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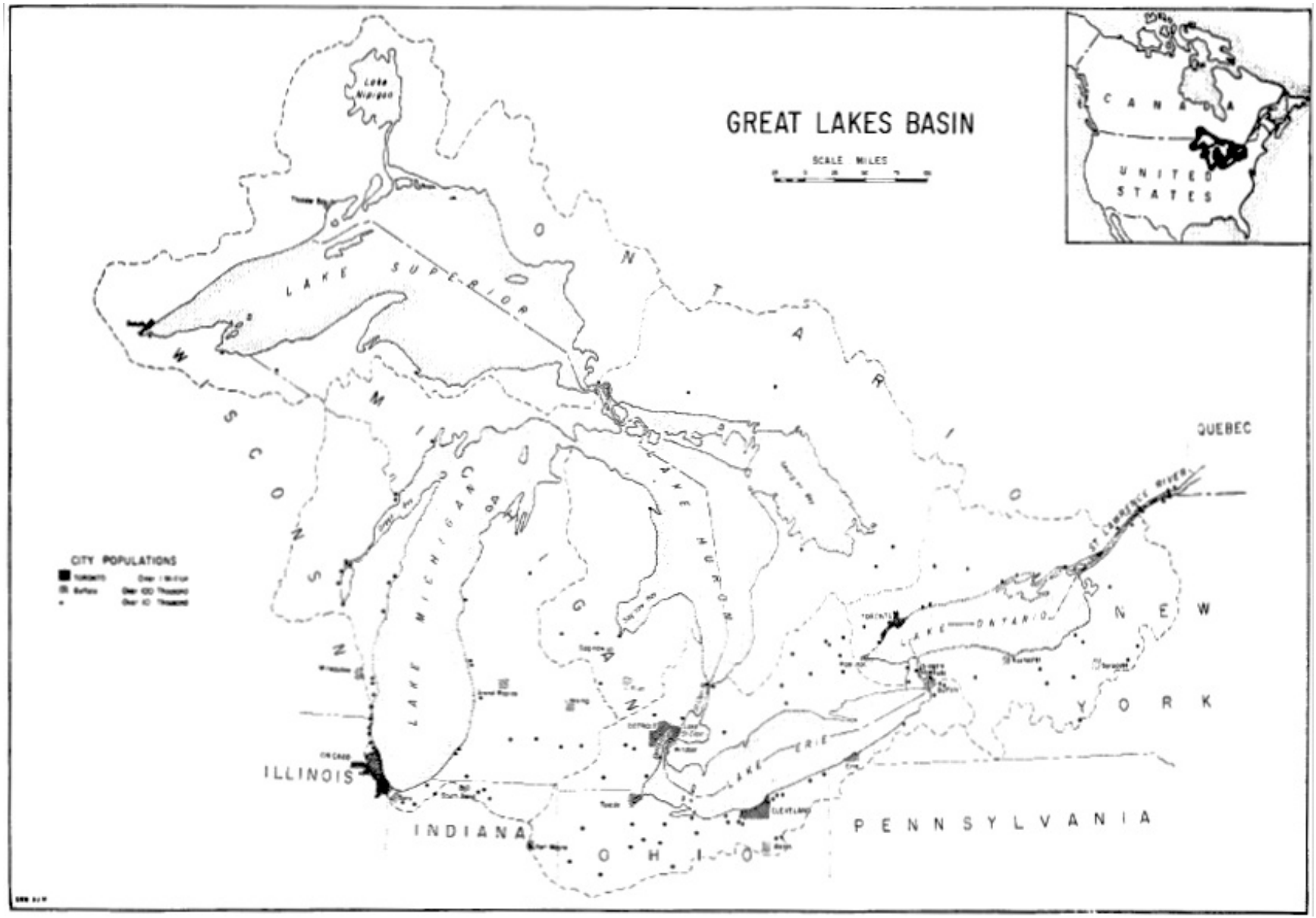
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Chapter I

INTRODUCTION

Volume II of the New and Revised Great Lakes Water Quality Objectives contains the scientific summary and other factors upon which the recommended objectives were based.

The work presented here was initially begun by the Water Quality Objectives Subcommittee of the Great Lakes Water Quality Board in 1973. As this Subcommittee became more and more deeply involved in developing an approach to the establishment and use of water quality objectives and determining specific objectives, it became apparent that the scientific expertise available from the Great Lakes Research Advisory Board was essential. Thus the Research Advisory Board's Scientific Basis for Water Quality Criteria Committee, now the Task Force on the Scientific Basis for Water Quality Criteria, was called upon to assist. This assistance has continued, not only through periodic joint meetings, but also by members undertaking literature searches and other reviews on behalf of the Committees.

This volume is a compilation and consolidation of the work of these Committees which appeared in Appendix "A" of the Water Quality Board's Reports for 1974 and 1975. The Commission has accepted in principle, the water quality objectives and supporting rationale. The views expressed by the Committees are retained in this volume and therefore individual comments within the text may not necessarily reflect the views of the Commission.

Chapter II
APPROACH TO THE ESTABLISHMENT OF WATER QUALITY OBJECTIVES

The development of common water quality objectives for the Great Lakes is recognized as one of the primary program elements of the Canada-United States Great Lakes Water Quality Agreement of 1972. These common objectives will provide direction for all water quality surveillance programs and will be of critical importance in evaluating the success of remedial programs. The objectives should also ensure against future losses of the beneficial uses which the Parties desire to secure and protect when implemented in concert with limitations on the extent of mixing zones or zones of influence and localized areas as designated by regulatory agencies.

In the process of assessing, refining and recommending objectives which would accomplish what was interpreted as the intention of the Parties, the Water Quality Objectives Subcommittee and the Scientific Basis for Water Quality Criteria Committee evolved and adopted what they considered to be a scientifically defensible framework. The proposed objectives are predicated on this framework which is drawn partly from the Agreement, partly from the recommended revisions to the Agreement and partly from guidance received from the Water Quality Board and the International Joint Commission. The Committees believed that adoption of the framework would aid the jurisdictions in protecting against future losses of the beneficial uses which the Parties desire to secure and protect. The framework can be outlined as follows:

1. *In developing specific water quality objectives the philosophy of protecting the most sensitive use was employed.*

   As considered by the Water Quality Objectives Subcommittee and the Scientific Basis for Water Quality Criteria Committee, water quality objectives describe, in part, a minimum quality of water which will not only provide for, but also protect any designated use. In most cases, the recommended objectives are proposed to protect aquatic life or their consumers, because they are in those cases the most sensitive use. Protecting the public water supply is employed next in frequency, in the case that human health is most sensitive. Aesthetic and/or recreational uses are most sensitive for a few parameters.

2. *Adoption of objectives does not preclude the need for studying the aquatic environment and effects of conditions on related organisms and uses. Because infinite combinations of water quality characteristics may occur, the objectives could not take into account antagonistic, synergestic and additive effects.*

   Each objective alone should provide protection from effects of that specific condition. Within each objective a safety factor is used which may be very small for some conditions and unknown for others. It cannot be assumed that when two or more minimum conditions (specific numerical objectives) occur simultaneously that protection of use is assured. Antagonistic, additive or synergistic effects may occur. Considering the infinite combinations of water quality characteristics, it may never

be possible to predict the effects of these combinations even for adult organisms, much less for their life history stages and processes.

Furthermore, local biota and local natural or ambient water quality characteristics coupled with a particular objective can result in a different response than that assumed by the Committees. In local conditions therefore an objective may be more restrictive than necessary and conversely, regulatory agencies should not assume that meeting the general and specific objectives guarantees protection of uses.

3. *Because new data may lead to modified recommendations, the objectives should be subject to continual review.*

The water quality criteria on which the present objectives are based were drawn from a data bank which is constantly changing due to the extensive ongoing interest and subsequent research in aquatic toxicology.

4. *Because no adequate scientific data base exists for establishing scientifically justifiable numerical objectives for certain unspecified non-persistent toxic substances and complex wastes, criteria for developing an objective for local situations have been recommended.*

To provide a reasonable degree of protection from the potential effects of such substances and discharges these criteria recommend that the local jurisdiction conduct specified bioassay tests on the most sensitive, important local species, and apply a stipulated application factor to toxicity data so derived. Such criteria may be termed procedural objectives.

5. *Biological effect levels were recognized as well as the concentration of a substance or level of physical effect.*

The preamble to the Agreement specifically identifies serious concern for trans-boundary effects of water quality deterioration and calls for development and implementation of new and more effective cooperative actions to restore and enhance water quality in the Great Lakes System. An objective for a substance or physical effect is designed to protect uses, for example, aquatic organism communities, by limiting acceptable levels. An objective for biological effects also should be designed to protect uses, for example, aquatic organism communities, by recognizing "a maximum or minimum desired limit." Examples include microbiological water quality characteristics and prevention of nuisance growths of algae, weeds and slimes. The Committees assumed that the Parties intended originally to consider the level of biological effect of the common resource which may be tolerated without damage to the system. Inclusion would permit development of objectives to protect passive organisms, especially fish larvae, as the result of entrainment at water intakes. The Committees identified this phenomenon as potentially of great significance with the increased use of

nearshore waters of the Great Lakes for cooling purposes.

6. *The objectives should serve as a minimum target wherever water quality objectives currently are not being met.*

This is in accordance with Articles IV, V and X of the Great Lakes Water Quality Agreement, dealing with regulatory requirements, remedial programs and implementation.

For those contaminants which are non-point source related, or the result of human activity, and do not meet objectives, regulation of the activity itself should be considered in remedial programs.

7. *For jurisdictionally-designated areas which have outstanding natural resource value and existing water quality better than the objectives, the existing water quality should be maintained or enhanced.*

The Great Lakes Water Quality Agreement establishes a non-degradation philosophy of taking "all reasonable and practicable measures" to maintain water quality where it is better than the prescribed objectives.

Carrying the present non-degradation philosophy a step further, the Committees agreed that "all reasonable and practicable measures" should be taken not only to maintain existing water quality which is better than the objectives, but that the potential for and the desirability of enhancement should be recognized and provided for. This small alteration in approach encourages further improvement, particularly in the open waters of the lakes.

The Committees recognized that any jurisdiction could move toward a more positive non-degradation policy than that provided by taking "all reasonable and practicable measures."

8. *Specific water quality objectives were designed to be met at the periphery of mixing zones. This assumes that water quality conditions better than the objectives will result beyond the mixing zones. The objectives should be implemented in concert with limitations on the extent of mixing zones or zones of influence and localized areas as designated by the regulatory agencies.*

The establishment of water quality objectives alone may not ensure against further losses of the beneficial uses which the Parties desire to secure and protect.

The Agreement describes a mixing zone, in part, as an area within which specific water quality objectives shall not apply. Since specific water quality objectives describe the minimum quality of water which will provide for and protect any designated use, it follows that a mixing zone represents encroachment in most cases, a loss of use, a loss of value or trade off. The Committees were extremely

reluctant to propose specific water quality objectives when no well-defined international-interstate mechanism existed to limit the present and future loss of value to mixing zones, not only locally but on a waterbody-wide scale.

In its present form the Great Lakes Water Quality Agreement restricts mixing zones to the "vicinity" of outfalls, urges limiting localized areas to a "minimum" and establishes a non- degradation philosophy of taking "all reasonable and practicable measures" to maintain water quality where it is better than the prescribed objectives. The Committees believed that these definitions were inadequate to prevent excessive areas of the Great Lakes from remaining in non-compliance or to prevent excessive areas from being downgraded to the objectives in the future.

The biological value of the Great Lakes is most vulnerable to encroachment by mixing zones because of the fragility and interdependency of the interlocking parts which make up the whole of the Great Lakes ecosystem and the ecosystem of each waterbody. It is obvious to even the casual observer that there is *a* limit to the loss of fish spawning sites, nursery areas, and feeding grounds before ecosystem imbalance and ultimate collapse of a population occur. Ecosystems are not compartmentalized by jurisdictional boundary lines. If excessive encroachment upon an ecosystem is allowed by one jurisdiction, the loss may affect neighbouring jurisdictions sharing the ecosystem.

To protect the biological integrity of the system, the Committees thoughtfully considered a concept designed to limit biological effects by allocating value loss in mixing zones.

The Committees did not recommend adoption of the biological effect concept which is in the development stage, but strongly encouraged further study for possible adoption in the future. The institutional framework of the International Joint Commission provides a forum in which decisions can be made which are critical to the success of this cooperative system to recognize desired limits to biological effects on a waterbody or portion thereof.

The "desired limit" of biological effect agreed upon should be worked out by the Parties, provinces and states. A high degree of international and interagency cooperation is required and the first reaction of agencies may be to brush it aside as unworkable and too complicated. Of those who have this reaction, the Committees asked, "What other scientifically justifiable alternative do we have?" The method is simplistic when compared with the complexity of the ecosystem.

To further encourage consistent management by the various enforcement agencies, the Committees developed broad guidelines for mixing zones based upon principles of good water management which describe desirable conditions within and desirable locations for these zones.

9. *In recommending objectives to protect raw public drinking water supplies, it has been assumed that a minimum level of treatment is provided before distribution to the public for consumption.*

The Board did not design these objectives to protect Great Lakes waters for domestic use without treatment. This minimum level of treatment includes coagulation, sedimentation, rapid sand filtration and disinfection. Often, a numerical objective specified for a contaminant to protect raw public water supplies is the same as an established drinking water standard because:

1) there is inadequate information on the effect of the defined treatment process on contaminant removal; or

2) the defined treatment process is inconsistent in contaminant removal; or

3) the defined treatment process is ineffective in contaminant removal.

As of 1974-75, the Canadian Drinking Water Standards were under review by a National Working Group composed of federal and provincial environmental health and environmental water quality officials. This Working Group is responsible for a complete update of the standards by March, 1978.

The United States Congress passed the Safe Drinking Water Act (PL 93-523) in December, 1974. Under the Act, the United States Environmental Protection Agency (EPA) published National Interim Primary Drinking Water Regulations on March 14, 1975. After a period of review the Regulations were promulgated in December 1975 to take effect in June 1977. Also required by the Safe Drinking Water Act was a report, released to EPA in May 1977, by the National Academy of Sciences on recommended maximum contaminant levels for drinking water. Following a review, proposed Revised Primary Drinking Water Regulations (Health) will be promulgated. The National Secondary Drinking Water Regulations (Aesthetics) are undergoing an initial review schedule which is less stringent.

Jurisdictions currently protect their raw public water supplies by state, provincial or federal standards. Until the recommendations of the Parties work groups are known, existing regulations represent the best information currently available. For purposes of surveillance and monitoring to establish indications of non-compliance, the Committees recommended using the most restrictive of the raw public water supply standards of each country.

Chapter III

SPECIFIC OBJECTIVES

CHEMICAL CHARACTERISTICS

(A) PERSISTENT TOXIC SUBSTANCES

(1) ORGANIC

RECOMMENDATION

It is recommended that the following numerical objectives for Persistent Organic Contaminants be adopted to replace the existing interim objective in Annex I, paragraph 2(c) of the Water Quality Agreement:

> Persistent pest control products and other persistent organic contaminants that are toxic or harmful to human, animal or aquatic life should be substantially absent in the waters. Recognizing that such substances are present in the Great Lakes, the following objectives are recommended for the known persistent organic contaminants for which scientific data exist.

Compounds	Specified concentrations	
	Water	Tissue
	µg/l	µg/g
Phthalate esters		
Dibutyl phthalate	4.0	U*
Di-(2-ethylhexyl)phthalate	0.6	U*
Other phthalate esters	0.2#	U*
Total PCBs	U*	0.1
Total DDT/DDE	0.003#	1.0
Total Aldrin/Dieldrin	0.001#	0.3+
Total Heptachlor/Heptachlor epoxide	0.001#	0.3+
Endrin	0.002#	0.3+
Toxaphene	0.008	U*
Chlordane	0.060	U*
Lindane	0.010	0.3+
Methoxychlor	0.040	U*

U* - undetermined at present
\# - recommended quantification limit
\+ - based upon United States Food and Drug Administration guidelines for edible portions of fish.

*For other organic contaminants, the levels of which are not specified but which can be demonstrated to be persistent and are likely to be toxic, it is recommended that the concentrations of such compounds in water or aquatic organisms be sub*stantially *absent* and *less* than *the detection. level as determined by the best scientific methodology available at the time.*

Note: Where waters are found to be contaminated as defined by exceeding the appropriate objective, all reasonable and practicable measures should be taken by the regulating agencies to reduce the input of the persistent organic contaminant to any part of the Great Lakes system.

EXISTING OBJECTIVE

The recommended numerical objectives are intended to replace the existing interim objective in Annex I, paragraph 2 (c) of the Agreement, which states:

> "Persistent Organic Contaminants. Persistent pest control products and other persistent organic contaminants that are toxic or harmful to human, animal or aquatic life should be substantially absent in the waters".

RATIONALE

Synthetic organic contaminants entering surface waters may be broadly divided into persistent and non-persistent compounds. The distinction is important since the organic contaminants known to present the greatest hazard to human, animal and aquatic life are those which are resistant to degradation and which are thus available for dispersion in the environment and for incorporation into biological tissues. Concentrations of persistent organic contaminants in the aquatic environment seldom achieve acutely toxic proportions except in localized situations by accident or misuse. Of much greater concern are effects from long-term exposure at sublethal levels and bioconcentration of residues resulting in tissue accumulations of increasing magnitude with each higher level in the aquatic food chain. Thus persistent contaminants must be considered separately from those which are readily decomposed to non-toxic constituents.

Persistent organic contaminants are, according to the Great Lakes Water Quality Agreement, to be "substantially absent" from Great Lakes waters. While the Committees would like to have interpreted this as completely absent from the aquatic ecosystem, they were constrained by the need to justify selected levels based upon protection of all potential uses. Consequently, they recommended levels based upon (1) protection of all aspects of aquatic life and human health as measured by water and tissue levels, (2) quantification limits for water concentrations, and (3) drinking water and food standards when these levels were limiting. Persistent organic compounds in water, with the possible exception of oil (covered under a separate objective), do not limit recreational uses of water. Levels of persistent organic contaminants in water suitable for agricultural use are also likely to provide adequate protection for aquatic life.

Persistence

A persistent compound is defined as one which either (a) by itself or as its transformation product, has a half-life for degradation under natural environmental conditions of more than eight weeks, or, (b) by itself or as its transformation product, on entering surface waters may bioconcentrate in the biota of the receiving system.

Persistence is the property of chemical compounds, measured in units of time, which describes their ability to resist structural alteration under specific physical and chemical conditions. Under similar environmental conditions, different compounds exhibit different persistencies depending on their molecular configuration. Since no standard test of persistence has yet been developed, the term lacks precise definition. This is reflected in the common practice of arbitrarily classifying environmental contaminants as persistent when their presence can be demonstrated in different substrates several days or weeks after release.

It is desirable to develop a standard test of persistence before introducing an objective for persistent contaminants in water. However, such a test cannot be arbitrarily selected, will require careful research and evaluation, and must be generally acceptable to the scientific community before its application for regulatory purposes. Whereas such research should be stimulated at the earliest opportunity, it is not possible to develop an acceptable test for "persistence" for incorporation into the impending revision of the Great Lakes Water Quality Agreement. Thus if the word "persistence" is to appear in the objectives it must include the present broad meaning indicated in (a) of the above definition.

A laboratory study of eight organochlorine, ten organophosphorus and seven carbamate compounds showed marked differences in persistence in river water over an eight week period (1). The results indicated, however, that 50% or more of the initial concentrations (10 µg/l) of all major environmental contaminants studied remained at the termination of the study. Consequently a half-life of eight weeks is a reasonable criterion for separating persistent and non-persistent compounds in water.

In keeping with the intent of the objectives that persistent organic contaminants be substantially absent, the definition was modified to include bioconcentration potential thereby providing for the distinct possibility that levels in tissue may accumulate from water concentrations below those which can be detected. This effect is provided for in part (b) of the definition.

While the problem of bioconcentration is real and is the reason for including tissue levels in the objectives, bioconcentration factors are not standardized, sometimes derived by combining dietary and direct water uptake data, and sometimes from systems considerably above the solubility of the compound under examination. As a result of these and other difficulties in determining this factor, no defensible objective can presently be based primarily upon this influence. Future work in this area may change the situation.

Aquatic Life

Body burdens of persistent organic contaminants in aquatic biota and those of their predators may limit species survival. Most of these compounds are classified by organic chemists as "non-polar" and as such they are very insoluble and liable to occur predominantly as adsorbed material on the particulate load. From there it may be ingested along with the particulates, thus entering the food chain, or it may be deposited in the sediment from where it can enter via benthic organism. Fish may also absorb these compounds directly through the gills. In higher trophic organisms, the persistent material usually ends up in the liver or in the adipose tissue due to its preferential solubility in fats and oils rather than in aqueous fluids. In many cases, bioconcentration also occurs because the material may not readily be excreted by the organism. Because of these considerations, sediments, plankton, fish tissue and predators of fish are probably better indicators of the presence of persistent organic contaminants than water. Avian and other non-aquatic predators are of concern since they feed on aquatic life and their body burdens may accumulate to toxic levels as a result. Since fish are the important food source for these predators, levels are largely set for fish tissue, but where it can be shown that detrimental effects occur in the predators the tissue level objective should also be extended to include them.

The dynamics of adsorption also give rise to increased levels of persistent organic contaminants in the sediments. However, sediments are prone to movements over large distances through current action. Moreover, present technology cannot determine their deposition rates on a useful time scale for these monitoring purposes. Hence, sediments presently can indicate presence of the contaminants but cannot easily be related to detrimental effects. It is recommended that studies he undertaken into sampling methods to permit the use of sediment levels for measuring contamination. For the present however, criteria are not recommended for this compartment of the ecosystem.

Specific recommendations have been made for those cases where chronic toxicity experiments have determined "safe" levels for representative fish and invertebrate species. In cases where subtle and deleterious effects were noted at the lowest chronic dose level (e.g., a partial reduction in hatchability of eggs), an arbitrary safety factory of 0.2 was applied to estimate the "safe" level. Where acute toxicity studies indicated that some species of fish were more sensitive than those actually investigated, an experimentally determined application factor for fish for the compound in question was used to estimate a "safe" level for the more sensitive species. Data for invertebrate studies were handled in the same fashion.

When field studies of toxic materials at chronic levels were available which documented water concentrations and used intensive ecological analyses, they were given greater weight than laboratory studies. Concentration factors for pesticides from water to aquatic life were found to be too variable (often greater than an order of magnitude) to be utilized meaningfully in the establishment of water quality criteria. Therefore, body burdens of various persistent chemicals in fish were used directly when appropriate information existed. Protection of wildlife which consumes aquatic life is based on chronic feeding studies of sensitive species and calls for restrictions on body burdens.

It is the intent of the Agreement to protect boundary waters of the Great Lakes system as a raw public water supply which will produce safe drinking water after treatment. In addition, fish for human consumption should be protected. Existing standards for most of the toxic persistent organic contaminants are inadequate to protect aquatic life. Protection of fish as a resource is provided by guidelines of the United States Food and Drug Administration for three persistent organics (see recommendations). As new standards relating to raw water supplies or drinking water which are lower than recommended water concentrations are developed and adopted by Canadian or United States federal agencies, they should also be adopted as part of the specific objectives, as should new edible tissue guidelines from the Food and Drug Administration or the Canadian Food and Drug Directorate.

Quantification Limits

The water quality objectives proposed for persistent organic contaminants are based on the intent expressed in the existing Agreement that such materials should be "substantially absent" within the boundary waters of the Great Lakes. The philosophy assumed, which is perpetuated here, is that a danger exists in allowing persistent materials of unknown fate or biological significance to be added to surface waters within arbitrarily established limits because there is no assurance that bioconcentration will not occur and reach unacceptable levels.

Corrective action may come too late to offset serious environmental consequences. Therefore, the philosophy of substantial absence of these substances is endorsed. In a practical sense this is a concentration below which no amount of the material can be detected by the most sensitive analytical techniques. This philosophy should be adopted particularly for proven carcinogens. In a survey of ten laboratories in the Great Lakes region currently doing routine determinations of pesticides and other persistent organic contaminants, the following means and ranges of quantification limits were reported (Table 1).

Table 1

PERSISTENT ORGANIC CONTAMINANTS QUANTIFICATION LIMITS

Compound	Mean µg/l	Range µg/l	Recommended quantification limit µg/l
Lindane	0.004	0.001-0.010	0.001
Heptachlor	0.004	0.001-0.010	0.001
Heptachlor Epoxide	0.004	0.001-0.010	0.001
p,p'-DDD	0.012	0.001-0.050	0.002
p,p'-DDE	0.011	0.001-0.050	0.002
p,p'-DDT	0.011	0.001-0.125	0.003
o,p'-DDT	0.014	0.001-0.045	0.003
Aldrin	0.004	0.001-0.010	0.001
Dieldrin	0.008	0.001-0.025	0.001
Endrin	0.008	0.001-0.020	0.002
Chlordane	0.005	0.002-0.010	0.002
Total PCB	0.03541	0.010-0.100#	0.010
p,p' Methoxychlor	0.020	0.010-0.050	0.010
Phthalate esters	0.6	0.1 -1.5	0.2

- does not include a single high value of 1.5 µg/l.

The third column is the mean of the lowest three quantification limits reported. Since it is desirable to encourage the development of more sensitive procedures, and not to condone insensitive determinations, these means are recommended. They are employed, where appropriate, to specify concentrations for which experimental data are not available to produce "safe" water levels but where there are data to establish "safe" tissue levels.

Where an organic compound can be demonstrated to be persistent and likely toxic and for which data are unavailable to establish either "safe" water or tissue concentrations, it is recommended that its concentration in water or aquatic organisms be limited to the detection level as determined by the best scientific methodology available at the time.

These quantification and detection limits, however, should not be accepted as permanent substitutes for experimentally determined "safe" concentrations. instead, it is intended that they should stimulate research on safety evaluations and analytical methods, plus provide a mechanism for action in the case of newly observed contaminants.

LITERATURE CITED

1. Eichelberger, J.W., and J.J. Lichtenberg. "Persistence of pesticides in river water". Env. Sci. Tech. 5(6): 541-544 (1971).

(a) Pesticides

(i) Aldrin/Dieldrin

RECOMMENDATION

It is recommended that the following numerical objective for aldrin/ dieldrin be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2 (c) of the Water Quality Agreement:

> *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 (2,6), and nonaquatic organisms (1,3). It has also been shown that the toxicity to aquatic organisms of both aldrin and dieldrin are similar (4,5). Consequently the recommendation has been expressed in terms of the total concentrations of dieldrin and aldrin.

Based on carcinogenicity studies, a standard of 0.00014 µg/l total aldrin plus dieldrin was recommended in the proposed United States drinking water standards and guidelines (11). This standard, which was not included in the Environmental Protection Agency's National Interim Primary Drinking Water Regulations promulgated in December 1975, is lower than any objectives which can be obtained from acute or chronic effect levels for freshwater aquatic organisms. The lowest effect levels for freshwater species were observed in the stonefly and the sailfin molly. The stonefly naiad was observed to have a 20-30 day LC_{50} of 0.2 µg/l (5), 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 (7) - 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 to protect the stonefly and possibly other species. Aldrin and dieldrin have recently been shown to be carcinogenic (12), hence the recommended concentration is the present recommended quantification limit based on the lowest three reported values in the laboratory survey (Table 1, p.14).

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 (8) showed no ill effects over 90 days to 2 years at dietary levels of 0.5 µg/g while Hungarian partridges (10) exhibited adverse reproduction effects 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 (9). The allowable edible fish tissue concentration under the United States Food and Drug Administration guidelines of 0.3 µg/g is recommended.

LITERATURE CITED

1. Bann, J.M, T.J. DeCino, N.W. Earle, and Y.P. Sun. "Fate of aldrin and dieldrin in the animal body". J. Agr. Food Chem. 4: 937 (1956).

2. Gakstatter, J.H. "Rates of accumulation of ^{14}C - dieldrin residues in tissues of goldfish exposed to a single sub-lethal dose of ^{14}C - aldrin". J. Fish. Res. Board Can. 25: 1797 (1968).

3. Gianotti, O., R. L. Metcalf, and R.B. March. "The mode of action of aldrin and dieldrin in *periplaneta Americans".* Ann. Ent. Soc. Amer. 49: 588 (1956).

4. Henderson, C., Q.H. Pickering, and C.M. Tarzwell. "Relative toxicity of ten chlorinated hydrocarbon insecticide to four species of fish". Trans. Amer. Fish. Soc. 88: 23 (1959).

5. Jensen, L.D. and A.R. Gaufin. "Acute and long term effects of aquatic insecticides on two species of stonefly naiads". J. Water Poll. Cont. Fed. 38: 1273 (1966).

6. Khan, M.A.Q., A. Kamal, R.J. Wolin, and J. Runnels. *"In vivo* and *in vitro* epoxidation of aldrin by aquatic food chain organisms". Bull Environ. Contam. Toxicol. 8: 219 (1972).

7. Lane, C.E. and R.J. Livingstone, "Some acute and chronic effects of dieldrin on the sailfin molly, *Poecilia Zatipinna".* Trans. Amer. Fish. Soc. 99: 489 (1970) .

8. Lehman, A.J. "Toxicity of pesticides". Assoc. of Food and Drug Officials of the U.S., Topeka, Kan., 1965.

9. Mehrle, P.M. and R.A. Bloomfield. "Ammonia detoxifying mechanisms of rainbow trout altered by dietary dieldrin". Toxicol. Appl. Pharmacol. 27: 355 (1974).

10. Neill, D.D., J.V. Schutze, and H.D. Mueller. "Pesticide effects on the fecundity of the gray partridge". Bull. Environ. Contam. Toxicol. 6: 546 (1971).

11. U.S. Environmental Protection Agency. "Proposed drinking water standards and guidelines". 1974.

12. Walker, A.I.T., E. Thorpe, and D.E. Stevenson. "The toxicity of dieldrin (HEOD): long term oral toxicity experiments in mice". Turnstall Laboratory Report 13, 1970.

(ii) Chlordane

RECOMMENDATION

It is recommended that the following numerical objective for chlordane be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2(c) of the Water Quality Agreement:

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

RATIONALE

Cardwell (1) 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 0.08 to less than 0.3 µg/l, and corresponding 96-hr LC_{50} values varied 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 (2) 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 (1). 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 1,000 µg/l (1,3,4,5,6). 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.

LITERATURE CITED

1. Cardwell, R.D., D.G. Foreman, T.R. Payne, and D.J. Wilbur. "Acute and chronic toxicity of chlordane to fish and invertebrates". (In preparation). U.S. Environmental Protection Agency Ecol. Res. Series. 1974.

2. "Effects of pesticides on fish and wildlife: 1964 research findings of the Fish and Wildlife Service". U.S. Fish and Wildlife Serv. Circ. 226, 1965.

3. Konar, S.K. "Experimental use of chlordane in fishery management". Progr. Fish-Cult. 30(2): 96-99 (1968).

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

5. Sanders, H.O. and 0.B. Cope. "Toxicities of several pesticides to two species of cladocerans". Trans. Amer. Fish. Soc. 95(2): 165-169 (1966).

6. Sanders, H.O. "Toxicity of pesticides to the crustacean, *Gammarus Zacustris".* U.S. Bur. Sport Fish. and Wildlife Tech. Paper 25, 1969.

(iii) DDT and Metabolites

RECOMMENDATION

It is recommended that the following numerical objective for DDT and metabolites be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2(c) of the Water Quality Agreement:

> *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 fish (wet weight basis) should not exceed 1.0 micrograms per gram for the protection of fish-consuming aquatic birds.*

RATIONALE

Egg shell thinning has been reported in the American kestrel after chronic experimental feeding with 2.8 µg/g DDE (7); mallard (2.8 µg/g DDE, converted from dry basis) (2); black duck (3.3 µg/g DDE, converted from dry basis) (4); and other species (6). It is assumed that similar intake levels will produce detrimental effects on reproduction in some species 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 subtle. As such, an arbitrary 0.2 safety factor was applied to estimate the "safe" level. This would produce an estimated "safe" body burden of 0.56 µg/g DDE in fish consumed by aquatic birds. This metabolite has been found to constitute 50 to 90% of the residue of DDT (1,3,5). Therefore the permissible body burden in fish was set at 1 µg/g total DDT to protect aquatic birds.

The United States Food and Drug Administration and the Canadian Food and Drug Directorate administrative action guidelines for DDT in edible portions of fish are 5 µg/g. This may be adequate for human consumption, but considering 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 since 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 the laboratory survey (Table 1, p.14).

LITERATURE CITED

1. Frank, R., A.E. Armstrong, R. G. Boelens, H.D. Braun, and C.E.W. Douglas. "Organochlorine insecticide residues in sediment and fish tissues, Ontario, Canada." Pestic. Monit. J. 7(¾): 165-180 (1974).

2. Health, G.B., J. Spann, and J.F. Kreitzer. "Marked DDE impairment of mallard reproduction in controlled studies". Nature 224: 47-48 (1969).

3. Klaassen, H.E. and A.M. Kadoum. "Pesticide residues in natural fish population of the Smokey Hill River of western Kansas, 1967-69". Pestic. Monit. J. 7(1): 53-61 (1973).

4. Longcore, J.R., F.B. Samson, J.F. Kreitzer, and J.W. Spaner. "DDT thins eggshells and lowers reproductive success of captive black ducks". Bull. Environ. Contam. Toxicol. 6 (6): 485-490 (1971).

5. Reinert, R.E. and H.L. Bergman. "Residues of DDT in lake trout *(Salvelinus namaycush)* and coho salmon *(Oncorhyrchus kisutch)* from the Great Lakes". J. Fish. Res. Board Can. 31(2): 191-199 (1974).

6. Stickel, L.F. "Pesticide residues in birds and mammals", in Environmental pollution by pesticides. Plenum, New York, 1973.

7. Wiemeyer, S.M. and R. D. Porter. "DDE thin eggshells of captive American kestrels". Nature 227: 737-738 (1971).

(iv) Endrin

RECOMMENDATION

It is recommended that the following numerical objective for endrin be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2 (c) of the Water Quality Agreement:

> 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 gram for the protection of human consumers of fish.*

RATIONALE

While considerable data are available on the acute toxicity (96-hour LC_{50}) of endrin for fish at approximately 0.5 µg/l (1,3,4), no experimental data are available which would permit the translation of these concentrations to "safe" levels for aquatic organisms. There is a reported 30-day LC_{50} for the stonefly naiad of 0.035 µg/l (2), so "safe" levels to protect all aquatic organisms must lie below this value. In addition to the absence of appropriate chronic toxicity 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 quantification limit as derived from the survey of laboratories (Table 1, p. 14) 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 (5); however, they are not fish-eating predators. The United States Food and Drug Administration guideline of 0.3 µg/g for residues of this compound in edible fish tissue is recommended for the protection of consumers of fish.

LITERATURE CITED

1. Henderson, C., Q.H. Pickering, and C.M. Tarzwell. "Relative toxicity of ten chlorinated hydrocarbon insecticides to four species of fish". Trans. Amer. Fish. Soc. 88: 23 (1959).

2. Jensen, L.D. and A.R. Gaufin. "Acute and long-term effects of organic insecticides on two species of stonefly naiads". J. Water Poll. Cont. Fed. 38:1273 (1966).

3. Katz, M. "Acute toxicity of some organic insecticides to three species of salmonids and to three spine stickleback". Trans. Amer. Fish. Soc. 90: 264 (1961).

4. Mount, D.I. "Chronic effects of endrin on blunt nose minnows and guppies". U.S. Fish and Wildlife Serv. Res. Report 58, 1962.

5. Stickel, L.F. Personal communication from Patuxent Wildlife Research Laboratory, Patuxent, Md., 1975.

(v) Heptachlor

RECOMMENDATION

It is recommended that the following numerical objective for heptachlor be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2(c) of the Water Quality Agreement:

> *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 gram for the protection of human consumers of fish.*

RATIONALE

Epoxidation of heptachlor yields heptachlor epoxide. Epoxidation is a reaction which is facile in the aquatic environment (1,2,4,7,9). Since the epoxidized form is at least as toxic as the parent compound (5,8), 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 heptachlor in water. Because the lowest available LC_{50} of 1.1 µg/l for stoneflies (6) cannot be translated into "safe" levels, a quantification limit is recommended. The limit of 0.001 µg/l total in water is the mean of the lowest three reported values in the survey cited in Table 1, p.14.

For tissues, the minimal or no-effect dietary level for rats and dogs is reported at 0.5 µg/g (3). The United States Food and Drug Administration guideline for this pesticide as a residue in edible fish tissue is 0.3 µg/g and in the absence of aquatic dosing experiments, the latter level is recommended for edible portions of fish in the Great Lakes.

LITERATURE CITED

1. Barthel, W.F., R. T. Murphy, W.G. Mitchell, and C. Corley. "Fate of heptachlor in the soil following granular application to the surface". J. Agr. Food. Chem. 8: 445 (1960).

2. Hannon, M.R., Y. Greichus, R. L. Applegate, and A.C. Fox. "Ecological distribution of pesticides in Lake Poinsett, South Dakota". Trans. Amer. Fish. Soc. 99: 496 (1970).

3. Lehman, A.J. "Toxicity of pesticides". Assoc. Food and Drug Officials of the U.S., Topeka, Kan., 1965.

4. Perry, A.S., A.M. Mattson, and A.J. Bruckner. "Metabolism of heptachlor by resistant and susceptible houseflies". J. Econ. Entomol. 51: 346 (1958).

5. Rudd, R.L. and R.E. Genelly. "Pesticides: their use and toxicity in relation to wildlife". 1956.

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

7. Stickel, W.J., D.W. Hayne, and L.F. Stickel. "Effects of heptachlor-contaminated earthworks on woodcock". J. Wildlife Manage. 29: 132 (1965).

8. U.S. Department of Health, Education and Welfare. "The toxic substances list". U.S. Govt. Print. Office, Washington, 1972.

9. Wisman, E.L., R.W. Young, and W.L. Beane. "Persistence of heptachlor in egg yolks". Poult. Sci. 46: 1606 (1967).

(vi) Lindane

RECOMMENDATION

It is recommended that the following numerical objective for lindane be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2(c) of the Water Quality Agreement:

> *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 gram for the protection of human consumers of fish.*

RATIONALE

Macek *et al.* (3) 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. With a 96-hour LC_{50} of 2.0 µg/l, the brown trout is apparently most sensitive to lindane on an acute basis amongst those species used in aquatic bioassays (4). 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.* (3) 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, in that the application factors for these invertebrates were much 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 (6) and Cope (2) reported 96-hour LC_{50} values of 1 µg/l for stoneflies. Sanders and Cope (5) reported an acute 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 stonefly, 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 exists on the accumulation of lindane in fish tissues. However, Macek *et al.* (3) observed whole-body (eviscerated) concentrations (wet weight) about 500 times the corresponding water concentrations in fathead minnows that had been exposed for several months. Butler (1) 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. As a result, the commended 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.

LITERATURE CITED

1. Butler, P.A. "Pesticide residues in estuarine mollusks", in Proceedings of the National Symposium on Estuarine Pollution, P.F. McCarty and R. Kennedy, ed. Stanford University Department of Civil Eng., Stanford, Ca., 1967: 107-121.

2. Cope, O.B. "Sport fishery investigation", in "Effects of pesticides on fish and wildlife: 1964 research findings of the Fish and Wildlife Service". U.S. Fish and Wildlife Serv. Circ. 226, 1965.

3. Macek, K.J., K.S. Burton, S.K. Derr, J.W. Dean, and S. Sauter. "Chronic toxicity of lindane to selected aquatic invertebrates and fish". U.S. Environmental Protection Agency Ecol. Res. Series 600/3-76-047. 1976.

4. Macek, K.J. and W.A. McAllister. "Insecticide susceptibility of some common fish family representatives". Trans. Amer. Fish. Soc. 99(1): 20-27 (1970).

5. Sanders, H.O. and 0.B. Cope. "The relative toxicities of several pesticides to naiads of three species of stoneflies". Limnol. Oceanogr. 13(1): 112-117 (1968).

6. Snow, J.R. "A preliminary report on the comparative testing of some of the newer herbicides". Proc. 11th Ann. Conf. Southeast. Assoc. Game Fish Comm. 1958: 25-132.

(vii) Methoxychlor

RECOMMENDATION

It is recommended that the following numerical objective for methoxychlor be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2(c) of the Water Quality Agreement:

The concentration of methoxychlor in water 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 mortality 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% in controls to 39% (2). Yellow perch seem to be less sensitive than fathead minnows.

Merna and Eisele (2) 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 (1) continuously dosed a small stream with 0.2 µg/l methoxychlor for one year. No insect or fish mortalities were observed and no invertebrate species were eliminated, although 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 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 exploring chronic effects under field conditions. The 0.2 µg/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 recommended "safe" concentration of 0.04 µg/l.

Because it degrades readily, methoxychlor may not conform to the definition of a persistent compound. The structure of its probable metabolites indicates that they too are not likely to persist. However, the actual rate of degradation of methoxychlor is not indicated in the literature and it has been considered under the category of persistent contaminants due to its organochlorine pesticide nature. If it were classified as non-persistent, consideration would be given to the lowest reported 96-hour LC_{50}

concentrations which pertain to crustaceans (0.8 to 5 µg/l) (4,5) and to insects (0.6 to 1.4 µg/l) (2,3). 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.

LITERATURE CITED

1. Eisele, P.J. "The effects of methoxychlor on aquatic invertebrate populations and communities". Ph.D. Thesis, University of Michigan, 1974.

2. Merna, J.W. and P.J. Eisele. "The effects of methoxychlor on aquatic biota". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-046. 1973.

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

4. Sanders, H.O. and O.B. Cope."Toxicities of several pesticides to two species of cladocerans". Trans. Amer. Fish. Soc. 95(2): 165-169 (1966).

5. Sanders, H.O. "Toxicity of pesticides to the crustacean *Gcvrmarus lacustri".* U.S. Bur. Sport Fish. and Wildlife Technical Paper 25, 1969.

(viii) Toxaphene

RECOMMENDATION

It is recommended that the following numerical objective for toxaphene be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2(c) of the Water Quality Agreement:

> *The concentration of toxaphene in water should not exceed 0.008 micrograms per litre for the protection of aquatic life.*

RATIONALE

Mayer *et al.* reported decreased reproduction of brook trout 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 *et al.* (6) also 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 (5) and 3.5 µg/l in soft water for bluegills (3). Acute toxicities were also reported for several species of fish by Macek (4), ranging from 2 µg/l for largemouth bass to 13 µg/l for black bullhead, and by Nagvi and Ferguson (7) for freshwater shrimp as 24-hr LC_{50}, ranging from 41 to 283 µg/l in four different lakes.

Schoettger and Olive (8) reported mortality of kokanee salmon when fed *Daphnia* which were exposed to sublethal concentrations of 10 and 20 µg/l of toxaphene over periods of 120 to 312 hours. Hughes (1) documented that lakes treated with 40 to 150 µg/l toxaphene 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 (6), and 1,000 to 2,000 for aquatic invertebrates (9) were observed. Bioconcentration of toxaphene in fathead minnows was found to be in the range of 77,000 to 108,000 (6). However, these factors have not been related to deleterious body burdens, thus, no recommendation for tissue concentrations of toxaphene can be set at this time.

LITERATURE CITED

1. Hughes, R.A. "Persistence of toxaphene in natural waters". M.S. Thesis, University of Wisconsin, 1969.

2. Hughes, R.A. and G.F. Lee. "Toxaphene accumulation in fish in lakes treated for rough fish control". Environ. Sci. Technol. 10: 934-939 (1973).

3. Lawrence, J.M. "Toxicity of some new insecticides to several species of pond fish". Progr. Fish-Cult. 12: 141-146 (1950).

4. Macek, K.H. "Insecticide susceptibility of some common fish family representatives". Trans. Amer. Fish. Soc. 99: 20-27 (1970).

5. Mandi, M.A. "Mortality of some species of fish to toxaphene at three temperatures". U.S. Bur. Sport Fish. and Wildlife Inves. in Fish Control 6: 1-10 (1966).

6. Mayer, F., P.M. Mehrle, Jr., and W.P. Dwyer. "Toxaphene effects on reproduction, growth, and mortality of brook trout". U.S. Environmental Protection Agency Ecol. Res. Series 600/3-75-013. 1975.

7. Nagvi, S.M. and D.E. Ferguson. "Levels of insecticide resistence in fresh water shrimp, *Palaemonetese kadiakensis".* Trans. Amer. Fish. Soc. 99:696-699 (1970).

8. Schoettger, R.A. and J.R. Olive. "Accumulation of toxaphene by fish-food organisms". Limnol. Oceanogr. 6: 216-219 (1961).

9. Terriere, L.C., V. Kelgemagi, A.R. Gerlach, and R.L. Borovicka. "The persistence of toxaphene in lake water and its uptake by aquatic plants and animals". J. Agr. Food Chem. 14: 66-69 (1966).

(b) Other Compounds

(i) Phthalic Acid Esters

RECOMMENDATION

It is recommended that the following numerical objective for phthalic acid esters be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex 1, paragraph 2(c) of the Water Quality Agreement:

> *The concentrations of dibutyl phthalate and di-(2-ethylhexyl) phthalate in water should not exceed 4.0 micrograms per litre and 0.6 micrograms 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 water 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 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, some of the more common ones are indicated:

Phthalic Acid Esters

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

The occurrence of PAE residues in North American environments was reviewed at a 1972 conference on PAE's sponsored by the National Institute of Environmental Health Sciences, by Mathur (6) and by Mayer *et al.* (9). Within aquatic ecosystems, PAE residues have been detected in fish, water and sediments; the most likely sources are municipal and industrial effluents (2,5,9,11). 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 in these tributaries is not well defined, but analyses of settleable solids showed residues ranging from 1 to 75 µg/g (dry weight). 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 (1) reported that laboratory-scale activated sludge processes degraded 91% 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 weight) (5). 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 (3,4). In aerobiosis studies, 98% of DBP was degraded after 5 days at 20°C, but only 50% 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 other factors. In any case, limited monitoring data (5) suggest that PAE's may occur in bottom sediments, 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 to 0.5 µg/g, and DEHP residues have been found as high as 3.2 µg/g (9,11). PAE residues in Great Lakes area fish range from undetected to 1.3 µg/g (5). 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 (8,9). Mayer and Sanders (8) 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 (3,4). However, Mayer and Rodgers (7) 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 (8). When fish and invertebrates containing PAE residues are placed in untreated water, they eliminate 50% 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 of DBP for fathead minnows, channel catfish *(Ictalurus punctatus),* rainbow trout *(Salmo gairdneri),* scud *(Gammarus pseudolimnaeus)* and crayfish *(Orconectesnais)* fall between 730 and 10,000 µg/l. (8). 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. Flowthrough tests used for scud *(G. fasciatus)* gave a 9-week LC_{50} value of 210 µg/l (10). 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 (10) 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 (8,10). The emergence of adult midges *(Chironomus tentans)* is reduced significantly at a DEHP concentration of 14 µg/l (7).

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 DEHP 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.

LITERATURE CITED

1. Graham, P.R. "Phthalate ester plasticizers: why and how they are used". Environ. Health Perspect. 3: 3 (1973).

2. Hites, R.A. "Phthalates in the Charles and Merrimack Rivers". Environ. Health Perspect. 3: 17 (1973).

3. Johnson, B.T. "Interaction of micro-organisms and pesticides". (Unpublished data). Project No. 221 in U.S. Fish and Wildlife Service, Fish-Pesticide Research Laboratory, Columbia, Mo., "Semi-annual progress report, October 1973 - March 1974": 19-21 (1974).

4. Johnson, B.T. Personal communication from U.S. Fish and Wildlife Service, Fish-Pesticide Research Laboratory, Columbia, Missouri, 1974.

5. "Lake Michigan Toxic Substances Committee Report". U.S. Environmental Protection Agency, Chicago, Illinois, 1974: 184.

6. Mathur, S.P. "Phthalate esters in the environment: pollutant or natural products"? J. Env. Qual. 3(3): 189-193 (1974).

7. Mayer, F.L., Jr., and C.A. Rodgers. "Residue dynamics of di-2-(ethylhexyl) phthalate in fathead minnows". (Unpublished data). U.S. Fish and Wildlife Service, Fish-Pesticide Research Laboratory, Columbia, Missouri, "Annual Progress Report 1972": 24-25.

8. Mayer, F.L., Jr., and H.O. Sanders. "Toxicity of phthalic acid esters in aquatic organisms". Environ. Health Perspect. 3: 153 (1973).

9. Mayer, F.L., Jr., D.L. Stalling, and J.L. Johnson. "Phthalate esters as environmental contaminants". Nature 238 (5364): 411-413 (1972).

10. McKim, J. Personal communication from Environmental Protection Agency, National Water Quality Laboratory, Duluth, Minnesota, 1974.

11. Stalling, D.L., J.W. Hogan and J.L. Johnson. "Phthalate ester residues: their metabolism and analysis in fish". Environ. Health Perspect. 3: 159 (1973) .

(ii) Polychlorinated Biphenyls

RECOMMENDATION

It is recommended that the following numerical objective for polychlorinated biphenyls be adopted to replace, in part, the existing interim objective for Persistent Organic Contaminants in Annex I, paragraph 2(c) of the Water Quality Agreement:

> *The concentration of total polychlorinated biphenyls in fish tissues (whole fish, calculated on a wet weight basis), should not exceed 0.1 micrograms per gram for the protection of fish-consuming birds and animals.*

Note: The Committees expressed concern that a water concentration objective for this ubiquitous contaminant is unavailable. Based upon poorly defined bioconcentration factors it was concluded that PCBs in water should not exceed 0.001 micrograms per litre. However, this level may not be adequate to provide protection to certain predators, and could not presently be enforced because of insufficiently sensitive quantification limits.

RATIONALE

In North America polychlorinated biphenyls (PCBs) are manufactured solely by the Monsanto Chemical Company and are distributed under the trade name AROCLOR . Each Aroclor is a mixture of various isomers of which 210 may occur in theory; the actual number of isomers formed chemically is probably closer to 100. In addition, it has been predicted that a significant percentage of these isomers exist in pairs of optically active forms (7).

Polychlorinated biphenyls are now known to be the third most widely distributed pollutant on earth, exceeded only by the chlorinated insecticides DDT and dieldrin. Similar to the latter compounds, PCB residues are found in the fat deposits of numerous warm and cold-blooded animals including man. Their persistence is generally considered to be greater than most chlorinated insecticides. In the aquatic environment, PCBs have been detected in water, sediments, invertebrates, fish and waterfowl with highest levels being recorded in predatory organisms high in the food chain. While greater quantities are found in areas close to heavy industrialization, substantial residues have been detected in fish from undeveloped localities suggesting that aerial transport may play a significant role in their distribution.

PCBs in Great Lakes Waters

Contamination of the Great Lakes by PCBs has been, and continues to be, extensive. Fifty-seven percent of water samples from 30 major tributaries analyzed by the Michigan Water Resources Commission (9) contained detectable concentrations of PCB ($\geq$0.01 µg/l). Mean values determined for Michigan tributaries of lakes Michigan, Huron, Superior, St. Clair and Erie were 0.023 µg/l, 0.228 µg/l, 0.010 µg/l, 0.081 µg/l, and 0.186 µg/l total PCB, respectively.

The Canada Centre for Inland Waters examined the open waters of lakes Ontario and Erie during 1971 and found measurable quantities in 60 and 63 percent of the samples respectively. Summaries of data from the three basins in each lake were presented in the Water Quality Board's report on PCBs in the Great Lakes environment (6). The highest mean value was 0.062 µg/l for bottom waters of Lake Ontario (western region), while the lowest was 0.012 µg/l for the surface of Lake Ontario (eastern region). Lakewide means of surface and bottom water samples were 0.030 µg/l and 0.032 µg/l, respectively for Lake Ontario, and 0.027 µg/l and 0.025 µg/l for Lake Erie. Samples taken from Hamilton Harbour (Lake Ontario) by the Ontario Ministry of Environment in 1972 showed ranges for PCBs of .035 to 0.095 µg/l in water and 0.2 to 10.1 µg/g in sediments (1).

PCBs in Biota

Fish contamination is also widespread. The United States Food and Drug Administration guideline of 5 µg/g in edible tissue has been exceeded in numerous species in Lake Michigan including lake trout, coho salmon, chinook salmon and chub. In Lake Huron, walleye, whitefish, and catfish are above the tolerance level and likewise smelt and coho salmon in Lake Ontario. PCB concentrations in Lake Erie fish are generally below 5 µg/g with the exception of white bass (6). Analyses by the Ontario Ministry of the Environment on fish from the St.Clair River revealed muscle concentrations of 4.3 to 12.3 µg/g in white bass, 0.1 to 6.8 µg/g in pike, 0.1 to 2.8 µg/g in white suckers and 1.5 to 4.7 µg/g in coho salmon. Perch and walleye from Lake St. Clair showed levels of 0.1 to 0.25 µg/g and 0.2 to 3.0 µg/g, respectively (1).

A serious situation exists for fish-eating birds in the vicinity of the lower Great Lakes. Severe reproductive failure has been identified in herring gull colonies around Lake Ontario. While eggshell thinning has been correlated with DDE content of the eggs, there is a positive correlation between early embryonic mortality and PCB contamination (3). Geometric means for PCBs in eggs of four fish-eating bird species are given below in Table 2 (5).

TABLE 2 - PCB RESIDUES IN BIRD EGGS

Location	(µg/g, dry weight basis) Herring gull	Ring-billed gull	Common tern	Double-crested cormorant
Lake Nipigon				77.5 (52)
Lake Huron	368 (5)*	113 (2)	81.7 (8)	140.0 (55)
Detroit River	520 (2)			
Lake Erie	300 (6)	243 (4)	156 (15)	63.7 (18)
Hamilton Harbour			258 (71)	
Lake Ontario	565 (16)	379 (4)	268 (20)	114 (7)

* Quantities in brackets indicate number of samples analyzed.

The major effect of PCBs on young birds is to produce symptoms of chick edema disease. The symptoms are subcutaneous pericardial and abdominal edema, prophyria, liver necrosis and high mortality (2,3,4). In herring gull chicks from Lake Ontario colonies poor hatching success is associated with these levels of edema, and increased prophyrin synthesis but not liver necrosis. These signs were associated with levels of PCBs of over 900 µg/g on a dry matter basis in the liver, amongst the highest levels in the world. Less severe signs were seen in Lake Erie chicks at about 600 µg/g, but were not completely absent in a control group at about 35 µg/g from outside the Great Lakes. Clearly, even the Lake Erie group is contaminated by more than an order of magnitude above these.

In summary there can be little doubt that the existing state of PCB contamination in the Great Lakes system is excessive. Of particular concern must be the higher forms of life in which the process of bioconcentration causes the greatest residues to be accumulated. At present, there are insufficient data to estimate water concentrations of PCBs which will assure protection of predatory fish, fish-eating birds and other predators; this will require greater understanding of the correlation of dietary intakes and bioconcentration factors.

Effects of PCBs on Biota

PCBs are toxic to aquatic life by direct exposure and are hazardous also to consumers of contaminated fish. Reproduction of midges and *Daphnia magna* was reduced at 0.45 µg/l (Aroclor 1254) and 1.3 µg/l, respectively (12). The highest concentration of Aroclor 1248 having no effect on the fathead minnow was about 0.3 µg/l (11), a concentration which resulted in tissue residues of about 90 µg/l or 18 times the guideline for human consumption recommended by United States and Canadian federal health authorities. This indicates a bioconcentration factor for fathead minnows of approximately 3×10^5. The factor for bluegills with Aroclors 1248 and 1254 has been estimated at 7.1×10^4 (15), while large Lake Michigan coho salmon have mean tissue values of about 15 µg/g (16) which is 1.5×10^6 times greater than the maximum open water concentration of around 0.010 µg/l (8).

Two µg/g PCBs in fish flesh have been shown to prevent survival of newborn commercial ranch mink (14) while reproduction was eliminated in mink fed a beef diet containing 0.64 pg/g Aroclor 1254 (13). While this is not a subtle effect, it is the lowest dietary concentration observed to produce a deleterious biological effect. The safety factor of 0.2 applied to this results in the recommended tissue level of 0.1 µ/g.

The recommendation for PCBs is designed to protect the aquatic biota as well as the consumer of aquatic life. A conservative bioconcentration factor of 10^5 could be used to calculate a water concentration for total PCBs which should prevent tissue levels greater than 0.1 pg/g. This would result in a PCB concentration in water of less than 0.001 µg/l, a concentration which would be beyond the present routine analytical sensitivities and therefore impossible to monitor or enforce. It is recommended then that the regulatory agencies undertake fish and bird monitoring programmes to determine compliance with the recommendation regarding tissue levels.

(iii) Other Organic Contaminants

SEE PAGES 10 to 15.

LITERATURE CITED

1. Berg, O.W., P.L. Diosady and G.V. Rees. "PCB contamination of the aquatic environment in Ontario". Ontario Ministry of Environment, Laboratory Branch Report. Toronto, Ontario, 1974.

2. Gilbertson, M. "Pollutants in breeding herring gulls in the Lower Great Lakes". Can. Field Natur. 88: 273-280 (1974).

3. Gilbertson, M., and Fox, G. "Pollutant associated embryonic mortality of Great Lakes herring gulls". Environ. Pollut. 12: 211 (1977).

4. Gilbertson, M., and R. Hale. "Early embryonic mortality in a herring gull colony in Lake Ontario". Can. Field Natur. 88: 354-358 (1974).

5. Gilbertson, M., and R. Reynolds. "DDE, PCB in Canadian birds 1969 - 1972". (Unpublished). Canadian Wildlife Service Occasional Paper No. 19, 1974.

6. Great Lakes Water Quality Board. "Polychlorinated biphenyls in the Great Lakes environment; a report to the International Joint Commission". (Unpublished). 1972.

7. Kaiser, K.L.E. "On the optical activity of polychlorinated biphenyls". Environ. Poll. 7: 93-101 (1974).

8. "Lake Michigan Toxic Substances Committee Report". U.S. Environmental Protection Agency, Chicago, Illinois, 1974.

9. Michigan Water Resources Commission. "PCBs in water intakes in Lake Michigan" in "Lake Michigan Toxic Substances Committee Report". U.S. Environmental Protection Agency, Chicago, Illinois, 1974.

10. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

11. National Water Quality Laboratory. Unpublished reports. Duluth, Minnesota, 1974.

12. Nebecker, A.V. and F.A. Puglisi. "Effect of polychlorinated biphenyls (PCBs) on survival and reproduction of *Daphnia, Gammarus,* and *Tanytarsus".* Trans. Amer. Fish. Soc. 103: 722-728 (1974).

13. Platonow, N.S. and L.H. Karstad. "Dietary effects of polychlorinated biphenyls on mink". Can. J. Comp. Med. 37: 391-400 (1973).

14. Ringer, R.K., R.J. Aulerich, and M. Zabik. "Effect of dietary polychlorinated biphenyls on growth and reproduction of mink". Amer. Chem. Soc. National Meeting, Preprints of Papers 12: 149-154 (1972).

15. Stalling, D.L. and F.L. Mayer, Jr. "Toxicities of PCBs to fish and environmental residues". Environ. Health Perspect. 1: 159-164 (1972).

16. Veith, G.D. "Chlorinated hydrocarbons in fish from Lake Michigan. Final Report". U.S. Water Quality Office, EPA Grant 16020 PBE. 1973.

(2) INORGANIC

(a) Metals

Introduction

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

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

All elements in surface waters are the result of weathering of rocks and soils (Table 3), industrial and municipal effluents and precipitation of airborne matter. In fact, it has been calculated that some large lakes with comparatively little human activity in the drainage basin may derive the major part of their metals from precipitation. Lake sediments, especially in shallow lakes, may also be an important source of trace metals. Such a recycling has been observed for mercury and is well known for phosphorus.

Chemistry

Dissolved Metals

In distilled water, dissolved metals largely exist in "free" ionic form; that is, as very weakly complexed hydroxy- or aquo- complexes. Because of their low complex formation constants, these elements are readily available for any chemical reaction and for biological uptake. Consequently, any uptake by organisms of such ionic metals from water will be rapid and proportionate to their concentration.

TABLE 3

AVERAGE CONCENTRATIONS OF METALS IN ROCKS IN mg/kg (1)

METAL	IGNEOUS ROCK	SHALES	SANDSTONES	LIMESTONES	SOILS	COAL
Aluminum	82,300	80,000	25,000	4,200	71,000	----
Arsenic	1.8	13	1	1	6	25
Cadmium	0.2	0.3	0.05	0.035	0.06	0.25
Chromium	100	90	35	11	100	60
Copper	55	45	5	4	20	300
Iron	56,300	47,200	9,800	3,800	38,000	----
Mercury	0.08	0.4	0.03	0.04	0.03	----
Lead	13	20	7	9	10	5
Nickel	75	68	2	20	40	35
Selenium	0.05	0.6	0.05	0.08	0.2	<7
Silver	0.07	0.07	0.05	0.05	0.1	0.1
Zinc	70	95	16	20	50	40

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

Organo-metallic Compounds

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

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

Particulate and Colloid Metals

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

Additionally, trace metals may be found as hydroxides and their dehydrated forms in very finely dispersed particulate matter of a few hundred to a few thousand molecular units. These aggregates or colloids are usually formed by precipitation of dissolved metals as a result of pH changes, oxidation or biological action. Processes of that nature occur primarily in effluents entering water of different quality. Because of the very small size of colloids, their inherently large surface area and high chemical and biological activity, they may be toxic to biota to a much higher degree than large size particulate matter of a similar chemical composition.

Analysis and Great Lakes Concentrations

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

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

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

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

It was difficult to obtain accurate data on concentrations of metals in Great Lakes waters. Unpublished raw data from monitoring often contained incorrect values due to sample contamination at some stage between collection and analysis. Since analyses are generally quite accurate, the problem is one of sample collection and storage. Consequently, metal concentrations in offshore Great Lakes waters in Table 4 include only summary statistics derived from well-screened raw data on specific metals from the Upper Lakes. Similar statistics are not available for lakes Michigan, St. Clair, Erie and Ontario. Other data on concentrations of metals in waters is from published sources and the accuracy of the data has not been assessed (Table 5).

Biological Effects and Monitoring Problems

In his review of the utility of bioassay results, Sprague (8), indicated that for fish at different times and places, "precipitated" zinc was less toxic, equally as toxic or more toxic than "ionic" zinc. This ambiguity was probably the result of various authors' inability to measure the various forms of zinc. On the other hand, there has been reasonable success in relating toxicity to specific forms of copper.

As a working method for some metals, fairly good correlations with biological availability and hence toxicity, have been obtained by assuming that soluble toxic forms pass a 0.45 µ filter while insoluble non-toxic forms do not.

TABLE 4

CONCENTRATIONS (pg/12) OF METALS IN FILTERED WATER SAMPLES
FROM THE EPILIMNION OF THE UPPER GREAT LAKES

These statistics, which represent values from many stations within a lake sampled several times within a year, are taken from the Upper Lakes Reference Croup, The Waters of Lake Huron and Lake Superior," v.2 and 3. Windsor, Ontario, 1977.

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

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

* Could not be determined from the data available.

TABLE 5

CONCENTRATIONS (µg/l) OF METALS IN FILTERED GREAT LAKES WATER SAMPLED FROM MUNICIPAL WATER INTAKES BETWEEN 1962 AND 1967.*

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

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

* Source: Kopp and Kroner. "Trace elements in waters of the United States, Oct. 1, 1962 - Sept. 30, 1967" (4).

It is recognized however, that the actual separation of these forms is not that simple. Forms which were retained by the filter could be a reservoir of potentially toxic forms which may readily redissolve under changing conditions. Pulse polarography has been used to measure "labile" and "non-labile" forms of copper, but lability has not been directly related to toxicity to algae (3). Specific ion electrodes were used to measure ion activity of copper (12). While the measured ion activity was roughly related to copper toxicity to salmon, an ion activity below 200 µg/l could only be determined by extrapolation. Shaw and Brown (7) also correlated copper toxicity to trout with ion activity as well as with estimated concentrations of carbonate-complexed and NTA-complexed copper. They concluded that toxicity was best characterized by the total of copper (II) ($\approx$ ion activity) and copper carbonate and not by a single form alone.

The standard chemical procedure of acidifying samples to pH2 solubilizes many loosely-bound forms of copper (= "acid extractable"). While this may be undesirable when carrying out toxicity tests, it is an essential procedure for assessing loadings and the potential harm of toxic forms and reservoirs of copper, as well as temporarily inactive forms of copper.

Removal of phyto- and zooplankton from a sample is probably unncessary because their metal concentrations are low and their contribution to total metal concentrations in water samples is minor. For example, copper concentrations in Lake Michigan phyto- and zooplankton were 6 and 5 mg/kg wet weight respectively (2). Assuming a Lake Erie seasonal maximum density of phytoplankton of 14 mg/l (10) and of zooplankton of 1 mg/l (11), the total copper in plankton would be equivalent to 0.089 µg/l of copper in the water. Copper concentrations in Lake Michigan water average 5 µg/l (2). Thus in whole water the maximum error in the metal concentration of the sample during plankton blooms would be about 2%. This value may be too high since plankton in a bloom might deplete the metal ions in the water being sampled rather than adding metal ions. There is also a possibility of zooplankton "swarms" with densities approaching one gram per litre. Such "swarms" might contribute significantly to metal concentrations but the problem could be avoided by not sampling under such extreme conditions. In addition, filtration to remove micro-organisms could be another problem in that the filter may add or remove ionic copper (5). A further problem may be anomalously high concentrations of metals in samples obtained from turbid inshore waters affected by shoreline erosion. These concentrations should be interpreted with caution. The measurement of metals in a sample that has been allowed to settle or that has been filtered could also give erroneous results if metals which are easily dissolved from particulate matter were removed.

Stiff (9) assembled a variety of methods and outlined an analytical routine for differentiating various forms of copper. However, results of this approach have yet to be correlated to toxicity tests in a variety of waters and are not suitable for application to routine monitoring. Nonetheless, it is hoped that future developments in the methodology for identifying the various forms of metals will allow for refinements of objectives. Obviously any such refinement in the determination of the chemical and physical specification of an element will also require more elaborate sampling and storage procedures.

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

Setting Objectives for Metals for Aquatic Biota

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

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

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

"Safe" concentrations may be derived from three measurements:

(a) The Maximum Acceptable Toxicant Concentration (MATC) as defined by Mount and Stephan (6) consists of two numbers: (1) the lowest concentration of a toxicant having a harmful effect on an organism (unsafe) and (2) the highest concentration not producing that effect (safe). The threshold of response occurs somewhere between these two concentrations.

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

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

The Water Quality Board intended to provide a quality of water in the Great Lakes to protect all water uses. Therefore, the following proposed objectives for metals are based on the most sensitive of the defined uses of these Great Lakes waters.

LITERATURE CITED

1. Bowen, H.J.M. Trace elements in biochemistry. Academic Press, London, 1966.

2. Copeland, R.A. and J.C. Ayers. "Trace element distributions in water, sediment, phytoplankton, zooplankton and benthos of Lake Michigan: a baseline study with calculations of concentration factors and buildup of radioisotopes in the food web". Special Report No. 1, Environ. Res. Group, Inc., Ann Arbor, Mi., 1972.

3. Gächter, R., K. Lum-Shue-Chan and Y.K. Chau. "Complexing capacity of the nutrient medium and its relation to inhibition of algal photosynthesis by copper". Schweiz Z. Hydrol. 35: 252-261 (1973).

4. Kopp, J.F. and R.C. Kroner. "Trace elements in waters of the United States, Oct. 1, 1962 - September 30, 1967". Federal Water Pollution Control Administration, Cincinnati, Ohio, 1970.

5. Marvin, K.T., R. R. Proctor, Jr., and R.A. Neal. "Some effects of filtration on the determination of copper in freshwater and saltwater". Limnol. Oceanogr. 15: 320-325 (1970).

6. Mount, D.I. and C.E. Stephan. "A method for establishing acceptable toxicant limits for fish: malathion and the butoxyethanol ester of 2, 4-D". Trans. Amer. Fish. Soc. 96: 185-193 (1967).

7. Shaw, T.L. and V.M. Brown. "The toxicity of some forms of copper to rainbow trout". Water Res. 8: 377-382 (1974).

8. Sprague, J.B. "Measurement of pollutant toxicity to fish. II. Utilizing and applying bioassay results". Water Res. 4: 3-32 (1970).

9. Stiff, M.J. "The chemical states of copper in polluted freshwater and a scheme of analysis to differentiate them". Water Res. 5: 585-599 (1971).

10. Vollenweider, R.A., M. Munawar, and P. Stadelmann. "A comparative review of phytoplankton and primary production in the Laurentian Great Lakes". J. Fish. Res. Board Can. 31: 739-762 (1974).

11. Watson, N.H.F. "Zooplankton of the St. Lawrence Great Lakes: species composition, distribution and abundance". J. Fish Res. Board Can. 31: 783-794 (1974).

12. Zitko, V., W.V. Carson and W.G. Carson. "Prediction of incipient lethal levels of copper to juvenile Atlantic salmon in the presence of humic acid by cupric electrodes". Bull. Env. Contam. Toxicol. 10: 265-271 (1973).

(i) Arsenic

RECOMMENDATION

It is recommended that the following numerical objective for arsenic be adopted in compliance with Annex I, paragraph 7(a), and to replace, in part, the existing interim objective for Mercury and Other Toxic Heavy Metals in Annex I, paragraph 2(b) of the Water Quality Agreement:

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

EXISTING OBJECTIVE

The above objective is recommended to replace the existing interim objective in Annex I, paragraph 2(h) of the Agreement, which states:

"Mercury and Other Toxic Heavy Metals. The aquatic environment should be free from substances attributable to municipal, industrial or other discharges in concentrations that are toxic or harmful to human, animal or 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_20_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 (7). Arsenic is also found in a variety of salt forms including 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 3, p.42).

Other important sources of arsenic contamination are the burning of fossil fuels such as coal and oil, and various pesticides, for example, herbicides, applied directly to water (8,20). Arsenic also comes from various cleansing compounds in which levels as high as 35 µg/g have been measured (22). About 9,000 kg of arsenic were used in the Great Lakes basin in 1968, primarily as As_2O_3, for metallurgy (6).

Arsenic levels in surface waters, from natural or man-made contamination, vary considerably. Ferguson and Gavis (7) reported levels between 0 and 10 µg/l in fresh water, while in Germany levels of 2 to 3 µg/l are normally found (10). Concentrations of arsenic in the Great Lakes are uniformly 1 µg/l, or less in offshore waters (4) but were found to be as high as 58 µg/l in a water intake at Massena, New York (Table 3). The Moira River, flowing into the Bay of Quinte, contains high levels of arsenic from 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 (14).

Arsenic has no known nutritive value for plants (3) and its role in animal nutrition has not yet been proven. However, arsenic in the forms of arsanilic acid, 4-nitrophenylarsonic acid, 3-nitro-4-hydroxy-phenylarsonic acid and phenyl-arsenoxide are proven growth stimulants for pigs and poultry (17).

Arsenic was classified by Bowen (3) 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 (6).

In accordance with the "Safe Drinking Water Act", (PL 93-523), the United States Environmental Protection Agency promulgated National Interim Primary Drinking Water Regulations on December 24, 1975. The proposed maximum contaminant level for arsenic is 50 µg/l, the same value as in the existing standards. A maximum level of 100 µg/l, total arsenic was recommended in "Water Quality Criteria 1972" (13) "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, currently under review, for raw water in Canada specify an acceptable arsenic level of 10 µg/l, and a maximum permissible level of 50 µg/l, (5). For livestock an upper limit of 200 µg/l of arsenic in water is recommended (13).

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 (8), while others have used arsenite as arsenic trioxide (9). The lethal concentrations of both arsenate and arsenite for some algae fall between 2,000 and 10,000 µg/l (21).

The three week LC_{50} of sodium arsenate to *Daphnia magna* was 2,850 µg/l while the concentrations causing 50% and 16% impairment of reproduction were 1,400 and 520 µg/l, respectively (2). 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 (1). Holland (9) noted 22% initial mortality of young pink salmon exposed to 5,300 µg/l arsenic, but mortality in the survivors continued for an additional 20 days. Speyer (16) 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 (11) investigated the effect of arsenic trioxide on fish production using ponds stocked with bluegills. At 4,000 µg/l and 8,000 µg/l, reduction of bottom organisms as compared to the controls was 34% and 45%, respectively. The weight of fish harvested was also substantially reduced in the treated ponds. Conditioned avoidance behaviour of goldfish was significantly impaired by 100 µg/l, arsenic as sodium arsenate but not by 50 µg/l (19).

Gilderhus (8) 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 ovaries. Average residues in Great Lakes fish vary from 3 to 43 µg/kg on a whole weight basis (12), <50 to 700 µg/kg on a dressed fish basis (18), and 6 to 80 µg/kg on a liver basis (12). These values are considerably below those observed experimentally.

Concentrations of arsenic considered safe for public drinking water supplies are substantially lower than those required to protect aquatic life. Consequently, the objective for arsenic should be 50 µg/l in keeping with the approved concentration for the protection of human health. However, to protect aquatic life, the Province of Ontario specifies that "an environmental level of 10 µg/l should not be exceeded under any circumstances" (15). This guideline is not well supported by scientific evidence.

LITERATURE CITED

1. Alderdice, D.F. and J.R. Brett. "Toxicity of sodium arsenite to young chum salmon". Fish. Res. Board Can. Progress Report 108: 27-29 (1957).

2. Biesinger, K.E. and G.M. Christensen. "Effect of various metals on survival, growth, reproduction and metabolism of *Daphnia magna".* J. Fish. Res. Board Can. 29: 1691-1700 (1972).

3. Bowen, H.J.M. Trace elements in biochemistry. Academic Press, London, 1966.

4. Canada Centre for Inland Waters. Unedited data from the STAR file. (Unpublished). Burlington, Ont.

5. "Canadian drinking water standards and objectives 1968". Prepared by the Joint Committee on Drinking Water Standards of the Advisory Committee on Public Health Engineering and the Canadian Public Health Association. Department of National Health and Welfare, Ottawa, 1969.

6. Fenwick, P.D. "The use of selected metals and metallic compounds by industry in the Great Lakes basin". (Unpublished). Canada Centre for Inland Waters, Burlington, Ont., 1972.

7. Ferguson, J.F. and J. Gavis. "A review of the arsenic cycle in natural waters". Water Res. 6: 1259-1274 (1972).

8. Gilderhus, P.A. "Some effects of sub-lethal concentrations of sodium arsenite on bluegills and the aquatic environment". Trans. Amer. Fish. Soc. 95: 289-296 (1966).

9. Holland, G.A., J.E. Lasater, E.D. Neumann and W.E. Eldridge. "Toxic effects of organic and inorganic pollutants on young salmon and trout". State of Washington Dept. of

Fish Research Bull. 5: 188-195 (1960).

10. Hutchinson, G.E. A treatise on limnology, v. 1, Geography, physics, and chemistry: 153. John Wiley and Sons, London, 1957.

11. Lawrence, J.M. "Recent investigations on the use of sodium arsenite as an algicide and its effects on fish production in ponds". Proc. 11th Ann. Conf. Southeast. Assoc. Game Fish Comm.: 281-287 (1958).

12. Lucas, H.F., Jr., D.N. Edgington and P.J. Colby. "Concentrations of trace elements in Great Lakes fishes". J. Fish. Res. Board Can. 27: 677-684 (1970).

13. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

14. Ontario Ministry of the Environment. Water Quality Branch. "Water quality data for Ontario lakes and streams 1970-71, Vol. VI, Part 2". Toronto, Ont., 1971.

15. Ontario Water Resources Commission. "Guidelines and criteria for water quality management in Ontario 1970".Toronto, Ont., 1970.

16. Speyer, M.R. "Some effects of combined chronic arsenic and cyanide poisoning n the physiology of rainbow trout". M.Sc. Thesis, Concordia University, Montreal, 1975.

17. Underwood, E.J. Trace elements in human and animal nutrition. 3d ed. Academic Press, New York, 1971.

18. Uthe, J.F. and E.G. Bligh. "Preliminary survey of heavy metal contamination of Canadian freshwater fish". J. Fish. Res. Board Can. 28: 786-788 (1971).

19. Weir, P.A. and C.H. Hine. "Effects of various metals on behaviour of conditioned goldfish". Arch. Env. Health 20: 45-51 (1970).

20. Wiebe, A.H. "Notes on the exposure of young fish to varying concentrations of arsenic." Trans. Amer. Fish. Soc. 60: 270-278 (1930).

21. Wong, P.T.S. "Toxicity of trace elements on algae". (Unpublished literature review).Canada Centre for Inland Waters, Burlington, Ont., 1975.

22. Zwick, D. and M. Benstock. Water wasteland. Grossman Publishers, New York, 1971.

(ii) Cadmium

RECOMMENDATION

It is recommended that the following numerical objective for cadmium be adopted in compliance with Annex I, paragraph 7(a), and to replace, in part, the existing interim objective for Mercury and Other Heavy Metals in Annex I, paragraph 2(b) of the Water Quality Agreement:

> *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 lead and zinc. There is no specific mining activity for cadmium; it is obtained principally as a by-product of zinc mining (30).

The properties of cadmium make it important in electroplating, in solders, in pigments, as a catalyst in photography, lithography and the electronics industry, and in the manufacture of glass, alloys, biocides, lubricants and storage batteries (13,30). 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 (30). There is considerable use in the automotive and metallurgical industries of the Lower Great Lakes region. Cadmium may enter Great Lakes waters as a result of all these processes. Additional inputs are 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 (22) derived theoretical limiting equilibrium solubilities for the carbonate and hydroxide complexes of cadmium in specific waters. He found that cadmium concentrations in surface waters of the United States, as reported by various authors, were much lower than the maximum permitted by the solubility product of the carbonate, the least soluble salt. He attributed the difference to the action of other complexing and adsorbing materials. Hahne and Kroontje (20) 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 (18) measured the degree of complexation of cadmium in synthetic solutions and natural river waters containing varying amounts of carbonate, sewage $effluent_s$ 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 found that, of 1,000 µg/l added cadmium, 29-89% occurred as Cd , and the proportion was generally in excess of 50% (18). Suspended solids originating from bottom muds will also adsorb cadmium (19). The degree of adsorption depended on the type of solid, state of subdivision, concentration of metal ion, time of contact and concentration of other complexing ligands. Humic materials again appeared to be the major important component

of mud. After these laboratory studies however, Gardiner (19) 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 (36) measured the metal content of coarse particulate suspended solids (Svedberg coefficient* (S) > 20000), in colloidal particulate suspended solids (100 < S < 20000) and in dissolved solids. The mean cadmium concentrations in these fractions were 18, 519 and 12 µg/g, 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 total amount of cadmium (≈98%) occurred in the dissolved solids. Presumably these materials would include humic acids, carbonates, chlorides, etc. Total cadmium in these waters ranged from 2 to 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 4). In Lake Superior a small proportion of concentrations are between 0.2 and 0.6 µg/l. In Lake Erie, concentrations of cadmium in offshore filtered water never exceeded 1 µg/l, the detection limit at that time (12,21), but in a water intake at Buffalo concentrations ranged as high as 12 µg/l and the mean was 7 µg/l, (Table 5, p. 46). In Lake Michigan, concentrations never exceeded 1 µg/l in 1970 although some tributaries were slightly higher (21). In a 1974 survey of American nearshore waters, cadmium was always less than 2 µg/l (detection limit) in lakes Superior and Huron (33).

Cadmium is extremely toxic to mammals. Acute toxicity to humans includes severe nausea, salivation, vomiting, diarrhea, abdominal pains and myalgic. Liver and/or kidney damage may follow acute poisoning and respiratory distress may also occur (17). Chronic toxicity includes damage to liver, kidney, hematopoietic tissue and the respiratory tract (17). Cadmium has been implicated in bone degeneration in Japan although these findings are controversial (40). Epidemiological and experimental evidence suggests that cadmium may also cause hypertension. In experimental animals cadmium causes testicular damage, kidney damage, increased incidence of tumours and reduced growth (17). The biochemical bases for these effects may be the interaction of cadmium with thiol groups of enzymes or with phosphatidylethanolamine and phosphatidylserine mono- layers (49). As a result, many enzymatic reactions are inhibited by cadmium, and toxic effects occur in mitochondria, kidney tubules and nerve membranes (49). The daily uptake of cadmium by an adult human from drinking water has been estimated as 15 µg, as compared to 200 µg in food and 1 µg in air (35). Of the total cadmium taken in, only 1 to 2% is retained and the rest is excreted in faeces and urine. To limit intake from water to 200 µg/day, a drinking water limit of 10 µg/l cadmium has been recommended (34). 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, (9). To protect livestock 50 µg/l is recommended (34).

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

Cadmium is not a nutrient for plants and was classified by Bowen (7) as highly toxic (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 (34).

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 (24). *Selenastrum capricornutum* is somewhat less sensitive since 80 µg/l caused complete growth inhibition, while 50 µg/l, caused a slight inhibition (2). In a comparative study, Burnison *et* al. (8) found that the concentrations of cadmium in Lake Ontario water causing 70% inhibition of primary productivity *of Scenedesmus quadricauda, Chlorella pyrenoidosa, Ankistrodesmus falcatus* and *Chlorella vulgaris* were 20, 100, 1,000 and 1,000 µg/l, respectively. A macrophyte, *Najas quadulepensis,* was also affected by cadmium. Severe effects were observed at 90 µg/l while 7 µg/l caused reduced chlorophyll, turgor and stolon development (10).

The acute toxicity of cadmium to zooplankton varies considerably with the species tested. In water from Lake di Monate, Italy, the 48-hour LC_{50}'s for *Cyclops abyssorum prealpinus, Eudiaptomus padanus padanus* and *Daphnia hyalina* were 3,800, 550 and 55 µg/l, respectively (3). The 48-hour LC_{50} for *Daphnia magna* in Lake Superior water was 65 µg/l (6), a value close to that of *Daphnia hyalina.* The 3-week LC_{50} for *Daphnia magna* was 5 µg/l while 0.17 µg/l, caused 16% impairment of reproduction. (6). The 96-hour LC_{50} of the freshwater shrimp *Paratya tasmaniensis* at 10 mg/l hardness was 60 µg/l (47). A 96-hour exposure of these shrimp to 30 µg/l cadmium caused a change in the ultrastructure of the gills (26).

Aquatic insects are less sensitive than zooplankton. At a hardness of 44 mg/l, the 96-hour LC_{50}'s of cadmium for *Acroneuria lycorias* (stonefly), *Ephemerella subvaria* (mayfly) and *Hydropsyche betteni* (caddisfly) were >32,000, 2,000 and >32,000 µg/l, respectively (50). 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 (38). The species of caddisfly was unidentified and appeared 10 times more sensitive than that tested by Warnick and Bell (50). The 96-hour LC_{50}'s of a caddisfly, a damsel fly and a mayfly of Tasmania in water of 10 mg/l hardness were 2,000, 250,000 and 840 µg/l, respectively (47). Amphipods are much more sensitive since the 96-hour LC_{50} of *Australochiltonia subtennis* was 40 µg/l (47) while that of a scud *(Gammarus* sp.) was 70 µg/l (38).

The 96-hour LC_{50}'s for a gastropod snail were 3,800 µg/l for eggs and 8,400 µg/l for adults (38). 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 (27). 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 (38), while that of the rotifer *Philodina* sp. was about 100 µg/l (45). *Tetrahymena pyriformis,* a protozoan, showed a growth depression at 15,000 µg/l cadmium and slower swimming at 1,000 µg/l (5).

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 (37). 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 3-10 µg/l (1). Kumada et al. (25) observed a similar 10-day LC_{50} for rainbow trout of 5 to 7 µg/l, cadmium. The 96-hour LC_{50}'s for bluegills *(Lepomis macrochirus),* Florida flagfish (*Jordanella floridae),* dace *(Triborodon hakonensis)* and striped bass *(Monrone saxatilis)* were 17,200 to 24,200, 2,500, 56 to 100, and 2 µg/l, respectively (15,23,25,44).

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 (37). 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 (16). 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 (15) 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 (11) found that bluegill survival was not affected at 80 µg/l cadmium but 100% mortality occurred at 850 µg/l after 5 months. The principal difference between Eaton's study (15) and that of Cearley and Coleman (11) 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 (11) 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 (11).

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. (44).

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 haemorrhagic necrosis of the testes of male trout (42,43). 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 2 weeks earlier than controls (41). 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 (14).

These results correspond fairly well with the effects of cadmium on reproduction and survival of brook trout measured by Benoit *et al.* (4). 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 (25).

Cadmium residues in fish are fairly uniform. Lovett *et* al. (28) 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 pg/kg, the detection limit, with the one exception of 60 µg/kg in rainbow smelt from Lake Erie (48). Using neutron activation, Lucas *et al.* (29) measured cadmium concentrations of 62 to 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 suggesting that the liver concentrates cadmium. Lake Michigan fish (presumably whole) contained 100 to 300 µg/kg cadmium without any variation in feeding habits (21).

In experimental systems, bass and bluegills had total body accumulations of 8 to 15 and 6 to 20 times the concentration in water, depending on that concentration (11). Uptake and concentration in tissues levelled off within 2 months and the greatest accumulation occurred in internal organs. Kumada *et al.* (25) found that concentrations in rainbow trout exposed to cadmium in water reached a plateau in 10 to 20 weeks with maximum concentrations in the kidneys. Concentrations in whole fish were about 10 to 80 µg/kg in control fish and increased only at cadmium concentrations above 1 µg/l. Concentrations in whole fish reached a maximum of 960 µg/kg after 30 weeks in 4.8 µg/l and declined to 440 µg/kg after 10 weeks in clean water. Similar increases in cadmium content were seen in rainbow trout and dace fed food containing up to 100,000 µg/kg of cadmium. Cadmium levels in whole trout fed this maximum amount reached 1,600 µg/kg after 12 weeks and declined dramatically to 70 µg/kg after 6 weeks on a clean diet (25). The dramatic decrease was seen at all concentrations and indicates that cadmium taken in with the food is cleared faster than cadmium taken in from water. This could be illusory if the gills of fish exposed to cadmium in water contain high concentrations that are slowly released to the rest of the body after transferral to clean water.

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

Despite accumulation of cadmium, there is little evidence for bioconcentration up food chains. Mathis and Cummings (31) 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. Similarly, 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 (32). 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 (46). The model may not be useful since data on all trophic levels below fish are inadequate. However, future use of such models, based on adequate data, may give a clearer indication of the potential for bioconcentration. Therefore, because of the extreme sensitivity of reproduction of trout and zooplankton to cadmium, an objective for cadmium in the Great Lakes of 0.2 µg/l is recommended for an unfiltered water sample.

LITERATURE CITED

1. Ball, I.R. "The toxicity of cadmium to rainbow trout *(Salmo gairdneri* Richardson)". Water Res. 1: 805-806 (1967).

2. Bartlett, L., F.W. Rake, and W.H. Funk. "Effects of copper, zinc and cadmium on *Selenastrum capricornutum".* Water Res. 8: 179-185 (1974).

3. Beaudoin. M.F. and P. Scoppa. "Acute toxicity of various metals to freshwater zooplankton". Bull. Env. Contam. Toxicol. 12: 745-751 (1974).

4. Benoit, D.A., E.N. Leonard, G.M. Christensen, and J.G. Fiandt. "Chronic effects of cadmium on three generations of brook trout *(Salvelinus fontinalis)".* Trans. Amer. Fish. Soc. 105(4):55-560 (1976).

5. Berquist, B.L. and E.C. Bovee. "Some adverse effects of cadmium on growth and locomotion of *Tetrahymena pyriformis".* J. Protozool. 20: 497 (1973).

6. Biesinger, K.E. and G.M. Christensen. "Effects of various metals on survival, growth, reproduction and metabolism of *Daphnia magna".* J. Fish. Res. Board Can. 29: 1691-1700 (1972).

7. Bowen, H.J.M. Trace elements in biochemistry. Academic Press, London, 1966.

8. Burnison, G., P.T.S. Wong, Y.K. Chau and B. Silverberg. "Toxicity of cadmium to freshwater algae". Proc. Cdn. Fed. of Biol. Sci. 18(182): 46 (1975).

9. "Canadian drinking water standards and objectives 1968". Prepared by the Joint Committee on Drinking Water Standards of the Advisory Committee on Public Health Engineering and the Canadian Public Health Association. Department of National Health and Welfare, Ottawa, 1969.

10. Cearley, J.E. and R.L. Coleman. "Cadmium toxicity and accumulation in southern naiad". Bull. Env. Contam. Toxicol. 9: 100-101 (1973).

11. Cearley, J.E. and R.L. Coleman. "Cadmium toxicity and bioconcentration in largemouth bass and bluegill". Bull. Env. Contam. Toxicol. 11: 146 (1974).

12. Chawla, V.K. and Y.K. Chau. "Trace elements in Lake Erie" in Proc. 12th Conf. Great Lakes Res., Ann Arbor, Mi., May 5-7, 1969: 760-765.

13. Cheremisinoff, P.N. and Y.H. Habib. "Cadmium, chromium, lead, mercury: plenary account for water pollution. I. Occurrence, toxicity and detection". Water and Sewage Works. 119: 70-73 (1972).

14. Christensen, G.M. "Biochemical effects of methyl mercuric chloride, cadmium chloride, and lead nitrate on embryos and alevins of the brook trout, *Salvelinus fontinalis".* Toxicol. Appl. Pharmacol. 32: 191-197 (1975).

15. Eaton, J.G. "Chronic cadmium toxicity to the bluegill *(Lepomis macrochirus* Rafinesque)". Trans. Amer. Fish. Soc. 103: 729-735 (1974).

16. Eaton, J.G. "Chronic toxicity of a copper, cadmium and zinc mixture to the fathead minnow *(Pimephales promelas* Rafinesque)". Water Res. 7: 1723-1736 (1973) .

17. Flick, D.F., H.F. Kraybill, and J.M. Dimitroff. "Toxic effects of cadmium: a review". Environ. Res. 4: 71 (1971).

18. Gardiner, J. "The chemistry of cadmium in natural water. 1. A study of cadmium complex formation using the cadmium specific ion electrode". Water Res. 8: 23-30 (1974).

19. Gardiner, J. "The chemistry of cadmium in natural water. II. The adsorption of cadmium on river muds and naturally occurring solids". Water Res. 8:157 (1974).

20. Hahne, H.C.H. and W. Kroontje. "Significance of pH and chloride concentration on behaviour of heavy metal pollutants: mercury (II), cadmium (II), zinc (II) and lead (II)". J. Env. Qual. 2: 444 (1973).

21. "Heavy metals in surface water sediments and fish in Michigan". Report to the Michigan Water Resources Commission, Bureau of Water Management, Dept. of Natural Resources. Lansing, MI., 1972.

22. Hem, J.D. "Chemistry and occurrence of cadmium and zinc in surface water and groundwater". Water Resources Res. 8: 661-679 (1972).

23. Hughes, J.S. "Acute toxicity of thirty chemicals to striped bass *(Morone saxatilis)".* Presented at the Western Association of State Game and Fisheries Commissioners in Salt Lake City, Utah, 1973.

24. Klass, E., D. W. Rowe and E.J. Massaro. "The effect of cadmium on population growth of the green alga *Scenedesmus quadricauda".* Bull. Env. Contam. Toxicol. 12: 442-445 (1974).

25. Kumada, H., S. Kimura, M. Yokote and Y. Matida. "Acute and chronic toxicity, uptake, and retention of cadmium in fresh water organisms". Bull. Freshwater Fish Res. Lab. Tokyo. 22: 157-165 (n.d.).

26. Lake, P.S. and V.J. Thorp. "The gill lamellae of the shrimp *Paratya tasmaniensis* (Atyidae:Crustacea); normal ultrastructure and changes with low levels of cadmium", in Eighth International Congress on Electron Microscopy, Canberra, Vol. II: 448-449 (1974).

27. Leidel, G. and E. McLaughlin. "The effects of cadmium chloride on the embryonic development of the freshwater snail *Helisoma* sp". J. Ala. Acad. Sci. 44:178 (1973).

28. Lovett, R.J., W.H. Gutenmann, I.S. Pakkala, W.D. Youngs, D.J. Kisk, G. E. Burdick, and E.J. Harris. "A survey of the total cadmium content of 406 fish from 49 New York State fresh waters". J. Fish. Res. Board. Can. 29: 1283-1290 (1972).

29. Lucas, H.F., Jr., D.N. Edgington and P.J. Colby. "Concentrations of trace elements in Great Lakes fishes". J. Fish. Res. Board Can. 27: 677-684 (1970).

30. Lymberner, D.B. "Environmental contaminants inventory study. No. 2. The production, use and distribution of cadmium in Canada." Environment Canada, Inland Waters Directorate, Report Series No. 39. Burlington, Ont., 1974.

31. Mathis, B.J. and T.F. Cummings. "Selected metals in sediments, water and biota in the Illinois River". J. Water Poll. Cont. Fed. 45: 1573-1583 (1973).

32. Mathis, B.J. and N.R. Kevern. "Distribution of mercury, cadmium, lead, and thallium in a eutrophic lake". Hydrobiologia 46: 207-222 (1975).

33. Michigan Department of Natural Resources. Bureau of Water Management. "Summary of physical and chemical water quality of Michigan's nearshore waters of Lake Superior and Lake Huron". (Unpublished report prepared for the Upper Lakes Reference Group, International Joint Commission). Lansing, MI., 1974.

34. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

35. Nilsson, R.M. "Aspects on the toxicity of cadmium and its compounds: a review". Swedish Natural Science Research Council, Ecological Research Committee Bulletin No. 7. Stockholm, Sweden, 1970.

36. Perhac, R.M. "Distribution of cadmium, cobalt, copper, iron, manganese, nickel, lead and zinc from two streams in Tennessee". J. Hydrol. 15: 177-186 (1972).

37. Pickering, Q.H. and M. H. Gast. "Acute and chronic toxicity of cadmium to the fathead minnow *(Pimephales promelas)".* J. Fish. Res. Board Can. 29: 1099-1106 (1972).

38. Rehwoldt, R., L. Lasko, C. Shaw and E. Wirkowski. "The acute toxicity of some heavy metal ions towards benthic organisms". Bull. Env. Contam. Toxicol. 10: 291 (1973).

39. Rowe, D.W. and E.J. Massaro. "Cadmium uptake and time dependent alterations in tissue levels in the white catfish, *Ictalurus catus* (Pisces:Ictaluridae)". Bull. Env. Contam. Toxicol. 11: 244 (1974).

40. Sandi, E. Personal Communication from Food Directorate, Health Protection Branch, Dept. of National Health and Welfare, Ottawa.

41. Sangalang, G. B. and H.C. Freeman. "Effects of sublethal cadmium on maturation and testosterone on 11-keto-testosterone production *in-vivo* in brook trout". Biol. Reprod. 11: 429-435 (1974).

42. Sangalang, G.B. and M. J. O'Halloran. "Adverse effects of cadmium on brook trout testis and on *in-vitro* testicular androgen synthesis". Biol.. Reprod. 9: 394-403 (1973).

43. Sangalang, G.B. and M. J. O'Halloran. "Cadmium-induced testicular injury and alterations of androgen synthesis in brook trout." Nature 240 (5382): 470-471 (1972).

44. Spehar, R. L. "Cadmium and zinc toxicity to *Jordanella floridae* (Goode and Bean)". U.S. Environmental Protection Agency Ecol. Res. Series 600/J-76-024.1976.

45. Sullivan, G.W., A.L. Buikema and J. Cairns, Jr. "Acute bioassays for the assessment of heavy metal pollution using the fresh water littoral rotifer *Philodina* sp". Va. J. Sci. 24: 120 (1973).

46. Thomann, R.V., D.S. Szumski, D.M. DiToro and D.J. O'Connor. "A food chain model of cadmium in Western Lake Erie". Water Res. 8: 841-849 (1974).

47. Thorp, V.J. and P.S. Lake. "Toxicity biossays of cadmium on selected freshwater invertebrates and the interaction of cadmium and zinc on the freshwater shrimp, *Paratya tasmaniensis* Riek". Aust. J. Mar. Freshwat. Res. 25: 97-104 (1974).

48. Uthe, J.F. and E. G. Bligh. "Preliminary survey of heavy metal contamination of Canadian freshwater fish". J. Fish. Res. Board Can. 28: 786-788 (1971).

49. Vallee, B.L. and D. D. Ulmer. "Biochemical effects of mercury, cadmium and lead". Ann. Rev. Biochem. 41: 91-128 (1972).

50. Warnick, S.L. and H.L. Bell. "The acute toxicity of some heavy metals to different species of aquatic *insects".* J. Water Poll. Cont. Fed. 41: 280-284 (1969).

(iii) Chromium

RECOMMENDED

It is recommended that the following numerical objective for chromium be adopted in compliance with Annex I, paragraph 7(a), and to replace in part, the existing interim objective for Mercury and Other Toxic Heavy Metals in Annex I, paragraph 2(b) of the Water Quality Agreement:

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

RATIONALE

Chromium as Cr(VI) can enter aquatic ecosystems from the production and use of explosives, paper dyes, paints, plated materials and tanning. As Cr(III), chromium is present in glass, ceramics, photography processes and textile dying mordants (5). Up to 1,700 mg/l of chromium as dichromate are also added to cooling tower waters to prevent corrosion; this amount is discharged directly to water courses (16). Chromium occurs at very low concentrations in Great Lakes waters. Offshore, the average recorded concentrations are less than 0.2 µg/l, the detection limit, and 95% of samples contain less than 0.6 µg/l (Table 4, p.45). At water intakes, average concentrations are shown to be less than 10 µg/l and maxima less than 20 µg/l (Table 5). However, concentrations of chromium in water intakes in the St. Lawrence River appear to be much higher (Table 5, p.46). Since Cr(III) is probably complexed as an insoluble hydrated oxide above pH 5 (10), most dissolved chromium in Great Lakes waters is probably in the Cr(VI) valence state. However, Schroeder and Lee (15) clearly demonstrated that Cr(III) added to natural lake waters is converted very slowly to Cr(VI) and that the conversion is slower at low temperatures. Consequently, significant concentrations of Cr(III) could exist in lake water for many days. Cr(VI) can potentially be reduced by H2S at the interface of aerobic and anaerobic waters (15). 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 (15).

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 (11). In mammals, chromium interacts with insulin to increase glucose tolerance and some diabetic conditions are alleviated by chromium treatment (3,17). A 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" (11).

At high concentrations, chromium in air causes respiratory damage and cancer in mammals, while contact with the skin can cause ulcers, scars and allergic effects (11). The effects on humans of chromium in drinking water are unknown but a standard of 50 µµg/l total chromium has been set in the United States to limit total daily intake (10). In Canada, the maximum permissible concentration is 50 µg/l (4).

The toxicity of chromium to aquatic biota is quite variable depending on the species tested. Hervey (7) used a subjective measurement of unicellular algal growth inhibition to demonstrate that some diatoms were sensitive to 320 µg/l, but not to 32 µg/l of chromium. Using ^{14}C fixation to estimate growth, Wium-Anderson (19) estimated that 650 µg/l, of Cr(VI) caused 50% inhibition of photosynthesis by the diatom, *Nitaschia palea.* Patrick *et al.* (13) indicated that 2.08 µg/l of Cr(III) also caused 50% reduction of photosynthesis of *N. palea.* Based on cell counts, 150 µg/l allowed very little growth after 4 days exposure at low cell densities (19). *Daphnia magna* reproduction and activity were inhibited by 330 and 320 µg/l chromium, respectively (2,19). Another invertebrate, *Philodina roseola,* was shown to be 10 times less sensitive than *Daphnia magna* since its life cycle was affected between 3,400 and 4,600 µg/l (14).

A series of unpublished studies by Benoit and Pickering, reported in "Water Quality Criteria 1972" (10), demonstrated "safe" concentrations, based on reproduction, of 300, 600, and 1,000 µg/l of hexavalent chromium for rainbow trout *(Salmo gairdneri),* brook trout *(Salvelinus fontinalis),* and fathead minnow *(Pimephales promelas),* respectively. The "safe" concentration of trivalent chromium for fathead minnows was 1,000 µg/l. Therefore, both valence states of chromium appear equally toxic on a sublethal basis. However, Olson (12) observed that chinook salmon fingerlings *(Oncorhynchus tshawytscha),* after 12 weeks exposure, had higher mortality rates (>50%) and lower growth rates in 200 µg/l Cr(VI) than in 200 µg/l Cr(III) or in control tanks. The fish in Cr(III) had mortality and growth rates identical to those of the control fish. Therefore, on an acute basis, Cr(III) appears less toxic than Cr(VI).

Chromium concentrations in fish tissue are low. Lucas and Edgington (9) measured chromium by neutron activation and found that average whole body concentrations in alewife, spottail shiner and trout perch ranged from 0.9 to 1.6 µg/g. Chromium was also measured by neutron activation in dressed samples of whitefish, northern pike, smelt and perch. The concentrations ranged from <0.017 µg/g to 0.034 µg/g wet weight (18). These results are quite low compared to those in whole fish, suggesting that chromium is not retained by muscle. In addition, there was no variation in the chromium concentration in the fish within species from Lake Erie and from Moose Lake, Manitoba, which is free from industrial activity. Experimental exposures indicate that Cr(VI) was taken up from water at concentrations as low as 1 µg/l (6). At 2,500 µg/l, uptake was via the gills and the metal occurred in the spleen, posterior gut, pyloric caeca, stomach and kidney (8). 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 (11). Also, the residues reported in the uptake experiments were not associated with any damage to the fish. Therefore, no objective for chromium concentrations in fish tissues is recommended at this time.

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

LITERATURE CITED

1. Anderson, B.G. "The toxicity thresholds of various sodium salts determined by the use of *Daphnia magna".* Sewage Works 18: 82(1946).

2. Biesinger, K.E. and G.M. Christensen. "Effects of various metals on survival, growth, reproduction and metabolism of *Daphnia magna."* J. Fish. Res. Board Can. 29: 1691-1700 (1972).

3. Bowen, H.J.M. Trace elements in biochemistry. Academic Press, London, 1966.

4. "Canadian drinking water standards and objectives 1968". Prepared by the Joint Committee on Drinking Water Standards of the Advisory Committee on Public Health Engineering and the Canadian Public Health Association. Department of National Health and Welfare, Ottawa, 1969.

5. Cheremisinoff, P.N. and Y.H. Habib. "Cadmium, chromium, lead, mercury: a plenary account for water pollution. I. Occurrence, toxicity and detection." Water and Sewage Works. 119: 73 (1972).

6. Fromm, P.O. and R.M. Stokes. "Assimilation and metabolism of chromium by trout". J. Water Poll. Cont. Fed. 34: 1151 (1962).

7. Hervey, R.J. "Effect of chromium on the growth of unicellular chlorophyceae and diatoms". Bot. Gaz. 111: 1-2 (1949).

8. Knoll, J. and P.O. Fromm. "Accumulation and elimination of hexavalent chromium in rainbow trout". Physiol. Zool. 33:1-8 (1960).

9. Lucas, H.F., Jr., D.N. Edgington and P.J. Colby. "Concentrations of trace elements in Great Lakes fishes". J. Fish. Res. Board Can. 27: 677-684 (1970).

10. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

11. National Research Council. Committee on Biologic Effects of Atmospheric Pollutants. "Chromium". National Academy of Sciences, Washington, D.C., 1974.

12. Olson, P.A. "Comparative toxicity of Cr(VI) and Cr(III) in salmon". Hanford Biology Research Annual Report for 1957, Hanford Atomic Products Operation, Richland, Wash. 1958.

13. Patrick, R., J. Cairns and A. Scheier. "The relative sensitivity of diatoms, snails and fish to 20 common constituents of industrial wastes". Progr. Fish -Cult. 30: 137-140 (1968).

14. Schaefer, E.D. and W.O. Pipes. "Temperature and the toxicity of chromate and arsenate to the rotifer, *Philodina roseola".* Water Res. 7: 17811790 (1973).

15. Schroeder, D.C. and G.F. Lee. "Potential transformations of chromium in natural waters". Water Air Soil Pollut. 4: 355-365 (1975).

16. Shepherd, C.M. and R.L. Jones. "Hexavalent chromium: toxicological effects and means for removal from aqueous solution". Interim Report, Naval Research Laboratory, Washington, D.C., 1971.

17. Underwood, E.J. Trace elements in human and animal nutrition. 3d ed. Academic Press, New York, 1971.

18. Uthe, J.F. and E.G. Bligh. "Preliminary survey of heavy metal contamination of *Canadian* freshwater fish". J. Fish. Res. Board Can. 28: 786-788 (1971).

19. Wium-Anderson, S. "The effect of chromium on the photosynthesis and growth of diatoms and green algae". Physiol. Plant. 32: 308-310 (1974).

(iv) Lead

RECOMMENDATION

It is recommended that the following numerical objective for lead be adopted in compliance with Annex I, paragraph 7(a), and to replace in part, the existing interim objective for Mercury and Other Toxic Heavy Metals in Annex I, paragraph 2(b) of the Water Quality Agreement:

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

RATIONALE

Lead is released to aquatic ecosystems from the production and use of lead in gasolines, paints, glazes, pipes, roofing materials and ammunition, especially shotgun pellets. Lead is also released during metal mining and refining processes, recycling of used lead products, burning of fuels and recycling or disposal of used motor oils (17).

Lead generally occurs in very low concentrations in water because of its low solubility. Since carbonate, hydroxide, phosphate, chloride, etc. form insoluble salts with lead, any dissolved lead can be converted to an insoluble form and precipitated to the sediments. In Lake Ontario water, for example, it has been found that at concentrations above 100 mg/l lead, more than 98% is precipitated after 24 hours. Above 10 mg/l, 70% is precipitated and above 1 mg/l, 10% is precipitated. The precipitate does not appear to re-dissolve upon agitation (12). 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 (7). At a hardness of 24.0 mg/l, alkalinity of 22.8 mg/l and pH of 6.91, about 100% of lead below 100 mg/l is in a dissolved form. In water with a hardness of 353 mg/l, alkalinity of 243 mg/l and pH of about 7.9, dissolved lead was only 2% of a total of 3,240 mg/l. As the total concentration decreased, dissolved lead increased to 27% of a total of 40 µg/l lead (7). Lead solubility is strongly influenced by pH; above pH 8.0 the solubility is less than 10 µg/l, regardless of alkalinity (11).

Modal lead concentrations in the Upper Great Lakes are less than 1.0 µg/l offshore, and 95% of all samples contain less than 3.0 µg/l (Table 4, p.45) At water intakes, mean lead concentrations are as high as 34 µg/l with maxima at 55 µg/l or less (Table 5, p.46). The higher inshore concentrations probably reflect local inputs to the lakes.

Lead is not essential for plant and animal growth and is, in fact, quite toxic. Bowen (2) has rated lead as being very toxic to plants, that is, 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 (17).

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 (8). The same exposure also caused marked changes in enzyme activity of brain and liver tissue (8). The lethal dose is estimated as 5 to 6 lead pellets for a mallard and 15 to 25 for a Canada goose (17); toxicity varies with diet.

Lead toxicity to mammalian wildlife has not been reported but humans and some domestic animals are quite susceptible to lead. Domestic animals are exposed through ingestion of solid waste, for example, 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 (17). Many of these results are from experimental poisonings. The recommendation for lead in water for livestock in the United States is 100 µg/l (16).

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 (17). Lead poisoning or plumbism, has three aspects: mild or severe dysfunction of the alimentary tract, neuromuscular atrophy, and encephalopathy. Therefore, it has been recommended that total lead intake be limited to 0.6 mg/day by adults (16,17) and 0.3 mg/day by children (17). The recommendation for lead in drinking water in the United States is 50 µg/l (16) while in Canada the maximum permissible limit is 50 µg/l, less than 50 µg/l, is acceptable, and the objective is "not detectable" (4).

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 (22). Toxicity varies considerably between species and between growth media. The growth media factor is of considerable importance since toxicity of lead in natural waters is much greater than in artificial media. Growth of *Ankistrodesmus falcatus,* a green alga of the Great Lakes, was reduced 50% by about 10,000 µg/l lead in Chu 10 medium. In Lake Ontario water, a similar effect was seen between 10 and 100 µg/l (22). Temperature must also be considered, since toxicity increases with temperature (22) and most laboratory studies are conducted at 20°C.

Daphnia magna reproduction was inhibited by 30 µg/l lead (1). Conditioned behaviour of goldfish *(Carassius auratur)* was affected by 70 µg/l lead (21), 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 (9), while growth of guppies *(Lebistes reticulatus)* was reduced by continuous exposure to 1,250 µg/l (5,6).

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) (7). These effects which are probably due to neural damage 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 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. For trout exposed from the egg stage onwards and for parents exposed to lead for one year, the safe-unsafe range was 6 to 12 µg/l in soft water.

Interpolating from Davies and Everhart's results (7), safe-unsafe concentration ranges for total lead in the Great Lakes are as follows:

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

These results were confirmed by Goettl *et al.* (10) 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 (13). On a dissolved basis, this represented 39 and 84 µg/l. Thus, brook trout are apparently not as sensitive as rainbow trout.

Some lead accumulation occurs in aquatic biota. Phytoplankton accumulate large quantities, perhaps due to adsorption by the relatively large surface areas of algal cells, or to ion exchange (19). Leland and McNurmey (14) showed that concentrations of lead were always highest in periphyton of streams and decreased with increasing trophic level. Herbivorous fish had higher concentrations of lead than did carnivorous fish. All concentrations of lead in fish were less than 5.0 µg/g.

Lead concentrations in fillets of Great Lakes fish were found to be uniformly less than 0.5 µg/g, the detection limit, regardless of species or sample location (20). However, in a more recent survey, Brown and Chow (3) 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 (18). 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 to 2.93 µg/l lead, twice as much as in hatchery water. Lead may also occur in blood and accumulate in kidney tissue (12). The significance of these residues to fish health has not yet been determined.

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

Since lead may be methylated to tetramethyl lead by lake sediments (23), these objectives should be re-evaluated when the significance of methylation is defined.

LITERATURE CITED

1. Biesinger, K.E. and G.M. Christensen. "Effects of various metals on survival, growth, reproduction and metabolism of *Daphnia magna".* J. Fish. Res. Board Can. 29: 1691-1700 (1972).

2. Bowen, J.R. Trace elements in biochemistry. Academic Press, London, 1966.

3. Brown, J.R. and L.Y. Chow. "The comparison of heavy metals in fish samples taken from Baie du Dore, Lake Huron and Toronto Harbour, Lake Ontario". Presented at the International Conference on Heavy Metals in the Environment, Toronto, Canada, October 27-31, 1975.

4. "Canadian drinking water standards and objectives 1968". Prepared by the Joint Committee on Drinking Water Standards of the Advisory Committee on Public Health Engineering and the Canadian Public Health Association. Department of National Health and Welfare, Ottawa, 1969.

5. Crandall, C.A. and C.J. Goodnight. "Effects of sublethal concentrations of several toxicants on growth of the common guppy, *Lebistes reticulatus".* Limnol. Oceanogr. 7: 233-239 (1962).

6. Crandall, C.A. and C.J. Goodnight. "The effects of sublethal concentrations of several toxicants to the common guppy, *Lebistes reticulatus".* Trans. Amer. Microsc. Soc. 82: 59-73 (1963).

7. Davies, P.H. and W.H. Everhart. "Effects of chemical variations in aquatic environments. V. III. Lead toxicity to rainbow trout and testing application factor concept". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-011c. 1973.

8. Dieter, M.P. and M.T. Finley. "Lead and 5-ALAD enzyme in canvas backed ducks: a 3 year study". Presented at the International Conference on Heavy Metals in the Environment, Toronto, October, 1975.

9. Dorfman, D. and W.R. Whitworth. "Effects of fluctuations of lead, temperature, and dissolved oxygen on the growth of brook trout". J. Fish. Res. Board Can. 26: 2493-2501 (1969).

10. Goettl, J.P., J.R. Sinley and P.H. Davies. "Water pollution studies". Job Progress Report, Project No. 4-33-R-8, Colorado Division of Wildlife, 1973.

11. Hem, J.D. and W.H. Durum. "Solubility and occurrence of lead in surface water". J. Amer. Water Works Assoc. 65: 562-568 (1973).

12. Hodson, P.V. Unpublished data. Canada Centre for Inland Water, Burlington, Ont.

13. Holcombe, G.W.L., D.A. Benoit, E.N. Leonard and J.W. McKim. "Long-term effects of lead exposure on three generations of brook trout *(Salvelinus fontinalis)".* U.S. Environmental Protection Agency Ecol. Res. Series 600/5-76-022. 1976.

14. Leland, H.V. and J. McNurney. "Transport of lead in a river ecosystem". Presented at the International Conference on Transport of Persistent Chemicals in Aquatic Ecosystems, University of Ottawa, Ottawa, May 1-3, 1974.

15. Lucas, H.F., Jr., D.N. Edgington, and P.J. Colby. "Concentrations of trace elements in Great Lakes fishes". J. Fish Res. Board Can. 27: 677-684 (1970).

16. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972." U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

17. National Research Council of Canada. Associate Committee on Scientific Criteria for Environmental Quality. "Lead in the Canadian environment". Publications NRCC/CNRC No. BY73-7 (ES). October, 1973.

18. Pagenkopf, G.K., and D.P. Neumann. "Lead concentrations in native trout". Bull. Environ. Contam. Toxicol. 12:70.

19. Shukla, S.S. and H.V. Leland. "Heavy metals: a review of lead". J. Water Poll. Cont. Fed. 45: 1319-1331 (1973).

20. Uthe, J.F. and E.G. Bligh. "Preliminary survey of heavy metal contamination of Canadian freshwater fish". J. Fish. Res. Board Can. 28: 786-788 (1971).

21. Weir, P.A. and C.H. Hine. "Effects of various metals on behaviour of conditioned goldfish". Arch. Env. Health 20: 45-51 (1970).

22. Wong, P.T.S., B. Silverberg and Y.K. Chau. "Lead and the aquatic biota". in Biogeochemistry of lead, J.O. Nriagu, ed. (In preparation).

23. Wong, P.T.S., Y.K. Chau, and P.L. Luxon. "Methylation of lead in the environment". Nature 253 (5489): 263-264 (1975).

(v) Mercury

RECOMMENDATION

It is recommended that the following numerical objective for mercury be adopted in compliance with Annex I, paragraph 7(a), and to replace, in part, the existing interim objective for Mercury and Other Toxic Heavy Metals in Annex I, paragraph 2(b) of the Water Quality Agreement:

> *The concentration of total mercury in a filtered water .sole 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 as well as fish--consuming birds.*

RATIONALE

The biologically significant form of mercury is methylmercury, the form in which the bulk of the mercury found in freshwater fish occurs (13,14).

Various forms of mercury may be methylated by at least two mechanisms (17, 26). The extent and rates of methylation are affected by many factors including concentration of mercury ions, availability of mercury ions, growth rate or metabolic activity of the methylating organisms, temperature, and pH (4). Methylmercury may also be demethylated by bacteria in sediments (23). 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. Consequently, the combination of the available mercury concentrations and the operations of both transformation processes are significant. Because fish concentrate methylmercury preferentially over other forms of mercury, and excrete methylmercury very slowly, they are a good indicator of long-term trends of the net methylation rate in an environment. Crayfish also accumulate significant amounts of methylmercury (3). 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 United States Food and Drug Administration and Canadian Food and Drug Directorate administrative guidelines for fish for human consumption are 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 (18).

It is nearly impossible to correlate environmental concentrations of total mercury in unfiltered water with concentrations of methylmercury which accumulate in fish. There are 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 (3,25). 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 (2), and dissolved mercury compounds. The proportion of methylmercury in this complex mixture is probably variable, but cannot be readily determined by present 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 (18).

Equilibrium concentrations were not reached during this exposure and were estimated to be significantly higher (>3 µg/g) by Hartung (10). However, background levels of total mercury in water have been reported to range from 0.05 to 0.1 µg/l (19), and these have been associated with concentrations of 0.01 to 0.2 Old mercury in fish. Thus there is a significant discrepancy between bioaccumlation data derived from experimental exposures to methylmercury when compared will those derived from experimental data. Consequently 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, summarized in Table 6, 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 on an evaluation of the amounts of mercury accumulated in aquatic. organisms.

On Lake St. Clair in 1970, great blue herons and terns were found with mercury levels up to 23 µg/g and 7.5 µg/g, respectively, in their flesh. Fish recovered from their stomachs contained up to 3.8 µg/g mercury (6). No mortalities or population effects were noted in these species. Keith and Gruchy (15) also reported finding elevated mercury residues in gull eggs without any effects on reproduction. The levels found in these instances are close 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 6

MERCURY TOXICITY STUDIES

Organism	Compound	Effect	Lowest Conc. Producing Effect	Highest No Effect Conc.	Remarks	Reference
Gammarus sp.	Hg^{++}	24hr LC_{50}	90 µg/l			(22)
		96hr LC_{50}	10 µg/l			
Nais sp.	Hg^{++}	24hr LC_{50}	1900 µg/l			
		96hr LC_{50}	1000 µg/l			
Caddis fly	Hg^{++}	24hr LC_{50}	5600 µg/l			
		96hr LC_{50}	1200 µg/l			
Damsel fly	Hg^{++}	24hr LC_{50}	3200 µg/l			
		96hr LC_{50}	1200 µg/l			
Chironomus sp.	Hg^{++}	24hr LC_{50}	60 µg/l			
		96hr LC_{50}	10 µg/l			
Amnicola sp.	Hg^{++}	24hr LC_{50}	1100 µg/l			
		96hr LC_{50}	80 µg/l			
Brook trout embryos	CH_3Hg^+	GOT (decreased)	1.03 µg/l	0.08 µg/l	adults exposed 7 mo. before spawning; offspring	
alevins	CH_3Hg^+	GOT (enhanced)	0.93 µg/l	0.08 µg/l	maintained at same conc.	
Rainbow trout	CH_3Hg^+	Decreased Hematocrit	10 µg/l		12 weeks exposure	(20)
		Plasma electolytes in vitro O_2 metabol.		10 µg/l 10 µg/l	" "	
Brook trout	CH_3Hg^+	Cough response	3 µg/l		5 day exposure	
Zebrafish	Phenyl mercuric acetate	No. eggs spawned	1 µg/l	0.2 µg/l	19-25 day exposure	(16)
		% hatching	0.2 µg/l			

TABLE 6 (cont'd)

MERCURY TOXICITY STUDIES

Organism	Compound	Effect	Lowest Conc. Producing Effect	Highest No Effect Conc.	Remarks	Reference
Rainbow trout	Hg^{++}	decreased activity	50 µg/l		4-6 day exposure	(1)
Brook trout	CH_3Hg^+	deformities, deaths in 2nd gen.	0.93µg/l	0.29 µg/l	3 generation exposure	(18)
Cat	CH_3Hg^+	C.N.S. deaths	0.25 mg/kg/day		55-96 feeding of synthetic or "natural" CH_3Hg^+	(5)
Japanese quail	$HgCl_2$	Egg shell thinning	1 µg/g (diet)	2 µg/g (diet)		(24)
Mallard	N-(ethyl mercury)-p-toluene-sulfonanilide	Egg shell thinning		200 µg/g	85 day exposure (contains 3.1% Hg)	(9)
American kestrel	CH_3Hg^+	Egg shell thinning		10 µg/g (diet)	3 months exposure	(21)
Ring dove	CH_3Hg^+	Egg shell thinning decreased egg laying	10 µg/g	10 µg/g	intramuscular	(21) (21)
Mallard	CH_3Hg^+	Decreased hatchling survival	3 µg/g (diet)	0.5 µg/g (diet)	21 week exposure	(11,12)
Mallard duckling	CH_3Hg^+	enhanced avoidance response	0.5 µg/g (diet)		hens fed prior to and during reproductive phase	(11,12)

Table 6 also lists the effects of feeding methylmercury to birds. Eggshell thinning was reported to occur in one study of Japanese quail at 1µg/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 effect found was hatchling survival in mallards at 3 µg/g, but not at 0.5 µg/g. The avoidance response of ducklings was enhanced slightly at 0.5 µg/g methylmercury fed to ducks prior to and during the reproductive phase. Since this effect was slight and may not be harmful, it is likely that the safe level for methylmercury in the diet of birds is close to 0.5 µg/g.

Thus, if the concentration of total mercury in whole fish does not exceed 0.5 µg/g, fish-eating birds should be protected. 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. Therefore, the simultaneous application of the proposed objectives for water and bioaccumulated mercury in fish should protect aquatic life as well as the consumers of aquatic life.

LITERATURE CITED

1. Alexander, D.G. "Mercury effects on swimming and metabolism of trout", in Proc. Int. Conf. on Transport of Persistent Chemicals in Aquatic Ecosystems, Ottawa, Ontario, May 1-3, 1974: 111-65.

2. Andren, A.W. and R.C. Harriss. "Observations on the association between mercury and organic matter dissolved in natural waters". Geochim. Cosmochim. Acta 39: 1253-1257 (1975).

3. Armstrong, F.A.J. and A.L. Hamilton. "Pathways of mercury in a polluted north-western Ontario lake", in Trace metals and metal-organic interactions in natural waters, P.C. Singer, ed. Ann Arbor Science, Ann Arbor, Mi., 1973.

4. Bisogni, Jr., J.J. and A.W. Lawrence. "Kinetics of mercury methylation in aerobic and anaerobic aquatic environments". J. Water Poll. Cont. Fed. 47: 135-152 (1975).

5. Charbonneau, S.M., I.C. Munro, E.A. Nera, R.F. Willes, T. Kuiper-Goodman, F. Iverson, C.A. Moodle, D.R. Stoltz, F.A.J. Armstrong, J.F. Uthe and H.C. Grice. "Subacute toxicity of methylmercury in the adult cat". Toxicol. Appl. Pharmacol. 27: 569-581 (1974).

6. Christensen, G.M. "Biochemical effects of methylmercuric chloride, cadmium chloride, and lead nitrate on embryos and alevins of the brook trout, *Salvelinus fontinalis".* Toxicol. Appl. Pharmacol. 191-197 (1975).

7. Drummond, R.A., G.F. Olson and A.R. Batterman. "Cough response and uptake of mercury by brook trout, *Salvelinus fontinalis,* exposed to mercuric compounds at different hydrogen ion concentrations". Trans. Amer. Fish. Soc. 103: 244-249 (1974).

8. Dustman, E.H., L.F. Stickel and J.B. Elder. "Mercury in wild animals, Lake St. Clair, 1970", in Environmental mercury contamination, R. Hartung and B. Dinman, ed. Ann Arbor Science, Ann Arbor, Mi., 1972.

9. Haegele, M.A., R.K. Tucker and R.H. Hudson. "Effects of dietary mercury and lead on eggshell thickness in mallards". Bull. Environ. Contam. Toxicol. 11: 5-11 (1974).

10. Hartung, R. "Pharmokinetic approaches to the evaluation of methylmercury in fish"., in Proc. of a Workshop on Toxicity to Biota of Metal Forms in Natural Water, Duluth, Minn., Oct. 7-8, 1975. Windsor, Ont., International Joint Commission, 1976.

11. Heinz, G. "Effects of low dietary levels of methylmercury on mallard reproduction". Bull. Environ. Contam. Toxicol. 11: 386-392 (1974).

12. Heinz, G. "Effects of methylmercury on approach and avoidance behaviour of mallard ducklings". Bull. Environ. Contam. Toxicol. 13: 554-564 (1975).

13. Johnels, A.G., T. Westermark, W. Berg, P.I. Persson and B. Sjöstrand. "Pike (*Esox lucius L.*) and some other aquatic organisms in Sweden as indicators of mercury contamination in the environment". Oikos 18: 323333 (1967).

14. Kamps, L.R., R. Carr and H. Miller. "Total mercury-methylmercury content of several species of fish". Bull. Environ. Contam. Toxicol. 8 (5): 273-279 (1972).

15. Keith, J.A. and I.M. Gruchy. "Mercury residues in Canadian wildlife", in Proc. Special Symposium on Mercury in Man's Environment, Royal Society of Canada, Ottawa, 1971.

16. Kihlstrom, J.E., C. Lundberg, and L. Hulth. "Number of eggs and young produced by Zebrafishes *(Brachydanio rerio)* spawning in water containing small amounts of phenylmercuric acetate". Env. Res. 4: 355-359 (1971).

17. Ladner, L. "Biochemical model for the biological methylation of mercury suggested from methylation studies *in vivo* with *Neurospora crassa".* Nature 230: 252-253 (1971).

18. McKim, J.M., G.F. Olson, G.W. Holcombe and E.P. Hunt. "Long term effects of methylmercuric chloride on three generations of brook trout *(Salvelinus fontinalis):* toxicity, accumulation, distribution, and elimination". (Manuscript). U.S. Environmental Protection Agency, Environmental Research Laboratory, Duluth, Minn., 1975.

19. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

20. O'Connor, D.V. and P.O. Fromm. "The effect of methyl mercury on gill metabolism and blood parameters of rainbow trout". Bull. Environ. Contam. Toxicol. 13: 406-411 (1975).

21. Peakall, D.D. and J.L. Lincer. "Methylmercury: its effect on eggshell thickness". Bull. Environ. Contam. Toxicol. 8: 89-90 (1972).

22. Rehwoldt, R., L. Laske, C.Shaw and E. Wirhowsky. "The acute toxicity of some heavy metal ions toward benthic organisms". Bull. Environ. Contam. Toxicol. 10: 291-294 (1973).

23. Spangler, W.J., J.L. Spigarelli, J.M. Rose and H.M. Miller. "Methylmercury bacterial degradation in lake sediments". Science 180: 192-193 (1973).

24. Stoewsand, G.S., J.L. Anderson, W.H. Guntenmann, C.A. Bache and D.J. List. "Eggshell thinning in Japanese Quail fed mercuric chloride". Science 173: 1030-1031 (1971).

25. Walters, Jr., L.R. and J.J. Waler. "Transfer of heavy metal pollutants from Lake Erie bottom sediments to the overlying water". Project completion report No. 421X, Water Resources Center, Ohio State University, 1974.

26. Wood, J.M., F.S. Kennedy and C.G. Rosen. "Synthesis of methylmercury compounds by extracts of a methanogenic bacterium". Nature 220: 173-174 (1968).

(vi) Selenium

RECOMMENDATION

It is recommended that the following numerical objective for selenium be adopted in compliance with Annex I, paragraph 7(a), and to replace in part, the existing objective for Mercury and Other Toxic Heavy Metals in Annex I, paragraph 2(b) of the Water Quality Agreement:

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

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

RATIONALE

Selenium is a common element appearing in the earth's crust at approximately 7 $x10^{-5}$ %. It is present largely as heavy metal selenides (together with sulphide minerals) but also occurs as selenates and selenites. In soils, excluding seleniferous soils not normally found in the Great Lakes region, it has been variously reported to be present at levels ranging from 0.l µg/g to less than 2 µg/g (12). Elevated levels of selenium are found in some sedimentary rock formations and their derived soils in central areas of Canada and the United States. There are no known mining activities for selenium and its production comes mostly as a by-product of copper and lead refining.

Commercial use of selenium was about 500 metric tons in 1968, mostly in the elemental form as red crystals or grey powder. It is used in electronics for rectifiers, photocells, and xerography, in steel and in pigments for paints, glass, and ceramics (11,24).

Selenium is usually present in water as selenate and selenite; the elemental form is insoluble but may be carried in suspension, Weathering of rocks and soil erosion is a major source of selenium in water. On a world basis, approximately 10,000 metric tons yearly is 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 (38). 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 (24). Use of fossil fuel puts about 450 tons per year of selenium (SeO_2) into the atmosphere, about 4.5% of the amount eroded naturally (38).

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 (16).

Concentrations in water are usually low. The literature has been reviewed in several places (e.g. 28), 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 (41). Surface waters in a province of Germany averaged 4 µg/l (17). The normal concentration in sea water is only 0.4 µg/l (9). 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 co-precipitation with ferric hydroxide (41). Selenium concentrations in the Great Lakes are below 1 µg/l offshore and mean concentrations are 0.2 µg/l or less.

Lake sediments seem to act as reservoirs or sinks; in the northern United States they contained from 1.0 to 3.5 µg/g dry weight of selenium, considerably more than the usual concentration in soils (45). Small experimental ecosystem experiments showed that of the total amount of selenium in rain which fell on soil, 75% stayed in soil and 25% ran off into an aquatic system. Thirty-six percent of the selenium entering the aquatic system ended in the sediments while most of the rest was in the biota (19).

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 (28) 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 (23), an amount normally found in an adequate diet (28).

Selenium poisoning of livestock has been divided into two classes: the acute type termed blind staggers and the chronic, called alkali disease. Acute poisoning is associated with ingestion of highly seleniferous plants containing 1,000 µg/g or more of selenium, while the chronic type is associated with grains and plants which contain 5 to 20 µg/g of selenium (27). The extensive literature on natural poisoning of livestock from selenium in their food plants agrees, in general, that 5 µg/g or more can cause death in the herbivore, and that such levels in plants result from soil concentrations in the range 0.5 to 6 µg/g (25,28,42). Also, a diet containing 3 µg/g of selenium in selenite form, in a lifetime study killed rats (37). 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 pallor red tainting of fingers, teeth and hair, dental caries, debility, depression and irritation of nose and throat. Acute toxicity in humans may be characterized by nervousness, vomiting, cough, dyspnea, convulsions, abdominal pain, diarrhea hypotension and respiratory failure (28,38). No recognized cases of non-industrial chronic selenium poisoning in man have been reported (35).

The carcinogenic potential of selenium has been widely investigated (38). Recent critical evaluations made of these early studies concluded that insufficient high quality data exist to assess the carcinogenicity of selenium compounds (34, 46). No suggestion that selenium is carcinogenic in man can be found in the available data (46).

Antagonism between toxicity of selenium and other metals has been pointed out. Levander (23) reviewed the action of arsenic in counteracting selenium toxicity and several cases in which cadmium poisoning is decreased by selenium were listed in Pakkala *et al.* (33) and "The Selenium Paradox" (40). The action against mercury toxicity was mentioned by Koeman *et al.* (20). There are other aspects such as the interrelationship with vitamin E and possible teratogenic effects (40).

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 three months (5).

"Water Quality Criteria 1972" (28) 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 the World Health Organization, the United States, Canada and the U.S.S.R., although some European countries such as France use a 50 µg/l limit.

The National Academy of Sciences and National Academy of Engineering recommended an upper limit of 50 µg/l for selenium in water given to livestock (28). This figure is also used by the Ontario Ministry of the Environment (32).

Bowen (6) described selenium as moderately toxic to plants (toxic effects at concentrations between 1 and 100 mg/l in the nutrient solution). Apparently this applies to freshwater algae as well. The concentrations of selenite causing 95% growth inhibition of *Anabaena variabilis* and *Anacystis nidulans* were 20 and 70 mg/l, respectively (22). Selenate produced the same results with these species at 30 and 50 mg/l, respectively. Kumar (21) showed that growth of *Anacystis nidulans,* a blue-green 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 (7).

Little information is available on the toxicity of selenium to invertebrates, but *Daphnia* sp. is as sensitive as *Scenedesmus* sp. with a lethal threshold of 2.5 mg/l (7).

Niimi and LaHam (29,30) published the most comprehensive studies to date on toxicity of selenium to fish. Acute studies (29) indicated that lethality of selenium to zebrafish larvae *(Brachydanio rerio)* varied with the selenium salt used. The 96-hour and 10-day LC_{50}'s (Table 7) indicated that selenate salts are less toxic than selenite salts.

Table 7

ACUTE TOXICITY OF SELENIUM SALTS TO ZEBRAFISH LARVAE (29)

	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 to a loss of compounds from the solution perhaps because of biological action. This 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.

Studies on the toxicity of selenium dioxide to zebrafish embryos showed that they were quite resistant and concentrations up to 10 mg/l had no effect on hatching (30). This was due probably to the extreme 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 (15). Ellis *et al.* (15) showed that 2 mg/l of the same salt killed goldfish in 1,846 days. Weir and Hine (44) found 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 (44) also discovered that 0.25 mg/l could significantly affect learning behaviour as compared to controls. A concentration of 0.15 mg/l had no meaningful 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 (8).

Concentrations of selenium in fish tissues vary from 0.16 to about 0.6 µg/g, wet weight, in a wide range of locations in fresh and ocean water. This is true for Canadian dressed fish from industrial and isolated locations (0.17 to 0.38 µg/g) (43); for a large series of freshwater fish from New York (0.2 to 0.5 µg/g) (33); for ocean and freshwater fish in Finland (0.2 to 0.58 µg/g) (36); seafoods (about 0.32 to 0.56 µg/g) (26); the edible portion of trout (about 0.28 to 0.68 µg/g) (1); and for samples of marine food fish obtained in Ontario markets (0.16 to 0.4 µg/g) (2). In a very large series of fish from central Canada, concentrations in muscle samples averaged about 0.26 µg/g, and most of the fish fell in the range mentioned above (4). 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 to 2.0 , 0.42 to 1.15, 0.10 to 0.75 and 0.06 to 0.96 µg/g, respectively.

Fish mortality in a Colorado reservoir reported by Barnhart (3) was 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, animals were shown to have higher residues than 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 (36). In an experimental system, Sandholm *et* al. (36) 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 *(Pontius arulius)* absorbed selenium principally from food and showed little uptake from inorganic and organic forms in water. Copeland (13) reported that concentrations of selenium from Lake Michigan zooplankton were highest downwind of industrialized areas, although this was not reflected in the sediments where concentrations were uniformly less than 0.5 µg/g. Concentrations in zooplanKton however, 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 between selenium concentration and size, sex or age of fish (33). Therefore, selenium may be excreted in a fashion similar to that 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 (39).

The discovery that livers of some seals contain from 46 to 134 µg/g selenium may be a serious cause for concern (20). These values are much higher than those of 0.5 to 1.3 µg/g found in the livers of land animals. Also, a 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, however Koeman *et* al. (20) considered that the high selenium might protect 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)(18). 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 seriously.

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. Chau *et al.* (10) demonstrated methylation of sodium selenite, sodium selenate, selenocystine, selenourea and seleno-DL-methionine by microbial action in lake sediments. All sediments that demonstrated microbial action were capable of methylating selenite and/or selenate. Three compounds, mono-, and dimethyl selenide, and an unknown were produced. Since the bacterial action may have produced an unknown selenium compound of high toxicity to fish (29), the selenium objective should be reviewed when the environmental significance of selenium methylation is better understood.

LITERATURE CITED

1. Arthur, D. "Selenium content of Canadian foods. Can. Inst. Food Sci. Technol. J. 5: 165-169 (1972).

2. Arthur, D., University of Guelph, Dept. of Nutrition. Personal communication.

3. Barnhart, R.A. "Chemical factors affecting the survival of game fish in a western Colorado reservoir." Colorado Co-operative Fisheries Research Unit, Quarterly Report, 4: 25-28 (1958). Cited from Sport Fishery Abstracts 3: Abstract No. 1673, p. 114 (1958).

4. Beal, A.R. "A study of selenium levels in freshwater fishes of Canada's central region". Environment Canada Fisheries and Marine Service, Tech. Rept. Ser. No. CEN/T-74-6. 1974.

5. Beath, O.A. "Selenium poisons Indians". Science News Letter 81: 254 (1962).

6. Bowen, H.J.M. Trace elements in biochemistry. Academic Press, London, 1966.

7. Bringmann, G. and R. Kuhn. "The toxic effects of waste water on aquatic bacteria, algae, and small crustaceans". Gesundheits-Ing. 80: 115-120 (1959). (In German, quoted indirectly).

8. Cardwell, R.D., D.G. Moreman, T.R. Payne, and D.J. Wilbur. "Acute toxicity of selected toxicants to six species of fish". (Unpublished). U.S. Environmental Protection Agency Contract No. 68-01-0748. Program Element 1Ba021. n.d.

9. Chau, Y.K. and J.P. Riley. "The determination of selenium in sea water, silicates and marine organisms". Anal. Chim. Acta 33: 36-49 (1965).

10. Chau, Y.K., P.T.S. Wong, B.A. Silverberg, P.L. Luxon and G.A. Bengert. "Methylation of selenium in the aquatic environment". (Submitted to Science, 1976).

11. Cooper, W.C. "Selenium toxicity in man", in Selenium in biomedicine, 0.H. Muth, J.E. Oldfield and P.H. Weswig, ed. Avi Pub. Co., Westport, Connecticut, 1967.

12. Cooper, W.C., K.G. Bennett and F.B. Croxton. "The history, occurrence, and properties of selenium" in Selenium, R.A. Zingar and W.C. Cooper, ed. Van Nostrand Reinhold, New York, 1974.

13. Copeland, R. "Selenium: the unknown pollutant". Limnos 3: 7-9 (1970).

14. Ellis, M.M. "Detection and measurement of stream pollution". Bull. U.S. Bur. Fish. 22:365--437 (1937).

15. Ellis, M.M., H.L. Motley, M.D. Ellis, and R.O. Jones. "Selenium poisoning in fishes". Proc. Soc. Exptl. Biol. Med. 36: 519-522 (n.d.).

16. Feldman, M.G. "Trace materials in wastes disposed to coastal waters: fates, mechanisms, ecological guidance and control": 107. National Technical Information Service Report No. PB 202346. 1974.

17. Heide, F. and P. Schubert. "The content of selenium in Saâle water". Naturwissenschaften 47: 176-177 (1960).

18. Hoffman, I., K.J. Jenkins, J.C. Meranger, and W.J. Pigden. "Muscle and kidney selenium levels in calves and lambs raised in various parts of Canada: relationship to selenium concentrations in plants and possibly Kumar, intakes". Can. J. Anim. Sci. 53: 61-66 (1973).

19. Huckabee, J.W. and B.G. Blaylock. "Microcosm studies on the transfer of mercury, cadmium and selenium from terrestrial to aquatic ecosystems" in Proc. 8th Ann. Conf. on Trace Substances in Environmental Health, 1974: 219-222. Abstracted in NSF-Rann Trace Contaminant Abstracts 283: 112 (July, 1974).

20. Koeman, J.H., W.H.M. Peeters, C.H.M. Koudstaal-Hol, P.S. Tjioe, and J.J.M. de Goeij. "Mercury-selenium correlation in marine mammals". Nature 342 (5425): 385-386 (1973).

21. Kumar, H.D. "Adaption of a blue-green alga to sodium selenate and chloramphenicol". Pl. Cell. Physiol. 5: 465-472 (1964).

22. Kumar, H.D. and G. Prakash. "Toxicity of selenium to the blue-green algae, *Anacystis nidulans* and *Anabaena variabilis".* Ann. Bot. 697-705 (1971).

23. Levander, 0.A. "Metabolic interrelationships and adaptations in selenium toxicity". Ann. N.Y. Acad. Sci. 192: 181-192 (1972).

24. Lymburner, D.B., and H. Knoll. "Selenium: its production and use in Canada, a background paper". (Unpublished). Canada Centre for Inland Waters, Burlington, Ontario, 1973.

25. McKee, J.E. and H.W. Wolf. "Water quality criteria." 2d ed. California State Water Quality Control Board Pub. 3A. Sacramento, 1963.

26. Morris, V.C. and O.A. Levander. "Selenium content of foods". J. Nutr. 100: 1383-1388 (1970).

27. Moxon, A.L. Trace elements. Academic Press, New York, 1958.

28. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

29. Niimi, A.J. and Q. N. LaHam. "Relative toxicity of organic and inorganic compounds to newly hatched zebrafish *(Brachydanio rerio)".* Can. J. Zool. 54: 501-509 (1976).

30. Niimi, A.J. and Q. N. LaHam. "Selenium toxicity on the early life stages of zebrafish, *Brachydanio rerio ".* J. Fish. Res. Board Can. 32: 803-806 (1975).

31. Ohio River Valley Water Sanitation Commission. Metal Finishing Industries Action Committee. Subcommittee on Toxicities. Report No. 3.Cincinnati, Ohio, 1950.

32. Ontario Ministry of the Environment. "Guidelines and criteria for water quality management in Ontario". Toronto, Ont., 1974.

33. Pakkala, Irene S., W. H. Gutenmann, D.J. Lisk, G.E. Burdick, and E.J. Harris. "A survey of the selenium content of fish from 49 New York State waters". Pestic. Monit. J. 6: 107-114 (1972).

34. Palmer, I.S. and Olson, 0.E. "Relative toxicities of selenite and selenate in the drinking water of rats". J. Nutrition 104: 306-314 (1974).

35. Sakurai, H. and Tsuchiya, K."Tentative recommendation for maximum daily intake of selenium". Env. Phys. Bi. 5(2): 107-118 (1975).

36. Sandholm, M., H.E. Oksanen, and L. Pesonen. "Uptake of selenium by aquatic organisms". Limnol. Oceanogr. 18: 496-498 (1973).

37. Schroeder, H.A. "Effects of selenate, selenite, and tellurite on the growth and early survival of mice and rats". J. Nutr. 92: 334-338 (1967).

38. Schroeder, H.A. The poisons around us; toxic metals in food, air, and water. Indiana Univ. Press, Bloomington, Indiana, 1974.

39. Schroeder, H.A., D.V. Frost, and J.J. Balassa. "Essential trace elements in man: selenium". J. Chron. Dis. 23: 227-243 (1970).

40. "The selenium paradox". Food Cosmet. Toxicol. 10: 867-874 (1972).

41. Standard methods for the examination of water and wastewater. 13th ed. Prepared by the American Public Health Assoc., American Water Works Assoc., and Water Pollution Control Fed. Washington, D.C., 1971.

42. U.S. National Technical Advisory Committee on Water Quality. "Water quality criteria; report to the Secretary of the Interior". Washington, 1968.

43. Uthe, J.F. and E.G. Bligh. "Preliminary survey of heavy metal contamination of Canadian freshwater fish". J. Fish. Res. Board Can. 28: 786-788 (1971).

44. Weir, P.A. and C. H. Hine. "Effects of various metals on behaviour of conditioned goldfish". Arch. Environ. Health 20: 45-51 (1970).

45. Weirsma, J.H. and G.F. Lee. "Selenium in lake sediments - analytical procedure and preliminary results". Environ. Sci. Technol. 5: 1203-1206 (1971).

46. World Health Organization. International Agency for Research on Cancer. Monograph on the Evaluation of Carcinogenic Risk of Chemicals to Man 9: 245-260 (1975).

(vii) Zinc

RECOMMENDATION

It is recommended that the following numerical objective for zinc be adopted in compliance with Annex I, paragraph 7(a), and to replace, in part, the existing interim objective for Mercury and Other Toxic Heavy Metals in Annex I, paragraph 2(b) of the Water Quality Agreement:

> *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 fabrication, metal coatings, batteries, paint and varnish, industrial chemicals, rubber, soaps, medicines and pulp and paper production. In 1968, over 1,356 million pounds was used for these purposes in the Great Lakes basin (10). Zinc may enter the Great Lakes as a result of these uses or 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 contributes soluble forms to water (10). Offshore in the Great Lakes, modal concentrations of zinc are less than 10 µg/l, and 95% of samples contain less than 40 µg/l (Table 4, p.45). 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 . The consistently high values here suggest local zinc outputs near the water intake. As noted in Table 5, p.46, high concentrations have also been observed at the St. Marys River (the outlet of Lake Superior), at Buffalo (Lake Erie) and at Massena (outlet of Lake Ontario). Because zinc use is so widespread, sample contamination may be a problem.

An essential element for both plants and animals, zinc is a constituent of many metalloenzymes and several proteins of unknown function (5). 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 resulting from hardening of the arteries (16).

Zinc toxicity to land plants is rare and is usually observed on soils enriched with zinc as a result of mining operations (5). Zinc is relatively nontoxic to man, although when zinc metal is heated, zinc oxide fumes may be produced that can cause "brass chills" or "brass founders ague". Direct doses of soluble zinc salts can cause nausea and vomiting (10). However, no harmful effects on humans have been reported from prolonged consumption of water containing up to 40,000 µg/l zinc (14). Consequently, the United *States* drinking water recommendation is based on taste and has been set at 5,000 µg/l (14). 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 (8).

Concentrations of zinc inhibiting growth of freshwater algae generally range between 1,000 and 10,000 µg/l (22). 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 (2,21).

Aquatic invertebrates are more sensitive to zinc than algae. *Daphnia magna* exposed to zinc for three weeks exhibited 50% mortality at 158 µg/l and 50% and 16% inhibition of reproduction at 102 µg/l and 70 µg/l, respectively (4). 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, (1).

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 (7). In similar water, flagfish *(Jordanella floridae)* were more sensitive than fathead minnows. Eighty percent mortality of flagfish larvae occurred at 85 µg/l zinc and only 10% at 51 µg/l. However, when 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 to 30% occurred at 139 µg/l and 0 to 20% occurred at 75 µg/l or less (18). Rainbow trout fry also died 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 to 251 µg/l (11). Reproduction of bluegills was also 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 (17).

Avoidance of zinc may prevent reproduction of Atlantic salmon. In the laboratory, juvenile salmon avoided 54 µg/l zinc, while in the field, with 19 µg/l copper in the water, migration of adults was prevented by about 240 µg/l zinc (19). 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 (3).

Sublethal toxicity to zinc may be enhanced when combined 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 (9). Therefore, a safe concentration of zinc for fathead minnows was 30 µg/l in soft water (7) and 27.3 µg/l in hard water in the presence of added copper and cadmium (9). However, in Eaton's study (9), it could not be stated that the observed effects 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 to 20 µg/g in fish fillets (20) and from 11 to 48 µg/g in fish livers (12). From these data there appeared to be little variation in zinc content in fish with location within species. In contrast, Brown and Chow (6) 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 exposure of fish to ^{65}Zn in water indicated maximum accumulation in the gills and kidney. Following injection, maximum accumulation occurred in body tissues including kidney, hepatopancreas, heart, intestine, gill and scales (15). Therefore, the route of uptake will affect distribution. Saiki and Mori (15) did not follow concentration or location beyond 48 hours of exposure, nor after transferral to clean water. Mount (13) found that the ratio of zinc in gills to zinc in bones was relatively constant in fish exposed to low levels of zinc. 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 are, 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.

LITERATURE CITED

1. Anderson, B.G. "The apparent thresholds of toxicity to *Daphnia magna* for chlorides of various metals when added to Lake Erie water". Trans. Amer. Fish. Soc. 78: 96-113 (1948).

2. Bartlett, L., F.W. Rabe, and W.H. Funk. "Effects of copper, zinc and cadmium on *Selenastrum capricornutum".* Water Res. 8: 179-185 (1974).

3. Bengtsson, B.E. "Effect of zinc on the growth of the minnow *Phoxinus phoxinus".* Oikos 25: 370-373 (1974).

4. Biesinger, K.E. and G.M. Christensen. "Effects of various metals on survival, growth, reproduction and metabolism of *Daphnia magna".* J. Fish. Res. Board Can. 29: 1691-1700 (1972).

5. Bowen, H.J.M. Trace elements in biochemistry. Academic Press, London, 1966.

6. Brown, J.R. and L.Y. Chow. "The comparison of heavy metals in fish samples taken from Baie du Dore, Lake Huron and Toronto Harbour, Lake Ontario". Presented at the International Conference on Heavy Metals in the Environment, Toronto, Ont., October 26-31, 1975.

7. Brungs, W.A. "Chronic toxicity of zinc to the fathead minnow, *Pimephales promelas* Rafinesque". Trans. Amer. Fish. Soc. 98: 272-279 (1969).

8. "Canadian Drinking water standards and objectives 1968". Prepared by the Joint Committee on Drinking Water Standards of the Advisory Committee on Public Health Engineering and the Canadian Public Health Association. Department of National Health and Welfare, Ottawa, 1969.

9. Eaton, J.G. "Chronic toxicity of a copper, cadmium and zinc mixture to the fathead minnow *(Pimephales promelas* Rafinesque)". Water Res. 7: 1723-1736 (1973).

10. Fenwick, P.D. "The use of selected metals and metallic compounds by industry in the Canadian Great Lakes Basin". (Unpublished). Canada Centre for Inland Waters, Burlington, Ont., 1972.

11. Goettl, J.P., J.R. Sinley and P.H. Davies. "Water pollution studies". Job Progress Report, Project No. 4-33-R-8, Colorado Division of Wildlife, 1973.

12. Lucas, H.F., Jr., D.N. Edgington and P.J. Colby. "Concentrations of trace elements in Great Lakes fishes". J. Fish. Res. Board Can. 27: 677-684 (1970).

13. Mount, D.I. "Chronic toxicity of copper to fathead minnows *(Pimephales promelas* Rafinesque)". Water Res. 2: 215-224 (1968).

14. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

15. Saiki, M. and T. Mori. "Studies on the distribution of administered radioactive zinc in the tissues of fishes". J. Bull. Jap. Soc. Sci. Fish. 21: 945-949 (1955).

16. Schroeder, H.A. The poisons around us; toxic metals in food, air and water. Indiana Univ. Press, Bloomington, Indiana, 1974.

17. Sparks, R.E., J. Cairns, Jr., and A.G. Heath. "The use of bluegill breathing rates to detect zinc". Water Res. 6: 895-912 (1972).

18. Spehar, R.L. "Cadmium and zinc toxicity to *Jordanella floridae* (Goode and Bean)". U.S. Environmental Protection Agency Ecol. Res. Series 600/3-76-024. 1976.

19. Sprague, J.B., P.F. Elson and R.L. Saunders. "Sublethal copper-zinc pollution in a salmon river: a field and laboratory study". Air Water Pollut. 9: 531-543 (1965).

20. Uthe, J.F. and E.G. Bligh. "Preliminary survey of heavy metal contamination of Canadian freshwater fish". J. Fish. Res. Board Can. 28: 786-788 (1971).

21. Whitton, B.A. "Toxicity of zinc, copper, and lead to *Chlorophyta* from flowing waters". Arch. Microbiol. 72: 353-360 (1970).

22. Wong, P.T.S. "Toxicity of trace elements on algae." (Unpublished literature review). Canada Centre for Inland Waters, Burlington, Ont., 1975.

(b) Others

(i) Fluoride

RECOMMENDATION

It is recommended that the following numerical objective for fluoride be adopted in compliance with Annex I, paragraph 7(a), of the Water Quality Agreement:

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

RATIONALE

Fluorine, chemically bound as fluoride, is the seventeenth most abundant element in the earth's crust. Occurring in both igneous and sedimentary rocks, it enters surface waters mainly through the weathering process of these rocks (20). The main fluorine-containing minerals are fluorspar (CaF_2), cryolite (Na_3AlF_6) and fluorapatite [$Ca_5F(PO_4)_6$].

The fluoride cycle involves passage to and from the atmosphere, hydrosphere, lithosphere and biosphere. It has been estimated that 6,000 tons of fluoride are contained in the 30 million tons of soil distributed in the atmosphere each year in the United States (28). Industrial inputs to the atmosphere and surface waters have steadily increased over the past hundred years with the processing of new materials from the earth's crust. These industrial sources include manufacture of aluminum, steel, brick and tile products, phosphorus fertilizer and coal fired electric power generation. Fluoride is also used as a pesticide and for many other commercial purposes. It has been estimated that approximately 120,000 tons of fluoride were emitted to the atmosphere in the United States in 1968 from industrial operations. A large portion of these particulates and gases are removed from modern plants practising good emission control by the use of filters, electrostatic precipitators and various wet- scrubbing systems (28).

Groth (13) estimated that the phosphate and aluminum industries discharge between 10,000 to 35,000 tons of fluoride into United States surface waters annually. He also estimated that fluoridation of municipal water supplies adds another 20,000 tons each year.

Fluoride in Water

In general, most fluoride salts formed with monovalent cations are water soluble (e.g., NaF, AgF and KF) but those formed with divalent cations are usually quite insoluble (e.g., CaF_2 and PbF_2) (37).

Natural or "background" fluoride levels in most freshwater streams are less than 0.2 mg/l (13,30). Concentrations of 13 mg/l are present in the Firehole and Madison rivers in Yellowstone National Park and in Pyramid and Walker lakes in Nevada (38). Many East African lakes contain more than 1,000 mg/l, the highest natural concentrations found anywhere (20). A 1970 "background" water quality survey of 23 streams in the urbanized southeast portion of Michigan's lower peninsula compared to 32 streams in the upper peninsula showed mean fluoride concentrations of 0.40 and 0.18 mg/l, respectively (23).

Fluoride concentrations in the Upper Great Lakes are below those predicted by the equilibrium constants of Kramer (21). These were based on the calcium-carbonate -phosphate-fluoride system which is believed to regulate the concentrations of fluoride in inland lakes. This regulating system was postulated as the explanation for the observation of uniform concentrations of fluoride (0.46 mg/l) at different depths and even in the interstitial water of 14 foot deep core samples from a meromictic lake (5). Comparison of Kramer's predicted fluoride levels for the Great Lakes with concentrations actually observed are given in Table 8:

Table 8

PREDICTED AND OBSERVED FLUORIDE LEVELS
(in mg/l)

		Average fluoride concentrations		
Lake	Predicted Levels	1961 to 1963 (21)*	1968 (46)	1971 (6)
Lake Superior	0.23	0.15	0.032	0.05
Lake Michigan	0.18	0.1	0.1	---
Lake Huron	0.43	---	0.074	0.08
Lake Erie	0.4	0.1	0.110	0.12
Lake Ontario	0.35	0.2	0.116	0.14

* Numbers in parentheses refer to references

Fluoride concentrations increase with high river flows (3), below municipal wastewater discharges (2), and in the vicinity of phosphate-mining operations (25).

Fluoride is considered to be of the main ligands responsible for keeping beryllium, aluminum, scandium, niobium, tantalum, iron and tin in solution in natural waters (33).

Drinking Water Supplies

Fluoride in drinking water generally has the same effect in domestic animals as man. While concentrations less than 2 mg/l usually have no effect, higher levels can cause mottling of teeth and skeletal fluorosis. Since food is the major source of fluoride intake by domestic animals, it has been suggested that concentrations in forage averaging 40 µg/g or less will not cause significant fluorosis (28). Four to 5 mg/l of fluoride in drinking water resulted in observable effects in cattle in the form of dental lesions, mottling, staining and abnormal wearing of the teeth. Thorough examination, however, established that these effects were insignificant in the health, vitality, reproduction or milk production of the animals (29). Even when cattle receive large amounts of fluoride it is not passed along the food chain to man. Instead the accumulated body burden is almost entirely in their bones (27). "Water Quality Criteria 1972" recommended an upper limit for fluoride in livestock drinking water of 2 mg/l to prevent excessive teeth mottling (26).

Up to 1.0 mg/l of fluoride is often added to domestic water supplies to prevent dental caries. Less than about 1 mg/l will seldom cause mottling of teeth even in the most susceptible children. Levels sufficient to cause other health problems could only be accumulated through a large intake of drinking water.

The World Health Organization's European drinking water standards (48) recommend an upper fluoride drinking water limit of 1.5 mg/l. The United States Public Health Service (41) and the Province of Ontario (32) specify a standard of 1.3 mg/l for drinking water for the Great Lakes Basin. The Canadian drinking water standards (7) specify a fluoride concentration of 1.2 mg/l.

Effects on Vegetation

All vegetation contains some fluoride due to uptake from soil and water ranging normally from 2 to 20 µg/g (dry weight). Plants also absorb soluble fluoride salts through their leaves. Information on the amount of fluoride in plant tissue derived from irrigation water is limited. One investigation showed that irrigation water containing 6.2 mg/l of fluoride increased the fluoride content of forage crops from 11 µg/g to 15 to 25 µg/g (34). A United States group of experts recommended a maximum of 1.0 mg/l for continuous use in irrigation water for all general soil applications and 15 mg/l for use over a 20-year period on neutral and alkaline fine textured soils (26).

Apparently no plant injury occurs from irrigation water containing 10 to 15 mg/l fluoride (4,22). No reduction in carbon dioxide uptake occurred in terrestrial mosses incubated in aqueous solutions containing 820 mg/l fluoride for 24 hours. Uptake was effectively stopped by 8,200 mg/l after 24 hours (18). Inglis and Hill (18) concluded that fluoride was relatively non-toxic to mosses.

Danilova (8) found aquatic plants contained higher (40.5 µg/g) concentrations of fluoride than terrestrial plants with 33.8 µg/g. However, no bioaccumulation was observed in either *Cladophora* or diatoms experimentally exposed for 72 days to fluoride concentrations of 52 mg/l (15). In a biossay using the alga *Chlorella pyrenoisda,* Smith and Woodson observed growth suppression at all levels between 4.2 to 4,000 mg/l of fluoride (39). They concluded that this anti-metabolite has its greatest effect between 420 and 4,200 mg/l where 86 and 98% inhibition occurred after 72 hours. Fluoride concentrations of 4.2 and 42 mg/l had equal inhibitory effects of 19% after 72 hours. However, measuring respiration instead of growth in the same algal species, Sargent and Taylor (36) did not detect inhibition at high levels of fluoride (1,680 mg/l). They did find that copper sulfate and fluoride acted more than additively in inhibiting respiration.

Kilman and Hecky (20) observed that the sedge *Cyperus papyrus* was absent in African lakes containing 5.4 and 6.6 mg/l of fluoride, but was abundant in lakes with 0.95 mg/l of fluoride.

Effect on Aquatic Animals

In a review of the available literature, Groth (13) concluded that there was a compelling case for dealing with fluoride as a pollutant with a great capacity for ecological harm. As part of his evidence Groth cited the fact that downstream concentrations of 0.5 to 3 mg/l fluoride can result from both industrial sources and municipal sewage. Concentrations are highest during summer months when biological activity is also at its peak. No ecological effects were correlated with these fluoride levels (2). Groth (13) stated that much additional research was needed on the effects of fluoride and indicated that adverse effects on aquatic life may have been masked in the past by far more severe effects of untreated sewage, industrial effluents and other major pollutants.

Bacterial species commonly associated with municipal wastewaters were unaffected by concentrations up to 800 mg/l fluoride during a 48-hour bioassay. No changes in growth or morphology were observed in *Escherichia coli, Pseudomonas fluorescens* and *Enterococcus* species grown in nutrient broth and mineral media with this concentration of fluoride. No changes in viability were observed in these species after 4 months storage in the fluoride solution (42). Paramecia, *Euglena* and rotifers continued to live, reproduce and were active in fluoride concentrations of 2 to 1,000 mg/l (45).

Using Lake Erie water as the diluent and fluoride as the toxicant, Anderson (1) found a 48-hour EC_{50} of 504 mg/l for *Daphnia magna.* The measure of acute toxicity used was the 48-hour median effective concentration (48-hour EC50) based on immobilization.

Indigenous populations of copepods were found in East African lakes containing 437 mg/l fluoride but not in lakes with 1,064 mg/l (20).

Studies with marine invertebrates indicated that only high fluoride concentrations were toxic to the bluecrab, *Callinectes sapidus* (greater than 20 mg/l) (25), and to oyster (greater than 128 mg/l) (24). However, Hemens and Warwick (15) found a 30% mortality in brown mussels *Prena perna* after 5 days exposure to approximately 7.2 mg/l, and 60% mortality at 41.6 mg/l. No deaths were observed in three species of estuarine fish after 96 hours in 100 mg/l fluoride test solution. Stewart and Cornick (40) found that 10 days exposure to 5 mg/l in sea water did not harm the lobster *Homarus americanus* at 2°C.

Reliable bioassay data for freshwater fish are very limited and some researchers have used soft water as a diluent. Since calcium is antagonistic to fluoride toxicity it may not be valid to apply bioassay data from low calcium water (less than 3 mg/l) to the Great Lakes which contain from 13 to 46 mg/2 calcium (38,43,47).

Neuhold and Sigler (30) determined a 20-day LC_{50} for rainbow trout *Salmo gairdneri* of 2.7 to 4.7 mg/l fluoride (95% confidence level) using softened dilution water (calcium less than 3 mg/l). They concluded that this is much lower than would occur in high calcium water. They also subjected rainbow trout to 30 different combinations of fluoride and calcium concentrations ranging from 0 to 25 mg/l fluoride and 0 to 25 mg/l calcium. From these bioassays they determined the antagonistic relationship between fluoride and calcium and expressed it in an equation. Applying their equation for calcium/fluoride antagonism to Lake Superior water with a calcium concentration of 13 mg/l, the LC_{50} for rainbow trout is 26 mg/l fluoride:

$$y = 2.33 + 2.03 X$$

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

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

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

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

$$5 = 2.33 + 2.03 (\text{Log F} - 0.11)$$

$$5 - 2.33 = 2.03 (\text{Log F}) - 0.11 \times 2.03$$

$$2.67 = 2.03 (\text{Log F}) - 0.22$$

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

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

$$1.42 = \text{Log F}$$

$$26.0 = \text{F conc. in mg/l}$$

The relationship between the concentrations of calcium and fluoride ions and the LC_{50} of rainbow trout subjected to varying combinations of calcium and fluoride was determined by plotting the log of the ratio of fluoride to calcium against the probit of responses to the varying combination of calcium and fluoride. A straight line relationship from which the LC_{50} can be determined was found (Figure 1). The LC_{50} was determined between 1.01 and 4.22 [fluoride] / [calcium] at the 95 percent confidence level. The sensitivity of the rainbow trout to the ratio of fluoride to calcium was between 1.71 and 2.35 probits of response and the log of the fluoride/calcium ratio (Figure 1) is expressed by the formula,

$$Y = 2.33 + 2.03 X$$

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

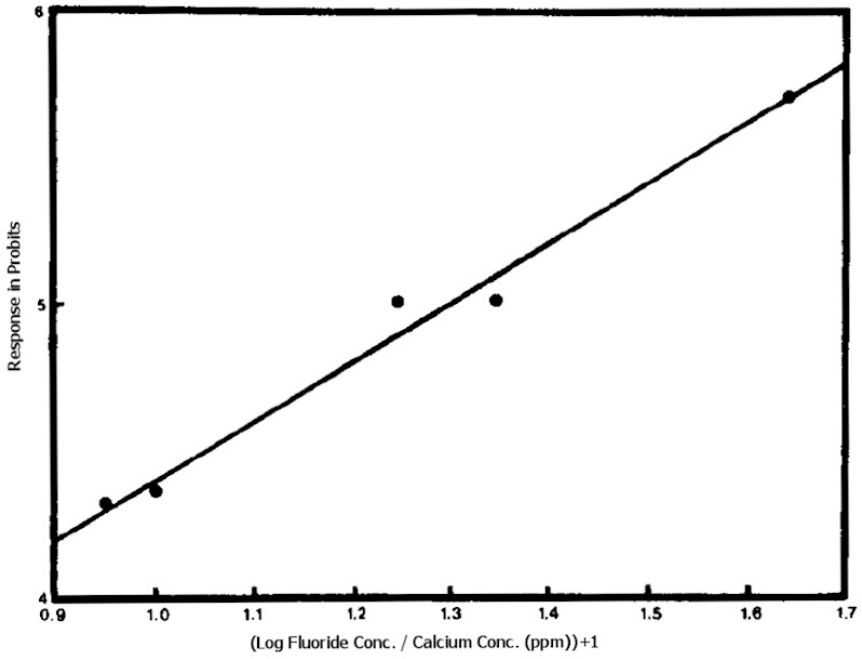

Figure 1. The response of rainbow trout to combinations of fluoride and calcium in the medium (expressed as the ratio between fluoride and calcium).

The LC_{50} Neuhold and Sigler determined for rainbow trout eggs (237 to 381 mg/l) was very high compared with earlier observations by Ellis *et al.* (11), indicating that l.5 mg/l delayed hatching and caused a poorer hatch. Neuhold and Sigler also found that rainbow trout embryos and fry are more sensitive than eggs to fluoride. The 34-day LC_{50} was between 61 and 85 mg/l. In bioassays of the more tolerant carp they found an LC_{50} between 71 and 91 (95% confidence level) at temperatures ranging between 18 and 24°C. The carp were from 10 to 33 cm in size.

Biossays by Herbert and Shurben (16) using rainbow trout showed a 96-hour LC_{50} of about 18 mg/l in very soft water (hardness 12 mg/l). However, the authors concluded that waters with a greater hardness significantly reduced the toxicity of fluoride. They also stated that 1.0 mg/l fluoride would have only a negligible toxic effect on a trout population.

Wallen *et al.* (44) found the mosquitofish *Gambusia affinis* survived fluoride concentrations of 560 mg/l and lower in turbid water with an alkalinity of less than 100 mg/l. The 96-hour LC_{50} was 925 mg/l. Ellis (10) reported that goldfish *Carassius auratus* survived in a concentration of 100 mg/l in hard water for four days (termination of experiment).

In a review, Sigler and Neuhold (38) noted that the response of fish to moderate fluoride concentrations (1.5 to 5 mg/l) is species dependent and related to acclimation, environmental variables such as calcium concentrations and temperature. The healthy growing populations of trout in the Firehole River in Yellowstone National Park and Pyramid and Walker lakes in Nevada where fluoride concentrations reach 13 mg/l indicated that resistence to fluoride toxicity varies in fish. Yet their earlier tests showed that trout raised in low fluoride concentrations displayed LC_{50}'s of approximately 3 mg/l (30).

Bioaccumulation in Aquatic Animals

Fluoride concentrations in fish range from less than 0.1 to 24 µg/g (27). Most of the data available deal with marine fish and potential problems with high fluoride concentrations in fish flour (12). Fish-protein concentrate made in the United States was found to contain 169 µg/g fluoride (14). Hoskins and Loustaunau (17) analyzed fish-protein concentrate made from two marine species and one freshwater species and found that all were less than the United States Food and Drug Administration's 100 µg/g limitation and many were less than 25 µg/g.

Bioassays using fluoride concentrations ranging from 0.5 to 128 mg/l showed accumulations occurred at 2 mg/l and above in oyster tissues. Minimum levels in tissue, which were obtained after the first five days of exposure, were 100 µg/g exposed to solutions of 32 mg/l fluoride while 18 µg/g was found after the 2 mg/l exposure (24). The blue crab similarly reached a concentration of 50 µg/g in muscle after 90 days exposure to 20 mg/l fluoride while the control (0.1 to 1.5 mg/l fluoride) contained 10 µg/g (25).

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

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

LITERATURE CITED

1. Anderson, G.B. "The toxicity thresholds of various sodium salts determined by use of *Daphnia magna."* Sewage Works J. 18: 82-87 (1946).

2. Bahls, L.L."Diatom community response to primary wastewater effluent". J. Water Poll. Cont. Fed. 45: 134-144 (1973).

3. Baker, D.B. and J.W. Kramer. "Water quality control through flow augmentation". U.S. Environmental Protection Agency Ecol. Res. Series 16080 DF001/71. 1971. Abstracted in Algae Abstracts 2: 171.

4. Bower, C.A. and J.T. Hatcher. "Adsorption of fluoride by soils and minerals". Soil Sci. 103: 151-154 (1967).

5. Brunskill, G.J. and R.C. Harriss. "Fayetteville Green Lake, New York. IV. Interstitial water chemistry of the sediments". Limnol. Oceanogr. 14: 858-859 (1969).

6. Canada Centre for Inland Waters, Personal communication, 1975.

7. "Canadian drinking water standards and objectives 1968". Prepared by the Joint Committee on Drinking Water Standards of the Advisory Committee on Public Health Engineering and the Canadian Public Health Association. Department of National Health and Welfare, Ottawa, 1969.

8. Danilova, V.V. " The geochemistry of dispersed fluoride. II. Determination of fluorine in plants". Tran. Lab. Biogeochem. Acad. Sci. URSS 7: 83-85 1944. Abstracted in Chemical Abstracts 41: 1375 f. (1947).

9. Eagers, R.Y. Toxic properties of inorganic fluorine compounds. Elsevier, New York, 1969.

10. Ellis, M.M. 1937. "Detection and measurement of stream pollution". Bull. U.S. Bur. of Fish. 48: 365-437 (1937).

11. Ellis, M.M., B.A. Westfall, and M.D. Ellis. "Determination of water quality". U.S. Fish and Wildlife Service Res. Report 9, 1948.

12. Farkas, C.J. and E.J. Farkas. "Potential effect of food processing on the fluoride content of infant foods". Sci. Total Environ. 2: 399-405 (1974).

13. Groth, Edward. "Fluoride pollution". Environment 17: 29-38 (1975).

14. Hadjimarkos, D.M. "Fluoride in fish flour - effect on teeth". J. Pediat. 65: 782-784 (1964).

15. Hemens, J. and R.J. Warwick. "The effects of fluoride on estuarine organisms". Water Res. 6: 1301-1308 (1972).

16. Herbert, D.W.M. and D.S. Shurben. "The toxicity of fluoride to rainbow trout". Water and Waste Treatment J. 10: 141-142 (1964).

17. Hoskins, Fred H. and J. Loustaunau. "Fish protein concentrate as a mineral nutrient source". Food Tech. 28: 58-60 (1974).

18. Inglis, F. and D.J. Hill. "The effect of sulphite and fluoride on carbon dioxide uptake by mosses in light". New Phytol. 73: 1207-1213 (1974).

19. Josephson, R.K. and K.W. Flessa. "Cryolite: a medium for the study of burrowing aquatic organisms". Limnol. 0ceanogr. 17: 134-135 (1972).

20. Kilham, Peter. and Robert E. Hecky. "Fluoride: geochemical and ecological significance in East African waters and sediments". Limnol. 0ceanogr. 18:932-945 (1973).

21. Kramer, J.R. "Theoretical model for the chemical composition of fresh water with application to the Great Lakes" in Proc. 7th Conf. Great Lakes Res., Toronto, 0nt., April 6-7, 1964: 147-160.

22. McKee, J.E. and H.W. Wolf. "Water quality criteria". 2d ed. California State Water Quality Board Pub. No. 3-A. Sacramento, 1963.

23. Michigan Dept. of Natural Resources. "A survey of background water quality in Michigan's streams". (Unpublished). Lansing, Mi., 1970.

24. Moore, D.J. "A field and laboratory study of fluoride uptake by oysters". Research Rept. No. 20, North Carolina Univ. State College, Raleigh, Water Resources Research Institute, 1969.

25. Moore, D.J. "The uptake and concentration of fluoride by the blue crab *Callinectes sapidus".* Chesapeake Sci. 12: 1-13 (1971).

26. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-003. 1973.

27. National Research Council. Subcommittee on Fluorosis. "Effects of fluorides in animals". National Academy of Sciences, Washington, D.C., 1974.

28. National Research Council. Committee on Biologic Effects of Atmospheric Pollutants. "Fluorides". National Academy of Sciences, Washington, D.C., 1971.

29. Neeley, K.L. and F.G. Harbaugh. "Effects of fluoride ingestion on a herd of dairy cattle in the Lubbock, Texas area". J. Amer. Vet. Med. Assoc. 124: 344-350 (1954).

30. Neuhold, J.M. and W.F. Sigler. "Effects of sodium fluoride on carp and rainbow trout". Trans. Amer. Fish.Soc. 89:358 (1960).

31. O'Brien, R.D. Insecticides, actions and metabolism. Academic Press, New York, 1967.

32. Ontario Ministry of the Environment. "Guidelines and criteria for water quality management in Ontario". Toronto, Ont., 1974.

33. Pitwell, L.R. "Metals coordinated by ligands normally found in natural waters". J. Hydrol. 21: 301-304 (1974).

34. Rand, W.E. and H.J. Schmidt. "The effect upon cattle of Arizona waters of high fluoride content". Amer. J. Vet. Res. 13: 50-61 (1952).

35. Sanders, H.O. and O.B. Cope. "Toxicities of several pesticides to two species of cladocerans". Trans. Amer. Fish. Soc. 95: 165-169 (1966).

36. Sargent, D.F. and C.P.S. Taylor. "The effects of cupric and fluoride ions in the respiration of *Chlorella".* Can. J. Bot. 50: 905-907 (1972).

37. Sienko, J.M. and R.A. Plane. Chemistry. McGraw Hill, New York, 1957.

38. Sigler, W.F. and John M. Neuhold. "Fluoride intoxication in fish: a review". J. Wildlife Dis. 8: 352-354 (1972).

39. Smith, A.0. and B.R. Woodson. "The effects of fluoride on the growth of *Chlorella pyrenoidosa".* Va. J. Sci. 16: l-8 (1964).

40. Stewart, J.E. and J.W. Carnick. "Lobster *(Homarus americanus)* tolerance for tris buffer, sodium fluoride and sea-water extracts of various woods". J. Fish. Res. Board Can. 21: 1549-1551.

41. U.S. Department of Health, Education and Welfare. Public Health Service. "Public Health Service drinking water standards". Rev. ed. PHS Pub. 956. 1962.

42. Vajdic, A.H. "The effect of sodium fluoride on the growth and survival of some bacterial species important in water quality measurement". Ontario Water Resources Commission Div. Res. Paper No. 2006. Toronto, Ont., 1966.

43. Vallin, S. "Toxicity of fluorine to fish". Vatten 24: 51-52. English summary abstracted in Wat. Poll. Abst. 42: 1261 (1969).

44. Wallen, I.E., W.C. Greer and R. Lasater. "Toxicity to *Gambusia affinis* of certain pure chemicals in turbid waters". Sewage and Indust. Wastes 29: 695-711 (1957).

45. Wantland, W.W. "Effect of various concentrations of sodium fluoride on parasitic and free-living protozoa and rotifera". J. Dent. Res. 35: 763-772 (1956).

46. Weiler, R.R. and V.K. Chawla. "Dissolved mineral quality of Great Lakes waters". in Proc. 12th Conf. Great Lakes Res., University of Michigan, Ann Arbor, May 5-7, 1969.

47. Weismann, L. and L. Svatarakova. "Toxicity of sodium fluoride on some species of harmful insects". Biologia (Bratislava) 29: 847-852 (1974).

48. World Health Organization. "European standards for drinking water". Geneva, Switzerland, 1970.

(ii) Total Dissolved Solids

RECOMMENDATION

It is recommended that the existing specific objective for Total Dissolved Solids stipulated in Annex 1, paragraph 1(c) of the Water Quality Agreement be retained:

> "Total Dissolved Solids. In Lake Erie, Lake Ontario and the International Section of the St. Lawrence River, the level of total dissolved solids should not exceed 200 milligrams per litre. In the St. Clair River, Lake St. Clair, the Detroit River and the Niagara River, the level should be consistent with maintaining the levels of total dissolved solids in Lake Erie and Lake Ontario and not to exceed 200 milligrams per litre. In the remaining boundary waters, pending further study, the level of total dissolved solids should not exceed present levels".

RATIONALE

The existing objective for Total Dissolved Solids (TDS) which is recommended for retention at this time is based upon a philosophy of non-degradation and does not comply with the Committees' definition of a specific water quality objective as the level of a substance which will provide for and protect a designated water use. There is no scientific evidence to demonstrate that the specified numerical objectives will interfere with any designated water use.

A review of the scientific literature on the potential effects of TDS on a variety of water uses indicated that a numerical objective far in excess of existing TDS levels would result if a defensible objective were to be established. The Committees were reluctant to pursue such a course as it would mock the non-degradation philosophy and provide an incorrect perspective to surveillance programmes.

While existing levels of TDS in the Great Lakes do not directly threaten any use of the waters, routine monitoring of levels of TDS has traditionally proven to be valuable to surveillance programmes in assessing trends in water quality. For this reason, and to comply with the non-degradation provisions in the Water Quality Agreement, the existing objective is endorsed to encourage the continued use of TDS as a monitoring tool.

On the basis of a recent report on the relationship of TDS and conductivity in the Great Lakes, the Great Lakes Water Quality Board's Surveillance Subcommittee recommended that the existing objective for TDS be replaced by an objective for conductivity. Using a standard conversion factor of 0.65, the numerical conductivity objective for the Lower Lakes would be 308 µmhos/cm. The Committees had no objection to measuring TDS by conductivity methods. It was anticipated that as the importance of individual components of TDS were identified, measurement of those individual components would be required.

The following commentary will provide an overview of the significance of TDS in the Great Lakes.

COMMENTARY

The existing objective in the Water Quality Agreement is consistent with that suggested in Chapter XII - "Remedial Measures" of the report on pollution of Lake Erie, Lake Ontario and the International Section of the St. Lawrence River (5). The report indicated that TDS levels of lakes Erie and Ontario increased from approximately 145 mg/l in 1910 to 185 mg/l in 1970. Furthermore, during that time these lakes experienced a three fold increase in chlorides and a two fold increase in sulfates as well as sodium plus potassium. The report also stated that "the build-up of TDS is not in itself at this time, a serious problem but indicates large accumulations of materials".

Dissolved Solids and Aquatic Life

Hart *et al.* (4) indicated that among the inland waters of the United States supporting a good mixed fish fauna, 95% had a dissolved solids concentration of under 400 mg/l. The main concern of TDS for aquatic life is the effect on an organism's ability to regulate the intake and elimination of water without diluting or concentrating body fluids. In freshwater fish, water is taken in through the mucous membranes of the gills which act like a semipermeable membrane. Body fluids of freshwater organisms are diluted as a result of osmosis. Nearly all freshwater and terrestrial plants, by virtue of their cellulose walls and active plasma membranes, maintain their cellular-fluid constituents, particularly their vacuolar sap, at concentrations higher than those of the fluids which bathe their tissues (11). The cells are continually more concentrated than the tissue fluids and hence turgid.

The range of environmental osmotic conditions tolerated by animals is great, whereas the tolerated range of internal osmotic conditions is much less. Typical values of osmoconcentrations for the freshwater aquatic environment and animal cells, expressed in terms of degrees Celsius lowering of the freezing point of water, are given in Table 9 (11).

TABLE 9

TOTAL DISSOLVED SOLIDS: OSMOCONCENTRATIONS

Environment	AT°C	Animal	OT°C
Fresh water	-0.01	Mussel	-0.08
		Pelomyxa*	-0.14
		Fish	-0.05 to -0.55
		Frog	-0.45
		Crayfish	-0.82
		Earthworm	-0.3 to -0.4

* A large amoeboid protozoan

The concept of freezing point reduction is used primarily because of its applicability in determining the molecular weights of non-volatile dissolved substances in dilute solutions such as body fluids. A water solution of a monovalent salt such as sodium chloride at a concentration of 0.1 molality (5800 mg/l) would cause a freezing point lowering of 0.35°C. (For dilute solutions of an electrolyte the osmotic concentration exceeds the molal concentration by a factor slightly less than 2 for monovalent salts, and slightly less than 3 for divalent salts based on activity coefficients). Table 10 shows the equivalent body fluid concentrations, expressed as sodium chloride, for the freshwater animals listed above.

TABLE 10

TOTAL DISSOLVED SOLIDS AS NaCl: BODY FLUID

Animal	mg/l	Milliosmoles
Mussel	1365	25
Pelomyxa	2388	41
Fish	8530 to 9380	147 to 162
Frog	7680	132
Crayfish	13,990	241
Earthworm	5118 to 6824	88 to 117

According to the report of the National Technical Advisory Committee on Water Quality (12), diatoms are extremely sensitive to changes in chlorides or other dissolved solids and a TDS limit of 50 milliosmoles for the protection of aquatic life is recommended.

In studying the toxicity of brine waters from oil wells, Clemens and Jones (2) found the 96-hr. median toxicity thresholds (equivalent to a 96-hr. LC_{50}) of TDS to 10 varieties of fish to be as listed in Table 11.

TABLE 11

TOTAL DISSOLVED SOLIDS: 96-HOUR LC_{50} VALUES FOR FISH

Fish	TDS-mg/l	
Plains killifish	23,000	
Gambusia	15, 240	
White crappie	12,570	
Bluegill	11,330	
Green sunfish	11,330	Median 11,200
Channel catfish	11,120	
Black bullheads	10,300	
Red shiner	10,506	
Largemouth bass	9,476	
Fathead minnows	8,858	

In addition, the 96-hr. median toxicity threshold of TDS for invertebrates based on dilution of brine waters, was reported to be as indicated in Table 12 (2):

TABLE 12

TOTAL DISSOLVED SOLIDS: 96-HOUR LC_{50} VALUES FAR INVERTEBRATES

Organism	TDS-mg/l	
Cambarus	17,900	
Libellulida	14,800	
Coenargrionidae	14,800	
Hexagenia	10,500	
Tubificidae	10,100	Median 8,950
Hyalella	7,830	
Baetidae	1,410	
Diaptomus	6,590	
Physa	6,400	
Daphnia	3,710	

A review of the above indicated a median value of 11,200 mg/l for fish and 8,950 mg/l for invertebrates. Since the major salt was calcium chloride, these values are equivalent to 101 and 81 milliosmoles, respectively, as calcium chloride. These data, in addition to those given in Table 10, tend to support the general belief that the toxicity of TDS to freshwater aquatic life is that level which has an osmoconcentration equal to that of the body fluids of the organism. It should be noted, however, that the osmoconcentrations of body fluids may differ for various species, as well as for life cycle, age, nutrition, and acclimation. The 2,000 mg/l as observed by McCarthy and Thomas (8) in Nebraska, tends to support this concept. The value given in Table 12 for *Daphnia* is equivalent to 33.4 milliosmoles, somewhat lower than the 50 milliosmoles value recommended in the report of the National Technical Advisory Committee (12).

In studies on the discharge of wastewaters from a soda ash manufacturer, the bioassay laboratory of the Ohio Environmental Protection Agency found the 96-hr. LC_{50} for fathead minnows was approximately 8,800 mg/l, whereas that for *Daphnia* was approximately 4,500 mg/l. Based on current knowledge, the laboratory recommended a safety factor of 0.3 to 0.5 be applied to derive appropriate levels for full life cycle protection of warm-water aquatic life in an inland stream.

In view of the above and the relatively low levels of dissolved solids in the Great Lakes, it appears that there is no scientific basis for establishing an objective for TDS for the protection of aquatic life that is in any way as low as existing or projected TDS levels.

The existing low level of dissolved solids in the Great Lakes limits their buffering capacity. To preserve this capacity there should be no significant change in the general chemical composition of the TDS levels.

Historical Trends: Their Value and Implications

Many investigators have measured the level of TDS in the Great Lakes for nearly 100 years. These measurements included an analysis of the major cation and anion composition of the TDS. This information has been collated by Beeton and Chandler (1), Kramer (7) and updated by Weiler and Chawla (13). Kramer (7) showed that a significant portion of the present TDS levels in each of the lakes results from the natural chemical equilibria between the water and the sediments. Lake Superior is considerably lower in dissolved solids and alkalinity because it lies outside of, or along the edge of a Paleozoic carbonate belt and its sediments have a lower concentration of carbonates. In contrast, the other four lakes lie entirely within this belt.

A review of the data presented by Kramer (7) and Weiler and Chawla (13) demonstrates that the TDS levels have significantly increased for lakes Erie and 0ntario since 1900, whereas only a moderate increase has taken place for lakes Michigan and Huron. In contrast, a slight decrease in TDS levels has occurred in Lake Superior.

Chloride

Chloride ion concentrations in the Lower Lakes have increased from 8 mg/l in 1900 for both lakes to nearly 25 mg/l for Lake Erie, and about 28 mg/l for Lake Ontario in 1970. During the same period there was a minimal increase in the other three lakes, ranging from zero for Lake Superior to 4 mg/l for lakes Huron and Michigan.

Based on an average outflow of 196,000 cfs for Lake Erie, a 1 mg/l increase is equivalent to the addition of 1.06×10^6 lbs. per day. Thus the increase from 8 mg/l to 25 mg/l represents an addition of 18.1×10^6 lbs. per day or 9050 tons per day of chlorides as Cl. The daily addition of chlorides to Lake Erie from human wastes plus an indication of impact of this load on the total load was estimated for 1970 as follows:

Per capita contribution of Cl = 0.0154 lbs./day (9)
Chloride load 11,000,000 x 0.0154 = 169,400 lbs./day
Per cent of total load 169,400/18,100,000 x 100 = 0.92%

The use of salt for deicing and its impact on Lake Erie has been investigated by the Federal Water Pollution Control Administration (FWPCA)(6), the Three Rivers Watershed District (the Cleveland and Akron Area) in 1969-70, the State University of New York at Buffalo in 1972-73, as well as 0wenby and Kee (10).

The FWPCA study indicated that in 1966, 3.12×10^6 lbs./day was used each year for deicing in the Lake Erie basin. During the winter of 1969-70, the Three Rivers Watershed District estimated 1.05×10^6 lbs./day of salt as chlorides was used annually in an area of 2.5 million persons. In the Greater Buffalo area which has a population of nearly 500,000, approximately 15,000 tons of salt as chloride was used for deicing during the winters of 1971-72 and 1972-73. Thus, the per capita use of salt for deicing varied from 0.312 to 0.420 lbs./day with a weighted average of 0.336 lbs./day. Using this weighted average the estimated total chloride load to Lake Erie is 1.52×10^6 lbs./day on an annual (150 day) average.

Another significant source of chloride loadings to Lake Erie is an Ohio manufacturer of soda ash which discharges nearly 2,000 tons/day of chlorides in approximately equal portions of sodium and calcium chloride. Other manufacturers of soda ash along the Detroit River plus other discharges contribute nearly 3,450 tons/day (Table 13).

TABLE 13
CHLORIDE LOADINGS TO LAKE ERIE

Source	Tons/day%	Total
Upper Lakes	2120	23.4
Human wastes	9	0.9
Deicing	760	8.4
Industrial	5450	60.2
Others	711	7.8

These percentages compare favorably with those of Owenby and Kee (10).

Sulfates

Human wastes contribute nearly 2.6 grams of sulfates per capita per day (3). Based on the Lake Erie drainage basin population of 11×10^6 persons, the total load of sulfates from human wastes would amount to 28.6×10^6 g/day, 28.6×10^3 kg/day or 62,900 lbs/day. Such a load would cause a minor increase of 0.05 mg/l in the sulfate concentration in Lake Erie compared with the long term increase of 11 mg/l reported by Beeton and Chandler (1). Therefore a significant portion of this increase can be attributed to the result of the 12 mg/l increase observed by Beeton and Chandler (1) in Lake Michigan.

In summary it should be noted that chloride and sodium concentrations are increasing in the Great Lakes at the expense of alkalinity and calcium ion concentrations.

Recognizing that the proposed water quality objectives may be adopted as standards by the regulatory jurisdictions, the Committees recommended that the objective for TDS not be translated into a standard but be retained as an objective for use as a monitoring tool.

The Committees approached the design of defensible objectives for TDS in two ways:

1. Investigation of the influence of TDS on species composition of phytoplankton communities; and

2. Investigation of effects of individual components of TDS such as sulfate in eutrophication and chlorides and sodium in drinking water.

LITERATURE CITED

1. Beeton, A.M., and D.C. Chandler. "The St. Lawrence Great Lakes", in Limnology in North America, D.G. Frey, ed. Univ. of Wisconsin Press, Madison, 1963.

2. Clemens, H.P., and W.H. Jones. "Toxicity of brine water from oil wells". Trans. Amer. Fish.Soc. 84: 97-109 (1954).

3. Deeds and Data. Water Pollution Control Federation. Jan. 1975.

4. Hart, W.B., P. Doudoroff, and J. Greenbank. "Evaluation of toxicity of industrial wastes, chemicals and other substances to fresh-water fish". Water Control Laboratory, Atlantic Refining Co., Philadelphia, Pa., 1945.

5. International Lake Erie Water Pollution Board and the International Lake Ontario - St. Lawrence River Water Pollution Board. "Pollution of Lake Erie, Lake Ontario and the International Section of the St. Lawrence River; report to the International Joint Commission", V.I., "Summary". Washington, D.C. and Ottawa, 1969.

6. Lake Erie Enforcement Conference Technical Committee. Proceedings of the Lake Erie Enforcement Conference, 1966. Chicago, FWPCA, 1966.

7. Kramer, J.R. "Theoretical model for the chemical composition of fresh water with application to the Great Lakes". Pub. No. 11, Great Lakes Research Division, The University of Michigan Press, Ann Arbor, Mi., 1964.

8. McCarthy, D.B., and R. Thomas. "Some ecological observations of fathead minnows in the alkaline waters of Nebraska". Trans. Amer. Fish. Soc. 97(l): 52-52 (1968).

9. Meridith, D.D., R. R. Runner, C.C. Chien, and R.P. Apman. "Chlorides in the Lake Erie Basin". Water Resources and Environmental Engineering Research Report No. 74.I, State University of New York at Buffalo, 1974.

10. Owenby, C., and D. Kee. "Chlorides in Lake Erie", in Proc. 10th Conf. Great Lakes Res., Toronto, Ont., April 10-12, 1967: 382-389.

11. Prosser, C.L., and F.A. Brown. Comparative animal physiology. Saunders, Philadelphia, Pa., 1966.

12. U.S. National Technical Advisory Committee on Water Quality. "Water quality criteria; report to the Secretary of the Interior". Washington, 1968.

13. Weiler, R.R. and V.K. Chawla. "Dissolved mineral quality of the Great Lakes," in Proc. 12th Conf. Great Lakes Res., University of Michigan, Ann Arbor, May 5-7, 1969.

(B) NON-PERSISTENT TOXIC SUBSTANCES

(1) ORGANIC
(a) Pesticides

(i) General Objective

RECOMMENDATION

It is recommended that the following numerical objective for unspecified, non-persistent pesticides be adopted in compliance with Annex I, paragraph 7(a) of the Water Quality Agreement:

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

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

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

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

The preceding approach will significantly restrict the number of specific pesticides regulated within this category of substances, since the scientific data base for most of them is too inadequate to permit the establishment of defensible numerical objectives. For this reason, the use of the arbitrary safety factor of 0.05 times the 96-hour LC_{50} is employed. Objectives based on this latter procedure may be inadequate to protect aquatic life from a variety of deleterious sublethal effects or conversely, they may be unduly restrictive.

Such a procedural objective is intended only as a temporary measure and not as a substitute for the requisite testing necessary to establish scientifically defensible objectives. The presence of some of the organophosphorus pesticides has been investigated in the Upper Great Lakes (1) but none have been observed. While many of the compounds are not "persistent", they may survive long enough in localized areas to cause deleterious effects either at acute levels or through accumulation of biological effects. The usage/discharge patterns are unknown for most of these substances and, to date, no pressing problems have been noted, at least on a basin-wide basis. It is possible, however, to conceive of changing patterns such that localized exposure to these compounds might lead to undesirable levels. Such exposure could come about through direct application in spraying programmes and accidental spillage, via surface runoff or leaching, and with discharges in manufacturing operations. It is to protect against these eventualities that objectives are being formulated here.

LITERATURE CITED

1. Glooschenko, W.A., W.M.J. Strachan and R.C.J. Sampson. "Distribution of pesticides and PCB's in water, sediments and seston of the Upper Great Lakes". Pest. Monit. J. 10(2): 61-67 (1976).

(ii) Diazinon

RECOMMENDATION

It is recommended that the following numerical objective for diazinon be adopted in compliance with Annex I, paragraph 7(a) of the Water Quality Agreement:

> *The concentration of diazinon in an unfiltered water sample should not exceed 0.08 micrograms per litre for the protection of aquatic life.*

RATIONALE

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

Available data indicate that the persistence of diazinon in aquatic ecosystems is greatly influenced by pH. Cowart *et al.* (4) demonstrated that the half-life of diazinon in water at a pH of 6.0 was 14 days. Miller *et al.* (7) reported that 320 µg/l applied to a cranberry bog disappeared completely within 6 days. Gomaa *et* al. (5) 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 to 9.0 are normally encountered in Great Lakes waters, it is possible that diazinon can persist 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 (that is, 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 it 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 50% of tissue residue was lost in less than one week (7). Allison and Hermanutz (1) 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 concentration.

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 (11).

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 (2). The 48-hour LC_{50}'s for rainbow trout at 13°C and

bluegills *(Lepomis macrochirus)* at 24°C were 170 µg/l and 96 µg/l, respectively (3). Mean 96-hr 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 (1).

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

Available data indicate that aquatic invertebrates are much more acutely sensitive to diazinon than fish. The 48-hour EC50 (immobilization value at 15°C) for water fleas *(Simocephalus serrulatus* and *Daphnia pulex)* exposed to diazinon was 1.8 µg/l and 0.90 µg/l, respectively (10). Sanders (9) reported that the 96-hr. LC_{50} for *Gammarus lacustris* was 200 µg/l. The 48-hour LC_{50} for the stonefly *(Pteryonarcys californica)* ranged from 6 µg/l (12) to 7.5 µg/l (3). The 96-hr. LC_{50} of diazinon for *Acroneuria lycorias* was reported to be 1.7 µg/l (8).

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 14 (8).

TABLE 14
TOXICITY OF DIAZINON TO AQUATIC INVERTEBRATES

Organism	30-day LC_{50} (µg/l)	30-day no effect (µg/l)
Gammarus pseudolimnaeus	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 indicated that an objective less than 0.20 µg/l would protect invertebrates from exposure to concentrations which are directly lethal. The unpublished work of Allison and Hermanutz (1) showed that 0.55 dig/l of diazinon was 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 applying 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 lycorias* (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 indicate that this objective should protect sensitive species of fish and aquatic invertebrates.

LITERATURE CITED

1. Allison, D.T., and R.O. Hermanutz. "Chronic toxicity of Diazinon to brook trout and fathead minnows". (Manuscript). U.S. Environmental Protection Agency, Environmental Research Laboratory, Duluth, Minnesota, n.d.

2. Cope, O.B. "Sport fishery investigations", in "Effects of pesticides on fish and wildlife; 1964 research findings of the Fish and Wildlife Service". U.S. Fish and Wildlife Serv. Circ. 226, 1965.

3. Cope, O.B. "Contamination of the freshwater ecosystem by pesticides". J. Appl. Ecol. 3 (Supplement on pesticides in the environment and their effects on wildlife): 33-44 (1966).

4. Cowart, R.P., F.L. Bonner and E.A. Epps. "Rate of hydrolysis of seven organophosphate pesticides". Bull. Environ. Contam. Toxicol. 6: 231-234 (1971).

5. Gomaa, H.M., I.H. Suffet, and S.D. Faust. "Kinetics of hydrolysis of diazinon and diazoxon". Residue Reviews 29: 171-190 (1969).

6. Martin, H. Pesticide manual. 2d ed. British Crop Protection Council, 1971.

7. Miller, C.W., B.M. Zuckerman, and A.J. Charig. "Water translocation of diazinon-^{14}C and parathion-^{35}S off a model cranberry bog and subsequent occurrence in fish and mussels". Trans. Amer. Fish. Soc. 95: 345-349 (1966).

8. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

9. Sanders, H.O. "Toxicity of pesticides to the crustacean, *Gammarus lacustris".* U.S. Bur. Sport Fish. and Wildlife Tech. Paper 25, 1969.

10. Sanders, H.O., and 0.B. Cope. "Toxicities of several pesticides to two species of cladocerans". Trans. Amer. Fish. Soc. 95: 165-169 (1966).

11. Stadnyk, L., and R.S. Campbell. "Pesticide effect on growth and ^{14}C assimilation in a freshwater alga". Bull. Environ. Contam. Toxicol. 6: l-8 (1971).

12. U.S. National Advisory Committee on Water Quality. "Water quality criteria; report to the Secretary of the Interior". Washington, 1968.

(b) Other Compounds

(i) Oil and Petrochemicals

RECOMMENDATION

It is recommended that the following revised objective for oil and petrochemicals be adopted in compliance with Annex 1, paragraph 7(a), and to replace the existing interim objective in Annex 1, paragraph 2(e) of the Water Quality Agreement:

Oil or petrochemicals should not be present in concentrations that:

(a) can be detected as a visible film, sheen, or discolouration on the surface;
(b) can be detected by odour;
(c) can cause tainting of edible aquatic organisms;
(d) can form deposits on shorelines and bottom sediments that are detectable by sight or odour, or are deleterious to resident aquatic organisms.

EXISTING OBJECTIVE

The above objective is recommended to replace the existing interim objective in Annex 1, paragraph 2(e) of the Agreement, which states:

> "Oil, Petrochemicals and Immiscible Substances. Waters should be free from floating debris, oil, scum and other floating materials attributable to municipal, industrial or other discharges in amounts sufficient to be unsightly or deleterious".

RATIONALE

Amenities, Waterfowl and Health

On the basis of general knowledge alone, all four objectives are required to protect aesthetic values, water and shoreline recreation.

The amount of oil required to produce a visible slick will vary with type of oil and weather condition. 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) (12).

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

Available information on occupational health and industrial hygiene indicates that any tolerable health concentrations for petroleum-derived substances far exceed 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. 0ils of animal or vegetable origins are usually non-toxic to humans and aquatic life.

Aquatic 0rganisms

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 napthenes 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, napthenes, 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 more persistent (1).

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 (14) and Atlantic salmon at 1700 µl/l, (13). Crude oil slicks exceeding concentrations equivalent to 500 mg/l killed young coho and sockeye salmon in laboratory tests (11). Diesel oil killed shad at 167 µl/l (14). 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 (15). 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 was reported in a provisional report by the United States National Water Quality Laboratory (2). Used crankcase oil, probably a major source of oil in the Great Lakes, was used in their tests. Floating oil killed fathead minnows at 11,000 µl/l, but mixed into water it killed these fish at 1,600 µl/l, and flagfish *(Jordanella floridae)* at 1,000 µl/l. In chronic tests with flagfish 338 µl/l affected reproduction although 93 µl/l did not. These are all nominal or "added" concentrations. The ratio of proven "safe" level to the LC_{50}, = 93/1,000 = 0.093, may be used as an application factor which is potentially useful in other situations.

Toxicity to marine animals apparently has 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 (8,9) plus shrimp (10). The same concentration of No. 2 fuel oil killed kelp crab larvae (6), while 10 µl/l of "oil" killed a copepod in 4 days (7). 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 ultraviolet spectrophotometry were only 18% 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 (16).

Some sub-lethal effects have also been documented in marine animals. Crude oil at 100 µl/l caused inactivity and death over 2 weeks of *Neopanope* (5). 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 (16). The ratio of this "safe" concentration to the 4-day LC_{50} is= 0.18, 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 and moulting was slowed at 12.5 mg/l, thereby 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 (16).

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

Use of application factors does seem warranted however. The three application factors obtained for a marine crustacean are close to the one calculated for flagfish in fresh water. Applying the application factor 0.09 calculated for freshwater fish to the mentioned average lethal concentrations, the estimated "safe" levels for freshwater fish are 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.

Control

The eventual fate of oil in water depends on the basic processes of weathering, dispersion and degradation. The natural processes causing the disappearance of oil in water include evaporation, solution, formation of emulsions, and sinking; none of these processes however, render the oil harmless to the aquatic environment. The ultimate destruction of oil depends upon its oxidation by bacteria, although some photo-oxidation takes place.

Numerous corrective measures such as mechanical means and the use of detergents have been found to clean up spilled oil. Mechanical means have proven quite successful, but the use of detergents has in many instances produced considerably more toxicity to aquatic life than the oil proper. These toxicity effects are covered by the section on Unspecified Non-Persistent Toxic Substances and Complex Effluents.

The only effective measure for the control of oil pollution of water is prevention of all spills and releases. 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 Erie Water Pollution Board (3) estimated that the input of oil and grease to the Detroit and St. Clair rivers exceeded 1,100 barrels per day which is about 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 (4) reported that 29 million pounds of oil was discharged to the Upper Niagara River. This is about 13,000 metric tons per year, almost equivalent to one "Arrow" wreck. Furthermore, the Board estimated that 40% 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 (3). Again, that is almost 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.

LITERATURE CITED

1. Butler, M.J.A. and F. Berkes. "Biological aspects of oil pollution in the marine environment." Marine Science Center Manuscript Report No. 22, McGill University, Montreal, Quebec, 1972.

2. Hedtke. S.F., F.A. Puglisi, G.D. Veith, J.E. Poldoski, D. W. Kuehl and L. F. Mueller. "The effects of waste oil on freshwater aquatic life". (Draft report). U.S. Environmental Protection Agency, National Water Quality Laboratory, Duluth, Minnesota, 1974.

3. International Lake Erie Water Pollution Board and the International Lake Ontario - St. Lawrence River Water Pollution Board. "Pollution of Lake Erie, Lake Ontario and the International Section of the St. Lawrence River; report to the International Joint Commission", V. 2, "Lake Erie". Ottawa and Washington, 1969: 252.

4. International Joint Commission. "Pollution of Lake Erie, Lake Ontario, and the International Section of the St. Lawrence River". Information Canada, Ottawa 1971:28.

5. Katz, L.M. "The effects of water soluble fraction of crude oil on larvae of the decapod crustacean *Neopanope texana* (Sayi)". Environ. Pollut. 5: 199-204 (1973).

6. Lichatovich, J.A., J.A. Strand, W.L. Templeton. "Development of toxicity test procedures for marine zooplankton". American Inst. Chemical Engr. 64th Annual Meeting, Preprint 15E: 372-378 (1971).

7. Mironov, 0.G. "Hydrocarbon pollution of the sea and its influence on marine organisms". Helgoland. wiss. Meerseunters 17: 335-339 (1968).

8. Mironov, O.G. "The effect of oil pollution upon some representatives of the Black Sea zooplankton". Zool. Zh. 48: 980-984 (1969). (In Russian with English summary).

9. Mironov, O.G. "Viability of larvae of some crustacea in sea water polluted with oil products". Zool. Zh. 48: 1734-1736 (1969). (In Russian with English summary).

10. Mills, E.R., and D.D. Culley Jr. "Toxicity of various offshore crude oils and dispersants to marine and estuarine shrimps". (Manuscript in preparation. Personal communication to A. Renzoni, 1972).

11. Morrow, J.E. "Effects of crude oil and some of its components on young coho and sockeye salmon". U.S. Environmental Protection Agency Ecol. Res. Series 660/3-73-018. 1973.

12. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

13. Sprague, J.B. and W.G. Carson. "Toxicity tests with oil dispersants in connection with oil spills at Chadabucto Bay, Nova Scotia". Fish. Res. Board Canada Technical Report Series No. 201, 1970.

14. Tagatz, M.E. "Reduced oxygen tolerance and toxicity of petroleum products to juvenile American shad". Chesapeake Sci. 2: 65-71 (1961).

15. U.S. Environmental Protection Agency. "Proposed criteria for water quality". (Unpublished). Washington, D.C. 20460, 1974.

16. Wells, P.G. "Toxicity of Venezuelan crude oil to larvae of the American lobster". PhD. Thesis, University of Guelph. 1975.

(ii) Unspecified Non-Persistent Toxic Substances and Complex Effluents

RECOMMENDATION

It is recommended that the following numerical objective for unspecified non-persistent toxic substances and complex effluents be adopted in compliance with Annex I, paragraph 7(a), and as a refinement of Annex I, paragraphs 2(b) and (c) of the Water Quality Agreement:

> *Unspecified non-persistent toxic substances and complex effluents of municipal, industrial or other origin should not be present in concentrations which exceed 0.05 of the lethal concentration (96-hour LC_{50}) for any sensitive local species to protect aquatic life.*

RATIONALE

This procedural objective was developed to limit the effects of: (1) unspecified non-persistent substances toxic to aquatic life 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 used in industrial processes, agriculture and 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. In addition, analytical procedures necessary for their identification and quantification have not been developed and there has been insufficient testing to establish a specific water quality objective. 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 Tests with Fish, Nacroinvertebrates and Amphibians" (2); 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 existing 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 in freshwater toxicity tests and are representative of sensitive important Great Lakes species.

To ensure that aquatic life within the receiving waters are given adequate protection

from acute toxicity of these materials, bioassays are required to establish the toxicity of individual substances or mixtures. Also necessary is the use of an application factor which should, in the majority of cases, reduce the concentration to that which is non-lethal for chronic exposure. Using an application factor will not preclude the possibility of sublethal effects occurring; however, 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 there are such effects, it should be evident that the application factor was inadequate to derive an objective which would provide for and protect the designated use.

In "Water Quality Criteria 1972" (1) it is proposed that the test species used to establish an objective should ideally correspond to the most sensitive important species existing in the locality where the objective will apply. 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, a large number of organisms must be evaluated. 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 warm water fish species as well as an important benthic invertebrate.

The large volume of acute toxicity data available from the scientific literature should be used 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) the dilution water quality used in the test was comparable to that existing at the intended point of application of the objective (boundary of a mixing zone).

The choice of application factor is based on the recommendation put forward by the National Academy of Sciences and the National Academy of Engineering (l) for determining acceptable concentrations of toxicants for which comprehensive toxicological data are lacking. This recommendation for non-persistent and non-cumulative materials is a concentration 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, an application factor of 0.05 ($1/20^{th}$), apart from specialized cases, should provide adequate protection for the aquatic community. Notwithstanding this recommendation, it is strongly advised that where two or more unspecified toxicants are discharged simultaneously, the potential for synergistic or additive effects should be established through bioassay testing and the acceptable concentration should be based on $1/20^{th}$ of the net toxicity of the mixture.

LITERATURE CITED

1. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

2. U.S. Environmental Protection Agency. "Methods for acute toxicity tests with fish, macroinvertebrates and amphibians". Ecol. Res. Series 660/3-75-009. 1975.

(C) OTHER SUBSTANCES

(1) pH

RECOMMENDATION

It is recommended that the following revised objective for pH be adopted to replace the existing specific objective stipulated in Annex I, paragraph 1(e) of the Water Quality Agreement:

> *Values of pH should not exceed the range 6.5 to 9. 0, nor should discharges change the pH at the boundary of the designated mixing zone more* than *0.6 units* from *the ambient*

EXISTING OBJECTIVE

The above objective is recommended to replace the existing specific objective in Annex I, paragraph 1(e) in the Agreement, which states:

> "pH. Values should not he outside the range of 6.7 to 8.5."

RATIONALE

In natural waters, pH, which is a measure of hydrogen ion concentration, results from the equilibrium achieved by the various dissolved compounds, salts and gases. The primary system regulating pH in natural waters is the carbonate system; its role has been described in some detail by Stumm and Morgan (7).

Public Water Supplies

The pH of a raw water supply is significant because it may adversely affect water treatment process and contribute to corrosion of water works structures, distribution lines and household plumbing fixtures, by adding such constituents as iron, copper, lead, zinc and cadmium to the water (6). Adjustment of pH within the range of 5.0 to 9.0, the common range of pH values in natural waters, is relatively simple. "Water Quality Criteria 1972" (6) notes that since "the defined treatment process can cope with natural waters within the pH range of 5.0 to 9.0, but becomes less economical as this range is extended, it is recommended that the pH of public water supplies be within 5.0 to 9.0". The "defined treatment process" includes sedimentation, rapid sand filtration, and disinfection with chlorine.

Primary Contact Recreation

"Water Quality Criteria 1972" states that "for most bathing and swimming waters, eye irritation is minimized and recreational enjoyment enhanced by maintaining the pH within the range of 6.5 and 8.3 except for those waters with a low buffer capacity where a range of pH between 5.0 and 9.0 may be tolerated"(6). Subsequent investigations on the level of protection afforded recreational users of Great Lakes waters indicated that the characteristics of water in the Great Lakes are such that no eye irritation would be expected if the pH did not exceed 9.0

(5).

Fresh Water Aquatic Life

Based upon present evidence (3,6), a pH range of 6.5 to 9.0 will provide adequate protection for the life processes of freshwater fish and bottom dwelling invertebrates. Outside this range, most aquatic organisms suffer adverse physiological effects of increasing severity as the degree of deviation increases.

A pH fluctuation not only produces acid or alkaline conditions, it can also increase the toxicity of various components in the waters. Reductions in pH caused by the addition of acids can liberate dissolved CO_2 amounts which may be toxic (1). The acute toxicity of a metallocyanide complex increased by a factor of a thousand when pH values were reduced by approximately 1.5 units (2). Conversely, increases in pH can cause concentrations of unionized ammonia to increase to toxic levels. Unionized ammonia has been demonstrated to be ten times more toxic at pH 8.0 than at pH 7.0 (4).

Because of such effects, pH changes of more than 0.5 pH units should be avoided.

Aesthetic Consideration

The solubility of calcium carbonate in natural waters is influenced by levels of pH. Where levels of dissolved calcium carbonate exceed saturation, a pH approaching 9.0 may cause precipitation of this compound thereby creating a milky tinge.

However, as the recommended upper pH limit of 9.0 will protect all other designated uses of the water, the range of 6.5 to 9.0 has been endorsed, and the objective for suspended solids should be used to control local situations where precipitation of calcium carbonate may aesthetically degrade water quality.

LITERATURE CITED

1. Doudoroff, P., and M. Katz. "Critical review of literature on the toxicity of industrial wastes and their components to fish. 1. Alkalies, acids and organic gases". Sewage Industrial Wastes 22: 1432-58 (1950).

2. Doudoroff, P., G. Leduc, and C.R. Schneider. "Acute toxicity to fish of solutions containing complex metal cyanides, in relation to concentrations of molecular hydrocyanic acid." Trans. Amer. Fish. Soc. 95(I): 6-22 (1966).

3. European Inland Fisheries Advisory Commission. Working Party on Water Quality Criteria for European Freshwater Fish. "Report on extreme pH values and inland fisheries". Water Res. 3: 593 (1969).

4. European Inland Fisheries Advisory Commission. Working Party on Water Quality Criteria for European Freshwater Fish. "Report on ammonia and inland fisheries". Water. Res. 7: 1011 (1973).

5. Mood, E., Yale University, Personal Communication, 1975.
6. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

7. Stumm, W., and J.J. Morgan. Aquatic chemistry. John Wiley and Sons, New York, 1970.

(2) TAINTING SUBSTANCES

RECOMMENDATION

It is recommended that the following revised objective for tainting substances be adopted to replace the existing specific objective in Annex I, paragraph 1(d) of the Water Quality Agreement:

1) Raw public water supply sources should be essentially free from objectionable taste and odour for aesthetic reasons.

2) Levels of phenolic compounds should not exceed 0.001 milligrams per litre in raw public water supplies to protect against taste and odour in domestic water.

3) Substances entering the water as the result of human activity that cause tainting of edible aquatic organisms should not be present in concentrations which will lower the acceptability of these organisms as determined by organoleptic tests.

EXISTING OBJECTIVE

The above objectives are recommended to replace the existing specific objective in Annex I, paragraph 1(d) in the Agreement, which states:

> "Taste and Odour. Phenols and other objectionable taste and odour producing substances should be substantially absent."

RATIONALE

Raw Water Supply

Municipal wastewater discharges, especially those serving urban areas, and an array of industrial discharges have the capacity to impart objectionable taste and odour to water. There is also a number of naturally occurring materials in aquatic environments, as well as the metabolic by-products of micro-organisms that create taste and odour problems at water treatment plants. Taste and odour are primary factors which influence a consumer in determining acceptability of water for domestic use. Since these factors cannot be directly correlated with the safety of the water supply, protection from objectionable odour and taste is based on aesthetics.

The defined water treatment process is inconsistently effective in removing taste and odour producing properties of raw water, and data identifying threshold levels of individual materials are extremely variable. The objective therefore, is narrative rather than numerical, with the exception of the objective for phenolic compounds. For more specific information on taste and odour in raw public water supplies see references 1, 2, 15, 22 and 23.

Phenolic compounds are defined (24) as hydroxy derivatives of benzene and its

condensed nuclei. Local major sources of phenolic compounds to the water environment include municipal wastewaters and a variety of industrial wastes, especially those of oil refineries and chemical plants. Phenolic compounds may also occur naturally in aquatic ecosystems. Some phenolic compounds are sufficiently resistant to degradation to be transported long distances in the water environment.

Major problems associated with phenolic compounds are their taste and odour producing properties in water and edible aquatic animals. Threshold odour levels in water are often below 1 mg/l, e.g. 0.555 mg/l p-cresol, 0.25 mg/l m- cresol, and 0.26 mg/l o-cresol (16). Generally, phenolic compounds are not removed efficiently by the defined treatment process including chlorination. If phenolic compounds are present in waters chlorinated for disinfection prior to distribution to consumers, chlorophenols can be formed. The odour threshold concentrations for chlorophenols are generally much lower than for the unchlorinated phenols, for example, 4.2 mg/l phenol (16) and 0.002 mg/l 2-chlorophenol (6).

"Water Quality Criteria 1972" (13) states that the development of criteria for specific phenolic compounds is hampered by the lack of sensitive standard analytical techniques for their detection. However it is widely believed that protection against taste and odour due to phenolic compounds in water treated by the defined process requires that no more than 0.001 mg/l (1 µg/l) phenolic compounds be present. Variations of this recommendation incorporating the same number appear in California's "Water Quality Criteria" (12), the "European Standards for Drinking Water" (30), "Water Quality Criteria 1972" (13), and the United States "Public Health Service Drinking Water Standards" (28). The "Canadian Drinking Water Standards and 0bjectives" (7), established a limit of 2 µg/l in the raw water supply unless reduced to this limit by applied treatment.

Water quality criteria for toxic effects of phenol and phenolic compounds to aquatic life reveal that requirements for protecting water supplies are considerably more stringent. A review of these requirements has been given by the European Inland Fisheries Advisory Commission (9).

Protection of Aquatic Life

When edible portions of aquatic life or wildlife are tainted to a degree which lowers the desirability and acceptability of the organism for use as determined by organoleptic (sensory) tests, this represents loss of a resource. Commercial and recreational harvest and their associated economic roles have been negatively affected by tainting (26).

Many of the same compounds and wastes which cause objectionable taste and odour in domestic water supplies can be taken up by aquatic organisms and detected by the consumer. In addition, the appearance, colour and consistency of an organism or its edible portions can become less acceptable through exposure to a variety of contaminants and conditions. Such tainting can occur in waters with concentrations of the offending material lower than those recognized as being harmful to the organism. "Water Quality Criteria 1972" (13) reviewed the subject in detail and summarized the literature on concentrations of wastewaters found to have lowered the palatability of fish flesh and concentrations of chemical compounds in water that can produce identifiable taste in fish flesh. (Tables 15 and 16).

Table 15

WASTEWATERS FOUND TO HAVE LOWERED THE PALATABILITY OF FISH FLESH (13)

Wastewaters	Concentration in water affecting palatability of fish	Species	Reference
2,4-D mfg. plant	50 to 100 ml/l	Trout	
Coal-coking	0.02 to 0.1 ml	Freshwater fish	(3)
Coaltar	0.1 ml/l	Freshwater fish	(3)
Kraft process (untreated)	1 to 2% by vol.	Salmon	
Kraft process (treated)	9 to 12% by vol.	Salmon	
Kraft and neutral sulfite process	----------	Trout	(14)
Municipal dump runoff	----------	Channel catfish	(27)
Municipal untreated sewage (2 locations)	----------	Channel catfish	(27)
Municipal wastewater treatment plants (4 locations)	----------	Channel catfish	(27)
Municipal wastewater treatment plant (Primary)	11 to 13% by vol.	Freshwater fish	
Municipal wastewater treatment plant (Secondary)	20 to 26% by vol.	Freshwater fish	(21)
Oily wastes	----------	Trout	(31)
Refinery	----------	Trout	(10)
Sewage containing phenols	0.1 ml/l	Freshwater fish	(3)
Slaughterhouses (2 locations)	----------	Channel catfish	(27)

Table 16

CONCENTRATIONS OF CHEMICAL COMPOUNDS IN WATER FOUND TO CAUSE TAINTING OF THE FLESH OF FISH

(Modified from "Water Quality Criteria 1972")

Chemical	Estimated threshold level in water (mg/l)	Reference
acetophenone	0.5	11*
acrylonitrile	18	20
cresol	0.07	20
m-cresol	0.2	20
o-cresol	0.4	20
p-cresol	0.12	20
cresylic acid (meta para)	0.2	11
N-butylmercaptan	0.06	20
o-sec. butylphenol	0.3	11
p-tert. butylphenol	0.03	11
p-chlorophenol	0.0001 to 0.015	4,11,17
p-chlorophenol	0.01 to 0.05	11,20,17
2,3-dichlorophenol	0.084	20
2,4-dichlorophenol	0.001 to 0.014	11,18,20
2,5-dichlorophenol	0.023	20
2,6-dichlorophenol	0.035	20
2-methyl, 4 chlorophenol	0.075	20
2-methyl, 6 chlorophenol	0.003	20
o-phenylphenol	1	11
2,4,6-trichlorophenol	0.003 to 0.05	20
phenol	1 to 10	11,17
phenols in polluted river	0.02 to 0.15	3
diphenyloxide	0.05	11
B,B-dichlorodiethyl ether	0.09 to 1.0	11,20
o-dichlorobenzene	0.10	11
ethylbenzene	0.25	11
ethanethiol	0.24	20
ethylacrylate	0.6	20
formaldehyde	95	20
gasoline	0.005	5
kerosene	0.1	11
kerosene plus kaolin	1	29
isopropylbenzene	0.25	11
naphtha	0.1	11
napthalene	1	3
naphthol	0.5	3
2-naphthol	0.3	20
dimethylamine	7	20
a-methylstyrene	0.25	11

Table 16 continued

Chemical	Estimated threshold level in water (mg/l)	Reference
oil, emulsifiable	15	11
pyridine	5 to 28	3,20
pyrocatechol	0.8 to 5	3,20
pyrogaliol	20 to 30	3
quinoline	0.5 to 1	3
p-quinone	0.5	3
styrene	0.25	11
toluene	0.25	11
outboard motor fuel, as exhaust	2.6 gal/acre-foot	8,25
guaiacol	0.082	20

* Fetterolf published the results of A.W. Winston, Jr. of the Dow Chemical Company. The data are also available in an undated mimeographed release of the company.

These numbers should not be used as specific objectives since chemical analytical techniques were not described in many of the reports, many of the sensory tests were conducted in uncontrolled environments, and the nature of the tests was often subjective. There is great variability in the threshold of sensory detection. In many cases the purposes of the tests were not to define a threshold level or nondetection level, but simply to provide information to a discharger on potential or existing problems associated with operating the facility. The numbers should be used as guidelines for identifying possible sources when tainting problems are investigated.

LITERATURE CITED

1. American Water Works Association. Water quality and treatment. 3d ed. McGraw-Hill, New York, 1971.

2. American Water Works Association. Committee on Tastes and Odors. "Committee report: research on tastes and odors". J. Amer. Water Works Assoc. 62(l): 59-62 (1970).

3. Bandt, H.J. "Fischereischaden durch phenolobwasser." Wasserwitrisch Wassertech. 5(9): 290-294 (1955).

4. Boetius, J. "Foul taste of fish and oysters caused by chlorophenol". Medd. Dan. Fisk. Havunders. 1(4): 1-8.

5. Boyle, H.W. "Taste/odor contamination of fish from the Ohio River". U.S. Federal Water Pollution Control Administration, Cincinnati, Ohio, 1967.

6. Burtschell, R.H., A.A. Rosen, F.M. Middleton, and M.B. Ettinger. "Chlorine derivatives of phenol causing taste and odor". J. Amer. Water Works Assoc. 51(2): 205-214 (1959).

7. "Canadian drinking water standards and objectives 1968". Prepared by the Joint Committee on Drinking Water Standards of the Advisory Committee on Public Health Engineering and the Canadian Public Health Association. Department of National Health and Welfare, Ottawa, 1969.

8. English, J.N., G.N. McDermott, and C. Henderson. "Pollution effects of outboard motor exhaust--laboratory studies." J. Water Poll. Cont. Fed. 35(7): 923931 (1963).

9. European Inland Fisheries Advisory Commission. Working Party on Water Quality Criteria for European Freshwater Fish. "Report on mono-hydric phenols and inland fisheries". Water Res. 7: 929 (1973).

10. Fetterolf, C.M. "Investigation of fish off flavor, Muskegon lake." Bureau of Water Management, Michigan Department of Natural Resources, Lansing, Mi., 1962.

11. Fetterolf, C.M. "Taste and odor problems in fish from Michigan waters" in Proc. Purdue

Indust. Waste Conf. 115: 174-182 (1964).

12. McKee, J.E. and H.W. Wolf. "Water quality criteria". 2d ed. State Water Quality Control Board, Pub. No. 3-A, Sacramento, Ca., 1963.

13. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-033. 1973.

14. Newton, M.E. "Fish tainting tests, Manistee Lake." Bureau of Water Management, Michigan Dept. of Natural Resources, Lansing, Mi., 1967.

15. Rosen, A. A. "Taste and odors: joint discussion. Recent developments in sensory testing." J. Amer. Water Works Assoc. 58(6): 699-702 (1966).

16. Rosen, A.A., J.B. Peter, and F.M. Middleton. "Odor thresholds of mixed organic chemicals." J. Water Poll. Cont. Fed. 34(1): 714 (1962).

17. Schulze, E. "The influence of phenol-containing effluents on the taste of fish." Int. Rev. Gesamten Hydrobiol. 4(1): 84-90 (1961).

18. Shumway, D.L. "Effects of effluents on flavor of salmon flesh." Agricultural Experiment Station, Oregon State University, Corvallis, Oregon, 1966.

19. Shumway, D.L. and G.G. Chadwick. "Influence of kraft mill effluent on the flavor of salmon flesh." Water Res. 5(11): 997-1003 (1971).

20. Shumway, D.L. and J.R. Palensky. "Impairment of the flavor of fish by water pollutants." Environmental Protection Agency Ecol. Res. Series R3-73-010. 1973.

21. Shumway, D.L. and J.R. Palensky, Oregon State University, Dept. of Fisheries and Wildlife, Corvallis, Oregon. Unpublished data. 1971.

22. Silvey, J.K.G. "Newer concepts of tastes and odors in surface water supplies. Water and Sewage Works 100(11): 426-429 (1953).

23 Silvey, J.K.G., J.C. Russel, D.R. Redden and W.C. McCormick. "Actinomycetes and common tastes and odors." J. Amer. Water Works Assoc. 42(11): 10181026 (1950).

24. Standard methods for the examination of water and wastewater. 13th ed. Prepared by the American Public Health Assoc., American Water Works Assoc., and Water Pollution Control Fed. Washington, D.C., 1971.

25. Surber, E.W., J.N. English, and G.N. McDermott. "Tainting of fish by outboard motor exhaust wastes as related to gas and oil consumption," in "Biological problems in water pollution," C.M. Tarzwell, ed. U.S. Public Health Service Pub. 999-WP-25, 1956.

26. Thomas, N.A. "Assessment of fish flesh tainting substances," in Biological methods for the assessment of water quality, J. Cairns and K. Dickson ed. American Soc. for Testing and Materials Special Tech. Pub. 528. Philadelphia, Pa., 1973.

27. Thomas, N.A. and D.B. Hicks. "Effects of waste water discharges on the flavor of fishes in the Missouri River (Sioux City, Iowa to Waverly, Missouri)," in "Everyone can't live upstream." United States Environmental Protection Agency, Office of Water Programs, Kansas City, Mo., 1971.

28. U.S. Department of Health, Education and Welfare. Public Health Service. "Public Health Service drinking water standards". Rev. ed. PHS Pub. 956, 1962.

29. Westman, J.R. and J.G. Hoff. "Flavor studies of Raritan Bay fish". Interstate Sanitation Commission, 10 Columbus Circle, New York, 1963.

30. World Health Organization. "European standards for drinking water." Geneva, Switzerland, 1970.

31. Zillich, J.A. "A secondary fish taint test, Muskegon Lake, with special emphasis on fish and water near Continental Motors Corporation." Bureau of Water Management, Michigan Department of Natural Resources, Lansing, MI., 1969.

PHYSICAL CHARACTERISTICS

(A) SETTLEABLE AND SUSPENDED SOLIDS AND LIGHT TRANSMISSION

RECOMMENDATION

It is recommended that the following revised objective for settleable and suspended solids and light transmission be adopted to replace the existing interim objective in Annex I, paragraph 2(d) of the Water Quality Agreement:

> *For the protection of aquatic life, waters should be free from substances attributable to municipal, industrial or other discharges resulting from human activity that will settle to form putrescent or otherwise objectionable sludge deposits or that will alter the value of the Secchi disk depth by more than 10 per cent.*

EXISTING OBJECTIVE

The above objective is recommended to replace the existing interim objective specified in Annex 1, paragraph 2(d) of the Agreement, which states:

> "Settleable and Suspended Materials. Waters should be free from substances attributable to municipal, industrial or other discharges that will settle to form putrescent or otherwise objectionable sludge deposits, or that will adversely affect aquatic life or waterfowl".

RATIONALE

Materials present in a lake absorb, scatter, and reflect light as it passes through the water (11). 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, colour, refractive index, and specific gravity.

The turbidity of the water can have a 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 (1,11,14). This has been clearly demonstrated in the Great Lakes by Chandler's work in western Lake Erie. Chandler showed that the algal productivity is high when turbidity is low and vice versa (2,6). His studies indicated that the composition, size, duration and emergence of phytoplankton pulses in this area are influenced by turbidity (3,4,5,6). Since 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 of turbidity may be entirely natural. Such mechanisms as wave-induced shoreline erosion and resuspension of bottom sediments, and the bloom of algal cells under favourable 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 creating a large and usually unfavourable 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. Not only can large blooms of algae lead to taste and odour problems in public water supplies but they can also make 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.

The National Academy of Sciences and the National Academy of Engineering recognized this need for all aquatic environments in the United States in their recommendations on water quality criteria (13):"The combined effect of colour and turbidity should not change the compensation point more than 10 per cent from its seasonally established norm, nor should such a change place more than 10 per cent of the biomass of photosynthetic organisms below the compensation point." 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 photo-synthesis 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 based upon light extinction as measured by Secchi disk, an easy and problem-free procedure, is recommended. Furthermore, it is generally accepted that the Secchi disk measurement bears an approximately constant relation to the lower limit at which the necessary light to carry on photosynthesis is available (e.g., 10).

The value of 10 per cent 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 a minor effect on photosynthesis. Thus the 10 per cent 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 its "Quality Criteria for Water" adopted the recommendation contained in "Water Quality Criteria 1972". The complete recommendation includes:

"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" (13).

The rationale presented below was taken primarily from the Environmental Protection Agency report.

"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 follows sedimentation to the bottom of the water body. Noted effects are similar for both fresh and marine waters.

[In a 1965 report, the European Inland Fisheries Advisory Commission (EIFAC) identified four ways suspended solids affect fish and fish food populations:]

(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; ..." (18).

While indicating that a "no-effect" level does not exist for inert suspended solids above which fisheries are not damaged, the EIFAC made the following conclusions assuming inert solids and otherwise satisfactory water quality:

"(a) there is no evidence that concentrations of suspended solids less than 25 mg/l have any harmful effects on fisheries;

(b) it should usually be possible to maintain good or moderate fisheries in waters that normally contain 25 to 80 mg/l suspended solids, other factors being equal, however, the yield of fish from such waters might be somewhat lower than from those in the preceding category;

(c) waters normally containing from 80 to 400 mg/l suspended solids are unlikely to support good freshwater fisheries, although fisheries may sometimes be found at the lower concentrations within this range;

(d) only poor fisheries are likely to be found in waters that normally contain more than 400 mg/l suspended solids" (13).

Available evidence indicates however, that the death rate for fish living in water containing 200 mg/l 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 (for example, coal washings and pulp wastes) may be substantially more toxic (8).

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

Settleable materials which blanket the bottom of waterbodies damage the invertebrate populations, block gravel spawning beds, and if organic, remove dissolved oxygen from overlying waters (7,8). In a study downstream from the discharge of a rock quarry where inert suspended solids were increased by 80 mg/l, the density of macroinvertebrate populations decreased by 60 per cent, regardless of the suspended solid concentrations (9). 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 thereby reducing organism density to only 7.3 per square foot *vs.* 25.5 per square foot upstream (17).

When settleable solids block gravel spawning beds containing eggs, high mortalities result although there is evidence that some species of salmonids will not spawn in such areas (8). It has been postulated that silt attached to eggs prevents sufficient exchange of oxygen and carbon dioxide between the eggs and the overlying water. The important variables are particle size, stream velocity and degree of turbulence (8).

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 as pollution sensitive forms disappear (12). Algae likewise flourish in such nutrient rich areas although forms may become less desirable (16).

Identifiable effects of suspended solids on irrigation use of water include the formation of crusts on top of the soil which inhibit 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 irrigation reservoir capacity, delivery canals and other distribution equipment (13).

LITERATURE CITED

1. Brylinsky, M. and K.H. Mann. "An analysis of factors governing productivity in lakes and reservoirs." Limnol. Oceanog. 18: 1-14 (1973).

2. Chandler, D.C. "Limnological studies of western Lake Erie. I. Plankton and certain physical-chemical data of the Bass Islands Region, from September, 1939, to November, 1939". Ohio J. Sci. 40: 291-336 (1940).

3. Chandler, D.C. "Limnological studies of western Lake Erie. II. Light penetration and its relation to turbidity". Ecology 23: 41-52 (1942).

4. Chandler, D.C. "Limnological studies of western Lake Erie. III. Phytoplankton and physical-chemical data from November, 1939, to November, 1940." Ohio J. Sci. 42: 24-44 (1942).

5. Chandler, D.C. "Limnological studies of western Lake Erie. IV. Relation of limnological and chemical factors to the phytoplankton of 1941." Trans. Amer. Microsc. Soc. 63: 203-236 (1944).

6. Chandler, D.C. and 0.B. Weeks. "Limnological studies of western Lake Erie V. Relation of limnological and meteorological conditions to the production of phytoplankton in 1942." Ecol. Monogr. 15: 435-456 (1945).

7. Edberg, N. and Hofstan, B.V. "Oxygen uptake of bottom sediment studied *in-situ* and in the laboratory." Water Res. 7: 1285 (1973).

8. European Inland Fisheries Advisory Commission. Working Party on Water Quality Criteria for European Freshwater Fish: "Report on finely divided solids and inland fisheries." Air Water Pollut. 9: 151 (1965).

9. Gammon, J.R. "The effect of inorganic sediment on stream biota". U.S. Environmental Protection Agency Water Poll. Cont. Res. Series 18050 DWC 12/70. 1970.

10. Holmes, R.W. "The Secchi disk in turbid coastal waters." Limnol. Oceanogr. 15: 688-694 (1970).

11. Hutchinson, G.E. A treatise on limnology, Vol. I., Geography, physics and chemistry. John Wiley and Sons, New York, 1957.

12. Mackenthun, K.M. "Toward a cleaner aquatic environment". U.S. Govt. Print. Office, Washington, D.C., 1973.

13. National Academy of Sciences and National Academy of Engineering. "Water quality criteria 1972". U.S. Environmental Protection Agency Ecol. Res. Series R3-73-003. 1973.

14. Pechlaner, R. "The phytoplankton spring outburst and its conditions in Lake Erken (Sweden)." Limnol. Oceanogr. 15: 113-130 (1970).

15. Standard methods for the examination of water and wastewater.13th ed. Prepared by the American Public Health Assoc., American Water Works Assoc. and Water Pollution Control Fed. Washington, D.C., 1971.

16. Tarzwell, C.M. and A. R. Gaufin. "Some important biological effects of pollution often disregarded in stream surveys," in Proc. 8th Purdue Indust. Waste Conf. Reprinted in "Biology of water pollution," U.S. Dept. of Interior, Washington, D.C., 1957.

17. Tebo, L.B. Jr. "Effects of siltation, resulting from improper logging, on the bottom fauna of a small trout stream in the southern Appalachians." Prog. Fish-Cult. 17:64. Reprinted in "Biology of water pollution", U.S. Dept. 'of Interior, Washington, D.C., 1957.

18. U.S. Environmental Protection Agency. "Quality criteria for water". (Draft report). Ecol. Res. Series 440/9-76-023. 1976.

19. U.S. Department of Health, Education and Welfare. Public Health Service. "Public Health Service drinking water standards". Rev. ed. PHS Pub. 956. 1962.

(B) ASBESTOS

RECOMMENDATION

It is recommended that the following objective for asbestos be adopted:

Asbestos should be kept at the lowest practicable levels and in any event should be controlled to the extent necessary to prevent harmful effects on health.

RATIONALE

At this time there is insufficient information to recommend a meaningful or defensible numerical asbestiform fibre objective for protection of aquatic organisms, raw public water supply or drinking water.

Asbestos is a generic name for several fibrous silicates. The reported background level of asbestiform fibre concentration in the Great Lakes varies from less than one million to ten million fibres per litre. Sources of asbestiform fibres are natural erosion, mining and processing operations, and man's use of the manufactured products. That inhaled asbestos is related to an increased incidence of cancer is reasonably well known, but the effects of ingested asbestiform fibres have only recently come under study and the demonstrable hazard to health is not defined. A major Lake Superior source is the Reserve Mining Company operation at Silver Bay, Minnesota. Eighty-seven million and two hundred fifty million fibres per litre have been reported from Lake Superior water in the Beaver Bay and Duluth, Minnesota areas, respectively. These elevated levels have focused attention on the desirability of defining threshold effect and safe levels for various water uses. The reader is referred to the Great Lakes Research Advisory Board report on asbestos in the Great Lakes Basin (l), the source of the above text, for more detailed information.

Research on health effects of ingested asbestiform fibres is ongoing through animal feeding studies conducted under the aegis of the United States Environmental Protection Agency and the United States Food and Drug Administration.

Very little is known about the effects of asbestiform fibres on aquatic organisms. Concern has been expressed over possible effects from (1) ingestion by fish which need water for osmoregulation; (2) inadvertent ingestion by particulate feeders; (3) decrease in buoyancy of planktonic organisms caused by accumulations on their surfaces; and (4) decreased gill-function efficiency.

Filter feeders, such as the fingernail clams, appear especially vulnerable to direct ingestion of asbestiform particles. Halsband (2) investigated the short term effects of asbestos intake on the mussel *(Mytilus edulis),* a marine mollusk filter feeder. He exposed mussels to the fine fraction of tailings from a process which separates asbestos fibres from ore whose source is the Ungava Peninsula of Canada on the northeastern shore of Hudson Bay. Mussels were exposed for 5 days in extremely high concentrations (10 to 100 mg/l). Some were removed after exposure and prepared for tissue examination while others were placed in "unpolluted" water for 7 days to provide an opportunity for purging before tissue examination.

Examination showed clearly that asbestos fibres penetrated the epithelial tissue of the stomach and intestinal tract of mussels in the variations of exposure and post-exposure. Apparently some mussels were allowed longer purging periods since, as Halsband states, "After several weeks exposure ... to unpolluted seawater these foreign bodies were not disposed of." He concluded that tissue damage had occurred, but offered no evaluation of effects.

Scientists at the Canada Centre for Inland Waters do not know of any experimental work in Canada dealing with effects of asbestos on aquatic organisms. The National Water Quality Laboratory at Duluth has begun a $644,000 project extending to 1979 on the "Environmental impact of asbestos on freshwater organisms." This project will determine the extent to which environmental contamination has occurred in the United States by extensively surveying all existing data; employ tracer, autoradiographic and other refined techniques to identify target organs and tissues in important fish species; develop better methods to determine asbestos content in water and tissue; develop response data relating effects of freshwater organisms exposed to asbestos fibres; and determine the extent to which previous water quality criteria data were affected through contamination of the laboratory's water supply by asbestiform minerals.

Examination of the research plan suggests that the emphasis will be on fish and larger invertebrate animals. Effects on planktonic organisms may be of equal or greater importance and might be more easily detected. Tests with these smaller organisms should be conducted.

LITERATURE CITED

1. Great Lakes Research Advisory Board. "Asbestos in the Great Lakes Basin with emphasis on Lake Superior; report to the International Joint Commission". International Joint Commission Great Lakes Regional Office, Windsor, Ont., 1975.

2. Halsband, E. "The effect of asbestos waste products on mussels *(Mytilus edulis);* report to the Fisheries Improvement Committee of the International Council for the Exploration of the Sea." Institut für Kusten-und Binnenfischerei der Bundesforschungsanstalt für Fischerei, Hamburg, Germany, 1974.

Chapter IV

BASIC CONCEPTS

(A) NON-DEGRADATION

RECOMMENDATION

It is recommended that the following revised statement regarding non- degradation be adopted to replace the existing statement which appears in Annex I, paragraph 3 of the Water Quality Agreement:

> Notwithstanding the adoption of specific water quality objectives, all reasonable and practicable measures shall be taken in accordance with paragraph 4 of Article III of the Agreement to maintain the levels of water quality existing at the date of entry into force of the Agreement in those areas of the boundary waters of the Great Lakes System where such *water quality is better than that prescribed by the specific water quality objectives.*

EXISTING OBJECTIVE

The above statement is recommended to replace the existing non-degradation statement in Annex I, paragraph 3 of the Agreement, which states:

> "Non-degradation. Notwithstanding the adoption of specific water quality objectives, all reasonable and practicable measures shall be taken in accordance with paragraph 4 of Article III of the Agreement to maintain the levels of water quality existing at the date of entry into force of the Agreement in those areas of the boundary waters of the Great Lakes System where such levels exceed the specific water quality objectives".

COMMENTARY

The existing statement is drawn from Article III, paragraph 4 of the Agreement and it should also be amended as above.

RATIONALE

A water quality objective as defined by the Committees is "that minimum quality of water which will provide for and protect any designated use". A primary purpose for establishing water quality objectives is to upgrade and enhance water quality that is already deteriorated from the objective. In many areas however, depending on the assigned use, the existing level of water quality is significantly better than the objective stated for that use. It became equally important then, that in order to maintain existing water uses, the water quality not be degraded beyond present levels.*

In the first sentence of the 1972 Amendments to the Federal Water Pollution Control Act, the United States Congress stated that "the objective of this Act is to restore and maintain the chemical, physical and biological integrity of the Nation's waters" (emphasis added). Similarly, "Guidelines and Criteria for Water Quality Management in Ontario" states, in part, that "water of a higher quality than that required by the standard will be maintained at that high quality..."(1). These statements clearly indicate the importance placed on preserving existing water quality, and are the basis on which non-degradation policies must be established for the boundary waters of the Great Lakes System.

The Water Quality Agreement presents a statement of non-degradation in Article III and again in Annex I. There are provisions in this statement which can result in technical misinterpretation of non-degradation, and water quality degradation of even the most sensitive areas of the Great Lakes.

Technical misinterpretations of the stated non-degradation policy centre upon the word "exceed". The statement indicates that water quality must be maintained at existing levels where such levels "exceed" the specific water quality objectives. It is generally understood that the drafters of the Agreement intended that water quality should be maintained at existing levels when those levels are within the limits of the stated objective. In a majority of cases, water quality objectives are concentrations of contaminants above which water uses will be restricted. In other cases, the objective is the lowest concentration which should be maintained to assure the stated use. Therefore, a strict interpretation of the word "exceed" could mean that if a particular parameter concentration was greater than the objective, maintenance at that level would be appropriate when actually the water quality objective was being violated. Maintaining water quality which is "better" than the stated objective more properly states the intent of the non-degradation policy.

Under current policies, degradation of the water quality of the boundary waters of Great Lakes system can occur provided that "all reasonable and practicable measures" are taken to maintain that quality. The implied intent of the Agreement is not to allow water quality degradation except in special circumstances and under very tight restrictions. That such deterioration should be allowed under warranted conditions may be necessary to preserve a resource of higher value.

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* Conformance with a non-degradation policy assumes a knowledge of baseline water quality. Other committees and study groups within the International Joint Commission are assigned the task of ascertaining existing levels of water quality. A non-degradation policy will necessarily be based upon agreement on the results.

The Natural Resources Defense Council recognized that fact in 1973 when recommending a policy on non-degradation to the United States Environmental Protection Agency for its adoption. That policy states in part:

> "After public hearings, all water segments in the state should be divided into two categories. Category I would be segments which should be kept in their present condition because they constitute an outstanding natural resource, for example, rivers in parks and other waters of great recreational or ecological significance. No degradation in these segments would be allowed.
>
> Category II would be all other segments. Water quality here would be allowed to degrade by a small predetermined percentage. The percentage would vary depending on the water quality parameter but in no case would the percentage be large enough to allow the waters to degrade significantly. Moreover, if existing water quality meets the 1983 interim standard expressed in Sections 101 (s) (2) and 302 of the Federal Water Pollution Control Act, the quality should in no case fall below that standard. The water quality required by 1983 is that which provides for the protection and propagation of fish, shellfish, and wildlife and which provides for recreation in and on the water."

Within the boundary waters of the Great Lakes System there are areas which have special significance because of their natural resources. These areas must receive the maximum amount of protection that current technology and legislation can provide. A non-degradation policy should reflect this protectionist philosophy for those unique areas of the Great Lakes that deserve this special attention. In order to assure that these areas are recognized, an effort must be made to designate areas of "outstanding natural resource value." The intent is not to prohibit all use, development, or discharge into such areas, but rather to assure that water quality is at least maintained at existing levels. The recommended revision provides management with a basis for maintaining or upgrading the existing water quality.

LITERATURE CITED

1. Ontario Ministry of the Environment. "Guidelines and criteria for water quality management in Ontario". Toronto, Ont., 1974.

(B) MIXING ZONES

RECOMMENDATION

It is recommended that the following statements regarding mixing zones be adopted to replace the existing statement which appears in Annex I, paragraph 5 of the Water Quality Agreement*:

> The responsible regulatory agencies may designate restricted mixing zones in the vicinity of outfalls within which the specific water quality objectives shall not apply. Mixing zones shall not be considered a substitute for adequate treatment or control of discharges at their source.
>
> *A mixing zone is an area, contiguous to a point source, where exceptions to water quality objectives and conditions otherwise applicable to the receiving waterbody may be granted. Thus, a mixing zone represents a loss in value.*

It is not prudent to provide blanket exemption from all water quality objectives within mixing zones. Therefore, exemption should be at the discretion of the regulatory authority on the basis of local conditions.

Because specific water quality objectives define minimum conditions to provide for and protect a use and because exemption to objectives may be granted within mixing zones, it is apparent that certain values are lost. There is a gradation of loss of values from greatest at the end of the pipe to least at the periphery. Mixing zones may increase recreation potential or production of desirable organisms in some instances and losses may occur only seasonnally. However, in allocation of loss of biological value it may be assumed that areas within mixing zones represent a potential total loss of the most sensitive value identified as being affected adversely.

The following guidelines should be used in the designation of mixing zones:

1. *Specific water quality objectives and conditions applicable to the receiving waterbody should be met at the boundary of mixing zones.*

It is important to recognize that this concept allows the plume of the effluent to be identifiable outside the mixing zone. It does not limit the extent of the plume, only that portion of the plume that is not required to meet water quality objectives.

2. *The size, shape and exact location of a mixing zone should be specified so that both the discharger and the regulatory agency know the bounds. The size should be minimized to the greatest possible degree.*

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* Where applicable, a rationale follows immediately after each new statement.

3. *Limitations on mixing zones should be established by the responsible regulatory agency on a case-by-case basis, where "case" refers to both* local *considerations and the waterbody as a whole, or segment of the waterbody.*

Guidelines cannot be substituted for knowledge of local areas or common sense but they can assist in identifying critical factors on which to base decisions. Mixing zones should be tailored to the characteristics of receiving systems, recognizing not only the local effect, but the cumulative effect of all mixing zones on the waterbody or segment thereof.

4. *Existing biological, chemical, physical and hydrological conditions should be known when considering location of a new mixing zone or limitations on an existing one.*

5. *Areas of extraordinary value should be designated off-limits for mixing zones.*

6. *When designing conditions to protect specific organisms it is necessary to know that the organisms would* normally *inhabit the area within the mixing zone. Zones of passage should be assured either by location or design of conditions within mixing zones. Mixing zones should not form a barrier to migratory routes of aquatic species or interfere with biological communities or populations of important species to a degree which is damaging to the ecosystem, or diminish other beneficial uses disproportionately.*

To prevent blocks to passage, less than half the stream width should be used as a mixing zone. Since dischargers may wish to use the other half in the future, good practice suggests limiting individual mixing zones to one-third of the width.

7. *No conditions within the mixing zone should be permitted which are either (a) rapidly* lethal *to important aquatic life (conditions which result in sudden fish* kills *and mortality of organisms passing through the mixing zone); or (b) which cause irreversible responses which could result in detrimental post-exposure effects; or (c) which result in bioconcentration of toxic materials which are harmful to the organism or its consumers.*

Rapid changes in water quality cause stress in aquatic life through shock effect, thus changes should be guarded against in the operational regime. Rapid dilution in mixing zones is desirable so that weak swimmers, such as planktonic organisms entrained in the plume at the discharge, will be exposed to the higher concentrations of constituents for short periods only.

8. *Concentrations of toxic materials at any point in the mixing zone where important species are physically capable of residing should not exceed the 24-to 96-hour* LC_{50}.

The mixing zone should be considered as a region in which organism response to water quality characteristics is time-dependent. Therefore, if organisms are exposed for short periods only, a greater concentration can be considered. Conversely, when it is known, or can be demonstrated, that the discharge is attracting and holding organisms for long periods, a reduction of concentrations of toxic materials to below the 96-hour LC_{50} should be considered.

9. Many of the general water quality objectives should apply to discharge-related materials within mixing zones. The zones should be free of:

(a) objectionable deposits;

(b) unsightly or deleterious amounts of flotsam, debris, oil, scum and other floating matter;

(c) substances producing objectionable colour, odour, taste or turbidity; and

(d) substances and conditions or combinations thereof at levels which produce aquatic life in nuisance quantities that interfere with other uses.

Objections of people to a point source discharge are often related to the impact on their aesthetic sensitivities. Aesthetically acceptable mixing zones create goodwill among the discharger, the public and the regulatory agency.

10. *Mixing zones may overlap unless the combined effects exceed the conditions set forth in other guidelines.*

11. *Municipal and other water supply intakes and recreational areas should not be in mixing zones as a general condition, but local knowledge of the effluent characteristics and the type of discharge associated with the zone could allow such a mixture of uses.*

EXISTING OBJECTIVE

The above statements are recommended to replace the existing mixing zone statement in Annex I, paragraph 5 of the Agreement, which states:

> "Mixing Zones. The responsible regulatory agencies may designate restricted mixing zones in the vicinity of outfalls within which the specific water quality objectives shall not apply. Mixing zones shall not be considered a substitute for adequate treatment or control of discharges at their source".

Appendix A

MEMBERSHIP LISTS

GREAT LAKES WATER QUALITY BOARD

AND

GREAT LAKES RESEARCH ADVISORY BOARD

AND

THEIR COMMITTEES

MEMBERSHIP

GREAT LAKES WATER QUALITY BOARD

These are the individuals who served on the Great Lakes Water Quality Board while the recommended objectives were being developed.

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MEMBERSHIP

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These are the individuals who served on the Standing Committee on Scientific Basis for Water Quality Criteria while the recommended objectives were being developed.

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