NH_3 have been reported to cause gill hyperplasia in fingerling chinook salmon in 6 weeks (Burrows, 1964).

Table 3: Ammonia Toxicity To Fish

Species	Length (cm.)	pH	Temp.(°C)	NH_3 Concentration (mg/L NH_3)	Dissolved Oxygen (% saturation)	Mortality (% of total fish)	Exposure Time (days)	Reference
Rainbow trout								
(salmo gairdnerii Richardson								
	15.2	7.86-8.22	10.5-11.6	0.50	280	50	2	Ball (1967)
	3-5	8.1-8.3	12-13	0.25 [a]		50	>4	Water Poll. Res.(1968)
	3-5	8.1-8.3	2-4	0.42 [a]		50	3	"
		7.8	5	< 0.25		50	4	"
		7.8	18	~0.75		50	4	"
				0.27		16	90	"
				0.07		5	90	"
				0.13		5	90	"
	13-14	7.45	13.6	0.70 (65) [b]		50	1 [d]	Herbert & Shurben (1965)
	13-14	7.81	13.6 (7)	0.49 (37) [b]		50	1 [e]	"
		7.8	17.5	~0.50(24.6 [c]	~100	50	2 [f]	Herbert & Shurben (1964)
		7.8	17.5	~0.45(30.1) [c]	47	50	2 [f]	"
		6.9	17.7	~0.65(193.) [c]	~100	50	2 [g]	"
	7.3	7.0-8.0	19.8	1.7	~ 15 to ~90	50	0.01-0.25	Downing & Merkens (1955)
	7.5	7.0-8.0	19.8	1.2	~ 15 to ~90	50	0.01-0.5	"
	7.3	7.0-8.0	19.8	0.7	~15 to ~35	50	0.01-0.8	"
Atlantic salmon		7.81	13.6 (7)	0.28 (15) [b]		50	1	Herbert & Shurben (1965)
Perch	10.1	7.75-8.12	9.6-11.1	0.35	≥92	50	4	Ball(1967)
Roach	8.6	7.86-8.30	10.0-13.2	0.42	≥90	50	4	"
Rudd	11.5	8.05-8.50	12.2-13.2	0.45	≥88	50	4	"
Bream	11.1	7.75-8.12	9.4-14.6	0.50	≥92	50	2 to 6	"
Striped bass	2.0-9.3	7.3-7.9	15	1.4	≥50	50	4	Hazel, Thomsen & Meith
	2.0-9.3	7.4-8.0	23	0.93	≥50	50	4	" (1971)
Sticklebach	3.2-6.0	6.8-7.2	15	1.0	≥50	50	4	"
(three splined)	3.2-6.0	7.0-7.3	23	0.88	≥50	50	4	"
Common carp		8.3-8.7	11	1.3		16	10	Flis (1968a)
		8.1-8.7	11	0.9		18	10	"
		8.05		0.11		8 and 0	35	Flis (1968b)

a - Water hardness 320 mg/L $CaCO_3$
b - Values taken from EiFAC (1970), original values (in brackets) as total mg/L N from NH_4Cl
c - Values calculated from Table; original values (in brackets) as total mg/L N from NH_4Cl
d - Water hardness 125 mg/L $CaCO_3$
e - Water hardness 248 mg/L $CaCO_3$
f - Water hardness 320 mg/L $CaCO_3$, water alkalinity 240 mg/L $CaCO_3$
g - Soft water

Rainbow trout have successfully spawned in the laboratory at 0.06 mg/L NH_3 and have produced significant numbers of viable fry (Thurston, 1974).

It is common practice, for the establishment of water quality objectives, to multiply 96-hr LC_{50} data by an application factor to arrive at recommendations for those objectives. According to Water Quality Criteria 1972 (NAS/NAE, 1974) application factors range, depending on the compound in question, from 0.1 (e.g. copper) to 0.005 (e.g. zinc). For ammonia, an application factor of 0.05 is recommended. With an average 96-hour LC_{50} value of about 0.50 mg/L NH_3 x 0.05, a safe concentration of 0.025 mg/L NH_3 is calculated. This value appears to be a safe concentration of un-ionized ammonia for the survival of rainbow trout fry and fingerlings. However, judging from the comparison of trout and salmon sensitivities by Herbert and Shurben (1965), salmon appear to be almost twice as sensitive to ammonia as trout. Therefore, with an application factor of 0.05 a limit of about 0.015 mg/L NH_3 could be calculated.

Both values of 0.025 and 0.015 mg/L NH_3 are in close proximity to the experimentally observed threshold for sub-lethal effect of ammonia on rainbow trout, reported as 0.02 mg/L NH_3 by Water Pollution Research (1971; 1972) and Smith and Piper (1974). Extreme deviations of the "mean" of 0.02 mg/L NH_3 may be represented by the value of 0.06 mg/L NH_3 for the successful spawning and apparently normal fry development of rainbow trout (Thurston, 1974) and by the observations of sub-lethal effect at un-ionized ammonia concentrations of 0.002 mg/L NH_3 to fingerling chinook salmon by Burrows (1964). Until it can be shown that exposure of biota to un-ionized ammonia concentrations of less than 0.02 mg/L NH_3 is indeed resulting in long term sub-lethal effect, and in view of the acute toxic levels of 0.2 mg/L NH_3 or higher, it is recommended that un-ionized ammonia should not exceed 0.02 mg/L NH_3.

AMMONIA IN RAW WATER SUPPLIES

In water treatment, (total) ammonia interferes with the water chlorination. If ammonia is present, chlorine will react with it first, producing chloramines. On continued chlorination, oxidation of intermediate chloramines to HCl and N_2 will occur. Chloramines also have bactericidal properties but are slower acting than free chlorine. In the past, chloramines, intentionally produced by adding ammonia to raw water, were used to prevent the reaction of chlorine with phenols. To destroy ammonia occurring in raw water, about 10 parts of chlorine to 1 part of ammonia nitrogen is required (Matheson, 1973). Because of these unwanted effects, the level of total ammonia for raw water supplies is desired to be less than 0.01 mg/L NH_3 (total). However, for practical purposes WHO (1963), NAS/NAE (1974), and Ministry of the Environment (1974), recommend 0.5 mg/L NH_3 (total) as the upper limit for raw water supplies. At concentrations above this value, problems associated with disinfection, and taste and odor are experienced.

In accordance with above recommendations and for the protection of the use of Great Lakes water as water supply, it is recommended that the objective for total ammonia should not exceed 0.5 mg/L NH_3 (total).

The conditions under which either the objective for un-ionized ammonia, for the protection of aquatic life, or the objective for total ammonia, for protection of public water supplies, becomes the limiting parameter is indicated in Table 2. At a low pH, the value for total a is s limiting; at a high pH, the value for un-ionized ammonia is the limiting parameter.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1974", pages 117-128 International Joint Commission, June 1975.

ARSENIC

OBJECTIVE

Concentrations of total arsenic in an unfiltered water sample should not exceed 100 micrograms per litre to protect aquatic life.

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 5).

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 we found to be as high as 58 µg/L in a water intake at Massena, New York (Table 6). 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 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_{5O} of sodium arsenate to *Daphnia magna was* 2,850 µg/L while the concentrations causing 50 to 16 percent 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 percent 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 percent and 45 percent, respectively. The weight of fish harvested was also substantially reduced in the treated ponds. Conditioned avoidance behaviour of goldfish was significantly impaired 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 an an experimental *basis.*

Applying an application factor of 1/100 to the algae data gives sore concentration from 20 to 100 µg/L. The same application factor applied to the chum salmon data gives 110 µg/L as a safe concentration. The avoidance behaviour affect on goldfish likely does not represent a major problem. In light of these data, a safe concentration of 100 µg/L in unfiltered water will protect aquatic life.

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SOURCE

Most of the above information were taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975", pages 18-21, International Joint Commission, 1975.

Additional rationale material prepared by staff of the Ministry of Environment appears in italics.

BERYLLIUM

OBJECTIVES

Concentration of total Beryllium in an unfiltered sample should not exceed 11 micrograms per litre in water of hardness less than 75 milligrams per litre of $CaCO_3$.

Concentrations of Beryllium in an unfiltered sample should not exceed 1100 micrograms per litre in water of hardness greater than 75 milligrams per litre.

RATIONALE

Beryllium is not likely to occur at significantly toxic levels in ambient natural waters (McKee and Wolf, 1963). Although the chloride and nitrate salts of beryllium are very water soluble, and the sulfate is moderately so, the carbonate and hydroxide are almost insoluble in cold water (Lange, 1961). Kopp and Kroner (1967) reported that for 1,577 surface water samples collected at 130 sampling points in the United States, 85 samples (5.4 percent) contained from 0.01 to 1.22 µg/L with a mean of 0.19 µg/L beryllium. The concentration of beryllium in sea water is 6×10^{-4} µg/L (Goldberg, *et al.*, 1971).

The major human toxic hasard potential of beryllium is via the inhalation of beryllium-containing fumes and dusts that might emanate from processing and fabrication operations. Beryllium could enter waters in effluents from certain metallurgical plants (NAS, 1974).

Contact dermatitis, characterized by itching and reddened, elevated, or fluid-accumulated lesions, which appear particularly on the exposed surfaces of the body, may occur either on an allergic basis or from primary irritation following contact with soluble beryllium salts (Van Ordstrand, *et al.*, 1945; McCord, 1951). A latent period is occasionally noted, indicating the development of delayed hypersensitivity (NIOSH, 1972).

Ocular effects may occur as inflammation of the conjunctiva in "splash burn" or in association with contact dermatitis (Van Ordstrand, *et al.*, 1945). Splashes may also cause corneal burns closely resembling those produced by acids and alkalies, and fluid accumulation and reddening around the eye socket are frequently noted (NIOSH, 1972).

Beryllium is demonstrably toxic by most routes of administration (NIOSH, 1972), but its oral toxicity is notably different from that by other routes. The sulfates, for example, while highly toxic by all other routes at a single dose level, is practically non-toxic by mouth at a level several thousand-fold greater by multiple dose (Reeves, 1965; Stokinger and Stroud, 1951).

Tarzwell and Henderson (1960) obtained 96-hour LC_{50} values ranging from 0.15 mg/L beryllium

(when tested as the nitrate and chloride) to 0.2 mg/L beryllium (when tested as the sulfate) for fathead minnows in soft water (20 mg/L $CaCO_3$, total alkalinity of 18 mg/L and pH of 7.4), and from 11 mg/L beryllium as sulfate) to 20 mg/L beryllium as nitrate) for fathead minnows in hard water (400 mg/L $CaCO_3$, total alkalinity of 360 mg/L, and pH of 8.2). For bluegill they obtained 96-hour LC_{50} values of 1.3 mg/L beryllium in soft water and 12 mg/L beryllium in hard water (both as sulfate).

Slonim (1973) obtained 96-hour LC_{50} static bioassay values of 0.19 mg/L beryllium for the common guppy, *Poecilia reticulate*, in soft water with a hardness of 20 to 25 mg/L as $CaCO_3$, total alkalinity of 16 to 18 mg/L, and pH of 6.3 to 6.5, and 20.3 mg/L beryllium in hard water with a hardness of 400 to 500 mg/L, alkalinity of 185 to 230 mg/L, and pH of 7.8 to 8.2.

Slonim and Slonim (1973) studied the influence of water hardness on the toxicity of beryllium sulfate to the common guppy, *Poecilia reticulate*, by simultaneously conducting static bioassays at 400, 275, 150 and 22 mg/L hardness as $CaCO_3$. The 96-hour LC_{50} values were 20.0, 13.7, 6.1, and 0.16 mg/L, respectively. In a water hardness of 22 mg/L, 80 percent of the test fish survived 0.1 mg/L beryllium for 96 hours, whereas 100 percent survived control conditions. In a water hardness of 150 mg/L, 70 percent survived 5 mg/L beryllium for 96 hours and 100 percent survived 2.5 mg/L beryllium.

Slonim and Ray (1975) studied the influence of water hardness on the toxicity of beryllium sulfate to two species of salamander larvae by conducting static bioassays at hardness levels of 400 mg/L and 20 to 25 mg/L as $CaCO_3$. In hard water, all of the test organisms survived exposure to 10 mg/L beryllium throughout the exposure period, whereas there was a significant decline in survival with time in soft water. The 96-hour LC_{50} values were 26.3 mg/L beryllium in hard water, and 4.7 mg/L beryllium in soft water.

Based on the fathead minnow and bluegill data of Taraweil and Henderson (1960), the observations of Slonim and Ray 11975 on salamander larva survival, and the observations of Slonim and Slants (1975) on guppy survival, the criterion for the protection of aquatic biota is established an 1.1 mg/L beryllium in hard fresh water. This value assumes an application factor of 0.1 of the 96-hour LC_{50} value for fathead minnows. For soft fresh water, in view of the reported approximate 100-fold increases in acute fish toxicity over that found in hard water, the criterion for the protection of aquatic biota is set at 0.011 mg/L beryllium.

Beryllium tas been reported to be concentrated 1000 times in marine organisms (Goldberg, *et al.*, 1971). The average concentration factors for marine benthic algae, phytoplankton, and zooplankton also have been reported as 110, 1000, and 15 mg/L, respectively (Lowman, *et al.*, 1971). Riley and Roth (1971) reported levels of 1.1 to 18 mg/L beryllium for various species of marine algae. The 96-hour TLm value for beryllium resulting from tests performed on the killifish, *Fundulus heteroclitus*, was 41 mg/L (Jackim, *et al.*, 1970). These data do not represent an adequate base upon which to establish a marine criterion.

Beryllium has been shown to inhibit photosynthesis in terrestrial plants (Bollard and Butler, 1966), and to be toxic to some varieties of citrus seedlings at 2.5 mg/L beryllium (Haas, 1932). Romney, *et al.* (1962) found that beryllium at 0.5 mg/L in nutrient solutions reduced the growth of bush beans, and Romney and Childress (1965) found that concentrations of 2 mg/L or greater in nutrient solutions reduced the growth of tomatoes, peas, soybeans, lettuce, and alfalfa plants. Additions of soluble beryllium salts at levels equivalent to 4 percent of the cation-adsorption capacity of two acid soils reduced the yields of ladino clover; beryllium carbonate and beryllium oxide at the same levels did not reduce yields, suggesting that beryllium in calcareous sails might be less active than in acid soils. Williams and LeRiche (1968) found that beryllium at 2 mg/L in nutrient solutions was toxic to mustard, whereas 5 mg/L was required for growth reduction in kale. In view of its toxicity in nutrient solutions in acid soils, the criteria for beryllium in irrigation waters are 0.10 mg/L for use on all soils except 0.50 mg/L for use on neutral to alkaline fine-textured soils.

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Source

The above rationale information was taken from "Quality Criteria for Water", US-EPA-440/9-76-023, pages 39-46, Environmental Protection Agency, Washington, D.C.

CADMIUM

OBJECTIVE

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 such 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^{2+}. 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^{2+} 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^{2+}, 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 concentrations 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 the metal content of coarse particulate suspended solids (Svedberg coefficient* (S) greater than 20,000), in colloidal particulate suspended solids (100 less than S and greater than 20,000) and in dissolved solids. The mean cadmium concentration 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 (approximately 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 7). In Lake Superior, a small proportion of concentrations are between 0.2 and 0.6 µg/L. In cake Erie, concentrations of cadmium in offshore filtered water never exceeded 1 µg/L, the detection limit at that time (Chawla and Chau, 1969; MARC, 1972) but in a water intake at Buffalo, concentrations ranged as high as 12 µg/L and the mean was 7 µg/L (Table 6). In Lake Michigan, concentrations never exceeded 1 µg/L in 1970 although some tributaries were slightly higher (MARC, 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 (MARC, 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, nematopoietic tissues 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, increased incidence of tumours and reduced growth (Flick *et al.*, 1971). The biochemical Oases for these effects may be the interaction of cadmium with thiol groups of enzymes or with phosphatidyiethanoiamine and phosphatidylserine monolayers (Vallee and Ulmer, 1972). As a result, many enzymatic reactions are inhabited 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 was been estimated as 15 µg, as compared to 200 ug in food and 1 ug in air (Nilsson, 1970). 0f the total cadmium taken in, only 1-2 percent is retained and the

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

rest is excreted in faeces and urine. To limit intake from water to 200 µg/L 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 (DOHS, 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/OlE, 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 percent inhibition of primary productivity of *Scenedesmus quadricauda, Chlorella oyrenoidosa, 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 hyaline* were 3,300, 550 and 55 µg/L respectively (Baudonin 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 hyaline*. A 3-week LC_{50} for *Daphnia magna* was 5 µg/L while 0.17 µg/L caused 16 percent 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 lyccrias* (stonefly), *Ephemerella subvaria* (mayfly) and *Hydrapsyche betteni* (caddisfly) were greater than 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 (Thorn 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 1050'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 Cast, 1972). Similarly, 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 (*Morose saxatilis*) were 17,200-24,200, 2,500, 56-100, and 2 µg/L respectively (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 percent mortality occurred at 850 µg/L 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/L) compared to the former 152 mg/L). In addition the chloride content of the water used by Cearley and Coleman (194) was 193 mg/L. Largemouth bass

(*Micropterus salmoides*) were more sensitive than bluegills. Significant mortality occurred at concentrations of 80 µg/L cadmium and behaviour was affected at 8 µg/L (Cearley and Coleman, 1974).

Survival of flagfish larvae in water of 44 mg/L hardness was affected at 8 µg/L cadmium and was normal at 4 µg/L. 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 of 25 µg/L for 24 hours or 10 µg/L for 21 days at 20 mg/L hardness caused extensive hemorhagic necrosis of the testes of male trout (Sangalang and O'Halloran, 1972, 1973). After about 4 months exposure, 1 µg/L 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 two weeks earlier than controls (Sangalang and Freeman, 19744). Brook trout alevins showed a decreased wet weight, increased protein content and increased acetylcholinesterase activity at 0.70 µg/L cadmium in water of 45 mg/L 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/L while no adverse effects were noted at 1.7 µg/L 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 lakes 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 two 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 10-20 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 1,60 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 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 percent of the dose (Rowe and Massaro, 1974). Within one hour, 75 percent of the cadmium in the body was s contained within the GI tract and 23 percent. was in the gills. The fact that 2 percent 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 percent of the cadmium was in the kidneys, 5 percent in the liver, about 56 percent 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 respectively. Similarity, 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 trophi 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 sensitively 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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Source

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975", pages 22-32, International Joint Commission, 1976.

CHLORINE

OBJECTIVE

Total residual chlorine, as measured by the amperometric (or equivalent) method, should not exceed 0.002 milligram per litre in order to protect aquatic life.

RATIONALE

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

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

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

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

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

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

METHODOLOGY

Many wastewater treatment plants are required to maintain a residual chlorine concentration of 0.5 to 2.0 mg/L. Most plant operators use the orthotolidine method which has been shown to be biased on the low side resulting in much higher concentrations than necessary for adequate disinfection. This compounds toxicity problems in receiving waters. Total residual chlorine concentrations in 20 Illinois effluents ranged from 0.98 to 5.17 mg/L (Snoeyink and Markus, 1974). A similar study at 22 plants in southern Wisconsin resulted in observed concentration of TRC between 0.13 and 10.3 mg/L (McKersie, 1974). Both studies demonstrated that the orthotolidine methods provided the poorest results when compared against better methods such as the amperometric titration technique. Other studies (Martens and Servizi, 1974; Servizi and Martens, 1974) reached the same conclusion that the commonly

used orthotolidine method is inadequate to determine TOC in wastewaters or receiving streams.

TOXICITY OF CHLORINATED WASTEWATERS

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

The Michigan Department of Natural Resources (1971) reported the effects on caged fish in several receiving streams below wastewater discharges. Fifty percent of the rainbow trout died within 96 hr (95-hr LC_{50}) at TRC concentrations of 0.014 to 0.029 mg/L; some fish died as far as 0.8 mile (1.3 km) below the outfall. These same discharges were studied when chlorination was temporarily interrupted, and no mortality was observed. In addition, dechlorination with sodium thiosulfate eliminated toxicity in 4-day tests with undiluted effluent.

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

Arthur *et al.* (1975) have studied the effect of chlorinated secondary wastewater treatment plant effluent containing only domestic sewage effluent on reproduction of fathead minnows, *Daphnia magna*, and the scud (*Gammarus pseudolimnaeus*). *Daphnia magna* apparently was the most sensitive invertebrate species and died at a TRC concentration of 0.014 mg/L, and acceptable reproduction occurred at 0.003 mg/L and below. Scud reproduction *was* reduced at concentrations above approximately 0.012 mg/L (1.2 percent effluent). No effects on any life cycle stage, including reproduction, of the fathead minnow was observed at a concentration of 0.014 mg/L; adverse effects were observed at 0.042 mg/L. Acute toxicity studies with eight species of fish, crayfish (*Orconectes virilis*), scud (*Gammarus pseudolimnaeus*), snails *(Physa integra* and *Campeloma decisum*), and stoneflies (*Acroneuria lycorias*) indicated that the

crayfish, snails, and stonefly larvae were least sensitive with 7-day LC_{50} values greater than 0.55 mg/L. Seven-day values for the other organisms were between 0.083 and 0.261 mg/L; coho salmon, brook trout, fathead minnow, white sucker, and walleye were the most sensitive (0.086 to 0.150 mg/L). Nearly 50 percent of the observed mortalities occurred in the first 12 hours of the acute tests indicating that the lethal effect of TRC occurs rapidly. Comparable acute and chronic tests with the effluent dechlorinated with sulfur dioxide indicated that most, if not all, of the toxicity of the chlorinated effluent was eliminated. Esvelt *et al.* (1971; 1973) and Krock and Mason (1971) completed an extensive study on the toxicity of chlorinated municipal wastewaters entering San Francisco Bay and surrounding areas. They observed a significant increase in toxicity following chlorination. Chlorine toxicity was still significant in aged (up to 3 days) chlorinated wastewater, in which TRC concentrations were as high as 25 percent of the initial level.

Rainbow trout was the most sensitive of the species tested, followed by the golden shiner and three-spined stickleback. A calculated chlorine residual of 0.03 mg/L, based on dilution of a measured concentration of 2.0 mg/L, reduced phytoplankton photosynthesis by more than 20 percent of the value obtained with a dilution of effluent having no chlorine residual. Dechlorination with sodium bisulfite also eliminated chlorine-related toxicity. One of the conclusions of the California study was that chlorination may be the largest single source of toxicity in San Francisco Bay.

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

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

Several reviewers of chlorine toxicity have recommended numerical objectives for concentrations of TRC that would not adversely affect aquatic populations when discharged continuously. Basch and Truchan (1974) recommended maximum concentrations of 0.02 and 0.005 mg/L for warmwater and coldwater intolerant fish, respectively. European Inland

Fisheries Advisory Commission (1973) has suggested objectives dependent upon pH and temperature with an acceptable upper limit of 0.004 mg HOCl/L (TRC from 0.004 mg/L at a pH of 6.0 and 5°C to 0.121 mg/L at a pH of 9.0 and 25°C). A third review by Brungs (1973) has recommended objectives of 0.01 mg/L for warmwater fish and 0.002 mg/L for coldwater species and the most sensitive fish food organisms. Since these recommendations, additional data on warmwater fish species (Arthur *et al.*, 1975; Tsai, 1973; Ward *et al.* 1976, 1977; Bogardus *et al.* 1976) do not support the distinction between coldwater and warmwater fish species, A mare recent recommendation (Brungs, 1976) supports the objective for total residual chlorine of 0.002 mg/L.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives, Great Lakes Water Quality 1976," pages 5-10, International Joint Commission, June 1977.

CHROMIUM

OBJECTIVE

Concentrations of total chromium in an unfiltered water sample should not exceed 100 micrograms per litre to protect aquatic life.

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 percent of samples contain less than 0.6 µg/L (Table 7). At water intakes, average concentrations are shown to be less than 10 µg/L and maxima less than 20 µg/L (Table 6). However, concentrations of chromium in water intakes in the St. Lawrence River appear much higher, (Table 6). 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 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 essential 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 essential 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 100 µg/L as CR (VI) and the acceptable limit is less than 100 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 cellular algal growth inhibition to demonstrate that some diatoms were sensitive to 320 µg/L but not to 32 µg/L of chromium. Wium-Anderson (1974), using C^{14} fixation to estimate growth, estimated that 650 µg/L of Cr (VI) caused 50 percent inhibition of photosynthesis by the diatom, *Nitzschia palea*. Patrick *et al.*, (1968) indicated that 208 µg/L of Cr (III) also caused 50 percent 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 roseolawas* 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 (*Pimeohales 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 *(Oncorhvnchus tshawytscha*), after 12 weeks exposure, had higher mortality rates (greater than 50 percent 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 0.9-16 µg/g. Chromium was also measured by neutron activation in dressed samples of whitefish, northern pike, smelt and perch. The concentrations ranged from less than 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 One 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. *Based on* the *algal growth response to Chromium and the* mortality *of chinook salmon,* a concentration of *100 µg/L should protect aquatic life.*

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SOURCE

The above rationale information was primarily taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975," pages 32-36, International Joint Commission, 1976.

Additional rationale information, prepared by staff of the Ministry of Environment appears in italics.

COPPER

OBJECTIVE

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

RATI0NALE

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^{++} 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 in electrode (Zitko *et al.*, 1973 and Andrew, 1975); and (c) anodic stripping voltammetry (Gachter *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 snowmelt. The modal copper concentration in the upper lakes offshore are less than 2.5 µg/L, and 95 percent 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 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 6). 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 percent 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 percent by only 20 µg/L copper (Nielsen *et al.*, 1969). Nielson *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 percent at 22 µg/L copper and by 50 percent 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 generations 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 37 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 Lame Ontario is 135 mg/L while that in the field study was 352 mg/L. 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 flowing 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 - 1.28 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 Raie 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/L. 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/L. Eaton (1973) found that fathead minnow spawning success and egg production were reduced between 5.3 and 6.7 µg/L copper when low concentrations of added cadmium and zinc were present. The cadmium and zinc concentrations were 3.9 and 27.3 µg/L, respectively, when no effect was observed and 7.1 and 42.3 µg/L, 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 7). 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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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975," pages 37-44, International Joint Commission, 1976.

CYANIDE

OBJECTIVE

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

RATIONALE

Cyanide is 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 scored for long periods to 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 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 in 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 zanthine 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 anaerobic 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 trotta* (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 concentrations) 70, 104, 150 and 180 µg/L 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 bio-assays 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 e 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 25°C to 8°C the lethal threshold concentration (LTC) for juvenile bluegills (*Lepomis machrochirus*) was determined to decrease linearly from 130 to 58 µg/L HCN. Should the above linearity persist to lower temperature - not as yet experimentally determined - the calculated LTC values at 5, 3, and 1°C would be 41, 32 and 23 µg/L HCN respectively.

In review it can be concluded that free cyanide concentrations in the range from 50 to 100 µg/L as cyanide have proven eventually fatal to many sensitive fishes and levels much above 200 µg/L 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 brood 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 (*Perna 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/L. Mean percentage hatch was significantly decreased at levels of 40 µg/L 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/L 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/L HCN effective reproduction was about 80, 50, and 30 percent of the controls respectively.

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

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes 'dater Quality 1975", pages 95-100, International Joint Commission, 1976.

DISSOLVED OXYGEN

OBJECTIVE

At no time should dissolved oxygen concentrations be Less than the values specified below:

	Dissolved Oxygen Concentration			
	Cold Water Biota		Warm Water Biota	
Temperature (°C)	% Saturation	mg/L	% Saturation	mg/L
0	54	8	47	7
5	54	7	47	6
10	54	6	47	5
15	54	6	47	5
20	57	5	47	4
25	63	5	45	4

In situations where additional physical and/or chemical stresses are present these minimum levels may prove inadequate and more stringent Objectives may be necessary.

In some hypolimnetic waters, dissolved oxygen is naturally lower than the above-specified concentrations. Such a condition should not be altered by adding oxygen demanding materials causing a depletion of dissolved oxygen.

RATIONALE

The presence of adequate dissolved oxygen is essential for the healthy functioning of aquatic ecosystems. Any reduction of the dissolved oxygen below saturation is likely to have some effect on the ecosystem and the magnitude of that effect will depend on the severity and duration of the reduction. The absence of dissolved oxygen will give rise to anaerobic decomposition with the production of noxious gases and the aesthetic degradation of water quality.

There is a large volume of literature pertaining to the effect of dissolved oxygen concentration on aquatic life, especially fish, and the concentrations below which fish will be affected. Comprehensive papers have been presented by Doudoroff and Shumway (1967, 1970) EIFAC (1973), NAS/NAE (1974) and Davis (1975). The intent of this rationale is not to reiterate the information which has been summarized in the above review papers. Rather, attention is given identifying dissolved oxygen response threshold and the effects of low oxygen levels on aquatic organisms. Much reliance has been placed on the review of oxygen requirements of aquatic life by Davis (1975) in selecting an objective.

Under conditions of reduced oxygen availability, fish attempt to maintain oxygen uptake by increasing the rate and amplitude of breathing and decreasing the heart rate (Holeton and Randall, 1967a, b; Garey 1967). The responses of fish to hypoxia represent a form of compensation or adjustment of bodily processes. Fish are able to tolerate short periods of reduced oxygen levels. The extent of this tolerance is dependent on the species, oxygen levels, and other environmental factors. The use of compensation mechanisms requires an expenditure of energy and will consequently reduce reserves needed for swimming, feeding, reproduction, avoiding predators and other activities.

Much work has been conducted in the laboratory to determine those concentrations of oxygen which are n some way harmful to fish maintained under quiescent conditions. However, Fry (1957) stated that "Any reduction of the oxygen content below the level where the active metabolic rate begins to be restricted is probably unfavourable to the species concerned. From the ecological point of view this "incipient limiting level" (the critical level under conditions of activity) can be taken as the point where oxygen content becomes unsuitable". Brett (1970) determined the oxygen requirements for fingerling sockeye salmon during various important activities. He found that a reduction in oxygen to 50% saturation (at 20°C) would severely limit energy expenditure for migrating or maximum feeding. Davis *et al.* (1963) reported that any reduction from ambient oxygen at 10-20°C usually reduced the maximum sustained swimming speed in coho and chinook salmon.

The ability of fish to avoid hypoxic water has been reported by Randall (1970). Whitmore *et al.* (1960) have observed a seasonal variability in behavioural sensitivity of juvenile chinook salmon to low dissolved oxygen. Juvenile fish avoided concentrations of 1.5 -4.5 mg/L in the summer, but showed little avoidance of 4.5 mg/L at lower temperatures in the fall.

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

Developing fish eggs and larvae show a number of responses to low oxygen including respiratory dependence, retarded growth, reduced yolk sac adsorption, developmental deformities, and mortality. It is apparent that hatching eggs and larval fish represent the most sensitive stage in the life history. Davis (1975) has calculated theme n threshold of incipient oxygen response for salmonid larvae to be 8.09 mg O_2/L.

In addition to the direct impact of low oxygen levels to fish, there is evidence that hypoxia enhances the lethal effect of toxicants by producing a metabolic stress, thus lowering the resistance of the animal, as well as increasing toxicant uptake as the result of elevated water flow as the gills. Alderice and Brett (1957) observed an apparent increase in the toxicity of kraft pulp mill waste to young sockeye salmon and the presence of low oxygen, while Lloyd (1961) reported an increase in the toxicity of an umber of chemical species (ammonia, lead, copper, zinc, phenols) to rainbow trout when oxygen levels fell below 60% saturation at 17.5°C. Downing (1954) demonstrated that any reduction in dissolved oxygen below 100% saturation (9.74 mg/L) at 17°C led to a significant enhancement of the lethal effect of cyanide to rainbow trout.

The ability of fish to acclimate to low dissolved oxygen is not clear, and there is lack of field data to demonstrate that this ability would markedly improve the survival of fish populations. The ability to acclimate would be useful if the transition to a lower DO regime were sufficiently slow to enable acclimation to occur without severe physiological stress. There is no question that acclimation is of no value if fish encounter a rapid downward shift of oxygen.

A great range of tolerances, responses and requirements for oxygen exist amongst aquatic invertebrates. Davis (1975) concludes that insufficient evidence exists at this time to allow meaningful dissolved oxygen criteria to be established for aquatic invertebrates. He suggests that any depression of natural oxygen conditions can result in a change in an aquatic invertebrate community. However, au many invertebrates are able to temporarily withstand periods of low oxygen, it is likely that establishment of objectives to protect fish will also ensure the protection to aquatic invertebrates.

Davis (1975) reviewed all of the oxygen data for aquatic organisms found in Canadian waters for the purpose of defining oxygen a concentrations which would represent "co-effect" levels.

The very large body of experimental data was reduced statistically to determine the oxygen concentrations at which the average member of a species in a fish community starts to exhibit symptoms of oxygen distress. These concentrations, called "Level B" by Davis were determined for different temperature. If oxygen concentrations are maintained at these values for long periods of time, some members of the fish community will experience some sublethal effects but no serious harm should occur. In most surface waters, it is quite reasonable to assume that if the level B concentrations are never violated, then for most of the time the concentrations will be above these values. The level B concentrations have been accepted as the Provincial objectives for surface waters.

Dissolved Oxygen Rationale

Many lakes experience oxygen concentration well below the level B in the hypolimnetic waters. Lake depth is a major factor in determining oxygen concentrations in the hypolimnetic waters (German, in press), but nutrient discharges from natural or man-made sources also result in oxygen depletion. However, as yet, neither of these cause-effect relationships has been accurately modelled.

Low hypolimnetic oxygen concentrations can represent a major stress to the fish populations in the lake, such that cold waters species may even be absent. The recommended remedial course such lakes is to prevent any discharge to the lake which will further reduce the oxygen concentration.

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SOURCE

The above rationale was prepared by staff of the Ministry of the Environment.

GASES, TOTAL DISSOLVED

OBJECTIVE

To protect aquatic organisms the total dissolved gas concentrations in water should not exceed 110 percent of the saturation value for gases at the existing atmospheric and hydrostatic pressures.

RATIONALE

Fish in water containing excessive dissolved gas pressure or tension are killed when dissolved gases in their circulatory system come out of solution to form bubbles (emboli) which block the flow of blood through the capillary vessels. In aquatic organisms this is commonly referred to as "gas bubble disease". External bubbles (emphysema) also appear in the fins, on the opercula, in the skin and in other body tissues. Aquatic invertebrates are also affected by gas bubble disease, but usually at supersaturation levels higher than those lethal to fish.

The standard method of analyzing for gases in solutions has been the Van Slyke method (Van Slyke, *et al.*, 1934); now gas chromatography also is used for determination of individual and total *gases.* For determination of total gas pressure, Weiss has developed the saturometer, a device based upon a thin-wall silicone rubber tube that is permeable to gases but impermeable to water; gases pass from the water through the tube, thus raising the internal gas pressure which is measured by manometer or pressure gauge connected to the tube (NAS, 1974). This method alone does not separate the total gas pressure into the separate components, but Winkler oxygen determinations can be run simultaneously, and gas concentrations can be calculated.

Total dissolved gas concentrations must be determined because analysis of individual gases may not determine with certainty that gas supersaturation exists. For example, water could be highly supersaturated with oxygen, but if nitrogen were at less than saturation, the saturation as measured by total gas pressure might not exceed one hundred percent. Also, if the water was highly supersaturated with dissolved oxygen, the oxygen alone might be sufficient to create gas pressures or tensions greater than the criterion limits, but one would not know the total gas pressure or tension, or by how much the criterion was exceeded. The rare and inert gases such a argon, neon, and helium are not usually involved in causing gas bubble disease as their contribution to total gas pressures is very low. Dissolved nitrogen (N_2), which comprises roughly 80 percent of the earth's atmosphere, is nearly inert biologically and is the most significant cause of gas bubble disease in aquatic animals. Dissolved oxygen, which is extremely bioactive, is consumed by the metabolic processes of the organism and is less important in causing serious gas bubble disease though it may be involved in initiating emboli formation in the blood (Nebeker, *et al.*, 1976a.).

Percent saturation of water containing a given amount of gas varies with the absolute temperature and with the pressure. Because the pressure changes, percent saturation with a given amount of gas changes with depth of the water. Gas supersaturation decreases by 10 percent per meter of increase in water depth due to hydrostatic pressure; a gas that is at 130 percent saturation at the surface would be at 100 percent saturation at 3 meters' depth. Compensation for altitude may be needed because a reduction in atmospheric pressure changes the water/gas equilibria resulting in changes in solubility of dissolved gases.

There are several ways that total dissolved gas supersaturation can occur:

(1) Excessive biological activity -- dissolved oxygen concentrations often reach supersaturation due to excessive algal photosynthesis. Renfro (1963) reported gas bubble disease in fishes resulting, in part, from algal blooms. Algal blooms often accompany an increase in water temperature and this higher temperature further contributes to supersaturation.

(2) Lindroff (1957) reported that water spillage at hydropower dams caused supersaturation. When excess water is spilled over the face of a dam it entrains air as it plunges to the stilling or plunge pool at the base of the dam. The momentum of the fall carries the water and entrained gases to great depths in the pool and, under increased hydrostatic pressure, the entrained gases are driven into solution causing supersaturation of dissolved gases.

(3) Gas bubble disease may be induced by discharges from power generating and other thermal sources (Marcelle, *et al.*, 1975). Cool, gas-saturated water is heated as it pastes through the condenser or heat exchanger. As the temperature of the water rises, percent saturation increases due to the reduced solubility of gases at higher temperatures. Thus the discharged water becomes supersaturated with gases and fish or other organisms living in the heated water may exhibit gas bubble disease (DeMont and Miller, 1972; Malouf, *et al.*, 1972; and Keup, 1975).

In recent years, gas bubble disease has been identified as a major problem affecting valuable stocks of salmon and trout in the Columbia River system (Rulifson and Abel, 1971). The disease is caused by high concentrations of dissolved atmospheric gas which "enter" the river's water during heavy spilling at hydroelectric dams. A report by Ebel, *et al.* (1975) presents results from field and laboratory studies on the lethal, sublethal, and physiological effects of gas on fish, depth distribution of fish in river (fish can compensate for some high concentrations of gas by moving deeper into the water column), detection and avoidance of gas concentrations by fish, intermittent exposure of fish to gas concentrations, and bioassays

of many species of fish exposed to different concentrations of gas. Several conclusions resulting from these studies are:

(1) When either juvenile or adult salmonids are confined to shallow water (1 m), substantial mortality occurs at and above 115 percent total dissolved gas saturation.

(2) When either juvenile or adult salmonids are free to sound and obtain hydrostatic compensation either in the laboratory or in the field, substantial mortality still occurs when saturation levels (of total dissolved gases) exceed 120 percent saturation.

(3) On the basis of survival estimates made in the Snake River from 1966 to 1975, it is concluded that juvenile fish losses ranging from 40 to 95 percent do occur and a major portion of this mortality can be attributed to fish exposure to supersaturation by atmospheric gases during years of high flow.

(4) Juvenile salmonids subjected to sublethal periods of exposure to supersaturation can recover when returned to normally saturated water, but adults do not recover and generally die from direct and indirect effects of the exposure.

(5) Some species of salmon and trout can detect and avoid supersaturated water; others may not.

(6) Higher survival was observed during periods of intermittent exposure than during continuous exposure.

(7) In general, in acute bioassays, salmon and trout were less tolerant than the non-salmonids.

Dawley and Ebel (1975) found that exposure of juvenile spring chinook salmon, *Oncorhynchus tshawytscha*, and steelhead trout, *Salsa gairdneri*, to 120 percent saturation for 1.5 days resulted in over 50 percent mortality; 100 percent mortality occurred in less than 3 days. They also determined that the threshold level where significant mortalities begin occurring is at 115 percent nitrogen saturation (111 percent total gas saturation in this test).

Rucker (1974), using juvenile coho salmon, *Oncorhynchus kisutch*, determined the effect of individual ratios of oxygen and nitrogen and established that a decrease in lethal effect occurred when the nitrogen content fell below 109 percent saturation even though total gas saturation remained at 119 percent saturation, indicating the importance of determining the

concentration of the individual components (O_2 and N_2) of the atmospheric supersaturation. Nebeker, *et al.* (1976a), using juvenile sockeye salmon, *Oncorhynchus nerka*, also showed that there was a significant increase in fish mortality when the nitrogen concentration was increased while holding the total percent saturation constant. They also showed that there was no significant difference in fish mortality at different CO_2 concentrations.

Research completed by Bouck, *et al.* (1975) showed that gas supersaturated water at and above 115 percent total gas saturation is acutely lethal to most species of salmonids, with 120 percent saturation and above rapidly lethal to all salmonids tested. Levels as low as 110 percent will produce emphysema in most species. Steelhead trout were most sensitive to gas-supersaturated water followed by sockeye salmon, *Oncorhynchus nerka*. Chinook salmon, *Oncorhynchus tshawytscha*, were intermediate in sensitivity. Coho salmon, *Orcorhynchus kisutch*, were significantly the more tolerant of the salmonids though still much more susceptible than non-salmonids like bass or carp.

Daphnia magna exhibited a sensitivity to supersaturation similar to that of the salmonids (Nebeker, *et al.*, 1975), with 115 percent saturation lethal within a few days; stoneflies exhibited an intermediate sensitivity similar to bass with mortality at 130 percent saturation; and crayfish were very tolerant, with levels near 140 percent total gas saturation resulting in mortality.

No differences are proposed in the criteria for freshwater and marine aquatic life as the data available indicate that there probably is little difference in overall tolerances between marine and freshwater species.

The development of gas bubble disease in menhaden, *Brevoortia* sp., and their tolerance to gas saturation in laboratory bioassays and in the field (Pilgrim Nuclear Power Station Discharge Canal) are discussed by Clay, *et al.* (1975) and Marcello, *et al.* (1975). At 100 percent and 105 percent nitrogen saturation, no gas bubbles developed externally or in any of the internal organs of menhaden. At 105 percent nitrogen saturation, however, certain behavioral changes became apparent. Fish sloughed off mucus, swam erratically, were more excitable, and became darker in color. Menhaden behavioral changes observed at 110 percent nitrogen saturation were similar to those noted at 105 percent. In addition, at 110 percent gas emboli were found in the intestines, the pyloric caeca, and occasionally the operculum.

The behavioral changes described were also observed at 115 percent, and clearly defined subcutaneous emphysema was observed in the fins and occasionally in the eye. At 120 percent and 130 percent nitrogen saturation, menhaden developed (within a few hours) classic symptoms of gas bubble disease. Externally, emboli were evident in all fins, the operculum, and within the oral cavity. Exophthalmia also occurred and emboli developed in internal organs.

The bulbous arteriosis and swim bladder were severely distended, and emboli were found along the length of the gill arterioles resulting in hemostasis. At water temperatures of 30°C, menhaden did not survive, regardless of gas saturation level. At water temperatures of 15°, 22°, and 25°C, 100 percent of the menhaden died within 24 hours at 120 percent and 130 percent gas saturation. Fifty percent died after 96 hours at 115 percent (22°C). Menhaden survival after 96 hours at 110 percent nitrogen saturation ranged from 92 percent at 22° and 25° to 83 percent at 15°C. Observations on the relationship between the mortality rate of menhaden and gas saturation levels at Pilgrim Station during the April 1975 incident suggest that the fish may tolerate somewhat higher gas saturation levels in nature.

It has been shown by Bouck, *et al.* (1975) and Dawley, *et al.* (1975) that survival of salmon and steelhead smolts in seawater is not affected by prior exposure to gas supersaturation while in fresh water. No significant mortality of juvenile coho and sockeye salmon occurred when they were exposed to sublethal concentrations of supersaturated water and then transferred to seawater (Nebeker, *et al.*1976b).

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SOURCE

The above rationale information was taken from "Quality Criteria for Water", U. S. EPA-440/9-76-023, pages 139-146. 9, Environmental Protection Agency, Washington, D.C., 1976.

HYDROGEN SULFIDE

OBJECTIVE

Concentration of undissociated hydrogen sulfide should not exceed 0.002 milligrams per litre, at any time, or place, to protect aquatic life.

RATIONALE

Hydrogen sulfide is a flammable, poisonous gas in the atmosphere with a characteristic odor. The gas is soluble in water to 4,000 mg/L at 20°C. H_2S present in the water may result from the decomposition of natural detritus and organic benthal deposits, from the discharge of anaerobic sewage, and as a result of sulfide wastes from the tanning, pulp and paper, textile, chemical, and gas manufacturing industries.

When sulfides are discharged to water, they react with the available hydronium ions to form HS^- or H_2S depending on the pH and temperature. At pH 9 about 99 percent of the sulfide is in the form of HS^-, and at pH 6.5 about 25 percent is in the form of HS^- with the remainder consisting of the toxic undissociated H_2S form. Figure 1 can be used to determine the amount of undissociated H_2S from analysis of the total sulfide. Rapid combination of H_2S with other materials present in the water, such as oxygen and iron, has until recently caused the material to be overlooked as a significant problem.

Toxicity to aquatic life caused by the presence of hydrogen sulfide is dependent on the temperature, pH and dissolved oxygen present. At a lower pH the amount of the toxic form (H_2S) will be greater. When temperatures are low, fish life exhibit a greater tolerance to H_2S. The presence of high dissolved oxygen levels promotes rapid conversion of the H_2S to sulfates, and therefore, maintaining high D.O. levels reduces the likelihood of H_2S toxicity problems. Fish also exhibit a strong avoidance reaction to sulfide, and it is hypothesized that such a reaction would occur before they were harmed (Jones, 1964).

Most recent investigations into H_2S toxicity have been as it relates to the presence near the sediment-water interface especially near organic sludge deposits. In this zone, within a few centimeters of the bottom, significant concentrations of H_2S can occur if the benthal material is undergoing anaerobic decomposition. It should be noted that certain types of sludge deposits, notably those from pulp and paper mills, are more likely to cause H_2S formation than others.

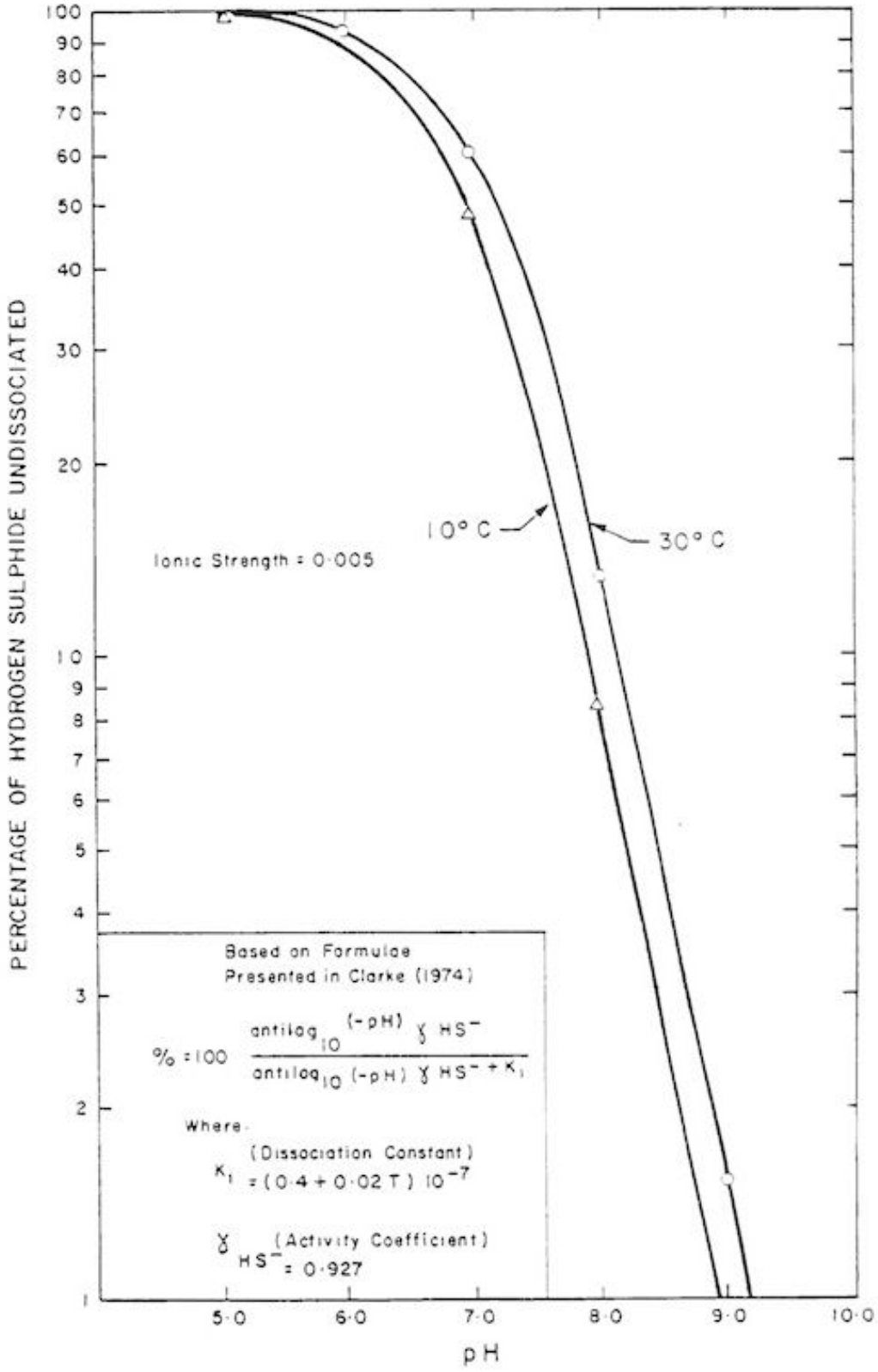

FIGURE 1: Percentage of Hydrogen Sulphide Undissociated as a Function of pH

Colby and Smith (1967) report that walleye eggs held in trays in zones where H_2S levels were commonly 0.1 to 0.02 mg/L did not hatch. The same study reports a 96-hour LC_{50} of 0.05 mg/L for walleye fry in a laboratory bioassay. Adelman and Smith (1970) report that "The maximum possible safe level of H_2S for (northern pike) eggs is between 0.014 and 0.018 mg/L and for sac fry between 0.004 and 0.006 mg/L for 96 hour exposure." Smith and Oseid (1973) report the 96-hour LC_{50} for juvenile brook trout, goldfish and walleye as 0.017, 0.090 and 0.020 mg/L respectively. They also indicated that the no-effect level for the growth of juveniles is also reported as 0.002 mg/L for bluegills, 0.004 mg/L for walleye and 0.003 mg/L for the fathead minnow.

On the basis of chronic tests evaluating growth and survival, the safe level for bluegill juveniles and adults was 0.002 mg/L and for fathead minnows was between 0.002 and 0.003 mg/L (NAS/NAE, 1974). Safe levels for various arthropods were between 0.002 and 0.003 mg/L (Smith, 1971). Oseid and Smith (1972) indicate that depending on other environmental factors H_2S levels of 0.0015 mg/L can reduce the physical capability of fish.

Oseid and Smith (1974) report that the maximum safe H_2S concentration for *Gammarus* is 0.002 mg/L in hard water. The mean 96-hour LC_{5O} for the same test was 0.022 mg/L indicating an approximate application factor of 10.

Clarke (1974) states that "A molecular (undissociated) H_2S concentration of 0.01 mg/L would appear to be an approximate safe level for sensitive fish development stages in water with a dissolved oxygen concentration of about 10-12 mg/L, and for periods of exposure of 100-200 hours." This proposed level is based on a slightly different method of calculating the undissociated H_2S from the total and is about 1.5 to 2 times greater than that reported in other works.

It should be noted that H_2S will occur in lakes where natural organic bottom material is decomposing. Under summer conditions the elevated levels will be limited to the hypolimnion, while in winter unsafe levels could spread throughout the profile. It is unlikely that this will occur in other than small lakes with little circulation. The primary area where H_2S may create problems in the Great Lakes is in relatively unmixed bays which received industrial wastes such as those from pulp and paper mills. Care must be exercised in interpreting H_2S data collected in the field, because a fish population could be thriving in the water overlying an area where the H_2S level at the mud-water interface is high. Where these H_2S levels are high near the decomposing benthal deposit, it is likely that dissolved oxygen conditions would also be low, thereby aggravating the problem. Although zones of elevated H_2S can occur, a transient fish population could be present, but the survival of eggs in the area is unlikely. The criterion proposed for H_2S will, as a secondary benefit, protect against the discharge of decomposable and settleable materials.

There is no criterion established for H_2S for either public or agricultural water supplies, because the unpleasant taste and odor would preclude use of that water at hazardous concentrations. The allowable levels of hydrogen sulfide in water for industrial use are greater than those necessary to sustain aquatic life.

Based upon the protection of a balanced population of complete aquatic life cycles, it is recommended that the maximum undissociated H_2S concentration be 0.002 mg/L.

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SOURCE

The above rationale information was taken from, "Appendix A Water Quality Objectives Subcommittee Report Great Lakes Water Quality 1974", pages 137-142. International Joint Commission. June, 1975.

IRON

OBJECTIVE

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 an 0.45 µ filter are less than 7 µg/L in 95 percent of all samples (Table 7). Inshore, iron in filtered samples average less than 50 µg/L in water intakes and concentrations never exceeded 200 µg/L (Table 6). 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 ln 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 greater than 16,000, approximately 320 and greater than 16,000 µg/L, respectively (Warnick and Bell, 1969). Mayflies were more sensitive than other species to iron and this was also true for 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 (Blesinger 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 I, 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 <u>total</u> iron in unfiltered water be 300 µg/L.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lake Water Quality 1975", pages 44-48, International Joint Commission, 1976.

LEAD

OBJECTIVE

The toxicity of lead is highly dependent on the alkalinity of the water. The toxicity declines as the alkalinity increases. The total lead concentration should not exceed the values given below:

Alkalinity (mg/L as $CaCO_3$)	Maximum Lead Concentration (µg/L)
up to 20	5
20 to 40	10
40 to 80	20
greater than 80	25

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 a ample, it has been found that at concentrations above 100 mg/L lead, more than 98 percent is precipitated after 24 hours. Above 10 mg/L, 70 percent is precipitated and above 1 mg/L, 10 percent 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 percent 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 percent of a total of 3,240 µg/L. As the total concentration decreased, dissolved lead increased to 27 percent 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 percent of all samples contain less than 3.0 µg/L (Table 7). At water intakes, mean lead concentrations are as high as 34 µg/L with maxima at 55 µg/L or less (Table 6). The higher inshore concentrations probably reflect local inputs to the lakes. *Total lead concentration in unpolluted inland Ontario waters are usually less than 10.0 µg/L.*

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 percent 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.*, 1978). Temperature must also be considered, since toxicity increases with temperature (Wong *et al.*, 1978) 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 it 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.* 1976). On a dissolved basis, this represented 39 and 84 µg/L. It would appear that brook trout are not as sensitive as rainbow trout.

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 Nuemen, 1971). 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 n blood and accumulate in kidney tissue (Hodson 1976; Holcombe *et al.* 1976 and Hodson *et al.* 1977). *The significance of these residues to fish health has not yet been completely 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. *These IJC lead objectives have been extrapolated to include the range of alkalinities measured in Ontario's inland Lakes:*

Alkalinity ($mg/L\ CaCO_3$)	Maximum Lead Concentration (µg/L)
up to 20	3
20 to 40	20
40 to 80	20
greater than *80*	*25*

Since lead may he 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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SOURCE

The above rationale information was primarily taken from "Appendix A, Water Quality Subcommittee, Great Lakes Water Quality 1975", pages 49-54, International Joint Commission, 1976.

Additional rationale information, prepared by staff of the Ministry of Environment, appear in italics.

MERCURY

OBJECTIVE

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).

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 (greater than 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 3.9 µg/g mercury (Dustman *et al.*, 1972). No moralities or population effects were noted in these species. Keith and Gruchy (1971) also reported finding gulls with

Table 4: Mercury Toxicity Studies

Organism	Compound	Effect	Lowest Conc. Producing Effect	Highest No Effect Conc.	Remarks	Reference
Gammarus sp.	Hg^{++}	24 hr LC_{50}	90 µg/L			Rehwoldt *et al.*
		96 hr LC_{50}	10 µg/L			
Nais sp.	Hg^{++}	24 hr LC_{50}	1900 µg/L			
		96hr LC_{50}	1000 µg/L			
Caddis fly	Hg^{++}	24 hr LC_{50}	5600 µg/L			
		96 hr LC_{50}	1200 µg/L			
Damsel fly	Hg^{++}	24 hr LC_{50}	3200 µg/L			
		96 hr LC_{50}	1200 µg/L			
Chironomus sp.	Hg^{++}	24 hr LC_{50}	60 µg/L			
		96 hr LC_{50}	10 µg/L			
Amnicola sp.	Hg^{++}	24 hr LC_{50}	1100 µg/L			
		96 hr 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 cone.	Christensen
alevins	CH_3Hg^+	GOT(enhanced)	0.93 µg/L	0.08 µg/L		

Table 4 cont'd: Mercury Toxicity Studies

Organism	Compound	Effect	Lowest Conc. Producing Effect	Highest No Effect	Remarks	Reference
Rainbow trout	CH_3Hg^+	Decreased Hematocrit	10 µg/L		12 weeks exposure	O'Connor & Fromm
		Plasma electolytes in vitro		10 µg/L	"	
		O_2 metabolism		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	1µg/L	0.2µg/L	19-25 day exposures	Kihlstrom *et al.*
		% hatching	0.2µg/L			
Rainbow trout	Hg^{++}	decreased activity	50 µg/L		4-6 day exposure	Alexander
Brook trout	CH_3Hg^+	deformities, deaths in 2nd generation	0.93 µg/L	0.29 µg/L	3 generation exposure	McKim *et al.*
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	$HgCl_2$	Eggshell thinning	1µg/g (diet)	2 µg/g (diet)		Stoewsand *et al.*

Table 4 cont'd: Mercury Toxicity Studies

Organism	Compound	Effect	Lowest Conc. Producing Effect	Highest No Effect	Remarks	Reference
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

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 in 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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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975", pages 55-61, International Joint Commission, 1976.

NICKEL

OBJECTIVE

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 9.9 million kg 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 percent of samples are less than 6.0 µg/L (Table 7). 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 6). 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 (Gong, 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 gives 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 greater than 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* greater than 700 µg/L (Anderson, 1948). Chronic three-week nickel exposures to *Daphnia magna* caused 50 percent mortality at 130 µg/L and 50 percent and 16 percent 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 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 is 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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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975", pages 62-65, International Joint Commission, 1976.

OIL AND PETROCHEMICALS

OBJECTIVE

- Oil or petrochemicals should not be present in concentrations that:
- can be detected as a visible film, sheen, or discolouration on the surface;
- can be detected by odour;
- can cause tainting of edible aquatic organisms;
- can form deposits on shorelines and bottom sediments that
- are detectable by sight or odour, or are deleterious to resident aquatic organisms.

RATIONALE

AMENITIES, WATERFOWL AND HEALTH

The four objectives are self-evident on the basis of general knowledge. However, to protect aesthetic values, water and shoreline recreation, all four objectives are required.

The amount of oil required to produce a visible slick will vary with type of oil and weather conditions. However, the American Petroleum Institute has estimated that the first trace of irridescence or colour is formed when about 15 millilitres of oil is spread over 100 square metres (= 100 U.S. gallons over one square mile, or a film about 0.15 microns thick) (NAS/NAE, 1974).

Surface slicks must also be prevented to protect water-birds. and aquatic mammals. The mortality of water-birds as a result of severe oil pollution is direct and immediate, and in major oil spills, deaths have been measured in the thousands. Birds that feed from the water or settle on it are vulnerable; diving ducks especially so. Plumage matted with oil allows water to displace air, causing the bird to lose both insulation and buoyance. Oil ingested during preening can have toxic effects. Less obvious, but long-continued small slicks such as from sewered oil, will in the end have similar debilitating effects on resident water-birds.

Available information on occupational health and industrial hygiene indicates that any tolerable health concentrations for petroleum-derived substances far exceeds the limits of taste and odour. Thus, any hazards to humans from drinking oil-polluted water will not arise because such substances become objectionable at concentrations far below their chronic toxicity levels. Oils of animal or vegetable origins are usually non-toxic to humans and aquatic life.

AQUATIC ORGANISMS

The toxicity of crude oils and their derived substances to aquatic life cannot be stated in simple terms because they contain many different organic compounds and inorganic elements. The major components of crude oil are a series of hydrocarbons from paraffins and naphthenes to aromatics, resins, asphaltenes, heterocyclic compounds and metallic compounds. The hydrocarbons make up the major group of acutely toxic compounds and there is agreement that their toxicity increases along the series paraffins, naphthenes, and olefins to aromatics. Within each series of hydrocarbons, the smaller molecules are more toxic than the larger molecules. However, the high carbon number aromatics are the more persistent (Butler and Berkes, 1972).

Among freshwater organisms some information is available for fish. Lethal levels of oils are in the hundreds or thousands of µL/L. Bunker oil is lethal to American shad at 2400 µL/L (Tagatz, 1961) and Atlantic salmon at 1700 µL/L (Sprague and Carson, 1970). Crude oil slicks exceeding concentrations equivalent to 500 mg/L killed young coho and sockeye salmon in laboratory tests (Morrow, 1974). Diesel oil kills shad at 167 µL/L (Tagatz, 1961). Some petroleum products appear to contain no soluble poisonous substances but when emulsified by agitation with water they prove deadly to fish. Agitated solutions of automobile gasoline and jet aviation fuel have been found to be lethal to fingerling salmon at concentrations of 100 and 500 mg/L respectively (EPA, 1974). Long-term effects would not be expected from these two fuels since they are volatile and would not remain in water for more than short periods, but short-term sublethal damage could occur.

An excellent set of tests has recently been reported in a provisional report by the U.S. National Water Quality Laboratory (Hedtke *et al.*, 1974). Used crankcase oil was used in their tests, and this is probably a major source of oil in the Great Lakes. Floating oil killed fathead minnows at 11,000 µL/L, but mixed into the water it killed these fish at 1,600 µL/L, and flagfish at 1,000 µL/L. In chronic tests with flagfish (*Jordanella floridae*, 338 µL/L affected reproduction, while 93 µL/L did not. These are all nominal or "added" concentrations. We may take the ratio of proven "safe" level to the LC_{50}, = 93/1,000 = 0.093, as an application factor which is potentially useful in other situations.

Toxicity to marine animals (i.e. living in sea water), seems to have been studied more extensively than toxicity to freshwater forms. Marine invertebrate larvae seem particularly sensitive to oils. About 100 µL/L of various crude oils were lethal to planktonic stages of crab larvae and several other invertebrates (Mironov, 1969a,b), shrimp (Mills and Gulley, 1972). The same concentration of No.2 fuel oil killed kelp crab larvae (Lichatovich *et al.*, 1971), while 10 µL/L of "oil" killed a copepod in 4 days (Mironov, 1968). Lobster larvae were killed in 4 days by 13 mg/L of dispersed crude oil, and in 30 days by only 0.78 mg/L. Those were nominal concentrations and the actual concentrations of oil estimated by measurement of the aromatics by u.v. spectrophotometry were only 18 percent of those values. That is, measured

concentrations in the lobster experiments were 4-day LC_{50} = 2.3 mg/L and 30-day LC_{50} 0.14 mg/L (Wells, in preparation).

Some sub-lethal effects have also been documented in marine animals. Crude oil at 100 µL/L caused inactivity and lack of survival over 2 weeks of *Neopanope* (Katz, 1973). For lobster larvae, the safe concentration of dispersed oil for rate of development and moulting was 0.72 mg/L nominal concentration, about the same as the 30-day LC_{50}. The measured concentration would be 0.13 mg/L (Wells, in preparation). The ratio of this "safe" concentration to the 4-day LC_{50} is 0.72 divided by 13 = 0.55, a value which may be used as an application factor. For floating crude oil, the 4-day LC_{50} for lobster larvae was 150 mg/L, moulting was slowed at 12.5 mg/L, yielding a similar application factor of 0.083. For floating No.2 fuel oil, the same values were 60 and 12.5 mg/L yielding an application factor of 0.21 (Wells, in preparation).

It is probable that the safe level of crude oils for sensitive Great Lakes crustaceans would be in the vicinity of 2-4 µL/L, as is the case for their marine cousins. However, such experiments have apparently not been done for freshwater invertebrates, and thus it is not warranted to use these low concentrations as criteria in the Great Lakes.

Use of application factors does seem warranted. The three application factors obtained for a marine crustacean are close to the one calculated for flagfish in fresh water. The median of the four application factors is 0.088, or close to 0.09. Applying this to the average lethal concentrations mentioned for freshwater fish, we may estimate "safe" levels for fish as follows:

Bunker oil	- 180 µL/L
Used crankcase oil	- 120 µL/L
Crude oil slicks	- 45 µL/L
Jet aviation fuel	- 45 µL/L
Diesel oil	- 15 µL/L
Automobile gasoline	- 9 µL/L

Those concentrations are nominal (added) ones, and would have to be related to the measured concentrations in the water, according to the chemical procedures used in any individual situation.

The approximate "safe" concentrations listed above, are higher than those which would be expected to cause problems of odour, amenities, etc. under the objectives. Therefore the safe concentrations for aquatic life have not been listed in the objectives, since other uses are more restrictive. The application factor has been put into the objectives. It seems likely that future research with sensitive organisms will show that in some situations, safe concentrations will be the most restrictive.

CONTROL

The eventual fate of oil in water depends on the basic processes of weathering, dispersion and degradation. The removal of hydrocarbons from water is accomplished by its breakdown into carbon dioxide and water. The natural processes that bring about the disappearance of oil in water include evaporation, solution, formation of emulsions, and sinking but none of these processes render the oil harmless to the aquatic environment. The ultimate destruction of oil depends upon its oxidation which is mainly by bacteria, although some photo-oxidation takes place.

There have been numerous corrective measures derived to clean up spilled oil, such as mechanical means and the use of detergents. Mechanical means have proven quite successful, but the use of detergent to disperse the oil has in many instances produced considerably more toxicity to aquatic life than the oil proper. The toxicity effects of detergent-oil mixtures are covered by the section on Unspecified Toxic Substances.

The only effective measures for the control of oil pollution of water is prevention of all spills and releases. In this connection it is not generally recognized that much more oil enters world waters from routine operations and dumping, than from spills. For example, the International Lake One Water Pollution Board (1969, page 252) has estimated that the input of oil and grease to the Detroit and St. Clair Rivers is in excess of about 1,100 barrels per day. This is in the vicinity of 64,000 metric tons of oil per year. Such a "normal" operation is equivalent to the amount of oil from 3 or 4 major tanker wrecks, every year, each the size of the "Arrow" disaster on the Canadian east coast.

Similarly, the International Niagara River Pollution Board (IJC, 1971, p. 26) reported that 29 million pounds of oils were discharged to the Upper Niagara River. This is about 13,000 metric tons per year, almost equivalent to one "Arrow" sized wreck. Furthermore, the Board estimated that 40 percent of the oil came from municipal treatment plants. Large quantities of oil are also contained in dredging spoils. Dredging operations In Cleveland harbour in 1966 and 1967 resulted in the disposal of over 16,000 metric tons of oil and grease to Lake Erie (International Lake Erie Water Pollution Board et al, 1969). Again, that is just about equal to the amount of oil in the wrecked "Arrow". It is evident that as much pollution-control effort should be devoted to these routine sources as to spills.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1974", pages 143-151. International Joint Commission, June, 1975.

PHENOL

OBJECTIVE

Concentration in waters should not exceed 1 microgram per litre to protect against tainting of edible fish flesh.

RATIONALE

Phenolic compounds include a wide variety of organic chemicals. The phenols may be classified into monohydric, dihydric, and polyhydric phenols depending upon the number of hydroxyl groups attached to the aromatic ring. Phenol itself, which has but one hydroxyl group, is the most typical of the group and is often used as a model compound. The properties of phenol, with certain modifications depending on the nature of the substituents on the benzene ring, are shared by other phenolic compounds. Phenolic compounds arise from the distillation of coal and wood; from oil refineries; chemical plants; livestock dips, human and other organic wastes; hydrolysis, chemical oxidation and microbial degradation of pesticides; and from naturally occurring sources and substances. Some compounds are refractory to biological degradation and can be transported long distances in water.

Phenolic compounds can affect freshwater fishes adversely by direct toxicity to fish and fish-food organisms; by lowering the amount of available oxygen because of the high oxygen demand of the compounds and by tainting of fish flesh (EIFAC, 1973). Shelford (1917) observed that a concentration of 1 cc per liter (purity of compound and concentration are unknown) was rapidly fatal to fish but solutions of one half to three quarters of this amount (i.e., 0.5 to 0.75 cc) would require up to one hour to kill fish. Subsequent studies have confirmed the toxicity of phenol to both adult and immature organisms (EIFAC, 1973). Decreased egg development in the oyster, *Crassostrea virginica*, has been found to occur at levels of 2 mg/L phenol (Davis and Hidu, 1969).

Various environmental conditions will increase the toxicity of phenol. Lower dissolved oxygen concentrations, increased salinity and increased temperature all enhance the toxicity of phenol (topic, 1973). It has been shown that phenol and o-cresol have 24-hour LC_{50}'s of 5 and 2 mg/L respectively for trout embryos (Albersmeyer and von Erichsen, 1959). Rainbow trout were killed in 7.3 mg/L phenol in 2 hours and in 6.5 mg/L phenol in 12 hours; at these concentrations there was rapid damage to gills and severe pathology of other tissues (Mitrovic, *et al.*, 1968). Pathologic changes in gills and in fish tissues were found at concentrations in the range of 20 to 70 µg/L phenol (Reichenback-Klinke, 1965).

McKee and Wolf (1963), following a review of world literature, concluded that phenol in a concentration of 1 µg/L would not interfere with domestic water supplies, 200 µg/L would not interfere with fish and aquatic life, 50 mg/L would not interfere with irrigation, and 1,000 mg/L

would not interfere with stock watering.

A major aesthetic problem associated with phenolic compounds is their organoleptic properties in water and fish flesh. Threshold odour levels range from 55 µg/L for p-cresol (Rosen, *et al.*, 1962) to 2 µg/L for 2-chlorophenol (Burttschell, *et al.*, 1959). The chlorinated phenols present problems in drinking water supplies because phenol is not removed efficiently by conventional water treatment and can be chlorinated during the final water treatment process to form persistent odour-producing compounds. Thus, odour problems are created in the distribution system. Boetius (1954), Fetterolf (1962), Schulze (1961), and Shumway (1966) estimated threshold fish flesh tainting concentrations for o-chlorophenol, p-chlorophenol, and 2,4 -dichlorophenol to range from 0.1 µg/L to 15 µg/L. The o-chlorophenol produced tainting at the lower concentration.

An objective of 1 µg/L phenol, which is about half of the chlorophenol odour effect level for a water supply and near the threshold fish flesh tainting concentration, should protect the freshwater environment for such users.

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SOURCE

The above rationale information was taken from "Quality Criteria for Water", U.S.-EPA-440/9-76-023, pages 347-351, Environmental Protection Agency, Washington, D.C., 1976.

RADIONUCLIDES

OBJECTIVE

Radiation exposure should be kept as low as reasonably achievable, economic and social factors being taken into account.

Radioactive materials should not be present in receiving waters as a consequence of the failure of an installation to exercise practical and economic controls to minimize releases.

Regardless of economic factors, the dose equivalent to individuals consuming the waters must not exceed the limits recommended for the appropriate circumstances by the Atomic Energy Control Board.

Water quality objectives based on these considerations will ensure that fish and other aquatic life will not receive hazardous doses of radiation.

Present Objectives are as follows:

Gross beta emitters	100 pCi/L
Radium-226	3 pCi/L

Where other radioisotopes occur, their significance should be assessed for each situation with respect to the potential hazard to humans and to aquatic life.

RATIONALE

The water quality objectives for radioactivity are established for the protection of aquatic life and recreation. It is assumed that this protection is guaranteed if the water is good enough for human consumption. This assumption is supported by many reports in the literature; the following excerpts are examples:

> "The doses that cause injury to plant and animal life are very much higher than would be permitted under any circumstances in which human safety must be considered" (Environmental Radioactivity, 1973).
>
> "Compared with the experimental data available for warm-blooded animals, only a meagre amount of information is available on chronic dose-effect relationships for aquatic forms. The preponderance of available data indicates, however, that no effects are discernible on either individual aquatic organisms or on populations of organisms at dose rates as high as several rads/week" (EPA, 1973).

Ra-226

The objective for the concentration of Radium-226 is derived from the recommendations of Committee 2 of the International Commission on Radiological Protection (ICRP). The Report of Committee Two states: "For a radionuclide or a mixture of radionuclides which does not have the total body or the gonads as critical organ, it is suggested that the average permissible level for large populations be one-thirtieth the continuous occupational value (168 hr/week), calculated according to the basic rules (stipulated in the Report)". For radium-226, this occupational value is 100 pCi/L. Thus the Provincial objective is 100/30 pCi/L. This is written as 3 pCi/L.

Gross -B

Results of gross-B analysis cannot be related directly to the dose equivalent to an individual consuming the water or to an aqueous species. They are useful, however, for detecting trends in the water quality of any particular location.

The 1000 pCi/L objective represents an arbitrary investigation level. If it is exceeded, more detailed analysis of the water is required to determine the radionuclides responsible for the activity.

N.B. The objectives for radium and other radionuclides are currently being reassessed in the light of knowledge gained since the publication, in 1959, of the Report of Committee Two of the ICRP.

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SOURCE

The above rationale was prepared by staff of the Ministry of Environment.

SELENIUM

OBJECTIVE

Concentrations of total selenium in an unfiltered water sample should not exceed 100 micrograms per litre to protect aquatic life.

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 x 10^{-5} percent. 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 rook 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 rooks 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 percent 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 6).

Lake sediments seem to act as reservoirs or sinks; in the northern United States they contain 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 percent stayed in soil and 25 percent 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 rats (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 sufficient 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 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 93 percent 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 Latham, 1976) indicated that lethality of selenium to zebraflsh larvae *(Brachydanio rerio*) varied with the selenium salt used. The 96-hour and 10-day LC_{50}'s (Table 8) indicate that selenate salts are less toxic than selenite salts.

TABLE 8: 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	approx. 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 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 Latham (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 5 day 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) fcund a 7 day 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; Aurthur, 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 selenate 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.

*Based on the toxicity t*o zebra fish and *gold fish, a concentration of* selenium *of 100 µg/L should protect* aquatic life.

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SOURCE

The above rationale information was taken primarily from "Appendix A, Water Quality Objectives Subccmmittee Report, Great Lakes Water Quality, 1975", p. 66-74, International Joint Commission, 1976.

Additional rationale information, prepared by staff of the Ministry of Environment, appear in italics.

SILVER

OBJECTIVE

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

RATIONALE

Silver occurs in the native state as a constituent of various natural alloys and in a great variety of minerals combined with sulfur, antimony, arsenic tellurium and selenium (Boyle, 1958). The average silver content of soils is about 0.10 mg/kg and certain coals contain considerable amounts of silver (2-10 mg/kg in the ash). The silver halides, such as silver iodide, are relatively insoluble in water in contrast to silver nitrate, the most common salt. A very comprehensive review of the sources, use, distribution, losses to the environment and human health aspects of silver has been prepared by Carson and Smith (1975).

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

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

Copeland and Ayers (1972), found an average of 0.3 µg/L silver in Lake Michigan waters. In a survey of trace metals in the waters of the United States, Kopp and Kroner (1967) indicated that silver was detectable in less than 7 percent of all samples with a mean observed value of 2.6 µg/L. The greatest occurrenoe of silver was in the Colorado River basin where it was observed in 18 percent of the samples at a mean concentration of 5.8 µg/L. In the St. Lawrence River, concentrations in water from inshore intake pipes ranged from non-detectable to 6 µg/L. The mean concentration was 2.6 µg/L. These concentrations are suspiciously high in light of toxicity data and the much lower values observed by Copeland and Ayers (1972) and Bard *et al*, (1976). They suggest either local inshore contamination or deficiencies in the analytical procedures. Silver has not been included in past monitoring of offshore water quality in the Great Lakes.

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

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

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

Invertebrates are also sensitive to silver. After 24 hours, 50 percent or more of *Daphnia magna* were dead or obviously incapacitated at a concentration of 19 µg/L silver (Bringmann and Kuhn, 1959). After 64 hours, 50 percent of *Daphnia magna* were immobilized at a concentration of silver of 3.2 µg/L (Anderson, 1948). Fifty percent of a planarian population (*Polycelis nigra*) were killed within 2 days at an exposure to silver of 150 µg/L (Jones, 1940). Nehring (1973) tested the response of several aquatic insects to silver. The 15-day LC_{50} for silver for mature stonefly naiads (*Pteronarcys californlca*) was 8.8 µg/L. Immature naiads of the same species were more sensitive with a 7-day LC_{50} of less than 4 µg/L. Nearly all mayflies *(Ephemerella grandis*) were killed in concentrations of silver as low as 9 µg/L after 10 days. The largest bioconcentration factor was about 300 times with a tissue level of 20 mg/kg. The 96-hr. LC_{50}'s for *Mercenaria mercenaria* (hard clam) and *Crassostrea virginica* (oyster) were 21 and 5.8 µg/L respectively in seawater at a temperature of 26°C. (Calabrese and Nelson, 1974; Calabrese and Collier, 1973).

"Effective control" of the nematode *Pratylenchus penetrans*, a plant parasite, was achieved with 10 µg/L silver, although other nematodes were less sensitive (Pitcher and McNamara, 1972). First stage larvae of a marine crustacean, *Palaemon serratus*, changed their orientation to light at 200-500 µg/L silver, concentrations similar to the 96-hr. LC_{50}. Larvae of *Carcinus maenus* were more sensitive with a 96-hr. LC_{50} of 10-100 µg/L and a change in photokinesis at 1 µg/L (Amiard, 1976).

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

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

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

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

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

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

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

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

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

CONCLUSION

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

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SOURCE

The above rationale information was taken from "Appendix A. Water Quality Objectives Subcommittee Report, Great Lakes Water Quality. 1975", pages 28-34., International Joint Commission, 1976.

TEMPERATURE

OBJECTIVE

1) General

The natural thermal regime of any body of water shall not be altered so as to impair the quality of the natural environment. In particular, the diversity, distribution and abundance of plant and animal life shall not be significantly changed.

2) Waste Heat Discharge

a) Ambient Temperature Changes

The temperature at the edge of a mixing zone shall not exceed the natural ambient water temperature at a representative control location by more than 10°C (18°F). However, in special circum stances, local conditions may require a significantly lower temperature difference than 10°C (18°F). Potential dischargers are to apply to the Ministry of the Environment for guidance as to the allowable temperature rise for each thermal discharge. This Ministry will also specify the nature of the mixing zone and the procedure for the establishment of a representative control location for temperature recording on a case-by-case basis.

b) Discharge Temperature Permitted

The maximum temperature of the receiving body of water, at any point in the thermal plume outside a mixing zone, shall not exceed 30°C (86°F) or the temperature at a representative control location plus 10°C (18°F) or the allowed temperature difference, whichever is the lesser temperature. These maximum temperatures are to be measured on a mean daily basis from continuous records.

c) Taking and Discharging of Cooling Water

Users of cooling water shall meet both the Objectives for temperature outlined above and the "Procedures for the Taking and Discharging of Cooling Water" as outlined in the Implementation Procedures for Policy 3.

RATIONALE

The temperature rise and the maximum temperature permitted at the edge of an approved mixing zone at the maximum safe limit for the protection of habitat and organisms in the receiving water. Most Ontario fish species can move into water 10°C above their acclimation temperature, to a maximum temperature of 30°C, for short periods without suffering impairment. [(1)] However, certain fish species acclimated to high temperatures, particularly in

winter, cannot safely move into water more than 10°C below their acclimation temperature.[1][2] For this reason the "Procedures for the Taking and Discharge of Cooling Water" [3] recommend that fish residency in the mixing zone be minimized.

The maximum temperature of 30°C is near the upper incipient lethal temperature for many Ontario fish species when they are acclimated to natural, summer water temperatures. Most Ontario fish species can tolerate brief exposures to 30°C, but for some more sensitive species exposure of less than one hour are potentially lethal. Avoidance of high temperatures by mobile species, and the small area influenced by temperatures close to 30°C, minimizes the potential for damage to juvenile and adult fish. Protection of fish habitat and the benthic community is achieved by the specifioation of a rapidly mixing discharge in the "Procedures for the Taking and Discharge of Cooling Water" [3] and the provision of Policy 5 - Mixing Zones, [3] that calls for the size of the mixing zone to be kept as small as possible. These two policies are designed to ensure that the smallest practicable area is disturbed by the thermal discharge.

The temperature rise of 10°C and the maximum temperature of 30°C, rather than lower temperatures, were chosen for the edge of the mixing zone to encourage a reduction in the volume of water pumped through cooling water systems. This reduction will lessen the damage done to aquatic life by entrainment and impingement. The higher temperature increases resulting from this volume reduction cause higher rates of entrainment mortality due to temperature alone. However, lower total mortality results since fewer organisms are exposed to the overall trauma of entrainment which includes mechanical and chemical, as well as thermal stress. [4]

To achieve this goal of reducing the volume of water pumped, the highest temperature rise through the cooling water system that is technologically and economically feasible is recommended.

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SOURCE

The above rationale information was prepared by staff of the Ministry of Environment.

TOTAL DISSOLVED SOLIDS

OBJECTIVE

Dissolved solids must not be added to increase the ambient concentrations by more than ⅓ of the natural concentrations to protect aquatic life. The added solids should not significantly alter the overall ionic balance of the receiving waters.

RATIONALE

Water devoid of dissolved materials is intolerable in nature because pure water will not support aquatic life. Natural waters contain endless varieties of dissolved materials in concentrations that differ widely from one locality to another as a well as from time to time. Many of these dissolved materials are essential for growth, reproduction. and the general well-being of aquatic organisms. The chlorides, carbonates, and silicates of sodium, potassium, and magnesium are generally the most common salts present. Traces of most other essential substances are also found.

Aquatic organisms live in different concentrations entrations of dissolved substances but productivity declines as the concentrations move from the optimum. Seldom, if ever, are the dissolved substances the optimum concentrations as we know them. The range of tolerance may be relatively wide, but when the concentrations reach too low or too high a level, organisms degenerate and die. Different organisms live and thrive under variations from the optimum. Some organismsare equally at home in sea water and in fresh water. Other organisms will tolerate only one or the other.

Any of the substances necessary to aquatic organisms has a range of concentration that is both essential and tolerable. The tolerance one for any osubstance vary depending on the concentration of other substances present. The presence of certain substances synergizes the effects of some materials but antagonizes the effeots of others. Under optimal concentrations, the synergistic and antagonistic effects are in balance and relatively high concentrations can be tolerated without adverse effects.

Although several measuresof dissolved materials are available, no measure in itself is adequate as an index of optimum concentration nor is any single measure adequate to express the range of tolerance. The biological effects depend on the concentrations of some individual solutes, someof which are tolerated in terms of grams per litre but others only in nanograms per litre. Some exert considerable osmotic pressures, but for others the osmotic effect is negligible. Some substances contribute greatly to conductivity. while others have little or no effect.

In general, the concentrations of dissolved materials in natural fresh waters are below the optimum for maximum productivity. In many instances, therefore, the addition of any of a large number of substances will be beneficial. In this way, many watercourses have a capacity to absorb materials to advantage. But the addition of what may be considered beneficial substances must be controlled so that they will exceed favourable limits.

The osmotic concentration of the body fluids of a fresh water animal is generally the maximum concentration of dissolved material that the animal will tolerate. In some animals, notably some of the fresh water mollusks, the body fluids have an osmotic concentration as low as 50 milliosmoles (the equivalent *of* about 0.025 molar or 1,500 mg/L sodium chloride). If the dissolved materials are relatively innocuous, having only an osmotic effect, it is judged that the total dissolvedmaterials in a watercourse may be increased to a certain extent but they should not exceed 50 milliosmoles if the fauna is to be maintained.

Many species of diatoms are very sensitive to changes in chloride and other salt concentrations. Some species, such as those in mountain streams and in black water streams of the coastal plains, can live only in waters with extremely low concentrations of salts.

The addition of salts to such streams will eliminate many desirable species of diatoms and permit undesirable species to flourish. Such changes may reduce the desirable food sources and bring about nuisance problems as well. It is believed that the total dissolved material in a watercourse should not be increased by more than one-third of that which is characteristic of the natural conditions of such a watercourse.

The toxicity of substances added to natural waters often depends on the substances already present in the receiving waters. With synergism, the toxicity increases, and with antagonism it decreases. Again the reaction of the toxic substances may produce, in some cases, new products of greater toxicity, and in others, products of lesser toxicity.

In view of the many factors that become involved in the disposal of soluble materials in natural waters, it is vident that no simple answer is available. Therefore, bioassays should be used to determine the amounts of the materials that may be tolerated without reducing the productivity of the watercourse in question.

SOURCE

The above rationale information was taken from "Water Quality Criteria, Report of the National Technical Advisory Committee to the Secretary of the Interior" pages 39-40, Federal Water Pollution Control Administration, Washington, D.G., 1968.

TOTAL PHOSPHORUS

GUIDELINE

Current scientific evidence issufficient to develop a firm objective at this time. Accordingly, the following phosphorus concentrations should only be considered as general guidelines which should be supplemented by site-specific studies:

To avoid nuisance concentrations of algae in lakes, average total phosphorus concentrations for the ice-free period should not exceed 20 µg/L;

A high level of protection against aesthetic deterioration will be provided by a total phosphorus concentration for the ice-free period od of 10 µg/L or less. This should apply to all lakes naturally below this value;

Excessive plant growth in rivers and streams should be eliminated at a total phosphorus concentration below 30 µg/L.

RATIONALE

The natural algal and macrophyte growths in any lake or river are mainly influenced by the morphology of the water body and the supply of nutrients. The amounts and species of aquatic plants play a major role in establishing the physical appearance and the total biological community of a waterway. For example, lakes with little algal growth will generally have clear water, low primary productivity and low fish productivity. Conversely, lakes with greater algal growth will be more turbid, have higher primary productivity and higher fish productivity.

It has been shown that phosphorus limits the algal and plant growth in many lakes and rivers (Schindler 1977, Wetsel 1975, Golterman 1975, Painter *et al.* 1976) and thus to a large extent controls the species present (Nicholls *et al.* 1977), the productivity and appearance of waterways.

It should be noted that phosphorus in the elemental form is very toxic to aquatic life (Zitko *et al.* 1970) but this form does not occur naturally. Elemental phosphorus is produced in some manufacturing processes and it would my be found in urface waters as an industrial discharge. Fortunately, this is a very rare occurrence.

Phosphorus in chemically combined form is not toxic to aquatic life and thus does not need to be controlled to protect aquatic life from any direct negative effects. Phosphorus concentrations must be controlled, however, to prevent any undesirable changes in the aquatic ecosystem due to increased algal growth which will inevitably result from phosphorus inputs to surface waters.

Increased algal growth resulting from phosphorus inputs to surface waters can be a benefit in some cases by providing more e food for fish production, (Oglesby 1977, Smith 1968, 1969). On the negative side, increased algal growth increases turbidity, thus reducing the aesthetic appearance of the water. The increased production of organic matter requires more oxygen during decomposition and may adversely affect aquatic life in the bottom waters of lakes by reducing the oxygen to unacceptable levels (Lasenby, 1975). Fish habitat and spawning grounds may be destroyed and, while total fish production may stay the same or even increase, there can be changes to less desirable species.

LAKES

AESTHETICS

Changes in the aesthetic appearance of a lake are highly subjective. The turbidity of a lake may increase a measured by a Secchi disc but the change may not be perceived by the general public. However, it is reasonable to assume that the aesthetic value of a lake will not be seriously impaired unless algal scums form on the surface. Dillon and Rigler 1975, concluded that algal scums would not occur if the summer average chlorophyll a concentration did not exceed 5 µg/L which correspond to a spring maximum total phosphorus concentration of 18.5 µg/L. Data from Gravenhurst Bay following phosphorus reduction at a sewage treatment plant have demonstrated that algal scums have not formed once the summer average total phosphorus concentrations fell below 20 mg/m^3 (Dillon 1977).

Therefore it may be concluded that aesthetic impairment of a lake will be prevented if the total phosphorus concentration for the ice free period is less than 20 µg/L.

Dillon and Rigler 1975, pointed out that maximum concentrations in the spring of 18.5 mg/m^3 total phosphorus may not protect dissolved oxygen concentrations in the bottom waters of the lake. Therefore, for a higher level of protection, they recommend that the maximum spring concentration of total phosphorus should be 10 µg/L. It is implied in applying this value as a guideline that the ice free average concentration of total phosphorus will not exceed this value.

RIVERS

Macrophytes are the main form of plant growth of concern in rivers. Algal growth in not generally a problem simply because algae float away with the river flow. Macrophyte growth affects aesthetics and oxygen concentrations.

Wong and Clark, 1975 and Painter, Wong and Clark 1976 reported nutrient concentrations/plant growth relationships. They concluded that the headwaters of rivers which had total phosphorus concentrations of 30 µg/L or less did not support the problem species *Potamogeton pectinatus* but rather a mixed plant population.

Although the 30 µg/L total phorphorus concentration corresponded to relatively clear rivers, they could not support this value as being an objective for providing both desirable biomass and dissolved oxygen concentrations. However, in the absence of better data, the 30 µg/L concentration may be applied as an objective likely to prevent objectionable aesthetic conditions. This concentration may also provide protection against excessive daily oxygen fluctuations although site specific data would be required to suhstantiate that conclusion.

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SOURCE

The above rationale was prepared by the staff of the Ministry of Environment.

TURBIDITY

OBJECTIVE

Suspended matter should not be added to surface water in concentrations that will change the natural Secchi disk reading by more than 10 percent.

RATIONALE

Materials present is a lake absorb, scatter, and reflect light as it passes through the water (Hutchinson 1957). Dissolved materials absorb light but substantial reduction in light transmission more commonly results from the presence of suspended particles. In areas where such particles are high in concentration, their influence can be noted by the human eye and the water is called turbid. Moreover, the effect of the particles on the light depends not only on concentration but also on size, shape, color, refractive index, and specific gravity.

The turbidity of the water can have e great effect on the types and quantities of algae that grow in a lake by altering the quality and quantity of light available for photosynthesis (Brylinsky and Mann, 1973; Hutchinson, 1967; and Pechlaner, 1970). This has been clearly demonstrated in the Great Lakes by Chandler's work in western Lake Erie. Chandler shows that the algal productivity is high when turbidity is low and vice versa (Chandler 1940; 1945). His studies indicate that the composition, size, duration and emergence of phytoplankton pulses in this area are influenced by turbidity (Chandler, 1942a; 1942b; 1944; 1945). As the light energy fixed into organic matter by phytoplankton is the basis of almost all aquatic life, the turbidity-induced effects on these plants have ramifications throughout the ecosystem.

The ecological effects due to turbidity may be entirely natural. Such mechanisms as wave induced shoreline erosion and resuspension of bottom sediments, and the bloom of algal cells under favorable conditions may decrease light transmission to such an extent that the magnitude of photosynthesis is substantially curtailed. In addition, human activities may greatly alter turbidity and increase its fluctuations, thus having a large and usually unfavorable effect on the ecosystem. Besides the obvious effects on turbidity from direct addition of particulates, human activities can indirectly increase turbidity by adding nutrients that cause increased production and abundance of aquatic plants. In special circumstances human activities can also decrease turbidity by adding substances that cause the existing particles to aggregate and settle out of suspension faster than otherwise would occur. Even this effect could be detrimental to beneficial uses of the water by allowing much greater than usual algal production and by smothering benthic organisms and fish eggs. Large blooms of algae can lead to taste and odor problems in public water supplles as well as making the water aesthetically less suitable for such recreational activities as boating, water skiing, fishing, etc. Thus alterations in the ability of Great Lakes water to transmit light need to be strictly controlled.

This need was recognized for all aquatic environments in the U.S. by the National Academy of Sciences in its recommendations on water quality criteria (NAS/NAE, 1974). The Academy recommends "The combined effect of color and turbidity should not change the compensation point more e than 10 percent from its seasonally established form, nor should such a change place more than 10 percent of the biomass of photosynthetic organisms below the compensation point." The term compensation point signifies the depth at which the amount of light energy fixed by algae is balanced by the energy used during normal metabolic processes. At depths greater than this point more energy is used than the algal cells fix.

As a result the algae must use metabolic reserves in order to survive. This recommendation is intended to protect the naturally occurring photosynthetic capacity in the upper waters where photosynthesis takes place. The only problem with a criterion based on compensation point and biomass is the difficult and time-consuming nature of the measurement. For this reason an objective upon light extinction a measured by Secchi Disk, an easy and problem-free procedure, is being recommended. Furthermore, it is generally accepted that the Secchi Disk measurement ent bears an approximately constant relation to the lower limit at which the necessary light to carry on photosynthesis is available (e.g., Holmes, 1970).

The value of 10 percent recommended in this criterion is somewhat arbitrary in that any alteration in turbidity will affect light transmission and consequently photosynthesis. Small changes in turbidity are difficult to detect, however, and will usually have only aeffect on photosynthesis. Thus, the 10 percent value has been chosen as a level that can be detected quite easily and at which appreciable changes in algal production may begin to occur.

The United States Environmental Protection Agency in a late draft of report to be published in Spring of 1975 has adopted the recommendation of the NAS. The complete NAS recommendation includes (NAS/NAE, 1974):

"Aquatic Communities should be protected if the following maximum concentrations of suspended solids exist:"

High level of protection	25 mg/L
Moderate protection	80 mg/L
Low Level of protection	400 mg/L
Very low level of protection	over 400 mg/L

The rationale as presented below was taken primarily from the U.S. EPA late draft.

Fish and other aquatic life requirements concerning suspended solids can be divided into those whose effect occurs in the water column and those whose effect occurs following sedimentation to the bottom of the water body. Noted effects are similar for both fresh and marine waters.

EIFAC (1966) identified four categories of concern as suspended solids affect fish and fish food populations namely:

"(1) by acting directly on the fish swimming in water in which solids are suspended, and either killing them or reducing their growth rate, resistance to disease, etc.;
(2) by preventing the successful development of fish eggs and larvae;
(3) by modifying natural movements and migrations of fish;
(4) by reducing the abundance of food available to the fish;..."

While indicating that no sharp boundaries exist for inert suspended solids whereby fisheries are not damaged above that level, the EIFAC review yielded the following conclusions assuming inert solids and otherwise satisfactory water quality:

"(a) there is no evidence that concentrations of suspended solids less than 25 ppm (parts per million) have any harmful effects on fisheries;
(b) it should usually be possible to maintain good or moderate fisheries in waters which normally contain 25 to 80 ppm suspended solids. Other factors being equal, however, the yield of fish from such waters might be somewhat lower than from those in category "a";
(c) waters normally containing from 80 to 400 ppm suspended solids are unlikely to support good freshwater fisheries, although fisheries may sometimes be found at the lower concentrations within this range; and
(d) at the best, only poor fisheries are likely to be found in waters which normally contain more than 400 ppm suspended solids."

However, available evidence indicates that the death rate for fish living in water containing 200 ppm or more of suspended solids for long periods of time will be greater than for similar fish living in clear water, and that suspended material from industrial discharges (e.g., coal washings and pulp wastes) may be substantially more toxic (EIFAC, 1965).

The committee added a caveat that although exposure to several thousand parts per million for several hours or days may not kill fish, such excessive concentrations should be prevented in waters reserved for maintenance of good fisheries.

Settleable materials which blanket the bottom of water bodies damage the invertebrate populations, block gravel spawning beds, and if organic, remove dissolved oxygen from overlying waters (EIFAC, 1965; Edberg and Hofsten, 1973). In a study downstream from the discharge of a rock quarry where inert suspended solids were increased by 80 mg/L, the density of macro—invertebrate populations also decreased by 60 percent regardless of the suspended solid concentrations (Gammon, 1970). Similar effects have been reported downstream from an area which was intensively logged. Major increases in stream suspended solids (25 mg/L upstream vs. 390 mg/L downstream) caused smothering of bottom invertebrates, reducing organism density to only 7.3 per square foot versus 25.5 per equate foot upstream (Tebo, 1955).

When settleable solids block gravel spawning beds which contain eggs, high mortalities result although there is evidence that some species of salmonids will not spawn in such areas (EIFAC, 1965).

It has been postulated that silt attached to the eggs prevents sufficient exchange of oxygen and carbon dioxide between the egg and the overlying water. The important variables are particle size, scream velocity and degree of turbulence (EIFAC, 1965).

Deposition of organic materials to the bottom sediments can cause imbalances in stream biota by increasing bottom animal density, principally worm populations, and diversity is reduced aspollution sensitive forms disappear (Mackenthun, 1973). Algae likewise flourish in such nutrient rich areas although forms may become less desirable (Tarzwell and Gaufin, 1953).

Identifiable effects of suspended solids on irrigation use of water include the formation of crusts on top of the soil which inhibits water infiltration, plant emergence and impede soil aeration; the formation of films on plant leaves which block sunlight and impede photosynthesis and which may reduce the marketability of some leafy crops like lettuce; and finally the adverse effect on rigation reservoir capacity, delivery canals and ocher distribution equipment (NAS/NAE, 1974).

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1974", pages 163-171 , International Joint Commission, June 1975.

UNSPECIFIED NON-PERSISTENT SUBSTANCES AND COMPLEX EFFLUENTS

OBJECTIVE

For non-persistent compounds a mixture with no Objectives because of a lack of specific data, their concentrations should not exceed 0.05 of the 96 hour LC_{50} value for an approved test species.

RATIONALE

This procedural objective is developed to limit the effects of: (1) unspecified non-persistent substances toxic to aquatic life but which are not presently identified by a specific objective within Annex I of the Agreement, and (2) complex industrial and municipal effluents which are toxic to aquatic life and are discharged directly to the Great Lakes.

A large number of specialty chemicals are presently utilized i industrial processes, in agriculture and in the home. They include chemical reagents, disinfectants, pest control products, preservatives, emulsifiers, defoamers, floatation and chelation agents. In some cases treatment systems are either not utilized or are inadequate to reduce the toxicity of these materials before they are discharged to surface waters. Some of these substances combine with others in ways which have not been defined, analytical procedures necessary for their identification and quantification have not been developed and toxicity testing sufficient to permit establishment of a specific water quality objective has not been conducted. These substances may be discharged as components of complex effluents and their effects within the receiving water will be indistinguishable from the combined effects of the total discharge. In view of the unspecified nature and the lack of an adequate toxicological data base for these substances the objective recommends use of an application factor with acute toxicity data derived for approved test species.

Acute toxicity refers to 96-hour concentrations lethal to half of the test organisms (96-hour LC_{50}, the median lethal concentration) derived in accordance with "Methods for Acute Toxicity with Fish, Macroinvertebrates and Amphibians", U.S. Environmental Protection Agency (in press); or published acute toxicity data expressed as the median lethal concentration for a 96-hour exposure during which test conditions were such that chemical and physical characteristics of the dilution water are comparable to existent water quality conditions at the boundary of the mixing zone.

Approved test species means any sensitive, locally important Great Lakes species or life history stage selected by the regulatory agency on the basis of appropriateness, or those species which have been used successfully ssfully in freshwater toxicity tests which are representative of sensitive important Great Lakes species.

To ensure that aquatic life within the receiving waters are afforded adequate protection from acute toxicity of these materials, it is necessary to perform bioassays to establish the toxicity of individual substances or mixtures and to use an application factor which should, in the majority of cases, reduce the concentration to that which is non-lethal for chronic exposure. The use of an application factor will not preclude the possibility of sub-lethal effects occurring, but, since by definition these substances are non-persistent, exposure times will tend to be of short duration and effects outside mixing zones would not normally be expected. Where effects outside established mixing zones are demonstrated it should be evident that the application factor was inadequate to derive an objective which would provide for and protect the designated use.

The test species utilized for the establishment of an objective should ideally correspond to the most sensitive important species existing in the locality where the objective is to apply. This is the recommendation currently proposed in Water Quality Criteria 1972 (NAS-NAE, 1974). While this is scientifically sound, it presents a serious difficulty in practice. In order to determine which local species is most sensitive to a given introduced toxicant, it is necessary to evaluate a large number of organisms. Consequently, the objective recommends a choice of locally important test species which are known to tolerate laboratory test conditions. Selection of the approved test species should include representatives of cold and warmwater fish species as well as important benthic invertebrates.

Recognition should be given to the large volume of acute toxicity data available from the scientific literature since it is unreasonable to require additional testing of those substances previously bioassayed by reputable laboratories. The objective permits use of such data where a) the species tested conforms to the requirements for species selection accompanying the objective and b) where dilution water quality utllized in the test was comparable to that which exists at the intended point of application of the objectlve (boundary of mixing zone).

The choice of application factor is based on the recommendation put forward by (NAS-NAE, 1974) for determining acceptable concentrations of toxicants for which comprehensive toxicological data are lacking. This recommendation for non-persistent and non-cumulative materials is a conoentration not exceeding 0.1 of the 96-hour LC_{50} at any time or place after mixing with the receiving waters, while the 24-hour average should not exceed 0.05 of the LC_{50} after mixing.

Since the boundary of a mixing zone may be located further from the source than the point at which rapid mixing is completed, and since monitoring by regulatory agencies will more likely involve spatial sampling on an irregular schedule rather than intensive sampling during a 24-hour period, it is recommended that the objective be 0.05 of the 96-hour LC_{50} at the mixing zone boundary. Based on a broad assessment of the scientific literature relative to the differences between LC_{50} values and incipient lethal concentrations for a diverse array of toxicants, and between lethal and sub-lethal concentrations, it is considered that an application

factor of 0.05 (1/20th) will, apart from specialized cases, provide adequate protection to the aquatic community. Notwithstanding this recommendation, it is strongly advised that where two or more unspecified toxicants are discharged simultaneously, that the potential for synergistic or additive effects be established through bioassay testing and that the acceptable concentration be based on 1/20th of the net toxiclty of the mixture.

REFERENCE

NAS-NAE, 1974. Water quality criteria, 1972. National Academy of Sciences and National Academy of Engineering. EPA Publication No. R3-73-033, 594 p. Superintendent of Documents, Washington, D.C.

SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1974", pages 152-156. International Joint Commission, June 1975.

ZINC

OBJECTIVE

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 1958, 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/L, and 95 percent of samples contain less than 40 µg/L (Table 7). However, the mean zinc concentrations range from 1.8 to 28.2 µg/L. At water intakes, the mean zinc concentrations are generally less than 45 µg/L except in Lake Erie at Buffalo, where the mean is 178 µg/L. High concentrations have been observed at the St. Mary's River, the outlet of Lake Superior, at Buffalo (Lake Erie) and at Massera (outlet of Lake Ontario) (Table 6). 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 constitutent 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/L 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 net at 5,000 µg/L (NAS/NAE, 1973). The maximum permissible limit in drinking water in Canada is also 5,000 µg/L but the objective is less than 1,000 µg/L (DNHW, 1969).

Concentrations of zinc inhibiting growth of freshwater algae generally range between. 1,000 and 10,000 µg/L (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/L, 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 percent mortality at 158 µg/L and 50 percent and 16 percent inhibition of reproduction at 102 µg/L and 70 µg/L, respectively (Biesinger and Christensen, 1972). Water hardness and alkalinity were 45.3 and 43.3 mg/L, respectively. In Lake Erie water, with a hardness and alkalinity of 123 and 91 mg/L, respectively, the 64-hour EC_{50} for immobilization of *Daphnia magna* was less than 150 µg/L (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/L, alkalinity, 42 mg/L) caused reduced egg production during spawning at 180 µg/L. No effect was observed at 30 µg/L (Brungs, 1969). In similar water, hagfish (*Jordanella floridae*) were more sensitive than fathead marrows. Eighty percent mortality of larvae of flagfish occurred at 85 µg/L zinc and only 10 percent at 51 µg/L. 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/L, 20-30 percent occurred at 139 µg/L and 0-29 percent occurred at 75 µg/L or less (Spehar, unpublished manuscript). Rainbow trout fry also die at low concentrations. In water of 26 mg/L hardness and 25 mg/L alkalinity, unacclimated trout had a 120-hr LC_{50} of 135 µg/L while those pre-exposed as eggs had an LC_{50} greater than 526 µg/L.

Based on lingering mortality of pre-exposed trout, the safe-unsafe concentrations were 135-251 µg/L (Goettl, *et al.*, 1973). Reproduction of bluegills was affected by zinc. Decreased spawning and complete mortality of fry occurred at 235 µg/L, while no effect was seen at 76 µg/L. Hardness and alkalinity were 51 and 41 mg/L, respectively (Sparks *et al.*, 1972).

Avoidance of zinc may prevent reproduction of Atlantic salmon. In the laboratory, juvenile salmon avoided 54 µg/L zinc, while in the field; migration of adults was prevented by about 240 µg/L (Sprague *et al.*, 1965). In the field, there were also 19 µg/L 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/L alkalinity was reduced at 130 µg/L zinc but not at 50 µg/L (Bengtsson, 1974).

Sublethal toxicity to zinc may be enhanced when in combination with copper and cadmium. At a hardness of 207 mg/L, alkalinity of 154 mg/L, copper of 6.7 µg/L, and cadmium of 7.1 µg/L, 42.3 µg/L of zinc was associated with reduced spawning of fathead minnows. When copper, cadmium and zinc were 5.3, 3.9 and 27.3 µg/L, respectively, reproduction was unaffected (Eaton, 1973). Therefore, a safe concentration of zinc for fathead minnows was 30 µg/L in soft water (Brungs, 1969) and 27.3 µg/L 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, hepatopancreas, 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/L zinc is recommended for the Great Lakes.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1975', page 77-81, International Joint Commission, 1976.

Table 5: 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	0.2	0.3	0.05	0.035	0.06	0.25
Cr	100	90	35	11	100	60
Cu	35	45	5	4	20	300
Fe	56,300	47,200	9,800	3,800	38,000	---
Ng	0.08	0.4	0.03	0.04	0.03	---
Pb	13	20	7	9	10	5
Ni	75	68	2	20	40	35
Se	0.05	0.6	0.05	0.08	0.2	7
Ag	0.07	0.07	0.05	0.05	0.05	0.1
Zn	70	95	16	20	50	40

Table 6: Concentrations (mg/L) of metals in filtered Great Lakes water sampled from municipal water intakes between 1962 and 1961 (Kopp and Kroner, 1920).

	Detection Limits	Lake Superior				Lake Michigan				Lake Huron		Lake St. Clair		Lake Erie		St. Lawrence R.	
		at Duluth		at St. Mary's R.		at Milwaukee		at Gary		at Port Huron		at Detroit		at Buffalo		at Massena	
Metal	(µg/L)	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Aluminum	40	11	ND[5] -26	6	ND- 10	Not measured		21	ND-58	24	ND-65	29	ND-68	11	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	--	--	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.

Table 7: Concentrations (µg/L) of metals in filtered water samples from the epilimnion of the Upper Great Lakes. These statistics represent values from many sections within a lake sampled several times within a year. The statiatics are derived from an unpublished draft of the 1975 Report of the Upper Lakes Reference Croup, IJC. Data on Lake Huron, Georgian Bay end the North Channel are from Vol. 11. 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	I.0	10	2.0-5.0	6.0
Zinc	1.0	72	7 -10	40	I.0	2	3.0	6.0

Table 1 (cont'd)

	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	20	≤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

RATIONALE FOR SWIMMING AND BATHING USE OF WATER

AESTHETICS AND DISCHARGE OF WASTE MATERIALS

AESTHETICS AND DISCHARGE OF WASTE MATERIALS

OBJECTIVE

Water used for swimming, bathing and other recreational activities should be aesthetically pleasing. The water should be devoid of debris, oil, scum and any substance which would produce an objectionable deposit, colour, odour, taste or turbidity.

Discharge of waste and offensive materials due to land drainage or due to direct application to the water body must be curtailed or controlled in order to maintain recreational usage.

RATIONALE

Aesthetics is classically defined as the branch of philosophy that provides a theory of the beautiful. In this Section, attention will be focused on the aesthetics of water in natural and man-made environments and the extent to which the beauty of that water can be preserved or enhanced by the establishment of water quality recommendations.

Although perceptions of many farms of beauty are profoundly subjective and experienced differently by each individual, there is an apparent sameness in the human response to the beauties of water. Aesthetically pleasing waters add to the quality of human experience. Water may be pleasant to look upon, to walk or rest beside, or simply to contemplate. It may enhance values of adjoining properties, public or private. It may provide a focal poiht of pride in the community. The perception of beauty and ugliness cannot be strictly defined. Either natural or man-made visual effects may add or detract, depending on any surroundings. As one writer had said when comparing recreational values with aesthetics, "Of probably greater value is the relaxation and mental well-being achieved by viewing and absorbing the scenic grandeur of the great and restless Missouri. Many people crowd the 'high-line' drives along the bluffs to view this mighty river and achieve a certain restfulness from the proximity of nature" (Purges *et al.* 1952).

Similarly, aesthetic experience can be enhanced or detroyed by space relationships. Power boats on a two-acre lake are likely to be more hazardous than fun and the water will be so choppy and turbid that people will hardly enjoy swimming near the shore. On the other hand, a sailboat an Lake Michigan can be viewed with pleasure. If a designated scenic area is surrounded by a wire fence, the naturalness is obviously tainted. If animals can only be viewed in restricted pens, the enjoyment is likely to be Less than if they could be seen moving at will in their natural habitat.

MANAGEMENT FOR AESTHETICS

The management of water for aesthetic purposes must be planned and executed in the context of the uses of the land, the shoreline, and the water surfaces. People must be the ultimate consideration. Aesthetic values relate to accessibility, perspective, space, human expectations, and the opportunity to derive a pleasurable reaction from the senses.

Congress has affirmed and reaffirmed its determination to enhance water quality in a series of actions strengthening the federal role in water pollution control and federal support for water pollution control programs of state and local governments and industry. In a number of states, political leaders and voters have supported programs to protect or even restore water quality with aesthetics as one of the values.

The recognition, identification, and protection of the aesthetic qualities of water should be an objective of all water quality management programs. The retention of suitable, aesthetic quality ise likely to be achieved through strict control of discharges at the source than by excessive dependence on assimilation by receiving waters. Paradoxically, the values tha aesthetically pleasing water provide are most urgently needed where pollution problems are most serious as in the urban areas and particularly in the central portions of cities where population and industry are likely to be heavily concentrated.

Unfortunately, one of the greatest unknowns is the value of aesthetics to people. No workable formula incorporating a valid benefit-to-cost ratio has yet been devised to reflect tanglible and intangible benefits accruing to conflicting uses or misuses and the cost of providing or avoiding them. This dilemma could be circumvented by boldly stating that aesthetic values are worth the cost of achieving them. The present public reaction action to water quality might well support this position, but efforts in this area have not yet proceeded far enough to produce values worthy of wide acceptance.

BASIS OF RECOMMENDATIONS FOR AESTHETIC PURPOSES

All surface waters should be aesthetically pleasing. But natural conditions vary widely, and because of this a series of descriptive rather than numerical recommendations are made. The descriptions are intended to provide, in general terms, for the protection of surface waters from substances or conditions arising from other than natural sources that might degrade or tend to degrade the aesthetic quality of the water. Substances or conditions arising from natural sources may affect water quality independently of human activities. Human activities that augment degradation from natural sources, such as accelerated erosion from surface disturbances, are not considered natural. The recommendations are also intended to cover degradation from "discharges or waste", a phrase embracing undesirable inputs from all sources attributable to human activities whether surface flows, point discharges, or subsurface drainage.

The recommendations that follow are essentially finite criteria. The absence of visible debris, oil, scum, and other matter resulting from human activity is a strict requirement for aesthetic acceptability. Similarily, recommended values for objectionable colour, odour, taste, and turbidity, although less precise, must be measured as no significant increase over background. Characteristics such as excessive nutrients and temperature elevations that encourage objectionable abundance of organisms, e.g., a bloom of blue-green algae resulting from discharge of a waste with a high nutrient content and an elevated temperature, must be considered.

These recommendations become finite when applied as intended in the context of natural background conditions. Specific numbers would add little to the usefulness of the descriptive recommendations because of the varying acuteness of sensory perception and because of the variability of substances and conditions so largely depended on local conditions.

In addition to this general objective for aesthetics, there are specific objectives relating to substances which could cause a change in the aesthetic quality of the environment. These objectives are located elsewhere in this document under the appropriate substance, total phosphorus.

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SOURCE

The above rationale information was taken from "Water Quality Criteria 1972" U.S. Environmental Protection Agency and National Academy of Sciences, EPA R3, 73-033-March 1973, pages 11 and 12.

Additional rationale material prepared by staff of the Ministry of the Environment appears in italics.

pH

OBJECTIVE

Because both alkaline and acid waters may cause eye irritation, the pH of the water used for recreational purposes should preferably be within the range of 6.5 and 8.5.

RATIONALE

The amount of information available relating the factors of pH, temperature, and clarity to ill-effect in recreation water was found to be more limited than for the other factors of interest. In general, it may be stated that the criteria recommended by the National Technical Advisory Committee (NTAC) on Water Quality Criteria represent the most likely conclusions to be drawn from available data, and the views represented by the group have been largely used as the basis for this section.

E.W. Mood (1968) has reviewed the literature on the relation between pH and aquatic activity, and explored the bases for the establishment of criteria for those properties of water thay may cause eye irritation to bathers and swimmers. Since his review contains a concise and useful account of the relation of pH to recreation water, and little supplemental information was encountered in our survey, its major points are brought out below.

Knowledge about the characteristics of water that may cause irritation to the eyes of swimmers has been developed through research efforts of ophthalmologists and others in connection with investigations on the preparation of ophthalmic solutions. Since the ideal non-irritating solution should have physico-chemical properties similar to tears, studies were undertaken initially to determine the chemical composition of lacrimal fluid, particularly of its hydrogen-ion concentration, or pH, its buffer capacity, and its tonicity. Although early studies of the hydrogen-ion concentration of tears revealed values ranging from 6.3 to 8.6, Hind and Coyan (1947; 1949) found that lacrimal fluid has a pH of approximately 7.4.

Lacrimal fluid is only weakly buffered and has the capacity to bring the pH of an unbuffered solution from as low as 3.5 or s high as 10.5 to within tolerable limits in a very short time. However, if the pH of a solution in direct contact with the eyes is lower than 7.3 or higher than 7.5, pain may be elicited.

Since the most sensitive part of the body that may come in contact with a given water is the eye, the tonicity or salt concentration is another important aspect. Early studies by Hind and Coyan (1947) showed that a sodium chloride equivalent range of 0.5 to 2.0 percent concentration was well tolerated. Later, Riegelmann, *et al* (1955) and Riegelmann and Vaughan (1958) suggested that the range be narrowed to the equivalence of between 0.7 to 1.5 percent

sodium chloride.

Seawater has a sodium chloride equivalent tonicity of 3.5 percent. It is mildly irritating to most swimmers but in normal recreational activity, exposure is limited. Concentrations higher than 3.5 percent NaCl are rarely encountered. Thus, Mood (1968) concludes that tonicity of recreation waters is of much less importance than the hydrogen-ion concentration and the buffer capacity in preventing or reducing eye irritation to bathers and swimmers.

"In summary, when water quality standards are proposed for swimming, bathing, and other similar uses, consideration should be given to those physico-chemical properties that may cause or contribute to eye irritation, if principal importance is the hydrogen-ion concentration with codependence upon the buffer capacity of the water. Ideally, the pH of the water should be approximately the same as Lacrimal fluid, which is about 7.0 for most people; a range of pH values from 6.5 to 8.3 can be tolerated under average conditions.

If the recreation water is relatively free of dissolved solids and has a very low buffer capacity, pH values from 5.0 to 9.0 should be acceptable. However, for recreation water having pH less than 6.5 or greater than 8.3, waste discharge standards should include prohibition against releases that will increase the buffer capacity of the receiving waters and yet maintain the pH below 6.5 or greater than 8.3. Tonicity standards do not seem to have any practical value" (NTAC, 1968).

Since there is some variance between the exact pH range to be used depending on the author quoted and the buffering capacity of the water, the pH range used as an objective for recreational use was adjusted to coinide witn that recommended to protect aquatic organism, 6.5 to 8.5 (reported elsewhere in this document). This range is well within than considered acceptable for recreational water relatively free of dissolved solids and of very low buffer capacity. It is also within the range of values reported by Hind and Goyan (1947; 1949).

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SOURCE

The above rationale information was taken from "Water Quality Criteria Data Book, Volume 4. An Investigation into Recreation Water Quality" by B.J. Mechalas, R.K. Hekimian, L.A. Schnazi, and R.H. Dudley. U.S. Environmental Protection Agency Project No. 18040 DAZ 1972, pages 143 and 144.

Additional rationale material prepared by staff of the Ministry of the Environment appears in italics.

WATER CLARITY

OBJECTIVE

The water in bathing areas should be sufficiently clear to estimate depth or to see submerged swimmers who may require assistance. To achieve this degree of safety, water clarity should be such that, if the bottom of the bathing area is not visible, the water should have a Secchi disc transparency of at least 1.2 m.

RATIONALE

Clarity in recreational waters is highly desirable from m the standpoint of visual appeal, recreational enjoyment and safety.Absolute criteria are impossible due to the great variations in local conditions. However, turbidity due to human activities should be controlled where feasible in recreational areas.

The general clarity of a recreational water can be estimated from a measurement of the turbidity or by using a Secchi disc to measure the visibility depth in the water.

Light penetration into water is extremely variable and the depth of penetration in natural water bodies is influenced by suspended microscopic plants and animals, suspended m mineral particles, stains that impart colour, detergent foams, and dense mats of floating or suspended debris.

For bathing and swimming areas, it has been recommended (NTAC, 1968) that the clarity of the water should be such that a Secchi disc is visible at a minimum depth of 1.2 m (4 ft.) and visible from the bottom in areas designated as learn-to-swim areas. In diving areas the desirable water clarity will be dependent upon the height of the diving board or platform.

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SOURCE

The above rationale information was taken from "A Compilation of Australian Water Quality Criteria" by B.T. Hart. Australian Water Resources Council Technical Paper No. 7, Research Project No. 71/36, 1974, page 166.

PUBLIC HEALTH CONSIDERATIONS

OBJECTIVE

The use of water for swimming, bathing and other recreational activities requiring immersion of the user should not cause disease(s) or infection(s) in the human user. Such disease(s), which may occur in the gastrointestinal tract, the eye, ear, nose or throat or in the skin, could be caused by pathogens including bacteria, fungi, protozoa or viruses contained in the water.

A potential health hazard is defined as a situation where there is a high risk of contracting a disease from use of the water. In these situations: confirmation of the hazard should be sought; potential users should be notified by the appropriate authority; immediate corrective action should be started; and surveillance of the water quality should continue until the corrective action is completed and the water quality can be declared safe for recreational use.

Water quality impairment is defined as a situation where there is a risk of disease from use of the water but where a less restrictive course of action can be followed. In these situations: confirmation of the impairment should be sought; corrective action should be started; and surveillance of the water quality should be maintained.

Because inadequately treated sewage and fecal matter are a primary source of disease-causing organisms, a potential health hazard exists if a sanitary survey discloses that inadequately treated sewage, fecal matter or other hazardous substances are being or may be discharged into the water. The sanitary survey should consist of an on-site inspection of adjacent and upstream areas and of all water flows and potential sources of discharge. The survey should take into account the effects of rainfall, peak user loads and the danger of accidental spillage and sources from other jurisdictions.

Because epidemiological data and outbreak reports are a direct measure of the risk of contracting a disease, a potential health hazard exists when such information (available to the medical officer of health a other appropriate health authority) discloses the presence, within the community served, of an infectious disease which may be spread or is being spread by the use of the water for recreational purposes.

Because the occurrence of disease-causing organisms is being measured directly, a potential health hazard exists when pathogenic organisms (e.g. *Pseudomonas a ruzinosa*, *Salmonella typhii*. and Polio virus), can be enumerated and frequently isolated from the water.

Bacteriological water quality indicators are groups of bacteria whose densities in water can be quantitatively related to the presence of sewage or fecal matter, and therefore to the risk of contracting a disease from the pathogens contained therein. The fecal coliforms are one of

these indicators. A potential health hazard exists if the fecal coliform geometric mean density for a series of samples exceeds 100 per 100 ml.

The total coliforms are also water quality indicators. However, increasing evidence suggests that the total coliform group can also be derived from sources other than sewage and fecal matter. Therefore, water quality is considered impaired when the total coliform geometric mean density for a series of water samples exceeds 1000 per 100 ml.

The fecal streptococci are yet another water quality indicator group. However, they can best be used in conjunction with the fecal coliforms as an indication of the nature of the potential fecal source. The ratio of the geometric mean densities of the fecal coliforms to fecal streptococci at the point of discharge exceeds 4 if the source of the discharge is likely to be human in origin. A ratio of less than 0.7 suggests that the source is probably of non-human origin. Ratios between these values are difficult to interpret and may indeed be mixtures. For reliable ratio data, the fecal coliform density should be approaching or exceeding 100 per 100 ml. The ratio must be applied carefully as numerous environmental factors will influence the densities of both of the organisms.

Other groups of bacteria (e.g. *Pseudomonas, Staphylococcus*) could provide better information concerning the risk of eye, ear, nose, throat and skin infections since these organisms may be indicators of the presence of the causative agents for these diseases.

SAMPLING

Water samples for bacteriological examination must be collected in sterile bottles under aseptic conditions. Because the water samples are highly perishable, the samples should be kept cool or refrigerated and bacteriological analyses of the samples should be done within 6 hours. The time elapsing between collection and the start of examination in the laboratory should not exceed 24 hours.

A series of at least 10 samples per month per sampling location is recommended, but an increased sampling frequency will be required when the water is used for recreational purposes or when the water is subjected to contamination or discharge.

RATIONALE

Microbiological Considerations

All recreational waters should be sufficiently free of pathogenic bacteria so as not to pose hazards to health through infections, but this is a particularly important requirement for planned bathing and swimming areas. Many bodies of water receive untreated or inadequately treated human and animal wastes that are a potential focus of human infection.

There have been several attempts to determine the specific hazard to health from swimming in sewage-contaminated water. Three related studies have been conducted in this country, demonstrating that an appreciably higher overall illness incidence may be expected among swimmers than among non-swimmers regardless of the quality of the bathing water (Smith *et al.* 1951, Smith and Woolsey 1952, 1961). More than one-half of the illnesses reported were of the eye, ear, nose and throat type; gastrointestinal disturbances comprised up to one-fifth; skin irritations and other illnesses made up the balance.

Specific correlation between incidence of illness and bathing in waters of a particular bacterial quality was observed in two of the studies. A statistically significant increase in the incidence of illness was observed am ong swimmers who used a Lake Michigan beach on three selected days of poorest water quality when the mean total coliform content was 2,300 per 100 ml. However, only data concerning these three days could be used in the analysis and differences in illness were not noted in comparison with a control beach over the total season (Smith *et al.* 1951). The second instance of positive correlation was observed in an Ohio River study where it was shown that, despite the relatively low incidence of gastrointestinal disturbances, swimming in river water having a median coliform density of 2,700 per 100 ml. appears to have caused a statistically significant increase in illnesses among swimmers (Smith and Woolsey 1952). No relationship between illness and water quality was observed in the third study conducted at salt water beaches on Long Island Sound (Smith and Woolsey 1961).

A study in England suggested that sea water carries only a negligible risk to health even on beaches that were aesthetically unsatisfactory (Moore 1959). The minimal risk attending such bathing is probably associated with chance contact with fecal material that may have come from infected persons.

Neither the English nor the United States salt water beach studies indicated a causal or associated relationship between water quality and disease among swimmers and bathers. While the two United States fresh water studies suggested some presumptive relationship, the findings were not definite enough to establish specific values for microbiological water quality characteristics.

Tests using fecal coliform bacteria are more indicative of the possible presence of enteric pathogenic microorganisms from man or other warm-blooded animals than the coliform group of organisms. The data for total coliform levels of the Ohio River Study were reevaluated to determine comparable levels of fecal coliform bacteria (Geldreich 1966). This reevaluation suggested that a density of 400 fecal coliform organisms per 100 ml was the approximate equivalent of 2,700 total coliform organisms per 100 ml. Using these data as a basis, a geometric men of 200 fecal coliform organisms per 100 ml has been recommended previously as a limiting value that under normal circumstances should not be exceeded in water intended for bathing and swimming (U.S. Department of the Interior, FWPCA 1968).

There may be some merit to the fecal coliform index as an adjunct in determining the acceptability of water intended for bathing and swimming, but caution should be exercised in using it. Current epidemiological data are not materially more refined or definitive than those that were available in 1935. The principal value of a fecal coliform index is an indicator of possible fecal contamination from man or other warm-blooded animals. A study of the occurrence of salmonella organisms in natural waters showed that when the fecal coliform level was less than 200 organisms per 100 ml, this group of pathogenic bacteria was isolated less frequently (Geldreich 1970).

Salmonella organisms were isolated in 28 percent of the samples with a fecal coliform density less than the 200 value, but they were isolated in more than 85 percent of the samples that exceeded the index value of 200 fecal coliform per 100 ml, and in more than 98 percent of the samples with a fecal coliform density greater than 2,000 organisms per 100 ml.

In evaluating microbiological indicators of recreational water quality, it should be remembered that many of the diseases that seem to be causally related to swimming and bathing in polluted water are not enteric diseases or are not caused by enteric organisms. Hence, the presence of fecal coliform bacteria or of *Salmonella* sp. in recreational waters is less meaningful than in drinking water. Indicators other than coliform or fecal coliform have been suggested from time to time as being more appropriate for evaluating bathing water quality. This included the staphylococci (Favero, *et al.* 1964), streptococci and other enterococci (Litsky *et al.* 1953). Recently *Pseudomonas aeruginosa*, a common organism implicated in ear infection, has been isolated from natural swimming waters (Hoadely 1968) and may prove to be an indicator of health hazards in swimming water. Unfortunately, to date, none of the alternative microbiological indicators have been supported by epidemiological evidence.

When used to supplement other evaluative measurements, the fecal coliform index may be of value in determining the sanitary quality of recreational water intended for bathing and swimming. The index is a measure of the "sanitary cleanliness" of the water and may denote the possible presence of untreated or nadequately treated human wastes. But it is an index that should be used only in conjunction with other evaluative parameters of water quality such as sanitary surveys, other biological indices of pollution, and chemical analyses of water. To use the fecal coliform index as the sole measure of "sanitary cleanliness", it would be necessary ary to know the maximum "acceptable" concentration of organisms; but there is no agreed-upon value that divides "acceptability" from "unacceptability". Thus, as a measure of "sanitary cleanliness, an increasing value in the fecal coliform index denotes simply a decrease in the level of cleanliness of the water.

In Ontario's water quality objectives the total coliform and fecal coliform indices are supplemented by 1) findings of sanitary surveys, 2) epidemiology data and disease outbreak reports, 3) the direct enumeration of pathogenic organisms and 4) information concerning other potential indicator organisms. The use of this supplementary information follows the

recommendations of the inter-ministerial Task Force on Water Quality Guidelines for Bathing Beaches, Ontario Ministry of Health, 1973). Each of the types of supplementary information provides a different vantage point to examine the potential health hazards.

A sanitary survey shows, that, if a disease-causing organism is present in a waste or other source, the organism is being discharged into a recreationally used water body. Because changes in local condition may have a great influence on the characteristics and amounts of discharges, the sanitary survey should consider the effects. that these changes may have. Changes in local conditions may e due to climatic condition, e.g. rainfall (Cabell, 1979) to variable patterns of use, e.g. certain facilities or structures may only be used on certain days of the week or when the user population reaches a certain level, or to incidents brought about by accidental discharge.

Epidemiology data and disease outbreak reports show that a disease-causing organism is present in human population, therefore sanitary wastes from that population will cause a health risk upon discharge to a recreationally used water body. This type of information needs to be examined and evaluated by appropriately trained medical staff in order to avoid data misinterpretation.

The isolation of a disease-causing organism from a body of water shows that organism is likely present in population and is being discharged into the receiving water. The exact densities of these organisms are important but present data does not permit generation of numerical objectives. This kind of information must be interpreted with caution since there are a large number of factors which may have an effect on the disease-causing potential of the organisms.

A variety of other microbial indicator systems have been proposed (Hoadley and Dutka, 1977), but their use and interpretation -must be approached with caution since for a majority of these systems there is not sufficient background information to provide meaningful guidelines. However, their use in conjunction with the traditional methods of showing the existence of a health risk is recommended since they may provide a mechanism of confirming the risk, of determining the most likely source of discharge or suggesting a possible course of action.

In the preparation of the water quality guidelines which were the predecessor of the present objectives (Ministry of the Environment, 1974), the available scientific information was assessed including the rationales for existing guidelines or objectives. At that time, the water quality of a large number of bodies of water in Ontario was also assessed using actual water analyses. On the basis of information from these two sources, the decision was made to set a total coliform guideline of 1,000 per 100 ml, a fecal coliform guideline of 100 per 100 ml and a fecal streptococcus guideline of 20 per 100 ml.

The fecal streptococcus guideline has been withdrawn because high levels of this organism occur at certain locations in the apparent absence of a sanitary water quality problem and

because there is some scientific uncertainty about the sources of some of the species in this group (Clausen et al. 1977). The fecal streptococci have been maintained in these objectives as a group of organisms which can provide supplementary information concerning the possible source of bacterial contamination.

The total coliform and fecal co:iform guidelines have been maintained in the water quality objectives. The fecal coliform objective is lower than that reported in some other objectives. However, this lower objective provides an added safety factor in categorization of the water quality and is being maintained and is rarely exceeded at most locations in Ontario. The difference between an objective of 100 and 200 per 100 ml on a logarithmic scale (geometric mean) is usually within the error associated with the normal bacterial variation. In addition to this Ontario experience, these objectives follow closely the consensus recommendations concerning the quality requirements for recreational waters which are being promoted by the World Health Organization (Suess, 1977).

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SOURCE

The above rationale information was taken from Water Quality Criteria 1972" pages 31 and 32, U.S. Environmental Protection Agency and National Academy of Sciences, EPA R3-73-033 1973

Additional rationale material prepared by staff of the Ministry of the Environment appears in italics.

RATIONALE FOR PESTICIDES

GENERAL PESTICIDES

METHODS, RATE, AND FREQUENCY OF APPLICATION

Pesticides are used for a wide variety of purposes. Often they are categorized according to their use or intended target (e.g., insecticide, herbicide, fungicide), but their release in the environment presents an inherent hazard to many non-target organisms. Some degree of contamination and risk is assumed with nearly all pesticide use. The risk to aquatic ecosystems depends upon the chemical and physical properties of the pesticide, type of formulation, frequency, rate and methods of application, and the nature of the receiving system.

The pesticides of greatest concern are those that are persistent for long periods and accumulate in the environment; those that are highly toxic to man, fish, and wildlife; and those that are used in large volumes over broad areas. The majority of these compounds are either insecticides or herbicides used extensively in agriculture, public health, and for household or garden purposes. In the absence of definitive data on their individual behavior and their individual effect on the environment, some generalization about pesticides is required to serve as a guideline for establishing water quality criteria to protect aquatic life. In specific instances. however, each compound most be considered individually on the basis of information about its reaction in the environment and its effect on aquatic organisms.

SOURCES AND DISTRIBUTION

The major sources of pesticides in water are runoff from treated lands, industrial discharges, and domestic sewage. Significant contributions may also occur in fallout from atmosphreic drift and in precipitation. Applications to water surfaces, intentional or otherwise, will result in rapid and extensive contamination.

Many pesticides have a low water solubility that favors their rapid sorption on suspended or sedimented materials and their affinity to plant and animal lipids. Soluble or dispersed fractions of pesticides in the water rapidly decline after initial contamination, resulting in increased concentrations in the sediments. In streams, much of the residue is in continuous transport on suspended particulate material or in sediments. The distribution within the stream flow is non-uniform because of unequal velocity and unequal distribution of suspended materials within the stream bed. Seasonal fluctuations in runoff and use pattern cause major changes in concentration during the year, but the continuous downstream transport tends to reduce levels in the upper reaches of streams while increasing them in the downstream areas and eventually in major receiving basins (i.e., lakes, reservoirs, or estuaries). If applications in a watershed cease entirely, residues in the stream will gradually and continuously decline. A similar decline would be expected in the receiving basins but at a slower rate.

In lakes the sediments apparently act as a reservoir from which the pesticide is partitioned into the water phase according to the solubility of the compound, the concentration in the sediment and the type of sediment particulate material, and in sediments may be toxic to aquatic organisms or contribute to residue accumulation in them.

PERSISTENCE AND BIOLOGICAL ACCUMULATION

All organic pesticides are subject to metabolic and non-metabolic degradation in the environment. Specific compounds vary widely in their rate of degradation, and some form degradation products that may be both persistent and toxic. Most pesticides are readily degraded to nontoxic or elementary materials within a few days to a few months; these compounds may be absorbed by aquatic organisms, but the residues do not necessarily accumulate or persist for long periods. Concentrations in the organism may be higher than ambient water levels, but they rapidly decline as water concentrations are diminished. Examples of such dynamic exchange have been demonstrated with malathion (Bender 1969), methoxychlor and various herbicides.

If degradation in water is completed within sufficient time to prevent toxic or adverse physiological effects, these nonpersistent compounds do not pose a long-term hazard to aquatic life. However. degradation rates of specific pesticides are often dependent upon environmental conditions. Considerable variation in persistence may be observed in waters of different types.

Some pesticides, primarily the organochlorine compounds, are extremely stable, degrading only slowly or forming persistent degradation products. Aquatic organisms may accumulate these compounds directly by absorption from water and by eating contaminated food organisms. In waters containing very low concentrations of pesticides, fish probably obtain the greatest amount of residue from contaminated foods; but the amount retained in the tissue appears to be a function of the pesticide concentration in the water and its rate of elimination from the organism. The transfer of residues from prey to predator in the food chain ultimately results in residues in the higher trophic levels many thousand times higher than ambient water levels.

RESIDUES

Samples of wild fish have often contained pesticide residues in greater concentrations than are tolerated in any commercially produced agricultural products. Pesticide residues in fish or fish products may enter the human food chain indirectly in other ways, as in fish oil and meal used in domestic animal feeds.

Fish may survive relatively high residue concentrations in their body fats, but residues concentrated in the eggs of mature fish may be lethal to the developing fry.

In addition to the problem of pesticide residues in aquatic systems, ocher problems include the potential of resistant fish species to accumulate levels hazardous to other species; the potential for enhanced residue storage when fish are exposed to more than one compound; and the potential effect of metabolites not presently identified.

Levels of persistent pesticides in water that will not result in undesirable effects cannot be determined on the basis of present knowledge. Water concentrations below the practical limits of detection have resulted in unacceptable residues in fish for human consumption and have affected survival of aquatic life. Criteria based upon residue concentrations in the tissues of selected species may offer some guidance.

It should also be recognized cognized that residue criteria are probably unacceptable except on a total ecosystem basis. Residues in stream fish may meet some guidelines, but pesticides from that stream may eventually create excessive re sidues in fish in the downstream receiving basins. Until more is known of the effects of persistent pesticide residues, any accumulation must he considered undesirable.

TOXICITY

Concentrations of pesticides that are lethal to aquatic life have often occurred in local areas where applications overlap streams or lakes, in streams receiving runoff from recently treated areas, and where misuse of spillage has occurred. Applications of pesticides to water to control noxious plants, fish. or insects have also killed desirable species.

Pesticides are toxic to aquatic life over wide ranges. Great differences in susceptibility to different compounds exist between species and within species. For example. 96-hour LC_{50} values of 5 to 610,000 µg/L were reported for various fish species exposed to organophosphate pesticides. In addition to species' differences, the toxicity may be modified by differences in formulation, environmental conditions, animal size and age, and physiological condition. The effect of combinations of pesticides on aquatic organisms has not received sufficient attention.

Most data on pesticide effects on aquatic life are limited to a few species and concentrations that are lethal in short-term test. The few chronic tests conducted with aquatic species indicated that toxic effects occurred at much lower concentrations.

BASIS FOR PESTICIDE OBJECTIVES

The use of short-term toxicity data and application factors is the approach to develop pesticide objectives. In general 96 hr. LC_{50} are multiplied by an application factor of 0.01 to obtain the pesticide objectives. In subsequent sections of this document, detailed rationales are given for a number of pesticide objectives. Detailed rationales are not available for the following pesticide objectives however; these objectives were ended by the U.S. Environmental Protection Agency in the Publication "Water Quality Criteria 1922" (EPA.R3.72.033, March, 1973).

	Concentration in µg/L (unfiltered sample)
Dicamba (Banvel)	200.0
Diquat	0.5
Diuron	1.6
Dalapon	110.0
Simazine	10.0
2,4-D (BEE)	4.0
Chlorphyrifos(Dursban)	0.001
Fenthion (Baytex)	0.006
Pyrethrum	0.01
TDE	0.006

OTHER PESTICIDES

ALDRIN/DIELDRIN

OBJECTIVE

The sum of the concentrations of aldrin and dieldrin in water should not exceed the recommended quantification limit of 0.001 micrograms per litre. The sum of the concentrations of aldrin and dieldrin in the edible portion of fish should not exceed 0.3 micrograms per gram for the protection of human consumers of fish.

RATIONALE

Aldrin is readily metabolized to the epoxy form, dieldrin, by both aquatic (Gakstatter, 1968; Khan *et al.*, 1972) and nonaquatic organisms (Gianotti *et al.*, 1956; Bann *et al.*, 1956).It has also been shown Chat the toxicity to aquatic organisms of both aldrin and dieldrin are similar (Jensen and Gaufin, 1966; Henderson *et al.*, 1959) and consequently, the recommendation has been expressed in terms of the total concentration of dieldrin and aldrin.

The proposed United States drinking water standard (EPA, 1971) was recommended to be 0.00014 µg/L total aldrin plus dieldrin based upon carcinogenicity studies. This standard is lower than any water levels which can be obtained from acute or chronic effect levels for fresh water aquatic organisms but its status is uncertain at present. The lowest effect levels which have been observed for freshwater species pertain to the stonefly and to the sailfin molly. The stonefly naiad was observed to have a 20-30 day LC_{50} of 0.2 µg/L (Jensen and Gaufin, 1966) but there is no available experimental application factor to obtain "safe" concentrations for this sensitive species. The sailfin molly exhibited chronic effects - inhibition of growth and reproduction (Lane and Livingstone, 1970) - at 0.75 µg/L and use of the arbitrary safety factor of 0.2 results in a concentration of 0.25 µg/L. This level, however, is inadequate for the protection of the stonefly and possibly other species.

Aldrin and dieldrin have recently been shown to be carcinogenic (Walker *et al.*, 1970) and, hence, the recommended concentration is the present recommended quantification limit as based on the lowest three reported values in the laboratory survey.

There are several reports on dietary dosing of aldrin and dieldrin which have led to low level sub-acute responses for different organisms. Rats and dogs (Lehman, 1965) showed no ill effects over 90 days - 2 years at dietary levels of 0.5 µg/L and the Hungarian partridge (Neill, 1969) had adverse effects on reproduction when the dosage in their diet was 1 µg/g. In the aquatic field, 0.36 µg/g in the diet of the rainbow trout affected the biochemical processes of the fish (Mehrle and Bloomfield, 1974). Since the allowable edible fish tissue concentration under the United states Food and Drug Administration guidelines is 0.3 µg/g, this is recommended.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1974", pages 55-58, international Joint Commission, June, 1975.

CHLORDANE

OBJECTIVE

The concentration of chlordane in an unfiltered water sample should not exceed 0.06 micrograms per litre for the protection of aquatic life.

RATIONALE

Cardwell *et al.*, (1974) conducted long-term flow-through studies on the effects of chlordane including studies on the effect on reproduction of fathead minnows, bluegills and brook trout. "Safe" concentrations ranged from 0.8 to less than 0.3 µg/L, and corresponding 96-hr LC_{50} values ranged from 59 to 37 µg/L. The smallest application factor between acute and "safe" concentrations was less than 0.008 for brook trout. If this factor is applied to the lowest available 96-hr LC_{50} of 7.8 µg/L (Anon., 1965) for rainbow trout, then a derived "safe" concentration would be 0.06 µg/L.

The "safe" level for the midge *Chironomus* was found to be 0.7 µg/L by Cardwell *et al.*, (1974). No acute toxicity determination could be made for this species. The "safe" chlordane concentrations for *Daphnia magna* and *Hyalella azteca* were about 12 and 5 µg/L, respectively.

Reported acute toxicity concentrations of chlordane for invertebrates in general range from less than 1 to more than 1000 µg/L (Cardwell *et al.*, 1974; Konar, 1968, Sanders, 1969; 1972; Sanders and Cope, 1966 and 1968). It is likely that the desired "safe" concentration for fish will provide adequate protection to aquatic invertebrates as well. Therefore it is recommended that the concentration in water should not exceed 0.06 µg/L.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1974", pages 59-60, International Joint Commission, June, 1975.

DECHLORANE (MIREX)

OBJECTIVE

0.001 micrograms per litre for freshwater and marine aquatic life.

RATIONALE

Mirex is used ta control the imported fire ant *Solenopsis saevissima richteri* in the southeastern United States. Its use is essentially limited to the control of this insect and it is always presented in bait. In the most common formulation, technical grade mirex is dissolved in soybean oil and sprayed on corncob grits. The bait produced in this manner consists of 0.3 percent mirex, 14.7 percent soybean oil, and 85 percent corncob grits. The mirex bait often is applied at a rate of 1.4 kilograms per hectare, equivalent to 4.2 grams of toxicant per hectare.

Relatively few studies have been made of the effects of mirex on freshwater invertebrates. Of these, only Ludke, *et al.* (1971) report chemical analyses of mirex in the water. Their study reported effects on two crayfish species exposed to mirex by three techniques. First, field-collected crayfish were exposed to several sublethal concentrations of technical grade mirex solutions for various periods of time; second, crayfish were exposed to mirex leached from bait (0.3 percent active ingredient); and third, the crayfish were fed mirex bait.

Procambarus blandingi juveniles were exposed to 1 or 5 µg/L for 6 to 144 hours, transferred to clean water and observed for 10 days. After 5 days in clean water, 95 percent of the animals exposed to 1 µg/L for 144 hours were dead. Exposure to 5 µg/L for 6, 24, and 58 hours resulted in 26, 50, and 98 percent mortality 10 days after transfer to clean water. Crayfish, *Procambarus hayi*, were exposed to 0.1 and 0.5 µg/L for 48 hours. Four days after transfer to clean water, 65 percent of the animals exposed to 0.1 µg/L were dead. At the 0.5 µg/L concentration, 71 percent of the animals were dead after 4 days in clean water. Tissue residue accumulations (wet weight basis) ranged from 940- to 27,210-fold above water concentrations. In leached bait experiments, 10 bait particles were placed in 2 liters of water but isolated from 20 juvenile crayfish. Thirty percent of the crayfish were dead in 4 days and 95 percent were dead in 7 days. Water analysis indicated mirex concentrations of 0.86 µg/L. In feeding experiments, 108 crayfish each were fed one bait particle. Mortality was noticed on the first day after feeding and by the sixth day, 77 percent were dead. In another experiment, all crayfish were dead 4 days after having been fed two bait particles each. From this report it is obvious that mirex is extremely toxic to these species of crayfish. Mortality and accumulation increases with time of exposure to the insecticide. Concentrations as low as 0.1 µg/L or the ingestion of one particle resulted in death.

Research to determine effects of mirex on fish has been concentrated on species which have economic and sport fishery importance. Hyde, *et al.* (1974) applied mirex bait (0.3 percent mirex) at the standard rate (1.4 kg bait per hectare) to four ponds containing channel catfish, *Ictalurus punctatus*. Three applications were made over an 8-month period with the first application 8 days after fingerling (average weight 18.4 g) catfish were placed in the ponds. Fish were collected at each subsequent application (approximately 4-month intervals). Two and one-half months after the final application, the ponds were drained, all fish were measured, weighed, and the percent survival was calculated. Mirex residues in the fish at termination of the experiment ranged from 0.015 µg/g (ppm) in the fillet to 0.255 µg/g in the fat.

In another study, Van Valin, *et al.* (1968) exposed bluegills, *Lepomis macrochirus*, and the goldfish, *Carassius auratus*, to mirex by feeding a mirex-treated diet (1, 3, and 5 mg mirex per kg body weight) or by treating holding ponds with mirex bait (1.3, 100, and 1000 µg/L computed water concentration). They reported no mortality or tissue pathology for the bluegills; however, after 56 days of exposure, gill breakdown in goldfish was found in the 100 and 1000 µg/L contact exposure ponds, and kidney breakdown was occurring in the 1000 µg/L ponds. Mortality in the feeding experiments was not related to the level of exposure, although growth of the bluegills fed 5 µg/L mirex was reduced.

In laboratory and field test systems reported concentrations of mirex usually are between 0.5 and 1.0 µg/L (Van Valin, *et al.*, 1968; Ludke, *et al.*, 1971). Although mirex seldom is found above 1 µg/L in the aquatic environment, several field studies have shown that the insecticide is accumulated through the food chain. Borthwick, *et al.* (1973) reported the accumulation of mirex in South Carolina estuaries. Their data revealed that mirex was transported from treated land and marsh to the estuary animals and that accumulation, especially in predators, occurred. In the test area, water samples consistently were less than 0.01 µg/L. Residues in fish varied from non-detectable to 0.8 µg/g with 15 percent of the samples containing residues. The amount of mirex and the percent of samples containing mirex increased at higher trophic levels.

Fifty-four percent of the raccoons sampled contained mirex residues up to 4.4 µg/g and 78 percent of the birds contained residues up to 17 µg/g. Naqvi and de la Cruz (1973) reported average residues for molluscs (0.15 µg/g) fish (0.26 µg/g), insects (0.29 µg/g), crustaceans (0.44 µg/g), and annelids (0.63 µg/g). They also reported that mirex was found in areas not treated with mirex which suggests movement of the pesticide in the environment. Wolfe and Moment (1973) sampled an area for one year following an aerial application of mirex bait (2.1 g mirex/hectare). Crayfish residues ranged from 0.04 to 0.16 µg/g. Fish residues were about 2 to 20 times greater than the controls and averaged from 0.01 to 0.76 µg/g. Kaiser (1974) reported the presence of Mirex in fish from the Pay of Quinte, Lake Ontario, Canada. Concentrations range from 0.02 µg/g in the gonads of the northern long nose gar, *Lepistosteus osseus*, to 0.05 µg/g in the areal fin of northern pike, *Esox lucius*. Mirex has never been registered for use in Canada.

Mirex does not appear to be greatly toxic to birds, with LC_{50}'s for the young of four species ranging from 547 to greater than 1667 µg/g (Heath, *et al.*, 1972). Long-term dietary dosages caused no adverse effect at 3 µg/g with mallards and 13 µg/g with pheasants (Heath and Spann, 1973). However, it has been reported (Stickel, *et al.*, 1973) that the persistence of mirex in bird tissue exceeds that of all organochlorine compounds tested except for DDE. Delayed mortality occurred among birds subjected to doses above expected environmental concentration.

A summary examination of the data available at this time shows a mosaic of effects. Crayfish and channel catfish survival is affected by mirex in the water or by ingestion of the bait particles. Bioaccumulation is well established for a wide variety of organisms but the effect of this bioaccumulation on the aquatic ecosystem is unknown. There is evidence that mirex is very persistent in bird tissue. Considering the extreme toxicity and potential for bioaccumulation, every effort should be made to keep mirex bait particles out of water containing aquatic organisms and water concentrations should not exceed 0.001 µg/L mirex. This value is based upon an application factor of 0.01 applied to the lowest levels at which effects on crayfish have been observed.

Data upon which to base a marine criterion involve several estuarine and marine crustaceans. A concentration of 0.1 µg/L technical grade mirex in flowing sea water was lethal to juvenile pink shrimp, *Penaeus durorarum*, in a three-week exposure (Lowe, *et al.*, 1971). In static tests with larval stages (megalopal) of the mud crab. *Rhithropanoueus harrisii*, reduced survival was observed in 0.1 µg/L mirex (Bookhout, *et al.*, 1972). In three of four 28-day seasonal flow-through experiments, Tagatz, *et al.*, (1975) found reduced survival of *Callinectes sapidus, Penaeus durorarum*, and grass shrimp, *Palaemonetes pugio*, at levels of 0.12 µg/L in summer, 0.06 µg/L in fall, and 0.09 µg/L in winter.

Since two reports, Lowe, *et al.*, (1971) and Bookhout, *et al.*, (1972), reported that effects of mirex on estuarine and marine crustaceans were observed only after considerable time had elapsed, it seems reasonable that length of exposure is an important consideration for this chemical. This may not be the case water since the crayfish were affected within 48 hours. Therefore, a 3 to 4 week exposure might be considered "acute" and by applying an application factor of 0.01 to a reasonable average rage of toxic effect Levels asummarized above, a recommended criterion of 0.001 µg/L results.

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SOURCE

The above rationale information was taken from "Quality Criteria for Water" EPA-440/0-76-023, pages 312-319, U.S. Environmental Protection Agency, Washington, D.C., 1976.

DIAZINON

OBJECTIVE

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-isopropy1-6-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 percent 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/L 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/L at 13°C (Cope, 1965). Cope (1966) reported that the 48-hr. LC_{50} for rainbow trout at 13°C and bluegills (*Lepomis machrochirus*) at 24°C was 170 µg/L and 96/L, respectively. Mean 96-hour LC_{50} values for diazinon were reported to be 7,800, 460, 770 and 1,600 µg/L 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/L (lowest concentrations tested). Exposure of brook trout for 6-8 months to concentrations of diazinon varying from 0.55 - 9.6 µg/L resulted in equally reduced growth rates for progeny as well as adults. For fathead minnow, the hatch of progeny was reduced by 30 percent at a concentration of 3.2 µg/L. There is evidence that these effects on the progeny were the result of parental exposure alone, and not diazinon levees 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 15°C) for water fleas (*Simocephalus serrulatus* and *Daphnia pulex*) exposed to diazinon was 1.8 µg/L and 0.90 µg/L, respectively (Sanders and Cope, 1966). Sanders (1969) has reported that the 96-hr LC_{50} for *Gammarus lacustris* was 200 µg/L. The 48-hour LC_{50} for the stonefly (*Pteryonarcys californica*) has been demonstrated to range from 6 µg/L (FWPCA, 1968) to 7.5 µg/L (Cope, 1966). The 96-hr. LC_{50} of diazinon for *Acroneuria lycorias* has been reported to be 1.7 µg/L (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 8 (NAS/NAE, 1973).

Table 9: Toxicity of Diazinon to Aquatic Invertebrates (NAS/NAE, 1973)

Organism	30-day LC_{50} (µg/L)	30-day no effect (µg/L)
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/L would protect invertebrates from exposure a to concentrations which are directly lethal. The unpublished work of Allison and Hermanutz would indicate that 0.55 µg/L 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/L) is the most sensitive organism. Accordingly, it is recommended that concentrations of diazinon in water not exceed 0.08 µg/L 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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SOURCE

The above rationale information was taken from "Appendix A, ater Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975", pages 103-106, International Joint Commission, 1976.

DDT AND METABOLITES

OBJECTIVE

The sum of the concentrations of DDT and its metabolites in water should not exceed the recommended quantification limit of 0.003 micrograms per litre. The sum of the concentrations of DDT and its metabolites in whole fish (wet weight basis) should not exceed 1.0 micrograms per gram for the protection of fish consuming birds.

RATIONALE

Egg shell thinning has been reported in the American kestrel after chronic experimental feeding with 2.8 µg/g DDE Wiemeyer and Porter, 1970); mallard (2.8 µg/g DDE converted from dry basis) (Health *et al.*, 1969; black duck (3.3 µg/g DDE, converted from dry basis) (Longcore *et al.*, 1971); and other species (Stickel, 1973). It is assumed that similar levels of intake will produce some detrimental effects on reproduction inspecies of birds under natural conditions. The lowest experimentally determined level at which egg shell thinning was found was 2.8 µg/g DDE. The effect was considered a subtle effect. *As* a subtle effect, an arbitrary 0.2 safety factor was applied to estimate the "safe" level. This would produce an estimated "safe" body burden in fish which are consumed by aquatic birds, of 0.06 µg/g DDE. This metabolite has been found to constitute 50-90 percent of the residue of DDT (Klaassen and Kadoum, 1973; Frank *et al.*, 1974; Reinert and Bergman, 1974). Therefore, the permissible body burden in fish was set at 1 µg/g total DDT to protect aquatic birds.

The FDA and FDD administrative action guidelines for DDT in edible portions of fish are set at 5 µg/g. This may be adequate for human consumption, but in the light of the above information, it will not protect aquatic birds.

The concentration of DDT in water which is likely to produce unacceptable body burdens in fish cannot be estimated accurately, because concentration factors for DDT appear to differ among the various Great Lakes, possibly due to other water quality parameters. Water concentrations which are "safe" for fish appear to be higher than those which produce unacceptable body burdens. However, "safe" water concentrations for fish have not been established by chronic experiments measuring subtle effects on fish. Therefore, no "safe" water concentration of DDT can be established and, consequently, the concentration of DDT in water should not exceed the recommended quantification limit of 0.003 µg/L, based on the lower three reported values from laboratory survey.

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SOURCES

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality, 1974", pages 61-63, International Joint Commission, June 1975.

ENDOSULFAN

OBJECTIVE

The concentration of Endosulfan in an unfiltered water sample should not exceed 0.003 micrograms per litre.

RATIONALE

The acute toxicity of endosulfan (also known as thiodan) to different fish species varies widely. Macek, *et al.* (1969) exposed rainbow trout, *Salmo gairdneri*, to endosulfan at three temperatures and computed 24-hour and 96-hour LC_{50}. At 1.6°C, 7.2°C, and 12.7°C the 24-hour LC_{50}. were 13, 6.1, and 3.2 µg/L, respectively. The corresponding 96-hour LC_{50} values were 2.6, 1.7, and 1.5 µg/L. Schoettger (1970), however, reports the 96 -hour LC_{50} for rainbow trout to be 0.8 µg/L at 1.5°C and 0.3 µg/L at 10°C. He also determined the 96-hour LC for the western white sucker, *Castostomus commersoni*, to be 3.5 µg/L at 10°C and 3.0 µg/L at 19°C.

A massive fish kill in the Rhine River was attributed to a concentrations of 0.7 µg/L endosulfan (Greve and Wit, 1971). The 96-hour LC_{50} at 20°C in a static bioassay using the guppy, *Poecilia reticulate*, was 4.2 µg/L based on the computed concentration. The measured concentration was only 0.2 µg/L (Herzel and Ludemann, 1971).

The 24-, 48-, and 96-hour LC_{50} for the amphipod, *Gammarus lacustris*, were found to be 9.2, 6.4, and 5.8 µg/L (Sanders, 1969). Sanders and Cope (1968) determined the 24-, 48-, and 96-hour LC_{50}'s for niads of the stonefly, *Pteronarcys californica*, to be 24, 5.6, and 2.3 µg/L, respectively. The 96-hour LC_{50} for *Gammarus fascia* was found to be 6.0 µg/L (Sanders,.1972).

No data are available on the levels to which endosulfan could be expected to accumulate in tissues of aquatic organisms at various water concentrations. Residues in fish are not anticipated to pose a hazard to fish-eating predators because of endosulfan's low oral toxicity to birds (Heath, *et al.*, 1972) and mammals Lindquist and Dahm, 1957). The U.S. Food and Drug Administration has not set allowable limits for endosulfan in edible fish tissues.

A 0.01 application factor applied to the lowest measured 96-hour LC_{50} for the rainbow trout (which appears to be the most sensitive native freshwater organism) results in a freshwater criterion of 0.003 µg/L.

Portman and Wilson (1971) determined the acute toxicity of endosulfan to a marine fish and several invertebrates by means of static bioassays. The 48-hour LC_{50} for the pogge, a fish, *Agonus cataphractes*, was 30 µg/L; the 48-hour LC_{50} for a mussel, the European cockle, *Cardium edule*, was greater than 10,000 µg/L; the 48-hour LC_{50} for the shrimp, *Crangon crangon,* was 10 µg/L.

Butler (1963) reported a 48-hour EC_{50} death or loss of equilibrium of 0.2 µg/L for the brown shrimp, *Penaeus aztecus*, a 48-hour EC_{50} for juvenile blue crabs, *Callinectes sapidus*, of 35 µg/L; and a 48 hour EC50 of 0.6 µg/L for juvenile white mullet, *Mugil curema*. A concentration of 65 µg/L resulted in a 50 percent decrease in shell gowth of the American oyster, *Crassostrea virginica*, at 28° C and salinity of 22 o/oo. Korn and Earnest (1974) report a 96-hour LC_{50} of 0.1 µg/L for the striped bass, *Morone saxatilis*.

Use of an application factor of 0.01 times the 96-hour LC 50 of the most sensitive marine organism tested, the striped bass, results in a marine criterion of 0.001 µg/L.

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SOURCE

The above rationale information was taken from "quality Criteria for Water," pages 265-269, U.S. EPA - 440/9 -76-023, Environmental Protection Agency.

ENDRIN

OBJECTIVE

The concentration of endrin in water should not exceed the recommended quantification limit of 0.002 micrograms per litre. The concentration of endrin in the edible portion of fish should not exceed 0.3 micrograms per litre for the protection of human consumers of fish.

RATIONALE

While there are considerable data available on the acute toxicity (96-hour LC_{50}) of endrin towards fish at approximately 0.5 µg/L (Mount, 1962; Henderson, 1959; Katz, 1961), there are no experimental data available which would permit the translation of these concentrations to safe levels for aquatic organisms. There is reported 30-day LC_{50} for the stonefly naiad of 0.035 µg/L, (Jensen and Gaufin, 1966), so "safe" levels to protect all aquatic organisms must lie below this value. In addition to the absence of appropriate chronic toxicitv data, the guidelines for raw water do not provide protection for all aquatic organisms. Consequently, it is recommended that the concentration of endrin in water should not exceed the recommended quantificiation limit as derived from the survey of laboratories and mentioned in the general section on persistent organic contaminants. Because it is felt that low levels should be sought in situations where data are inadequate to support a higher level, the quantification limit is set at the mean of the lower three of those reporting in the above survey. The level recommended for water is therefore 0.002 µg/L.

Two values for tissue levels are appropriate for consideration. American kestrels showed adverse effects when fed a diet containing 0.5 µg/g of endrin (Stickel, 1975), however, American kestrels are not fish eating predators. The United States Food and Drug Administration guideline for residues of this compound in edible fish tissues is 0.3 µg/g and the latter is recommended for the protection of consumers of fish.

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SOURCE

The above rationale information was taken from "Appendix A. Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1974", pages 64-66, International Joint Commission, June 1976.

GUTHION

OBJECTIVE

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 azinphosmethly and properly, O,O-dimethyl-S-(4-oxo-1,2, 3benzotriazin-3(4H)-ylmethyl) phosphorodithioate. It is used to protect fruit, grain and vegetable products on 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 cccurs in aqueous media (Heuer *et al.*, 1974) at environmental pH's and temperatures with half-lives ($T_{1/2}$) 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 ($T_{1/2}$'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 Corrideau, 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 aotivity in bluegills (Weiss and Gakstatter, 1964). In other studies with fathead minnows, decreased spawning was observed during long-term exposures at concentraticns 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 *Gammares lacustris* and *Gammares 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 10.24 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 (*Cammarus fasciatus* at 0.1 µg/L) and should afford reasonable protection. The recommended objective is, therefore, 0.005 µg/L.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975", pages 107-109, International Joint Commission, 1976.

HEPTACHLOR AND HEPTACHLOR EPOXIDE

OBJECTIVE

The sum of the concentrations of heptachlor and heptachlor epoxide in water should not exceed the recommended quantification limit of 0.001 micrograms per litre. The sum of the concentrations of heptachlor and heptachlor epoxide in edible portions of fish should not exceed 0.3 micrograms per litre, for the protection of human consumers of fish.

RATIONALE

Epoxidation of heptachlor yields heptachlor epoxide, and this reaction is facile in the aquatic environment (Stickel et al, 1965; Hannon *et al.*, 1970; Wiseman *et al.*, 1967; Barthel *et al.*, 1960; Perry *et al.*, 1958). The epoxidized form of heptachlor is at least as toxic as the parent compound, (U.S. - H.E. & W., 1972; Rudd and Genelly, 1956); and as a consequence, heptachlor concentrations are expressed as the sum of heptachlor plus heptachlor epoxide.

On the basis of available evidence, no experimentally determinable safe" levels can be set for water. The lowest available LC_{50} of 1.1 µg/L for stoneflies (Sanders and Cope, 1968) cannot be translated into safe levels. The proposed U.S. Drinking Water Standard of 0.1 µg/L may give a sufficient margin of safety and hence a quantification limit a ascertained from the survey of laboratories is recommended. The mean of the lower three reported values in the survey was employed in setting the recommended level of 0.001 µg/L total for water.

For tissues, the minimal or no-effect dietary level for rats and dogs is reported at 0.5 µg/g (Lehman, 1965). The United States Food and Drug Administration guideline for this pesticide as a residue in edible fish tissue is given as 0.3 µg/g and in the absence of aquatic dosing experiments, the latter is the level recommended for edible portions of fish in the Great Lakes.

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SOURCE

The above rationale information was taken from Appendix A, "Water Quality Objectives Subcommittee Report; Great Lakes Water Quality 1974", pages 67-69, International Joint Commission, June 1975.

LINDANE

OBJECTIVE

The concentration of lindane in water should not exceed 0.01 micrograms per litre for the protection of aquatic life. The concentration of Lindane in edible portions of fish should not exceed 0.3 micrograms per Litre for the protection of human consumers of fish.

RATIONALE

Macek *et al.*, (1975) experimentally determined "safe" water concentrations for bluegills, brook trout, and fathead minnows to range from 8.8 to 9.1 µg/L. The LC_{50} concentrations for the latter three species range from 20 to 54 µg/L which when divided by the respective "safe" concentrations, result in application factors of 0.17 to 0.34 for fish. The brown trout is apparently the fish most sensitive to lindane on anacute basis among those species used in aquatic bioassays. The 96-hour LC_{50} for brown trout is 2.0 µg/L (Macek and McAllister, 1970). Utilizing the lowest experimentally determined application factor for lindane in fish (0.17), a "safe" concentration of 0.34 µg/L would be predicted for brown trout.

Macek *et al.*,(1975) determined the acute and chronic toxicities of lindane to the midge *Chironomus tentans, Daphnia magna,* and the scud *Gammarus faciatus*. The midge was the most sensitive of these species chronically, with 2.2 µg/L being the highest concentration producing no observable adverse effect. Daphnia were least sensitive as 11 µg/L was determined to be "safe" over three consecutive generations of exposure. The midge and Daphnia were significantly different from fish in one respect, however the application factors for these invertebrates were uch less than for fish. namely, 0.01 and 0.02 based on 48-hour LC_{50} values of 207 and 485 µg/L, respectively.

Two investigators (Snow, 1958; Cope, 1965) have reported 96-hour LC_{50} values of 1 µg/L for stoneflies. Sanders and Cope (1968) reported an cute LC_{50} for stoneflies of 4.5 µg/L Lindane. If the experimentally determined application factor for invertebrates for lindane of 0.01 is applied to the lowest reported 96-hour LC_{50} of the most sensitive species, the scone fly, then a predicted "safe" concentration of lindane in water for that species would be 0.01 µg/L. This, therefore, is the recommended level for water.

Little information is available on accumulation of lindane in fish tissues. However, Macek *et al.*, (1975) observed whole-body (eviscerated) concentrations (wet weight) that were about 500 times the corresponding water concentrations in fathead minnows that had been exposed for several months. Butler (1967) observed accumulations of up to 250 times exposure concentrations in marine mollusks. Such factors, at present, are not consistent enough to be useful in deriving tissue levels and therefore the recommended criterion is based on the 0.3 µg/g administrative guideline of the United States Food and Drug Administration for lindane in edible portions of fish.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Suhcommittee Report, Great Lakes Water Qualitv. 1974", pages 70-72, International Joint Commission, June 1975.

MALATHION

OBJECTIVE

The concentration of Malathion in an unfiltered water sample should not exceed 0.1 micrograms per litre for the protection of freshwater aquatic life.

RATIONALE

The freshwater fish most sensitive to malathion, an organophosphorus pesticide, appear to be the salmonids and centrarchids. Post and Schroeder (1971) report a 96-hour LC_{50} between 120 and 265 µg/L for 4 species of salmonids. Ranch and McAllister (1970) found a 96-hour LC_{50} range between 101 and 285 µg/L for 3 species of centrachids and 3 species of salmonids. Other 96-hour LC_{50}s are: rainbow trout, *Salmo gairdneri*, 68 µg/L (Cope, 1965); largemouth bass, *Micropterus salmoides*, 50 µg/L (Pickering, *et al.*, 1962) and chinook salmon, *Oncorhynchus tshawytscha*, 23 µg/L (Katz, 1961). All of the above tests were in static systems. Eaton (1970) determined a 96-hour LC_{50} for bluegill, *Lepomis macrochirus*, in a flow-through system at 110 µg/L. Macek and McAllister (1970) reported a similar 96-hour LC_{50} for the bluegill in a static exposure. Static 96-hour LC_{50}'s of 120 and 160 µg/L were reported by Post and Schroeder (1971) for brook trout, *Salvelinus fontinalis*. Bender (1969) indicated that the acute toxicity to fathead minnows. Pimephales promelas, is slightly greater (about 2.0 times) in a static system than in a flow-through system. The flow-through acute toxicity to fathead minnows reported by Mount and Stephan (1967) approximated the static acute toxicity reported by Henderson and Pickering (1958) and Bender (1969).

Many aquatic invertebrates appear to be more sensitive than fish to malathion. The 96-hour LC_{50} for *Gammarus lacustris* was 1.0 µg/L (Sanders, 1969); for *Pteronarcella hadia*, 1.1 µg/L (Sanders and Cope, 1968); and for *Gammarus fasciatus*, 0.76 µg/L (Sanders, 1972). The 48-hour LC_{50} for *Simocephalus serrulatus* was 3.5 µg/L and for *Daphnia pulex*, 1.8 µg/L (Sanders and Cope, 1966). *Daphnia* were immobilized in 50 hours in 0.9 µg/L (Anderson, 1960). The 24-hour LC_{50}'s for two species of midge larvae were 2.1 µg/L (Mulla and Khasawinah, 1969) and 2.0 µg/L (Karnak and Collins, 1974).

Safe life cycle exposure concentrations for the more sensitive invertebrates are not known. The most sensitive aquatic organisms probably have not yet been tested; safe concentrations for the most sensitive invertebrates exposed through a complete life cycle have not been determined; and effects of low concentrations on invertebrate behaviour are unknown.

The stability of malathion in water is dependent on the chemical and biological conditions of the water (Paris, *et al.*, 1975).Weiss and Gakstatter (1964) have shown that the half-life of malathion w reduced from about 5 months at pH 5 to one to two weeks at pH 8. Eichelberger and Lichtenberg (1971) found that only 10 percent remained in the Little Miami River (pH 7.3-8.0) after 2 weeks. Bender (1969) states that one of the malathion breakdown products

may be more toxic than the parent compound.

It has been shown that a measured concentration of 575 µg/L malathion in flowing seawater kills 40 to 60 percent of the marine fish, *Lagodon rhomboides*, in 3.5 hours and causes about 75 percent brain acetylcholinesterase (AChE) inhibition (Coppage, *et al.*, 1975). Similar inhibition of AChE and mortality were caused in pinfish in 24, 48, and 72 hours at measured concentrations of 142, 92 and 58 µg/L, respectively. A concentration of 31 µg/L caused 34 percent AChE inhibition in pinfish but no deaths in 72 hours. Coppage and Matthews (1974) demonstrated that death may be associated with reductions of brain AChE activity of four marine or fishes by 70 to 80 percent or more in short-term exposures to malathion. Coppage and Duke (1971) found that moribund mullet, *Mugil cephalus*, in an estuary sprayed with malathion (3 oz./acre) during a large-scale mosquito control operation had about 98 percent inhibition of brain AChE. This is in agreement with 70 to 80 percent or more inhibition of brain AChE levels at and below which some deaths are likely to occur in short-term exposure. Spot, *Leiostomus xanthurus*, and Atlantic croaker, *Micropogon undulatus*, also had substantial inhibition of brain AChE during the spray operation (70 percent or more inhibition).

Toxicity studies have been made on a number of marine animals. Eisler (1970) studied the 96-hour LC_{50} for several marine fishes in at 20°C in static, aerated seawater. The 96-hour LC_{50} values (in µg/L) were: *Menidia menidia*, 125; *Mugil cephalus*, 550; *Fundulus majalis*, 250; *Fundulus heteroclitus*, 240; *Sphaeroides maculatus*, 3,250; *Anguilla rostrata*, 82, and *Thalassoma bifasciatum*, 27. Katz (1961) reported the static 24 hour LC_{50} for *Gasterosteus aculeatus* in 25 o/oo saltwater *as* 76.9 µg/L active ingredient. The 96-hour LC_{50} for striped bass, *Morone saxatilis*, in intermittent flowing seawater has been reported as 14 µg/L (U.S. BSFW 1970).

Reporting on studies of the toxicity of malathion on marine invertebrates, Eisler (1969) found the 96-hour LC_{50} (static, 24 o/oo salinity aerated) to be 33 µg/L for sand shrimp, *Crangon septemspinosa*; 82 µg/L for grass shrimp, *Palaemonetes vulgaris*; and 83 µg/L for hermit crab, *Pagurus longicarpus*. Growth of oyster, *Crassostrea virginica*, was reduced 32 percent by 96-hour exposure to 1 mg/L (Butler, 1963). The 48-hour LC_{50} for fertilized eggs of oysters was estimated by Davis and Hidu (1969) to be 9.07 mg/L and the 14-day LC_{50} for larvae, 2.66 mg/L.

Malathion enters the aquatic environment primarily as a result of its application as an insecticide. Because it degrades quite rapidly in most waters depending on pH, its occurrence is sporadic rather than continuous. Because the toxicity iexerted through inhibition of the enzyme acetylcholinesterase (AChE) and because such inhibition may be additive with repeated exposures and may be caused by any of the organo-phosphorus insecticides, inhibition of AChE by more than 35 percent may be expected to result in damage to aquatic organisms.

An application factor of 0.1 is applied to the 96-hour LC_{50} date for *Gammares lacustris*, *G. fasciatis* and *Daphnia* (sic), which are all approximately 1.0 µg/L, yielding a criterion of 0.1 µg/L.

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SOURCE

The above rationale information was taken from "Quality Criteria for Water", pages 296-305, U.S. EPA-440/9-76-023, Environmental Protection Agency, Washington, D.C.

METHOXYCHLOR

OBJECTIVE

The concentration of methoxychlor in an unfiltered water sample should not exceed 0.04 micrograms per litre for the protection of aquatic life.

RATIONALE

Chronic exposures of fathead minnows to methoxychlor demonstrated no effects on weight gain below 0.5 µg/L during 4 months of exposure, and no effects on ortality below 0.25 µg/L. The number of eggs laid by fathead minnows was unaffected by a 4-month exposure to 0.125 µg/L, but the hatchability of the eggs was reduced from 69 percent in controls to 39 percent (Merna and Eisele, 1973). Yellow perch seem to be less sensitive than fathead minnows.

Merna and Eisele (1973) also did chronic exposures of several invertebrates for 28 days and monitored survival, pupation, and/or emergence. Emergence for *Stenonema* was unaffected at 0.25 µg/L. Pupation of *Cheumatopsyche* was unaffected at 0.125 µg/L, but the growth rate of this species was affected by the exposure.

Eisele (1974) dosed a small stream with 0.2 µg/L methoxychlor continuously for one year. No insect or fish mortalities were observed. No invertebrate species were eliminated, but populations of baetids, stoneflies, and scuds were reduced. Hydropsychids, blackflies, crayfish and dragonflies showed only temporary changes before returning to control levels when exposed to continued dosing. While some species increased, there was no change in the diversity or density of invertebrates. There was however, a slight reduction in biomass. Most effects were sufficiently subtle so that routine ecological surveys would not have uncovered them. Crayfish body burdens rose to approximately 100 µg/g methoxychlor, indicating a concentration factor of 500.

In evaluating the above data, most weight was placed on studies which explored chronic effects under field conditions. The 0.2 mg/L exposure produced subtle effects on some invertebrate populations.

Application of the arbitrary 0.2 safety factor to these subtle effects was used to estimate the "safe" concentration of 0.04 µg/L recommended.

Methoxychlor may not conform to the definition of a persistent compound. It degrades readily and the structure of its probable metabolites would not indicate that they are likely to be persistent either. However, the actual rate of degradation is not indicated in the literature and it has been considered sidered under the category of persistent contaminants due to it organochlorine pesticide nature.

If it were classified as non-persistent, consideration would be given to the lowest reported 96-hr LC_{50} concentrations which pertain to crustaceans (0.8 to 5 µg/L; Sanders, 1969, Sanders & Cope, 1966) and to insects (0.6 to l.4 µg/L; Sanders and Cope. 1968; Merna and Eisle, 1974). Regardless of application factors, since good experimental and field data exist for deriving "safe" levels for this compound, these should be employed in setting the recommended level.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1974", pages 76-78, International Joint Commission, June 1975.

PARATHION

OBJECTIVE

Concentration 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 Was 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 partition (and other organophosphate pesticides) are reportedly via suppression of acetylcholinesterase (AChE) activity and this persists long after the actual exposure. Subsection of bluegills to 100 µg/L of parathion over 24 hours resulted in a 75 percent 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 he 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 9, along with sub-acute levels for the same species.

TABLE 10: 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)
Gammarusfasciatus	0.40 (4 days)	0.07 (43 days)	Spacie (1975)

In the study by Spacie (1975) *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 acetylchlorinesterease 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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SOURCE

The above rationale information was taking from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1975", International Joint Commission, 1976.

PHTHALIC ACID ESTERS

OBJECTIVES

The concentration of dibutyl phthalate and di-(2-ethylhexyl)phthalate in water should not exceed 4.0 micrograms per litre and 0.6 micro grams per litre respectively, for the protection of aquatic life. Other phthalic acid esters should not exceed the recommended quantification limit of 0.2 micrograms per litre in waters for the protection of aquatic life.

RATIONALE

It is recognized that the phthalic acid esters (PAE's) are probably non-persistent in water and aerobic sediments. However, there is some evidence that some PAE's may persist in anaerobic sediments, and for that reason they are provisionally included with the persistent organic chemicals. Since they are distinct compounds, not necessarily occurring together, a list of some of the more common ones are indicated.

Phthalic Acid Esters

Di-(2-ethylhexyl) phthalate (DEHP)
Di-iso-octylphthalate (DIOP)
Di-octylphthalate (D0P)
Di-butylphthalate (DBP)
Di-ethylphthalate (DEP)
Di-methylphthalate (DMP)

The occurrence of PAE residues in North American environments was reviewed at a conference on PAE's sponsored by the National Institute of Environmental Health Sciences (1972), by Mathur (1974) and by Mayer *et al.* (1972). Within aquatic ecosystems, PAE residues have been detected in fish, water and sediments, and sources are - most likely municipal and industrial effluents (Mayer *et al.*, 1972; Stalling *et al.*, 1973; Hites, 1973; Lake Michigan Toxic Substances Committee, 1974). Monitoring surveys by several Great Lakes states showed that effluents of industrial and municipal waste treatment facilities contained PAE's in concentrations ranging from less than 1 to 1,200 µg/L and tributaries to Lake Michigan contained 1 µg/L or less. The fate of PAE's discharged into these tributaries is not well defined, but analyses of settleable solids showed residues ranging from 1 to 75 µg/g (dry wt.). These results suggest that PAE's may be adsorbed to particulate materials in streams and ultimately deposited in bottom sediments.

Whether PAE's such as DEHP and DBP are biologically degraded in waste treatment plants *or* sediments of natural ecosystems has not been fully investigated. Graham (1973) reported that laboratory-scale, activated sludge processes degraded 91 percent of DEHP within 38 hours. However, analyses of sewage sludge from 54 municipal sewage treatment plants showed DEHP residues of 17 to 884 µg/g (dry wt.) (Lake Michigan Toxic Substances Committee, 1974). Thus, either activated sludge processes are not efficient in degrading PAE's, or raw sewage contains

very large amounts of PAE's. Laboratory incubation of DEHP and DBP with pond hydrosoils suggests that natural micro-organisms do, in time, hydrolyze the ester linkage and decarboxylate the phthalic acid moiety (Johnson, 1974). In aerobiosis studies, 98 percent of DBP was degraded after 5 days at 20°C, but only 50 percent of DEHP was degraded at 14 days. Under anaerobiosis, degradation of both PAE's was significantly retarded. Thus, although there is laboratory evidence for some biological degradation of PAE's, little is known of the dynamics of PAE residues in natural sediments. These dynamics could be affected by continuous or intermittent input of PAE's, oxidation-reduction state of the sediment, temperature, type of sediment, and probably other factors. In any case, limited monitoring data (Lake Michigan Toxic Substances Committee, 1974) suggest that PAE's may occur in bottom sediments and, therefore, important bottom-dwelling macro- and micro-fauna could be exposed to significant PAE residues.

DBP residues in fish from several areas of North America range from less than detectable concentrations of 0.5 µg/g, and DEHP residues have been found as high as 3.2 µg/g (Mayer, *et al.* 1972; Stalling *et al.* (1973). PAE residues in Great Lakes area fish range from undetected to 1.3 µg/g (Lake Michigan Toxic Substances Committee, 1974). However, one third to one half again as much residue may also be present in fish in the form of the monoester or conjugates of the monoester and phthalic acid (Mayer *et al.*, 1972; Mayer and Sanders, 1973). Mayer and Sanders (1973) exposed fathead minnows (*Pimephales promelas*) to 1.9 µg/L of DEHP for 56 days and found that residues reached an equilibrium concentration of 2.6 µg/g within 28 days. This gave an accumulation factor of nearly 1,400, which agrees well with data for DEHP in bluegills (*Lepomis macrochirus*) exposed to 0.1 µg/L (Johnson, 1974). However, Mayer and Rogers (1972) found that the accumulation factor for DEHP in fathead minnows was reduced to 160 when the fish were exposed to a higher concentration of 60 µg/L.

Accumulation factors for DEHP and DBP in aquatic crustacea and insects are generally between 350 and 3,900 following exposures ranging from 0.08 to 0.3 µg/L (Mayer and Sanders, 1973). When fish and invertebrates containing PAD residues are placed in untreated water, they eliminate 50 percent of the residue within 3 to 7 days. Residues in fish and invertebrates have not as yet been correlated with untoward biological effects.

TOXICITY

The acute 96-hour LC_{50} values for DBP with fathead minnows, channel catfish (*Ictalurus punctatus*), rainbow trout (*Salmo gairdneri*), scud *(Gamarus pseudolimnaeus*) and crayfish (*Orconeotes nais*) fall between 730 and 10,000 µg/L (Mayer and Sanders, 1973). Although the toxicity of DEHP is more difficult to determine in static tests because it is less soluble in water, 96-hour LC_{50} values are estimated to be above 10,000 µg/L. Flow-thrcugh tests were used for scud (*G. fasciatus*) and gave a 9-week LC_{50} value of 210 µg/L (McKim, 1974). The acute toxicities of both DBP and DEHP are considerably below those of most organochlorine insecticides which are usually toxic between 0.1 and 50 µg/L.

The chronic toxicities of DEHP and DBP have not been as well defined as desired. However, the chronic studies so far completed suggest that both DEHP and DBP are biologically active at concentrations well below acutely toxic concentrations. McKim (1974) reported that growth of brook trout (*Salvelinus fontinalis*) was reduced significantly at a DBP concentration of 300 µg/L, but not at 90 µg/L. However, aquatic invertebrates appear to be more sensitive than fish. Reproduction in daphnids (*Daphnia magna*) is impaired by DBP and DEHP concentrations of 20 and 3 to 5 µg/L, respectively (Mayers and Sanders, 1973; McKim, 1974). The emergence of adult midges, (*Chironomus tentans*) is reduced significantly at a DEHP concentration of 14 µg/L (Mayer and Rodgers, 1972).

Employing the chronic data for daphnids and the safety factor of 0.2 recommended maximum levels of 4 µg/L for DBP and 0.6 µg/L for DEEP are obtained. It is further recommended that until such time as chronic data on other PAE'S become available, concentrations of other individual PAE's in water be restricted to the recommended quantification level of 0.2 µg/L.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report. Great Lakes Water Quality 1974", p.41-46, International Joint Commission, June, 1975.

POLYCHLORINATED BIPHENYLS

OBJECTIVE

The concentration of polychlorinated biphenyls in an unfiltered water sample should not exceed 0.001 micrograms per litre to protect aquatic life.

Every reasonable effort should be made to minimize human exposure.

INTRODUCTION

Polychlorinated biphenyls (PCB's) are a class of compounds produced by the chlorination of biphenyls and are registered in the United States under the trade name Aroclor ®. The degree of chlorination determines their chemical properties, and generally their composition can be identified by the numerical nomenclature, e.g., Aroclor 1242, Aroclor 1254, etc. The first two digits represent the molecular type and the last two digits the average percentage by weight of chlorine (NTIS, 1072).

Identification of PCB's in the presence of organochlorine pesticides such as DDT and DDE has been difficult in the past because of their similar chromatographic characteristics (Risebrough, *et al.*, 1968).

In PCB analysis today, the interference of organochlorine hydrocarbons is overcome by sequential column chromatography on Florisil and silica gel (Armour and Burke, 1970: FDA, 1971). Gas-liquid chromatography with highly sensitive and selective detectors has been employed successfully in the detection of PCB's at low levels (Nebeker and Puglisi, 1974).

PCB compounds are slightly soluble in water (25-200 µg/L at $25^{0}0$), soluble in lipids, oils, organic solvents, and resistant to both heat and biological degradation (NTIS, 1972: Nisbet, *et al.*, 1972). Typically, the specific gravity, boiling point, and melting point of PCB's increase with their chlorine content. PCB's are relatively non-flammable, have useful heat exchange and dielectric properties, and now are used orincipally in the electrical industry in capacitors and transformers.

RATIONALE

The acute and chronic effects of PCB's have been determined on a number of aquatic organisms.

Ninety-six-hour LC_{50} values for newly hatched fathead minnows, *Pimephales promelas*, were 15 µg/L for Aroclor 1242 and 7.7 µg/L for Aroclor 1254. In 60-day continuous flow bioassays 50 percent of the fathead minnows were killed in 8.8 µg/L Aroclor 1242 and in 4.6 µg/L Aroclor 1254 (Nebeker, *et al.*, 1974.) Nine-month continuous flow bioassay tests were conducted in

the same series of experiments reported by Nebeker, *et al.* (1974). The spawning of fathead minnows was significantly affected at concentrations of 1.8 µg/L Aroclor 1254; concentrations of Aroclor 1242 above 10 µg/L were lethal to newly hatched fry. Defoe, *et al.* (In Press) conducted similar flow-through acute and chronic studies with fathead minnows using Aroclor 1248 and 1260. The calculated 30-day TL_{50} for newly hatched fathead minnows was 4.7 µg/L for Aroclor 1248 and 3.3 µg/L for Aroclor 1260. Fathead minnows were able to reproduce successfully at PCB concentrations which were acutely lethal to the larvae.

Stalling and Mayer (1972) determined 96-hour LC_{50} values ranging from 1,170 to 50,000 µg/L for cutthroat trout, *Salmo clarki*, using Aroclors 1221-1268. With rainbow trout, *Salmo gairdneri*, and Aroclors 1242-1260 the acute toxicity was greater than 1500 mg/L. Fifteen-day intermittent-flow bioassays carried out with bluegills, *Lepomis macrcchirus*, and Aroclors 1242, 1248 and 1254 resulted in LC_{50} values of 54, 76 and 204 µg/L, respectively.

Johnson (1973), Mayer (1975) and Veith (1975) conducted bioassays which showed that the toxicity of Aroclor 1016, introduced recently to replace PCB's of the Aroclor 1200 series in many applications, was similar to that of Aroclor 1242.

Nebeker and Puglisi (1974) conducted bioassays with *Daphnia magna* exposed to concentrations of Aroclors 1221-1268. In continuous flow tests Aroclor 1254 was the most toxic with a 3-week LC_{50} of 1.3 µg/L; 100 percent mortality occurred at 3.5 µg/L and 100 percent reproductive impairment occurred at 3.8 µg/L. Stalling and Mayer (1972) and Mayer *et al.* (1975) conducted flow-through and static bioassay tests on freshwater invertebrates which likewise indicated that these organisms are generally more susceptible to acute toxic effects of PCB's than fish.

Studies of the Milwaukee River (Wisconsin) revealed PCB concentrations in ambient water of 2.0 to 2.8 µg/L and residues in fish as high as 405 µg/g (Veith and Lee, 1971). Open water Lake Michigan PCB concentrations have been reported to be less than 0.01 µg/L; mean residues in coho salmon, *Oncorhynchus kisutch*, were about 15 µg/g (Veith, 1973). Veith (1973) found that goldfish, *Carassius auratus*, in the lower Milwaukee River accumulated Arochlor 1248 approximately 0.7×10^5 to 2×10^5 times depending upon the ambient water concentration. From both laboratory and river system studies, many aquatic organisms appear to bioaccumulate PCB mixtures containing 3, 4, 5, and 6 chlorine atoms per molecule approximately 3×10^3 to 2×10^5 times the concentration in the water. Tissue residues in fathead minnows, *Pimephales promelas*, ranged from 0.7 µg/g of Aroclor 1248 in control fish to 1036 µg/g of Aroclor 1254 in fish held for 8 months in water containing 4.0 µg/L of Aroclor 1254 (Nebeker, *et al.*, 1974). The latter case indicates a bioaccumulation factor of 2.3×10^5, which is essentially independent of the PCB concentration in the water.

Bluegill sunfish, *Lepomis macrochirus*, exposed to Aroclors 1248 and 1254 exhibited a bioaccumulation factor of 7.1×10^4 (Stalling and Mayer, 1972). The bioaccumulation factor for gizzard shad, *Dorosoma cepedianum*, in the Saginaw River (Michigan) varied between 0.6×10^5 and 1.5×10^5 for Aroclor 1254 (Michigan Water Resource Commission, 1973).

On the basis of the FDA action level of 5 µg/g in fish tissue, and both laboratory and field derived bioaccumulation levels for fathead minnows, *Pimephales promelas*, and goldfish, *Carassius auratus*, in the range of 0.7×10^3, to 2.3×10^5, an ambient water concentration of no more than 0.022 µg/L would be permissible. However, lake trout from the Great Lakes larger than 12 inches in length generally contain more than 5 µg/g of PCB's and chub from the Great Lakes generally contain PCB's in amounts approaching or slightly exceeding 5 µg/g. Since monitoring data on the Great Lakes' waters consistently indicate concentrations equal to or less than 0.01 µg/L, a criterion of less than 0.01 µg/L for all fishes appears necessary.

A residue level of 2 µg/g in fish consumed by commercial ranch mink has been shown to preclude survival of mink offspring (Ringer, *et al.*, 1972). Reproduction was nearly eliminated in range mink fed a beef diet containing 0.64 µg/g of Aroclor 1254 (Platonow and Kalstad, 1973). This suggests that a tissue level limit of not more than 0.5 µg/g would be required to protect ranch mink, and by implication, other carnivorous mammals. These data, plus the fact that lake trout from the Great Lakes (in water with PCB levels equal to or less than 0.01 µg/L) already exceed the 5 µg/g FDA limit, justify a fresh water criterion of not greater than 0.001 µg/L.

Median PCB concentrations in whole fish of eight species from Long Island Sound obtained in 1970 were reported to be on the order of 1 µg/g, as were comparable concentrations found in fish off the coast of Southern California (Hays and Risebrough, 1972; Risebrough, 1969). Generally, residues in ocean fish have been below 1 µg/g (Risebrough, 1970).

Surveys of Escambia Bay (Florida) during the period September 1969 to December 1971 produced data on the pathways and effects of PCB's in the estuarine and marine environments. The sediment reservoir of Arochlor 1254 is thought to be a continuing source of PCB's to aquatic biota. The initial survey of Escambia Bay biota revealed fish, shrimp, and crabs with levels as high as 12 µg/g. Higher levels of PCB's were detected in higher trophic levels than shrimp, which could implicate a chain transfer from sediment to large animals (Duke, 1974).

From the Escambia Bay data, which include flow-through bioassays with residue analyses where possible, the following conclusions were reached: (1) all of the Aroclors tested are acutely toxic to certain estuarine organisms; (2) bioassays lasting longer than 96 hours demonstrated that Aroclor 1254 is toxic to commercial shrimp at less than 1 µg/L; (3) fish, particularly sheepshead minnows, *Cyprinodon variegatus*, are extremely sensitive to Aroclor 1254 with 0.1 µg/L lethal to fry; and, (4) acute toxicity of Aroclor 1016 to estuarine organisms is similar to that of other Aroclors but it appears less toxic to marine fish in long-term

exposures than does Aroclor 1254(Duke, 1974; Schimmel, *et al.*, 1974).

Oysters, *Crassostrea virginica*, were sensitive to Aroclor 1260 with growth diminished by 44 percent in concentrations of 10 µg/L and by 52 percent in 100 µg/L. Approximately 10 percent of the pink shrimp, *Penaeus duoraran*, died in 100 µg/L, but no apparent effects on pinfish, *Lagodon rhomboides*, were noted at that concentration. Aroclor 1254 had no apparent effect on juvenile pinfish at 100 µg/L in 48-hour flow-through tests, but killed 100 percent of the pink shrimp. At 100 µg/L of Aroclor 1254 for 96 hours, shell growth of oysters sas inhibited and was decreased 41 percent at levels of 10 µg/L. The toxicity of Aroclor 1248 and 1242 to shrimp and pinfish was similar to that of Aroclor 1254. Aroclor 1242 was toxic to oysters at 100 µg/L. Killifish, *Fundulus heteroclitus*, exposed to 25 µg/L of Aroclor 1221 suffered 85 percent mortality. In 96-hour bioassays, Aroclor 1016 was toxic to an estimated 50 percent of the oysters, *Crassostrea virginica*; brown shrimp, *Penaeus amtecus*; and grass shrimp, *Paleomonetes pugio*, at 10 µg/L; it was lethally toxic to 18 percent of the pinfish at 100 µg/L (Duke, 1974).

Young oysters, *Crassostrea virginica*, exposed to Aroclor 1254 in flowing sea water for 24 weeks experienced reduction in growth rates at 4.0 µg/L, but apparently were not affected by 1.0 µg/L. Oysters assimilated as much as 100,000 times the test-water concentration of 1.0 µg/L. General tissue alterations in the vesicular connective tissue around the diverticula of the hepatopancreas were noted in the oysters exposed to 5.0 µg/L. No significant mortality was observed in oysters exposed continuously to 0.01 µg/L of Aroclor 1254 for 56 weeks (Duke, 1974).

Blue crabs, *Callinectes sapidus*, apparently were not affected by 20 days' exposure to 5.0 µg/L of Aroclor 1254. Pink shrimp exposed under similar conditions experienced a 72 percent mortality. Is subsequent flow-through bioassays, 51 percent of the juvenile shrimp were killed by Aroclor 1254 in 15 days and 50 percent of the adult shrimp were killed at 3.0 µg/L in 35 days. From pathological examinations of the exposed pink shrimp, it appears that Aroclor 1254 facilitates or enhances the susceptibility to latent viral infections. Aroclor 1254 was lethal to grass shrimp, *Paleomonetes pugio*, at 4.0 µg/L in 16 days, to amphipods at 10 µg/L in 30 days, and to juvenile spot, *Leiostonus xanthurus*, at 5.0 µg/L after 20 to 45 days. Sheepshead minnows, *Cyprinodon variegatus*, were the most sensitive estuarine organisms to Aroclor 1254 with 0.3 µg/L lethal to the fry within 2 weeks. Aroclor 1016, in two different 42-day flow-through bioassays, caused significant mortalities of pinfish at 32 µg/L and 21 µg/L. Pathological examination of those exposed to 32 µg/L revealed several liver and pancreatic alterations. Sheepshead minnows in 28-day Aroclor 1016 flow-through bioassays were not affected by concentrations of 10 µg/L or less, but died at 32 and 100 µg/L (Duke, 1974). The bioaccumulation factors determined by the flow-through bioassays are:

Aroclor	Organism	Time	Accumulation Factors (as a multiplier of test) water concentrations
1254	Oyster (*Crassostrea virginica*)	30 days	1.01×10^5
1254	Bluecrab (*Callinectes sapidus*)	20 days	4×10^3
1254	Grass shrimp (*Paleomonetes pugio*)	7 days	3.2×10^3 to 11×10^3
1254	Spot (*Leiostomus xanthurus*)	14-28 days	37×10^3
1254	Pinfish (*Lagodon rhomboides)*	35 days	21.8×10^3
1016	Pinfish (*Lagodon rhomboides*)	42 days	11×10^3 to 24×10^3
1016	Sheepshead minnow (*Cyprinodon variegatus*)		2.5×10^3 to 8.1×10^3

Based upon an accumulation factor of 100,000 in the oyster, it is necessary to limit the marine water concentration of PCB's to a maximum of 0.01 µg/L to protect the human consumer. However, data on the toxicity of Aroclor 1254 to sheepsheadminnow fry mentioned earlier (Schimmel, *et al.*, 1974), which indicate lethality at 0.1 µg/L, justify lowering the latter concentration by a factor of 0.01 to obtain a marine criterion of 0.001 µg/L. This level is further supported by the evidence cited earlier suggesting that a food tissue level of 0.5 µg/g, or 0.1 times the FDA level for human consumption, is necessary to protect carnivorous mammals.

Evidence is accumulating that PCB's do not contribute to shell thinning of bird eggs *(NAS,* 1974). Dietary PCB's produced no shell thinning in eggs of mallard ducks (Heath, *et al.*, 1972). PCB's may increase susceptibility ceptibility to infectious agents such a viral diseases (Friend and Trainer, 1970), and ie the activity of liver enzymes that degrade steroids, including sex hormones (Risebrough, *et al.*, 1968; Street, *et al.*, 1968). Laboratory studies have indicated that PCB's with their derivatives or metabolites, cause embryonic death of birds (Voss and Koeman, 1970). Feeding PCB's to White Leghorn pullets at a level of 20 ppm caused a significant decrease in hatchability of the eggs and viability of the surviving progeny (Lillie, *et al.*, 1974; Lillie, *et al.* 1975); in many cases, the cause of embryo mortality was attributed to gross abnormalities which ranged from edema to malformed appendages (Cecil, *et al.*, 1974).

Exposure to PCB's is known to cause skin lesions (Schwartz and Peck, 1943) and to increase liver enzyme activity that may have a secondary effect on reproductive processes (Risebrough, 1969; Street, *et al.*, 1968; Wasserman, *et al.*, 1970).It is not clear whether the effects are due to the PCB's or their contaminants, the chlorinated dibenzofurans, which are highly toxic (Bauer, *et al.*, 1961; Schultz, 1968; Varrett, 1970). While chlorinated dibenzofurans are a by-product of PCB production, it is not known whether they are also produced by the degradation of PCB's (NAS, 1974).

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SOURCE

The above rationale information were taken from "Quality Criteria for Water" 1976. U.S. Environmental Protection Agency, Washington, D.C. 20460.

TOXAPHENE

OBJECTIVE

The concentration of toxaphene in an unfiltered water sample should not exceed 0.008 micrograms per litre for the protection of aquatic life.

RATIONALE

Mayer *et al.* (1975) report decreased reproduction of brook trout when exposed to concentrations of 0.068 µg/L of toxaphene in water. Body burdens associated with this exposure were 0.6 µg/g. In a chronic bioassay with brook trout, Mayer at al. (1975) found that toxaphene in water, at a level of 0.039 µg/L, affected the growth and development of brook trout fry over an exposure period of 90 days. With the application of the safety factor of 0.2, a "safe" concentration of toxaphene is calculated to be 0.008 µg/L.

Acute toxicity of toxaphene to fish has been reported as 4.3 µg/L for bullheads (Mandi, 1966). Lawrence (1950) reported the acute toxicity to bluegills as 3.5 µg/L in soft water. Acute toxicities have also been reported for several species of fish by Macek (1970), ranging from 2 µg/L for largemouth bass to 13 µg/L for black bullhead, and by Nagvi and Ferguson (1970) for freshwater shrimp as 24-hr LC_{50}, ranging from 41 to 283 µg/L in four different lakes.

Schoettger and Olive (1961) reported mortality of kokanee salmon when fed Daphnia which were exposed to sub-lethal concentrations of 10 and 20 µg/L of toxaphene over periods of 120 to 312 hours. Hughes (1968) reported that toxaphene treated lakes (40 to 150 µg/L) remained toxic to fish for periods of a few months to five years after treatment. The persistence of toxaphene, and its highly lipophilic character would suggest the potential for bioconcentration and transfer through the food chain to higher trophic levels. Bioconcentration factors of 5,000 to 21,000 for brook trout (Mayer *et al.*, 1975), and 1,000 to 2,000 for aquatic invertebrates (Terriere *et al.*, 1966) were observed.

Bioconcentration of toxaphene in fathead minnows was observed to be in the range of 77,000 to 108,000 (Mayer *et al.*, 1975). However, these factors have not been related to deleterious body burdens, and, therefore, no recommendation for tissue concentrations of toxaphene can be set at this time.

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SOURCE

The above rationale information was taken from "Appendix A, Water Quality Objectives Subcommittee Report, Great Lakes Water Quality 1974", pages 73-75, International Joint Commission, June 1975.