PB-263 943

QUALITY CRITERIA FOR WATER

U.S. ENVIRONMENTAL PROTECTION AGENCY
Washington, D.C. 20460

NOTICE

THIS DOCUMENT HAS BEEN REPRODUCED FROM THE BEST COPY FURNISHED US BY THE SPONSORING AGENCY. ALTHOUGH IT IS RECOGNIZED THAT CERTAIN PORTIONS ARE ILLEGIBLE, IT IS BEING RELEASED IN THE INTEREST OF MAKING AVAILABLE AS MUCH INFORMATION AS POSSIBLE.

CONTENTS

CONTENTS CONT'D

CONTENTS CONT'D

TABLES

FIGURES

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

26 JUL 1976

OFFICE OF WATER AND
HAZARDOUS MATERIALS

To the Reader:

Thousands of fine scientists throughout the country have contributed directly or indirectly to this publication on "Quality Criteria for Water." This volume represents a stock-taking effort on the part of this Agency to identify as precisely as possible at this time, on a national scale, the various water constituents that combine to form the concept of "Quality Criteria for Water". This process of definition will continue far into the future because research related to water quality is a never-ending evolutionary process, and the water environment is so complex that man's efforts to define it will never attain finite precision.

Water quality criteria do not have direct regulatory use, but they form the basis for judgment in several Environmental Protection Agency and State programs that are associated with water quality considerations. The criteria presented in this publication should not be used as absolute values for water quality. As it is stated in the chapter on "The Philosophy of Quality Criteria" there is variability in the natural quality of water and certain organisms become adapted to that quality, which may be considered extreme in other areas. These criteria represent scientific judgments based upon literature and research about the concentration-effect relationship to a particular water quality constituent upon a particular aquatic species within the limits of experimental investigation. They should be used with considered judgment and with an understanding of their development. The judgment associated with their use should include the natural quality of water under consideration, the kinds of organisms that it contains, the association of those species to the particular species described in this volume upon which criteria values have been placed, and the local hydrologic conditions.

It must be emphasized that national criteria can never be developed to meet the individual needs of each of the Nation's waterways--the natural variability within the aquatic ecosystem can never

be identified with a single numerical value. Water quality criteria will change in the future as our knowledge and perception of the intricacies of water improve. There is no question but that criteria for some constituents will change within a period of only two years based upon research now in progress. That is a mark of continuing progressive research effort, as well as a mark of a better understanding by man of the environment that he inhabits.

This, then, is the challenge for the future: to expand upon our present baseline of knowledge of the cause-effect relationships of water constituents to aquatic life and of the antagonistic and synergistic reactions among many quality constituents in water; and to mold such future knowledge into realistic, environmentally protective criteria to insure that the water resource can fulfill society's needs.

Eckardt C. Beck
Deputy Assistant Administrator
for Water Planning and Standards

FOREWORD

The Federal Water Pollution Control Act Amendments of 1972 require the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. Proposed Water Quality Criteria were developed and a notice of their availability was published on October 26, 1973 (38 FR 29646). This present volume represents a revision of the proposed water quality criteria based upon a consideration of comments received from other Federal agencies, State agencies, special interest groups, and individual scientists.

This volume, Quality Criteria for Water, addresses the effects of those basic water constituents and pollutants that are considered most significant in the aquatic environment in the context of our present knowledge and experience. The format for criteria presentation has been altered substantially from the proposed volume. It is believed that the alphabetical arrangement of the water quality constituents and the form in which the information is arranged will be of considerable help to the reader in using this volume. For each basic water constituent or pollutant there is a recommended criterion, an introduction, a rationale supporting the recommended criterion and a list of the references cited in the development of the recommendation.

The thrust of this volume is to recommend criteria levels for a water quality that will provide for the protection and propagation of fish and other aquatic life and for recreation in and on the water in accord with the 1983 goals of P.L. 92-500. Criteria also are presented for the domestic water supply use. Generally, these water uses are the highest achievable beneficial uses and water quality that supports these uses will also be suitable for agricultural and industrial uses. In those few exceptions, criteria are presented to provide a safe water quality for agricultural use, or water quality conditions associated with agricultural and industrial uses are discussed in the rationale supporting a criterion recommendation.

Guidelines to implement the consideration of criteria presented in this volume in the development of water quality standards, and in other water-related programs of this Agency, are being developed and will be available for use by the States, other agencies, and interested parties in the near future.

Russell E. Train
Administrator

PREFACE

The genesis of water quality criteria in the United States began in the early 1900's. Marsh[1/], in 1907, published on the effects of industrial wastes on fish. Shelford[2/], in 1917, published effect data on fish for a large number of gas-waste constituents. In this early publication he reiterated that the toxicity of waste differs for different species of fish and generally is greater for the smaller and younger fish. Powers[3/], working with Shelford, experimented with the goldfish as a test animal for aquatic toxicity studies.

A monumental early effort to describe and record the effects of various concentrations of a great number of substances on aquatic life was that of Ellis in 1937. Ellis[4/] reviewed the existing literature for 114 substances in a 72-page document and listed lethal concentrations found by the various authors. He provided a rationale for the use of standard test animals in aquatic bioassay procedures and he used the common goldfish, Crassius auratus and the entomostracan, Daphnia magna, as test species in which experiments were made in constant temperature cabinets.

Early efforts to summarize knowledge concerning water quality criteria

---

1/ M.C. MARSH, The effect of some industrial wastes on fishes. Water supply and irrigation paper No. 192, U.S. Geol. Sur., pp. 337-348 (1907).

2/ V.E. SHELFORD, An experimental study of the effects of gas wastes upon fishes, with especial reference to stream pollution. Bull. Illinois State Lab. for Nat. History, 11:381-412 (1917).

3/ E.B. POWERS, The goldfish (Carassius carassius) as a test animal in the study of toxicity. Illinois Biol. Mono., 4:127-193 (1917).

4/ M.M. ELLIS, Detection and measurement of stream pollution. Bull. U.S. Bureau of Fisheries, 48:365-437 (1937).

took the form of a listing of the concentration, the test organism, the results of the test within a time period, and the reference for a cause-effect relationship for a particular water contaminant. In early bioassay efforts insufficient attention was given to the quality of the dilution water used for the experiment and to the effects of such dilution water on the relative toxicity of the tested contaminant. As a result, conclusions from citations of such references were, at best, difficult to formulate and most often were left to the discretion of the reader.

In 1952, the State of California[5/] published a 512-page book on "Water Quality Criteria" that contained 1,369 references. This classic reference summarized water quality criteria promulgated by State and interstate agencies, as well as the legal application of such criteria. Eight major beneficial uses of water were described. Three-hundred pages of the document were devoted to cause-effect relationships for major water pollutants. The concentration-effect levels for the pollutant in question were discussed for each of the designed water uses.

The State of California's 1952 Water Quality Criteria edition was expanded and tremendously enhanced into a second edition edited by Messrs. Jack E. McKee and Harold W. Wolf and published in 1963 by the Resources Agency of California, State Water Quality Control Board[6/]. This edition, which included 3,827 cited references, was a monumental effort in bringing together under one cover the world's literature on water quality criteria as of the date of publication. Criteria were identified and referenced for a host of water quality

---

5/ Water Quality Criteria. State Water Pollution Control Board, Sacramento, California.

6/ J.E. McKEE and H.W. WOLF, Water Quality Criteria, State Water Quality Control Board, Sacramento, California, Pub. 3-A (1963)

characteristics according to their effects on domestic water supplies, industrial water supplies, irrigation waters, fish and other aquatic life, shellfish culture, and swimming and other recreational uses. Specific values were arranged in ascending order, with appropriate references, as they had been reported damaging to fish or as not harmful to fish in the indicated time and under the conditions of exposure. The results of such a tabulation presented a range of values and, as would be expected by those investigating such conditions, there was often an overlap in values between those concentrations that had been reported by others as harmful. Such an anomaly is due to differences in investigative techniques among investigators, the characteristics of the water used as a dilutent for the toxicant, the physiological state of the test organisms, and variations in the temperature under which the tests were conducted. Nevertheless, the tabulation of criteria values for each of the water quality constituents has been helpful through time to predict a range within which a water quality constituent would have a deleterious effect upon the receiving waterway.

In 1966 the Secretary of the Interior appointed a number of nationally recognized scientists to a National Technical Advisory Committee to develop water quality criteria for five specified uses of water: domestic water supply, recreation, fish and wildlife, agricultural, and industrial. In 1968 the report was published[7/]. This report constituted the most comprehensive documentation to date on water quality requirements for particular and defined water uses. The book was intended to be used as a basic reference by personnel in State water pollution control agencies engaged in water quality studies and water quality standards setting activities. In some respects, this volume represented

---

7/ Water Quality Criteria, A Report of the National Technical Advisory Committee to the Secretary of the Interior. U.S. Government Printing Office, Washington, D.C. (1968).

a marriage between the best available experimental or investigative criteria recorded in the literature and the judgments of recognized water quality experts with long experience in associated management practices. Its publication heralded a change in the concepts of water quality criteria from one that listed a series of concentration-effect levels to another that recommended concentrations that would ensure the protection of the quality of the aquatic environment and the continuation of the designated water use. When a specific aquatic life recommendation for a particular water pollutant could not be made because of either a lack of information or conflicting information, a recommendation was made to substitute a designated application factor based upon data obtained from a 96-hour bioassay using a sensitive aquatic organism and the receiving water as a diluent for the toxicity test.

The U.S. Environmental Protection Agency contracted with the National Academy of Sciences and the National Academy of Engineering to embellish the concept of the 1966 National Technical Advisory Committee's Water Quality Criteria and to develop a water quality criteria document that would include current knowledge. The result was a 1974 publication that presented water quality criteria as of 1972[8/].

The Federal Water Pollution Control Act Amendments of 1972 (P.L. 92-500) mandated that the Environmental Protection Agency publish water quality criteria accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water.

8/ Water Quality Criteria, 1972. National Academy of Sciences, National Academy of Engineering. U.S. Government Printing Office, Washington, D.C. (1974).

Section 304(a) of P.L. 92-500 states, "(1) The Administrator, after consultation with appropriate Federal and State agencies and other interested persons, shall develop and publish, within one year after October 18, 1972 (and from time to time thereafter revise) criteria for water quality accurately reflecting the latest scientific knowledge (A) on the kind and extent of all identifiable effects on health and welfare including, but not limited to, plankton, fish, shellfish, wildlife, plant life, shorelines, beaches, esthetics, and recreation which may be expected from the presence of pollutants in any body of water, including ground water; (B) on the concentration and dispersal of pollutants, or their byproducts, through biological physical, and chemical processes; and (C) on the effects of pollutants on biological community diversity, productivity, and stability, including information on the factors affecting rates of eutrophication and rates of organic and inorganic sedimentation for varying types of receiving waters.

"(2) The Administrator, after consultation with appropriate Federal and State agencies and other interested persons, shall develop and publish, within one year after October 18, 1972 (and from time to time thereafter revise) information (A) on the factors necessary to restore and maintain the chemical, physical, and biological integrity of all navigable waters, ground waters, waters of the contiguous zone, and the oceans; (B) on the factors necessary for the protection and propagation of shellfish, fish, and wildlife for classes and categories of receiving waters and to allow recreational activities in an on the water; and (C) on the measurement and classification of water quality; and (D) for the purpose of Section 303 of this title, on and the identification of pollutants suitable for maximum daily load measurement correlated with the achievement of water quality objectives.

"(3) Such criteria and information and revisions thereof shall be issued to the States and shall be published in the Federal Register and otherwise made available to the public."

Section 101(a)(2) of P.L. 92-500 states, "It is the national goal that wherever attainable, an interim goal of water quality which provides for the protection and propagation of fish, shellfish, and wildlife, and provides for recreation in and on the water, will be achieved by July 1, 1983."

The objectives of this volume are to respond to these sections of the Act and thus establish water quality criteria. The QCW will be expanded periodically in the future to include additional constituents as data become available. While the NAS/NAE 1972 Water Quality Criteria considered aluminum, antimony, bromine, cobalt, fluoride, lithium, molybdenum, thallium, uranium and vanadium, these presently are not included in this volume; however, they should be given consideration in the development of State Water Quality Standards and quality criteria may be developed for them in future volumes of the QCW. In particular geographical areas or for specific water uses such as the irrigation of certain crops, some of these constituents may have harmful effects. Until such time that criteria for the 10 aforementioned constituents are developed, information relating to their effects on the aquatic ecosystem may be found in the NAS/NAE 1972 Water Quality Criteria.

PREPARATION OF THIS VOLUME

A volume of this scope results from the efforts of many dedicated people and includes technical specialists located throughout the Agency's operational programs and in its research laboratories. The responsibility for coordinating compilation efforts and in preparing manuscript was assigned to the Criteria Branch of the Criteria and Standards Division within the Office of Water Planning and Standards, EPA.

THE PHILOSOPHY OF QUALITY CRITERIA

Water quality criteria specify concentrations of water constituents which, if not exceeded, are expected to support an aquatic ecosystem suitable for the higher uses of water. Such criteria are derived from scientific facts obtained from experimental or *in situ* observations that depict organism responses to a defined stimulus or material under identifiable or regulated environmental conditions for a specified time period.

Water quality criteria are not intended to offer the same degree of safety for survival and propagation at all times to all organisms within a given ecosystem. They are intended not only to protect essential and significant life in water, as well as the direct users of water, but also to protect life that is dependent on life in water for its existence, or that may consume intentionally or unintentionally any edible portion of such life.

The criteria levels for domestic water supply incorporate available data for human health protection. Such values are different from the criteria levels necessary for protection of aquatic life. The Agency's interim primary drinking water regulations (40 *Federal Register* 59566 December 24, 1975), as required by the Safe Drinking Water Act (42 U.S.C. 300f, *et seq.*), incorporate applicable domestic water supply criteria. Where pollutants are identified in both the quality criteria for domestic water supply and the Drinking Water Standards, the concentration levels are identical. Water treatment may not significantly affect the removal of certain pollutants.

What is essential and significant life in water? Do Daphnia or stonefly nymphs qualify as such life? Why does 1/100th of a concentration that is lethal to 50 percent of the test organisms (LC50) constitute a criterion in some instances, whereas 1/20 or 1/10th of some effect levels constitute a criterion in other instances? These are questions that often are asked of those who undertake the task of criteria formulation.

The universe of organisms composing life in water is great in both kinds and numbers. As in the human population, physiological variability exists among individuals of the same species in response to a given stimulus. A much greater response variation exists among species of aquatic organisms. Thus, aquatic organisms do not exhibit the same degree of harm, individually or by species, from a given concentration of a toxicant or potential toxicant within the environment. In establishing a level or concentration of a quality constituent as a criterion it is necessary to ensure a reasonable degree of safety for those more sensitive species that are important to the functioning of the aquatic ecosystem even though data on the response of such species to the quality constituent under consideration may not be available. The aquatic food web is an intricate relationship of predator and prey organisms. A water constituent that may in some way destroy or eliminate an important segment of that food web would, in all likelihood, destroy or seriously impair other organisms associated with it.

Although experimentation relating to the effects of particular substances under controlled conditions began in the early 1900's, the effects of any substance on more than a few of the vast number of aquatic organisms have not

been investigated. Certain test animals have been selected by investigators for intensive investigation because of their importance to man, because of their availability to the researcher and because of their physiological responses to the laboratory environment. As general indicators of organism responses such test organisms are representative of the expected results for other associated organisms. In this context Daphnia or stoneflies or other associated organisms indicate the general levels of toxicity to be expected among untested species. In addition, test organisms are themselves vital links within the food web that results in the fish population in a particular waterway.

The ideal data base for criteria development would consist of information on a large percentage of aquatic species and would show the community response to a range of concentrations for a tested constituent during a long time period. This information is not available but investigators are beginning to derive such information for a few water constituents. Where only 96-hour bioassay data are available, judgmental prudence dictates that a substantial safety factor be employed to protect all life stages of the test organism in waters of varying quality, as well as to protect associated organisms within the aquatic environment that have not been tested and that may be more sensitive to the test constituent. Application factors have been used to provide the degree of protection required. Safe levels for certain chlorinated hydrocarbons and certain heavy metals were estimated by applying an 0.01 application factor to the 96 hour LC50 value for sensitive aquatic organisms. Flow-through bioassays have been conducted for some test indicator organisms over a substantial period of their life history. In a few other cases, information is available for the organism's natural life or for more than one generation of the species. Such data may indicate a minimal effect level, as well as a no-effect level.

The word "criterion" should not be used interchangeably with, or as a synonym for, the word "standard." The word "criterion" represents a constituent concentration or level associated with a degree of environmental effect upon which scientific judgment may be based. As it is currently associated with the water environment it has come to mean a designated concentration of a constituent that when not exceeded, will protect an organism, an organism community, or a prescribed water use or quality with an adequate degree of safety. A criterion, in some cases, may be a narrative statement instead of a constituent concentration. On the other hand a standard connotes a legal entity for a particular reach of waterway or for an effluent. A water quality standard may use a water quality criterion as a basis for regulation or enforcement, but the standard may differ from a criterion because of prevailing local natural conditions, such as naturally occurring organic acids, or because of the importance of a particular waterway, economic considerations, or the degree of safety to a particular ecosystem that may be desired.

Toxicity to aquatic life generally is expressed in terms of acute (short-term) or chronic (long-term) effects. Acute toxicity refers to effects occurring in a short time period; often death is the end point. Acute toxicity can be expressed as the lethal concentration for a stated percentage of organisms tested, or the reciprocal, which is the tolerance limit of a percentage of surviving organisms. Acute toxicity for aquatic organisms generally has been expressed for 24- to 96-hour exposures.

Chronic toxicity refers to effects through an extended time period. Chronic toxicity may be expressed in terms of an observation period equal to the lifetime of an organism or to the time span of more than one generation. Some chronic effects may be reversible, but most are not.

Chronic effects often occur in the species population rather than in the individual. If eggs fail to develop or the sperm does not remain viable, the species would be eliminated from an ecosystem because of reproductive failure. Physiological stress may make a species less competitive with others and may result in a gradual population decline or absence from an area. The elimination of a microcrustacean that serves as a vital food during the larval period of a fish's life could result ultimately in the elimination of the fish from an area. The phenomenon of bioaccumulation of certain materials may result in chronic toxicity to the ultimate consumer in a food chain. Thus, fish may mobilize lethal toxicants from their fatty tissues during periods of physiological stress. Egg shells of predatory birds may be weakened to a point of destruction in the nest. Bird chick embryos may have increased mortality rates. There may be a hazard to the health of man if aquatic organisms with toxic residues are consumed.

The fact that living systems, i.e. individuals, populations, species and ecosystems can take up, accumulate, and bioconcentrate man-made and natural toxicants is well documented. In aquatic systems biota are exposed directly to pollutant toxicants through submersion in a relatively efficient solvent (water) and are exposed indirectly through food webs and other biological, chemical, and physical interactions. Initial toxicant levels, if not immediately toxic and damaging, may accumulate in the biota or sediment over time and increase to levels that are lethal or sublethally damaging to aquatic organisms or to consumers of these organisms. Water quality criteria reflect a knowledge of the capacity for environmental accumulation, persistence, and effects of specific toxicants in specific aquatic systems.

Ions of toxic materials frequently cause adverse effects because they pass through the semipermeable membranes of an organism. Molecular diffusion through membranes may occur for some compounds such as pesticides, polychlorinated biphenyls and other toxicants. Some materials may not pass through membranes in their natural or waste-discharged state, but in water they may be converted to states that have increased ability to affect organisms. For example, certain microorganisms can methylate mercury thus producing a material that more readily enters physiological systems. Some materials may have multiple effects; for example an iron salt may not be toxic, an iron floc or gel may be an irritant or clog fish gills to effect asphyxiation, iron at low concentrations can be a trace nutrient but at high concentrations it can be a toxicant. Materials also can affect organisms if their metabolic byproducts cannot be excreted. Unless otherwise stated, criteria are based on the total concentration of the substance because an ecosystem can produce chemical, physical and biological changes that may be detrimental to organisms living in or using the water.

In prescribing water quality criteria certain fundamental principles dominate the reasoning process. In establishing a level or concentration as a criterion for a given constituent it was assumed that other factors within the aquatic environment are acceptable to maintain the integrity of the water. Interrelationships and interactions among organisms and their environment, as well as the interrelationships of sediments and the constituents they contain to the water above, are recognized as fact.

Antagonistic and synergistic reactions among many quality constituents in water also are recognized as fact. The precise definition of such reactions and their relative effects on particular segments of aquatic life have not been identified with scientific precision. Historically, much of the data to support criteria development was of an ambient concentration-organism response nature. Recently, data are becoming

available on long-term chronic effects on particular species. Studies now determine carcinogenic, teratogenic, and other insidious effects of toxic materials.

Some unpolluted waters in the nation may exceed designated criteria for particular constituents. There is variability in the natural quality of water and certain organisms become adapted to that quality which may be considered extreme in other areas. Likewise, it is recognized that a single criterion cannot identify minimal quality for the protection of the integrity of water for every aquatic ecosystem in the nation. To provide an adequate degree of safety to protect against long-term effects may result in a criterion that cannot be detected with present analytical tools. In some cases, a mass balance calculation can provide a means of assurance that the integrity of of the waterway is not being degraded.

Water quality criteria do not have direct regulatory impact, but they form the basis for judgment in several Environmental Protection Agency programs that are derived from water quality considerations. For example, water quality standards developed by the States under Section 303 of the Act and approved by EPA are to be based on the water quality criteria, appropriately modified to take account of local conditions. The local conditions to be considered include actual and projected uses of the water, natural background levels of particular constituents, the presence or absence of sensitive important species, characteristics of the local biological community, temperature and weather, flow characteristics, and synergistic or antagonistic effects of combinations of pollutants.

Similarly, by providing a judgment on desirable levels of ambient water quality, water quality criteria are the starting point in deriving toxic pollutant effluent standards pursuant to Section 307(a) of the Act. Other EPA programs that make use of water quality criteria include drinking water standards, the ocean dumping program, designation of hazardous substances, dredge spoil criteria development, removal of in-place toxic materials, thermal pollution, and pesticide registration.

To provide the water resource protection for which they are designed, quality criteria should apply virtually to all of the nation's navigable waters with modifications for local conditions as needed. To violate quality criteria for any substantial length of time or in any substantial portion of a waterway may result in an adverse effect on aquatic life and perhaps a hazard to man or other consumers of aquatic life.

Quality criteria have been designed to provide long-term protection. Thus, they may provide a basis for effluent standards, but it is not intended that criteria values become effluent standards. It is recognized that certain substances may be applied to the aquatic environment with the concurrence of a governmental agency for the precise purpose of controlling or managing a portion of the aquatic ecosystem; aquatic herbicides and aquatic piscicides are examples of such substances. For such occurrences, criteria obviously do not apply. It is recognized further that pesticides applied according to official label instructions to agricultural and forest lands may be washed to a receiving waterway by a torrential rainstorm. Under such conditions it is believed that such diffuse source inflows should receive consideration

similar to that of a discrete effluent discharge and that in such instances the criteria should be applied to the principal portion of the waterway rather than to that peripheral portion receiving the diffuse inflow.

The format for presenting water quality criteria includes a concise statement of the dominant criterion or criteria for a particular constituent followed by a narrative introduction, a rationale that includes justification for the designated criterion or criteria, and a listing of the references cited within the rationale. An effort has been made to restrict supporting data to those which have either been published or are in press awaiting publication. A particular constituent may have more than one criterion to ensure more than one water use or condition, i.e., hard or soft water where applicable, suitability as a drinking water supply source, protection of human health when edible portions of selected biota are consumed, provision for recreational bathing or water skiing, and permitting an appropriate factor of safety to ensure protection for essential warm or cold water associated biota.

Criteria are presented for those substances that may occur in water where data indicate the potential for harm to aquatic life, or to water users, or to the consumers of the water or of the aquatic life. Presented criteria do not represent an all-inclusive list of constituent contaminants. Omissions from criteria should not be construed to mean that an omitted quality constituent is either unimportant or non-hazardous.

AESTHETIC QUALITIES

CRITERIA:

All waters free from substances attributable to wastewater or other discharges that:

(1) settle to form objectionable deposits;

(2) float as debris, scum, oil, or other matter to form nuisances;

(3) produce objectionable color, odor, taste, or turbidity;

(4) injure or are toxic or produce adverse physiological responses in humans, animals or plants; and,

(5) produce undesirable or nuisance aquatic life.

RATIONALE

Aesthetic qualities of water address the general principles laid down in common law. They embody the beauty and quality of water and their concepts may vary within the minds of individuals encountering the waterway. A rationale for these qualities cannot be developed with quantifying definitions; however, decisions concerning such quality factors can portray the best in the public interest.

Aesthetic qualities provide the general rules to protect water against environmental insults; they provide minimal freedom requirements from pollution; they are essential properties to protect the nation's waterways.

ALKALINITY

CRITERION:

20 mg/l or more as $CaCO_3$ for freshwater aquatic life except where natural concentrations are less.

INTRODUCTION:

Alkalinity is the sum total of components in the water that tend to elevate the pH of the water above a value of about 4.5 It is measured by titration with standardized acid to a pH value of about 4.5 and it is expressed commonly as milligrams per liter of calcium carbonate. Alkalinity, therefore, is a measure of the buffering capacity of the water, and since pH has a direct effect on organisms as well as an indirect effect on the toxicity of certain other pollutants in the water, the buffering capacity is important to water quality. Examples of commonly occurring materials in natural waters that increase the alkalinity are carbonates, bicarbonates, phosphates and hydroxides.

RATIONALE:

The alkalinity of water used for municipal water supplies is important because it affects the amounts of chemicals that need to be added to accomplish coagulation, softening and control of corrosion in distribution systems. The alkalinity of water assists in the neutralization of excess acid produced during the addition of such materials as aluminum sulfate during chemical coagulation. Waters having sufficient alkalinity do not have to be supplemented with artificially added materials to increase the alkalinity. Alkalinity resulting from naturally occurring materials such as carbonate and bicarbonate is not considered a health hazard in drinking water supplies, per se, and naturally occurring

maximum levels up to approximately 400 mg/l as calcium carbonate are not considered a problem to human health (NAS, 1974).

Alkalinity is important for fish and other aquatic life in freshwater systems because it buffers pH changes that occur naturally as a result of photosynthetic activity of the chlorophyll-bearing vegetation. Components of alkalinity such as carbonate and bicarbonate will complex some toxic heavy metals and reduce their toxicity markedly. For these reasons, the National Technical Advisory Committee (NTAC, 1968) recommended a minimum alkalinity of 20 mg/l and the subsequent NAS Report (1974) recommended that natural alkalinity not be reduced by more than 25 percent but did not place an absolute minimal value for it. The use of the 25 percent reduction avoids the problem of establishing standards on waters where natural alkalinity is at or below 20 mg/l. For such waters, alkalinity should not be further reduced.

The NAS Report recommends that adequate amounts of alkalinity be maintained to buffer the pH within tolerable limits for marine waters. It has been noted as a correlation that productive waterfowl habitats are above 25 mg/l with higher alkalinities resulting in better waterfowl habitats (NTAC, 1968).

Excessive alkalinity can cause problems for swimmers by altering the pH of the lacrimal fluid around the eye, causing irritation.

For industrial water supplies high alkalinity can be damaging to industries involved in food production, especially those in which acidity accounts for flavor and stability, such as the carbonated beverages. In other instances, alkalinity is desirable because water with a high alkalinity is much less corrosive.

A brief summary of maximum alkalinities accepted as a source of raw water by industry is included in Table 1. The concentrations listed in the table are for water prior to treatment and thus are only desirable ranges and not critical ranges for industrial use.

The effect of alkalinity in water used for irrigation may be important in some instances because it may indirectly increase the relative proportion of sodium in soil water. As an example, when bicarbonate concentrations are high, calcium and magnesium ions that are in solution precipitate as carbonates in the soil water as the water becomes more concentrated through evaporation and transpiration. As the calcium and magnesium ions decrease in concentration, the percentage of sodium increases and results in soil and plant damage. Alkalinity may also lead to chlorosis in plants because it causes the iron to precipitate as a hydroxide (NAS, 1974). Hydroxyl ions react with available iron in the soil water and make the iron unavailable to plants. Such deficiencies induce chlorosis and further plant damage. Usually alkalinity must exceed 600 mg/l before such affects are noticed, however.

TABLE I *

MAXIMUM ALKALINITY IN WATERS USED AS A SOURCE OF SUPPLY PRIOR TO TREATMENT

Industry	Alkalinity mg/l as $CaCO_3$
Steam Generation Boiler Makeup.....	350
Steam Generation Cooling.....	500
Textile Mill Products.....	50-200
Paper and Allied Products.....	75-150
Chemical and Allied Products.....	500
Petroleum Refining.....	500
Primary Metals Industries.....	200
Food Canning Industries.....	300
Bottled and Canned Soft Drinks.....	85

* NAS, 1974

REFERENCES CITED

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972, U.S. Government Printing Office, Washington, D. C.

National Technical Advisory Committee to the Secretary of the Interior, 1968. Water quality criteria, U.S. Government Printing Office, Washington, D. C.

AMMONIA

CRITERION:

0.02 mg/l (as Un-ionized Ammonia) for freshwater aquatic life.

Concentrations of Total Ammonia ($NH_3 + NH_4^+$) Which Contain an Un-ionized Ammonia Concentration of 0.020 mg/ℓ NH_3*

Temperature (°C)	pH Value								
	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0
5	160.	51.	16.	5.1	1.6	0.53	0.18	0.071	0.036
10	110.	34.	11.	3.4	1.1	0.36	0.13	0.054	0.031
15	73.	23.	7.3	2.3	0.75	0.25	0.093	0.043	0.027
20	50.	16.	5.1	1.6	0.52	0.18	0.070	0.036	0.025
25	35.	11.	3.5	1.1	0.37	0.13	0.055	0.031	0.024
30	25.	7.9	2.5	0.81	0.27	0.099	0.045	0.028	0.022

*[Abstracted from Thurston et al. (1974)]

INTRODUCTION:

Ammonia is a pungent, colorless, gaseous, alkaline compound of nitrogen and hydrogen that is highly soluble in water. It is a biologically active compound present in most waters as a normal biological degradation product of nitrogenous organic matter. It may also reach ground and surface waters through discharge of industrial wastes containing ammonia as a byproduct or wastes from industrial processes using "ammonia water".

When ammonia dissolves in water, some of the ammonia reacts with the water to form ammonium ions. A chemical equilibrium is established which

contains un-ionized ammonia (NH_3), ionized ammonia (NH_4^+), and hydroxide ions (OH^-). The equilibrium for these chemical species can be expressed in simplified form by the following equation:

$$NH_3 + H_2O \rightleftharpoons NH_3 \cdot H_2O \rightleftharpoons NH_4^+ + OH^-$$

In the above equation NH_3 represents ammonia gas combining with water. The term $NH_3 \cdot H_2O$ represents the un-ionized ammonia molecule which is loosely attached to water molecules. Dissolved un-ionized ammonia will be represented for convenience as NH_3. The ionized form of ammonia will be represented as NH_4^+. The term total ammonia will refer to the sum of these ($NH_3 + NH_4^+$).

The toxicity of aqueous solutions of ammonia is attributed to the NH_3 species. Because of the equilibrium relationship among NH_3, NH_4^+, and OH^-, the toxicity of ammonia is very much dependent upon pH as well as the concentration of total ammonia. Other factors also affect the concentration of NH_3 in water solutions, the most important of which are temperature and ionic strength. The concentration of NH_3 increases with increasing temperature, and decreases with increasing ionic strength. In aqueous ammonia solutions of dilute saline concentrations, the NH_3 concentration decreases with increasing salinity.

A table of percent NH_3 for aqueous ammonia solutions of zero salinity at different values of pH and temperature is given below. This percentage can be used to determine the amount of total ammonia which is in the most toxic (NH_3) form.

Percent Un-ionized Ammonia in Aqueous Ammonia Solutions*

Temperature (°C)	pH Value								
	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0
5	0.013	0.040	0.12	0.39	1.2	3.8	11.	28.	56.
10	0.019	0.059	0.19	0.59	1.8	5.6	16.	37.	65.
15	0.027	0.087	0.27	0.86	2.7	8.0	21.	46.	73.
20	0.040	0.13	0.40	1.2	3.8	11.	28.	56.	80.
25	0.057	0.18	0.57	1.8	5.4	15.	36.	64.	85.
30	0.080	0.25	0.80	2.5	7.5	20.	45.	72.	89.

*[Thurston, et al. (1974)]

RATIONALE:

It has been known since early in this century that ammonia is toxic to fishes and that the toxicity varies with the pH of the water. Chipman (1934) demonstrated that undissociated ammonia (NH_3) was the chemical species toxic to goldfish, amphipods, and cladocerans. He concluded from his studies that the toxicity of ammonium salts was pH-dependent and was directly related to the concentration of undissociated ammonia. Chipman's work was confirmed by Wuhrmann, et al. (1947) who concluded that the NH_3 fraction was toxic to fish and that the NH_4^+ fraction had little or no toxicity. Further studies by Wuhrmann and Woker (1948) and Downing and Merkens (1955) agreed with these earlier findings. Tabata (1962), however, has attributed some degree of toxicity to fishes and invertebrates to the NH_4^+ species (less than 1/50th that of NH_3).

In most natural waters, the pH range is such that the NH_4^+ fraction of ammonia predominates; however, in highly alkaline waters, the NH_3 fraction can reach toxic levels. Many laboratory experiments of relatively short duration have demonstrated that the lethal concentrations for a variety of fish species are in the range of 0.2 to 2.0 mg/l NH_3 with trout being the most sensitive and carp the most resistant. Although coarse fish such as carp survive longer in toxic solutions than do salmonids, the difference in sensitivity between fish species to prolonged exposure is probably less than the tenfold range given as the lethal range. The lowest lethal concentration reported for salmonids is 0.2 mg/l NH_3 for rainbow trout fry (Liebmann, 1960). The toxic concentration for Atlantic salmon smolts (Herbert and Shurben, 1965) and for rainbow trout (Ball, 1967) was found to be only slightly higher. Although the concentration of NH_3 below 0.2 mg/l may not kill a significant proportion of a fish population, such concentration may still exert an adverse physiological or histopathological effect (Flis, 1968; Lloyd and Orr, 1969; Smith and Piper, 1974). Fromm (1970) found that at total ammonia ($NH_3 + NH_4^+$) concentrations of 3 mg/l ammonia nitrogen, the trout became hyperexcitable; at 5 mg/l, ammonia excretion by rainbow trout was inhibited; and at 8 mg/l (approximately 1 mg/l NH_3), 50 percent died within 24 hours. Burrows (1964) found progressive gill hyperplasia in fingerling chinook salmon during a six-week exposure to a total ammonia concentration (expressed as NH_4) of 0.3 mg/l (0.002 mg/l NH_3), which was the lowest concentration applied. Reichenbach-Klinke (1967) also noted gill hyperplasia, as well as pathological effects on

the liver and blood of various species at a concentration of 0.27 mg/l NH_3. Flis (1968) noted that exposure of carp to sublethal NH_3 concentrations resulted in extensive necrotic changes and tissue disintegration in various organs.

Herbert and Shurben (1965) reported that the resistance of yearling rainbow trout to ammonia increased with salinity (i.e. dilution with about 30 percent sea water) but above that level resistance appeared to decrease. Katz and Pierro (1967) subjected fingerling coho salmon to an ammonia waste at salinity levels of 20, 25, and 29 parts per thousand (i.e. dilution with about 57-83 percent sea water) and also found that toxicity increased with increased salinity. In saline waters the NH_4^+/NH_3 ratio must be adjusted by consideration of the activity of the charged species and total ionic strength of the solution. In dilute saline waters this ratio will change to favor NH_4^+, and thereby reduce the concentration of the toxic NH_3 species. At higher salinity levels the reported toxic effects of ammonia to fish must therefore be attributed to some mechanism other than changes in the NH_4^+/NH_3 ratio. Data on the effect of ammonia on marine species are limited and the information on anadromous species generally has been reported in conjunction with studies on freshwater species.

Although the NH_3 fraction of total ammonia increases with temperature, the toxic effect of NH_3 vs. temperature is not clear. Burrows (1964) has reported that the recovery rate from hyperplasia in gill tissues of chinook salmon exposed first to ammonia at sublethal levels and then to fresh water was less at 6°C than at 14°C. In this experiment, comparison was made between two different age classes of salmon.

Levels of un-ionized ammonia in the range of 0.20 to 2 mg/l have been shown to be toxic to some species of freshwater aquatic life. To provide safety for those life forms not examined, 1/10th of the lower value of this toxic effect range results in a criterion of 0.020 mg/l of un-ionized ammonia. This criterion is slightly lower than that recommended for European inland fisheries (EIFAC, 1970) for temperatures above 5° C and pH values below 8.5. Measurement of values of total ammonia for calculation of values in the range of 0.020 mg/l NH_3 is well within current analytical capability.

REFERENCES CITED:

Ball, I.R., 1967. The relative susceptibilities of some species of freshwater fish to poisons - I. Ammonia. Water Res. 1: 767.

Burrows, R.E., 1964. Effects of accumulated excretory products on hatchery-reared salmonids. Bureau of Sport Fisheries and Wildlife Research Report 66. G.P.O., Washington, D.C.

Chipman, W.A., Jr., 1934. The role of pH in determining the toxicity of ammonium compounds. Ph.D. Thesis, University of Missouri, Columbia, Missouri.

Downing, K.M. and J.C. Merkens, 1955. The influence of dissolved oxygen concentration on the toxicity of un-ionized ammonia to rainbow trout (Salmo gairdnerii Richardson). Ann. appl. Biol., 43: 243.

European Inland Fisheries Advisory Commission, 1970. Water quality criteria for European freshwater fish. Report on ammonia and inland fisheries. EIFAC Technical Paper No. 11, 12 p; Water Res. 7: 1011 (1973).

Flis, J., 1968. Anatomicohistopathological changes induced in carp (Cyprinus carpio L.) by ammonia water. Part I. Effects of toxic concentrations. Acta Hydrobiol., 10: 205. Part II. Effects of subtoxic concentrations. Ibid., 10: 225.

Fromm, P.O., 1970. Toxic action of water soluble pollutants on freshwater fish. EPA Water Pollution Control Research Series No. 18050DST. G.P Washington, D.C.

Herbert, D.W.M. and D.S. Shurben, 1965. The susceptibility of salmonid fish to poisons under estuarine conditions - II. Ammonium chloride. Int. J. Air Wat. Pollut., 9: 89.

Katz, M. and R.A. Pierro, 1967. Estimates of the acute toxicity of ammonia-urea plant wastes to coho salmon, Oncorhynchus kisutch. Final Report, Fisheries Research Institute, University of Washington, Seattle: 15 p.

Liebmann, H.,.1960. Handbuch der Frischwasser- und Abwasserbiologie - II München.

Lloyd, R. and L.D. Orr, 1969. The diuretic response by rainbow trout to sublethal concentrations of ammonia. Wat. Res., 3: 335.

Reichenbach-Klinke, H.-H., 1967. Untersuchungen über die Einwirkung des Ammoniakgehalts auf den Fischorganismus. Arch. Fischereiwiss., 17: 122.

Smith, C.E. and R.G. Piper, 1975. Lesions associated with chronic exposure to ammonia. IN: The Pathology of Fishes, W.E. Ribelin and G. Migaki (Eds.), University of Wisconsin Press, Madison. pp 497-514.

Tabata, K., 1962. Toxicity of ammonia to aquatic animals with reference to the effect of pH and carbon dioxide. (In Japanese with English summary). Bull. Tokai Reg. Fish. Res. Lab., 34: 67.

Thurston, R.V., et al., 1974. Aqueous ammonia equilibrium calculations. Fisheries Bioassay Laboratory Technical Report No. 74-1, Montana State University, Bozeman, 18 p.

Wuhrmann, K., et al., 1947. Über die fischereibiologische Bedeutung des Ammonium- und Ammoniakgehaltes fliessender Gewässer. Vjschr. naturf, Ges. Zürich, 92: 198.

Wuhrmann, K. and H. Woker, 1948. Beitrage zur Toxikologie der Fische. II. Experimentelle Untersuchungen über die Ammoniak- und Bläusaurevergiftung. Schweiz. Z. Hydrol., 11: 210.

ARSENIC

CRITERIA:

50 ug/l for domestic water supplies (health);

100 ug/l for irrigation of crops.

INTRODUCTION:

Arsenic is a shiny, gray, brittle element possessing both metallic and non-metallic properties. Compounds of arsenic are ubiquitous in nature, insoluble in water and occur mostly as arsenides and arseno-pyrites. Samplings from 130 water stations in the United States have shown arsenic concentrations of 5 to 336 ug/l with a mean level of 64 ug/l (Kopp, 1969). Arsenic normally is present in sea water at concentrations of 2 to 3 ug/l.

Arsenic exists in the trivalent and pentavalent states and its compounds may be either organic or inorganic. Trivalent inorganic arsenicals are more toxic than the pentavalent forms both to mammals and aquatic species. Though most forms of arsenic are toxic to humans, arsenicals have been used in the medical treatment of spirochaetal infections, blood dyscrasias and dermatitis (Merck Index, 1968). Arsenic and arsenicals have many diversified industrial uses such as hardening of copper and lead alloys, pigmentation in paints and fireworks, and the manufacture of glass, cloth and electrical semiconductors. Arsenicals are used in the formulation of herbicides for forest management and agriculture.

RATIONALE:

Arsenic concentrations in most community drinking water supplies in the United States range from a trace to approximately 0.1 mg/l (McCabe, et al., 1970).

Inorganic arsenic is absorbed readily from the gastrointestinal tract, the lungs, and to a lesser extent from the skin, and becomes distributed throughout the body tissues and fluids (Sollman, 1957). It is excreted via urine, feces, sweat, and the epithelium of the skin (Ducoff, et al., 1948; Musil and Dejmal, 1957).

After cessation of continuous exposure, arsenic excretion may continue for as long as 70 days (DuBois and Geiling, 1959).

Since the early nineteenth century arsenicals have been suspected of being carcinogenic (Paris, 1820; Sommers and McManus, 1953; Boutwell, 1963; Hueper and Payne, 1963).

According to Frost (1967), the most toxic arsenicals are well tolerated at concentrations of 10 to 20 ppm arsenic in the diet. The least toxic arsenicals can be fed without injury at levels which contribute up to at least 1000 ppm arsenic in the diet. He concluded that arsenicals appear remarkably free of carcinogenic properties.

In man, subacute and chronic arsenic poisoning may be insidious and pernicious. The symptoms of mild chronic poisoning are fatigue

and loss of energy. In more severe intoxication the following symptoms may be observed: gastrointestinal catarrh, kidney degeneration, tendency to edema, polyneuritis, liver cirrhosis, bone marrow injury, exfoliate dermatitis and altered skin pigmentation (DiPalma, 1965; Goodman and Gilman, 1965). No true tolerance of arsenic has ever been demonstrated (Dubois and Geiling, 1959). During chronic exposure, trivalent arsenic accumulates mainly in bone, muscle, and skin and to a lesser degree in the liver and kidneys (Smith, 1967).

Reports from epidemiological studies in Taiwan indicate that 0.3 mg/l arsenic in drinking water resulted in increased incidences of hyperkeratosis and skin cancer with increased consumption of water (Chen and Wu, 1962; Tseng, et al., 1968; Yeh, et al., 1968). A similar situation has been reported in Argentina (Trelles, et al., 1970). Dermatological manifestations of arsenicism were noted in children in Antofagasta, Chile who used a water supply containing an arsenic concentration of 0.8 mg/l. A new water supply was provided and preliminary data showed that the quantity of arsenic in hair decreased (Borgono and Grieber, 1972).

Arsenicism affecting two members of a family whose well water concentration varied from 0.5 to 2.75 mg/l arsenic over a period of several months was reported in Nevada (Craun and McCabe, 1971). A study in California indicated that a greater proportion of the population had elevated concentrations of arsenic in hair when their drinking water had more than 0.12 mg/l arsenic than when concentrations were lower, but illness was not noted (Goldsmith, et al., 1972). In none of the cited incidents of apparent correlation of arsenic in drinking water with increased incidence of hyperkeratosis and skin cancer has there been any confirmed evidence that arsenic was the etiologic agent in the production of carcinomas.

It is estimated that the total intake of arsenic from food averages 900 ug/day (Schroeder and Balassa, 1966). At the concentration of 50 ug/l recommended for drinking water supply and an average intake of 2 liters of water per day, the intake from water could reach 100 ug/day or approximately 10 percent of the total ingested arsenic.

Although arsenic is concentrated in aquatic organisms, it is evidently not progressively concentrated along a food chain. In addition, arsenic consumed as an organically-bound species in flesh appears to have low toxicity (Ferguson and Gavis, 1972). Surber and Meehan (1931) found that fish-food organisms generally can withstand concentrations of approximately 1.73 mg/l of arsenious trioxide (1.3 mg/l arsenic) in a sodium arsenite solution. Concentrations of 4 mg/l sodium arsenite (2.3 mg/l as arsenic) in confined outdoor pools have been found to reduce survival and growth of fish and to reduce bottom fauna and plankton populations (Gilderhus, 1966).

Trivalent arsenic is highly toxic to invertebrates. Conversely, pentavalent arsenic is of relatively low toxicity. In Lake Erie water Daphnia were observed to exhibit initial symptoms of immobility at 18 to 31 mg/l of sodium arsenate or 4.3 to 7.5 mg/l as arsenic (Anderson, 1944, 1946). The lethal threshold of sodium arsenate for minnows has been reported to be 234 mg/l as arsenic at 16° C to 20° C (Wilber, 1969).

Ambient arsenic concentrations in sea water are reported to be accumulated by oysters and other molluscan shellfish (Sautet, et al., 1964;

Lowman, et al., 1971). Wilber (1969) reported concentrations of 100 mg/kg in shellfish.

Beginning in 1926 and for many years thereafter, sodium arsenite was used for the control of vascular aquatic plants in the public lakes of Wisconsin (Mackenthun, 1950). Dosages up to 10 mg/l of the white arsenic equivalent were used depending on the physical character of the area to be treated. Harm to fish life within the lakes or to fishing was not noted.

Lueschow (1964) found that Wisconsin lakes naturally may contain 10 ug/l of arsenic. Pewaukee Lake, Wisconsin, a lake of 2300 acres, received 215,174 pounds of arsenic for aquatic vegetation control in a 14-year period beginning in 1950. From 1959 to 1964, arsenic concentrations in the lake's outlet reached a maximum of 463 ug/l arsenic and consistently were above 100 ug/l. The mean arsenic concentration in lake bottom muds was 208 ug/g on a dry weight basis. A single sample of Cladophora sp. collected from Pewaukee Lake in 1962 contained 1258 ug/g arsenic (dry weight). Fresh shoots of mature Myriophyllum sp. stems contained 228 and 561 ug/g arsenic (dry weight).

Benthic invertebrates, Chaoborus punctipennis and Tendipedidae, were present in Pewaukee Lake in populations that reached 288 per square foot. Lueschow concluded that the trivalent inorganic arsenic was 10 to 15 times more toxic to Tendipedes plumosus than the pentavalent form. His studies indicate that the trivalent inorganic arsenic is converted to pentavalent arsenic within 30 days and that long-term survival of

typical benthic organisms would be normal at concentrations as high as 1,920 ug/g arsenic (dry weight) in lake muds.

The data cited above indicate that freshwater fish-food organisms are adversely affected by concentrations of arsenic as low as 1.3 mg/l, and the mobility of the freshwater crustacean Daphnia is impaired by a concentration of arsenic as low as 4.3 mg/l. However, these data are not considered to be sufficient to recommend any numerical criterion for freshwater aquatic life. Such data as do exist indicate that the 50 ug/l criterion established for domestic water supplies should be protective of aquatic life.

Rasmussen and Henry (1965) found that arsenic at 0.5 mg/l in nutrient solutions produced toxicity symptoms in seedlings of the pineapple and orange. Below this concentration, no symptoms of toxicity were found. Clements and Heggeness (1939) reported that 0.5 mg/l arsenic as arsenite in nutrient solutions produced an 80 percent yield reduction in tomatoes. Following a review of available literature the National Academy of Sciences (NAS, 1974) suggests that a concentration of 100 ug/l could be used for 100 years on sandy soils, and a concentration of 2 mg/l used for a period of 20 years or 0.5 mg/l used for 100 years on clayey soils with an adequate margin of safety. Because of these factors, a criterion of 100 ug/l for the irrigation of crops is recommended. The herbicidal properties of arsenic in water to aquatic vegetation also are recognized. Although data are not sufficiently precise to recommend a criterion, such data as do exist indicate that 100 ug/l would be protective of aquatic vegetation.

REFERENCES CITED

Anderson, B.G., 1944. The toxicity thresholds of various substances found in industrial wastes as determined by the use of Daphnia magna. Sewage Works J. 1616: 1156.

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

Borgono, J.M. and R. Grieber, 1972. Epidemiological study of arsenicism in the city of Antofagasta. Proceedings of the University of Missouri's Fifth Annual Conference on Trace Substances in Environmental Health (in press).

Boutwell, R.K., 1963. Feed additives: a carcinogenicity evaluation of potassium arsenite and arsanilic acid. Jour. Agr. Food Chem., 11: 381.

Chen, K.P. and H. Wu, 1962. Epidemiological studies on blackfoot disease: II: A study of source of drinking water in relation to the disease. Jour. Formosa Med. Assn., 61: 611.

Clements, H.F. and H.G. Heggeness, 1939. Arsenic toxicity to plants. Hawaii Agr. Exp. Sta. Report, 1940: 77.

Craun, G. and L.J. McCabe, 1971. Waterborne disease outbreaks, 1961-1970.. Presented at the annual meeting of the Amer. Water Works Assn. (June).

DiPalma, J.R., 1965. Drill's pharmacology in medicine, 3rd ed. McGraw-Hill Book Co., New York, p. 860.

DuBois, K.P. and E.M.K. Geiling, 1959. Textbook of toxicology. Oxford University Press, New York, p. 132.

Ducoff, H.S., et al., 1948. Biological studies with arsenic (A8, 76). II. Excretion and tissue localization. Proc. Soc. Exp. Biol. Med. 69: 548.

Ferguson, J.F. and J. Gavis, 1972. A review of the arsenic cycle in natural waters. Water Research, 6: 1259.

Frost, D.V., 1967. Arsenicals in biology--retrospect and prospect. Fed. Amer. Soc. for Experimental Biol., 26:194.

Gilderhus, P.A., 1966. Some effects of sublethal concentrations of sodium arsenite on bluegills and the aquatic environment. Trans. Amer. Fish. Soc., 95: 289.

Goodman, L.S. and A.Z. Gilman, eds., 1965. The pharmacological basis of therapeutics, 3rd ed. The McMillan Co., New York, p. 944.

Goldsmith, J.R., et al., 1972. Evaluation of health implications of elevated arsenic in well water. Water Research, 6: 1133.

Heuper. W.C. and W.W. Payne, 1963. Carcinogenic effects of adsorbates of raw and finished water supplies. Amer. Jour. Clin. Path., 39: 475.

Kopp, J.F., 1969. The occurrence of trace elements in water, In: Proceedings of the Third Annual Conference on Trace Substances in Environmental Health, edited by D.D. Hemphill, University of Missouri, Columbia, p. 59.

Lowman, F.G., et al., 1971. Accumulation and redistribution of radionuclides by marine organisms, In: Radioactivity in the Marine Environment, National Academy of Sciences, Washington, D.C., p. 161.

Lueschow, L.A., 1964. The effects of arsenic trioxide used in aquatic weed control operations on selected aspects of the bio-environment. M.S. Thesis, Univ. of Wisconsin, Madison, Wisconson.

Mackenthun, K.M., 1950. Aquatic weed control with sodium arsenite. Sew. and Ind. Wastes, 22: 1062.

McCabe, L.J., et al., 1970. Survey of community water supply systems. Jour. Amer. Water Works Assn., 62: 670.

Merck Index, 1968. An encyclopedia of chemicals and drugs, 8th ed. Merck and Co., Inc., Rahway, N. J.

Musil, J. and V. Dejmal, 1957. Experimental and clinical administration of radioarsenic (As^{76}), Casopis Lekari Ceskych, 96: 1543.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Govt. Print. Office, Washington, D. C.

Paris, J.A., 1820. Pharmacologia: Comprehending the art of prescribing upon fixed and scientific principles together with the history of medicinal substances, 3rd ed. W. Philips, London, p. 132.

Rasmussen, G.K. and W.H. Henry, 1965. Effects of arsenic on the growth of pineapple and orange seedlings in sand and solution nutrient cultures. Citrus Ind., 46: 22.

Sautet, J., et al., 1964. Contribution to the study of the biological fixation and elimination of arsenic by Mytilus edulis, Second Note. Ann. Med. Leg. (Paris), 44: 466.

Schroeder, H.A. and J.J. Balassa, 1966. Abnormal trace metals in man: Arsenic. Jour. Chronic Dis., 19: 85.

Smith, H., 1967. The distribution of antimony, arsenic, copper and zinc in human tissue. J. Forensic Sci. Soc. , 7:97.

Sollman, T.H., 1957. A manual of pharmacology and its application to therapeutics and toxicology, 8th ed. W. B. Sanders Co., Philadelphia. n.

Sommers, S.C. and R.G. McManus, 1953. Multiple arsenical cancers of skin and internal organs. Cancer, 6: 347.

Surber, E.W. and L.O. Meehan, 1931. Lethal concentrations of arsenic for certain aquatic organisms. Trans. Amer. Fish. Soc., 61: 225.

Trelles, R.A., et al., 1970. El problema sanitario de la aguas destinadas a la bebida humana, con contenidos elevados de arsenico, vanadio, y flour. Saneamiento, 34(217: 31-80).

Tseng, W.P., et al., 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. Jour. Nat. Cancer Inst., 40: 454.

Wilber, C.G., 1969. The biological aspects of water pollution. Charles C. Thomas, Publisher, Springfield, Illinois.

Yeh, S., et al., 1968. Arsenical cancer of the skin. Cancer, 21:312.

BARIUM

CRITERION:

1 mg/l for domestic water supply (health).

INTRODUCTION:

Barium is a yellowish-white metal of the alkaline earth group. It occurs in nature chiefly as barite, $BaSO_4$, and witherite, $BaCO_3$, both of which are highly insoluble salts. The metal is stable in dry air, but readily oxidized by humid air or water.

Many of the salts of barium are soluble in both water and acid, and soluble barium salts are reported to be poisonous (Lange, 1961; NAS, 1974). However, barium ions generally are thought to be rapidly precipitated or removed from solution by adsorption and sedimentation (McKee and Wolf, 1963; NAS, 1974).

While barium is a malleable, ductile metal, its major commercial value is in its compounds. Barium compounds are used in a variety of industrial applications including the metallurgic, paint, glass and electronics industries, as well as for medicinal purposes.

RATIONALE:

Concentrations of barium in domestic drinking water supplies generally range from less than 0.6 ug/l to approximately 10 ug/l with upper limits in a few midwestern and western states ranging from 100 to 3,000 ug/l (PHS, 1962/1963; Katz, 1970; Little, 1971). Barium enters the body primarily through air and water, since

appreciable amounts are not contained in foods (NAS, 1974).

The fatal dose of barium for man is reported to be 550 to 600 mg. Ingestion of soluble barium compounds may also result in effects on the gastrointestinal tract causing vomiting and diarrhea and on the central nervous system causing violent tonic and clonic spasms followed in some cases by paralysis (Browning, 1961; and Patty, 1962, cited in Preliminary Air Pollution Survey of Barium and Its Compounds, 1969). Barium salts are considered to be muscle stimulants, especially for the heart muscle (Sollmann, 1957). By constricting blood vessels, barium may cause an increase in blood pressure. On the other hand, it is not likely that barium accumulates in the bone, muscle, kidney, or other tissues because it is readily excreted (Browning, 1961; McKee and Wolf, 1963).

Stokinger and Woodward (1958) developed a safe concentration for barium in drinking water based on the limiting values for industrial atmospheres, an estimate of the amount absorbed into the blood stream, and daily consumption of two liters of water. From these factors they arrived at a limiting concentration of 2 mg/l for a healthy adult human population, to which a safety factor was applied to allow for any possible accumulation in the body. Since barium is not removed by conventional water treatment processes and because of the toxic effect on the heart and blood vessels, a limit of 1 mg/l is recommended for barium in domestic water supplies.

Experimental data indicate that the soluble barium concentration in fresh and marine water generally would have to exceed 50 mg/l before toxicity to aquatic life would be expected. In most natural waters, there is sufficient sulfate or carbonate to precipitate the barium present in the water as a virtually insoluble, non-toxic compound. Recognizing that the physical and chemical properties of barium generally will preclude the existence of the toxic soluble form under usual marine and freshwater conditions, a restrictive criteria for aquatic life appears unwarranted.

REFERENCES CITED:

Browning, E., 1961. Toxicity of industrial metals. Butterworths, London, England.

Katz, M., et al., 1970. Effects of pollution on fish life, heavy metals, Annual literature review, Jour. Water Poll. Cont. Fed., 42:987.

Lange, N.A., 1961. Handbook of chemistry. McGraw-Hill, Tenth Ed.

Little, A.D., 1971. Inorganic chemical pollution of fresh water. Water Quality Data Book, Vol. 2. Environmental Protection Agency, 18010 DPV, pp. 24-26.

McKee, J.E. and W.W. Wolf, 1963. Water quality criteria, California State Water Resources Control Board, Pub. No. 3-A.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

Patty, F.A. Industrial Hygiene and Toxicology, Vol. II (New York: Wiley, pp 998-1002, 1962) cited in U.S. Department of Health, Education and Welfare, 1969.

Sollmann, T.H., 1957. A manual of pharmacology and its applications to therapeutics and toxicology. 8th ed. W.B. Saunders Co., Phila. Pa.

Stokinger, H.E. and R.L. Woodward, 1958. Toxicologic methods for establishing drinking water standards. Jour. Amer. Water Works Assn., 50:515.

U.S. Department of Health, Education and Welfare, 1969. Preliminary Air Pollution Survey of Barium and Its Compounds, A Literature Review. National Air Pollution Control Administration Publication No. APT D 69-28, Raleigh, NC.

U.S. Public Health Service, 1962/63. Drinking water quality of selected interstate carrier water supplies. U.S. Dept. of Health, Education and Welfare, Wash., D.C.

BERYLLIUM

CRITERIA:

11 ug/l for the protection of aquatic life in soft fresh water; *

1,100 ug/l for the protection of aquatic life in hard fresh water; *

100 ug/l for continuous irrigation on all soils; except

500 ug/l for irrigation on neutral to alkaline fine-textured soils.

INTRODUCTION:

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

RATIONALE:

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

*See criteria for Hardness (p. 147). The beryllium concentration that will be protective of a given aquatic ecosystem can be obtained by conducting flow-through bioassays using ambient water and native species of fish and invertebrates.

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

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

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

Tarzwell and Henderson (1960) obtained 96-hour LC_{50} values ranging from 0.15 mg/l beryllium (when tested as the nitrate and chloride) to 0.2 mg/l beryllium (when tested as the sulfate) for fathead minnows in soft water (20 mg/l $CaCO_3$, total alkalinity of 18 mg/l, and pH of 7.4), and from 11 mg/l beryllium (as sulfate) to 20 mg/l beryllium (as nitrate) for fathead minnows in hard water (400 mg/l $CaCO_3$, total alkalinity of

360 mg/l, and pH of 8.2). For bluegill they obtained 96-hour LC_{50} values of 1.3 mg/l beryllium in soft water and 12 mg/l beryllium in hard water (both as the sulfate.)

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

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

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

The 96-hour LC_{50} values were 26.3 mg/l beryllium in hard water, and 4.7 mg/l beryllium in soft water.

Based on the fathead minnow and bluegill data of Tarzwell and Henderson (1960), the observations of Slonim and Ray (1975) on salamander larva survival, and the observations of Slonim and Slonim (1973) on guppy survival, the criterion for the protection of aquatic biota is established at 1.1 mg/l beryllium in hard fresh water. This value assumes an application factor of 0.1 of the 96-hour LC_{50} value for fathead minnows. For soft fresh water, in view of the reported approximate 100-fold increase in acute fish toxicity over that found in hard water, the criterion for the protection of aquatic biota is set at 0.011 mg/l beryllium.

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

Beryllium has been shown to inhibit photosynthesis in terrestrial plants (Bollard and Butler, 1966), and to be toxic to some varieties

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

REFERENCES CITED:

Bollard, E.G. and G.W. Butler, 1966. Mineral nutrition of plants. Ann. Rev. Plant Physiol., 17:77.

Goldberg, E.D., et al., 1971. Marine chemistry. In: Radioactivity in the marine environment. National Academy of Sciences, Washington, D. C., p. 137.

Haas, A.R.C., 1932. Nutritional aspects in mottleleaf and other physiological diseases of citrus. Hilgardia, 6:483.

Jackim, E., et al., 1970. Effects of metal poisoning for five liver enzymes in the killifish (Fundulus heteroclitus). Jour. Fish. Res. Bd. Can., 27:383.

Kopp, J.F. and R.C. Kroner, 1967. Trace metals in waters of the United States. U.S. Department of the Interior, FWPCA, Cincinnati, Ohio.

Lange, N.A., Ed., 1961. Handbook of chemistry, 10th ed. McGraw-Hill Book Co., New York.

Lowman, F.G., et al., 1971. Accumulation and redistribution of radionuclides by marine organisms. In: Radioactivity in the marine environment. National Academy of Sciences, Washington, D. C., p. 161.

McCord, C.P., 1951. Beryllium as a sensitizing agent. Ind. Med. Surg., 20:336.

McKee, J.E. and H.W. Wolf, 1963. Water quality criteria. State Water Quality Control Board, Sacramento, Calif., Pub. 3-A.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

National Institute for Occupational Safety and Health, 1972. Occupational exposure to beryllium. U.S. Department of Health, Education and Welfare, Health Services and Mental Health Admin., National Institute for Occupational Safety and Health. U.S. Government Printing Office, Washington, D. C.

Reeves, A.L., 1965. Absorption of beryllium from the gastrointestinal tract. A.M.A. Arch. Envir. Health, 11:209.

Riley, J.P. and I. Roth, 1971. The distribution of trace elements in species of phytoplankton grown in culture. Jour. Mar. Biol. Assn. U.K., 51:63.

Romney, E.M., et al., 1962. Beryllium and the growth of bush beans. Science, 135:786.

Romney, E.M. and J.D. Childress, 1965. Effects of beryllium in plants and soil. Soil Sci., 100:210.

Slonim, A.R., 1973. Acute toxicity of beryllium sulfate to the common guppy. Jour. Water Poll. Cont. Fed., 45:2110.

Slonim, A.R. and E.E. Ray, 1975. Acute toxicity of beryllium sulfate to salamander larvae (Ambystoma spp.). Bull. Envir. Contam. Toxicol., 13:307.

Slonim, C.B. and A.R. Slonim, 1973. Effect of water hardness on the tolerance of the guppy to beryllium sulfate. Bull. Envir. Contam. Toxicol., 10:295.

Stokinger, H.E. and C.A. Stroud, 1951. Anemia in acute experimental beryllium poisoning. Jour. Lab. Clin. Med., 38:173.

Tarzwell, C.M. and C. Henderson, 1960. Toxicity of less common metals to fishes. Ind. Wastes, 5:12.

Van Ordstrand, H.S., et al., 1945. Beryllium poisoning. Jour., Amer. Med. Assn., 129:1084.

Williams, R.J.B. and H.H. LeRiche, 1968. The effects of traces of beryllium on the growth of kale, grass, and mustard. Plant Soil, 29:317.

BORON

CRITERION:

750 ug/l for long-term irrigation on sensitive crops.

INTRODUCTION:

Boron is not found in its elemental form in nature; it is usually found as a sodium or calcium borate salt. Boron salts are used in fire retardants, the production of glass, leather tanning and finishing industries, cosmetics, photographic materials, metallurgy, and for high energy rocket fuels. Elemental boron also can be used in nuclear reactors for neutron absorption. Borates are used as "burnable" poisons.

RATIONALE:

Boron is an essential element for growth of plants but there is no evidence that it is required by animals. The maximum concentration found in 1,546 samples of river and lake waters from various parts of the United States was 5.0 mg/l; the mean value was 0.1 mg/l (Kopp and Kroner, 1967). Ground waters could contain substantially higher concentrations at certain places. The concentration in sea water is reported as 4.5 mg/l in the form of borate (NAS, 1974). Naturally occurring concentrations of boron should have no effects on aquatic life.

The minimum lethal dose for minnows exposed to boric acid at 20° C for 6 hours was reported to be 18,000 to 19,000 mg/l in distilled water and 19,000 to 19,500 mg/l in hard water (Le Clerc and Devlaminck, 1955; Le Clerc, 1960).

In the dairy cow, 16 to 20 g/day of boric acid for 40 days produced no ill effects (McKee and Wolf, 1963).

Sensitive crops have shown toxic effects at 1000 ug/l or less of boron (Richards, 1954). Bradford (1966), in a review of boron deficiencies and toxicities, stated that when the boron concentration in irrigation waters was greater than 0.75 mg/l, some sensitive plants such as citrus began to show injury. Biggar and Fireman (1960) showed that with neutral and alkaline soils of high absorption capacities, water containing 2 mg/l boron might be used for some time without injury to sensitive plants. The criterion of 750 ug/l is thought to protect sensitive crops during long-term irrigation.

REFERENCES CITED:

Biggar, J.W. and M. Fireman, 1960. Boron absorption and release by soils. Soil Sci. Soc. Amer. Proc., 24:115.

Bradford, G.R., 1966. Boron [toxicity, indicator plants], in diagnostic criteria for plants and soils. H.D. Chapman, Ed., University of California, Division of Agricultural Science, Berkeley, p. 33.

Le Clerc, E., 1960. The self-purification of streams and the relationship between chemical and biological tests. Proc. Second Symposium on Treatment of Waste Waters, Pergamon Press, London, England, p. 281.

Le Clerc, E. and F. Devlaminck, 1955. Fish toxicity tests and water quality. Bull. de Belge Condument Eaux., 28:11.

Kopp J.F. and R.C. Kroner, 1967. Trace metals in waters of the United States. U.S. Dept. of Interior, Federal Water Pollution Control Admin., Cincinnati, Ohio.

McKee, J.E. and H.W. Wolf, 1963. Water quality criteria. State Water Quality Control Board, Sacramento, Calif., Pub. 3-A.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Richards, L.A. (Ed.), 1954. Diagnosis and improvement of saline and alkali soils. Agriculture Handbook No. 60, U.S. Government Printing Office, Washington, D. C.

CADMIUM

CRITERIA:

10 ug/l for domestic water supply (health).

Aquatic Life:

Fresh Water

Soft Water*	Hard Water*	
0.4 ug/l	1.2 ug/l	for cladocerans and salmonid fishes;
4.0 ug/l	12.0 ug/l	for other, less sensitive, aquatic life.

Marine

5.0 ug/l

INTRODUCTION:

Cadmium is a soft, white, easily fusible metal similar to zinc and lead in many properties and readily soluble in mineral acids. Biologically, cadmium is a nonessential, nonbeneficial element recognized to be of high toxic potential. It is deposited and accumulated in various body tissues and is found in varying concentrations throughout all areas where man lives. Within the past two decades industrial production and use of the metal has increased. Concomitantly, there have been incidences of acute cases of clinically identifiable cadmiosis. Cadmium may function in or may be an etiological factor for various human pathological processes including testicular tumors, renal dysfunction, hypertension, arteriosclerosis, growth inhibition, chronic diseases of old age, and cancer.

*See Criteria for Hardness (p. 147). The cadmium concentration that will be protective of a given aquatic ecosystem can be obtained by conducting flow-through bioassays using ambient water and native species of fish and invertebrates.

Cadmium occurs in nature chiefly as a sulfide salt, frequently in association with zinc and lead ores. Accumulations of cadmium in soils in the vicinity of mines and smelters may result in high local concentrations in nearby waters. The salts of the metal also may occur in wastes from electroplating plants, pigment works, textile and chemical industries. Seepage of cadmium from electroplating plants has resulted in groundwater cadmium concentrations of 0.01 to 3.2 mg/l (Lieber and Welsch, 1954). Kopp and Kroner (1967) on one occasion reported 120 ug/l dissolved cadmium in the Cuyahoga River at Cleveland, Ohio. However, dissolved cadmium was found in less than 3 percent of 1,577 water samples examined for the United States, with a mean of slightly under 10 ug/l. Most fresh waters contain less than 1 ug/l cadmium and most analyses of sea water indicate an average concentration of about 0.15 ug/l (Fleischer, et al., 1974).

RATIONALE:

Cadmium has been shown to be toxic to man when ingested or inhaled. Exposure by the former route causes symptoms resembling food poisoning. A group of school children became ill after eating popsicles containing 13 to 15 mg/l cadmium (Frant and Kleeman, 1941), and this level, equivalent to 1.3 to 3.0 mg of cadmium ingested, commonly is considered the emetic threshold concentration.

In a specially designed study, five groups of rats were exposed to drinking water containing cadmium concentrations of 0.1 mg/l to 10 mg/l. Although no visible toxic effects were noted, the content of cadmium in the kidney and liver increased in direct proportion

to the dose at all levels of exposure. At the end of one year, tissue concentrations were approximately double those found after six months (Ginn and Volker, 1944). Later work confirmed that virtually no absorbed cadmium was eliminated (Decker, et al., 1958).

Drinking water containing excessive cadmium led to the occurrence of itai-itai disease among the Japanese (Kobayashi, 1970). Within 15 years 200 cases of this disease were recorded in the Jintsu River Valley, half of which resulted in death. The disease is characterized by rheumatic symptoms with intense pain in the bones caused by a loss of bone minerals with the bones becoming as flexible as soft tissues. Yamagata and Shigematsu (1970) estimated that a daily intake of 600 ug cadmium would not produce itai-itai disease in an endemic area.

Studies in animals (Ferm, et al., 1969; Chernoff, 1973) as well as observations in human fetuses (Chaube, et al., 1973; and Friberg, et al., 1974) indicate that the placenta is not a complete barrier against the transfer of cadmium. About 6 percent of the cadmium that enters the stomach via food or drink is absorbed into the body tissues and about 30 percent of the cadmium inhaled into the lungs by breathing and smoking is absorbed (EPA, In Press). Presently there are no known physiological needs for cadmium and no mechanism by which the body maintains cadmium at a constant safe level. Once absorbed, it is stored largely in the kidneys and liver and is excreted at an extremely slow rate.

Chronic kidney disease (renal tubular dysfunction) will begin to occur in an individual when the cadmium accumulated in the kidneys reaches a critical concentration. While the critical concentration varies from one individual to another, the threshold of observed effect is about 200 ppm of cadmium in the renal cortex (Friberg, et al., 1974). Individuals have been found with several times this level without evidence of kidney disease. At a 10 ug/l criterion, as recommended in drinking water, the maximum daily intake of cadmium would not exceed 20 ug from water, assuming a 2-liter daily consumption. The daily intake from other sources is up to 60 ug in the population of the United States (Schroeder, et al., 1961).

Pickering and Gast (1972) conducted two separate flow-through tests on the chronic toxicity of cadmium to the fathead minnow, Pimephales promelas, using water of 202 mg/l hardness, 157 mg/l alkalinity, and 7.7 pH. Five cadmium concentrations from 4 to 350 ug/l were delivered to the exposure chambers in each test over the life history of the fish. A concentration of 57 ug/l cadmium decreased survival of developing embryos. At levels from 4.5 to 37 ug/l no adverse effect on survival, growth, or reproduction was found. Eaton (1974b) exposed bluegill sunfish, Lepomis macrochirus, to five cadmium concentrations ranging from 31 to 2,140 ug/l for 11 months in a flow-through system using water of the same hardness as above. Nine of the 18 adult bluegill sunfish exposed to 80 ug/l died by the end of the test, while all of those exposed to 31 ug/l and the control survived. Although at 80 ug/l cadmium the hatchability of eggs was not measurably affected, the survival and growth of the resulting larvae were severely reduced after 60 days. Larvae exposed to 31 ug/l cadmium survived and grew about as well as the control fish. Sixty days

after hatching in hard water, growth and survival of channel catfish fry, Ictalurus punctatus, was reduced significantly at a cadmium concentration of 17 ug/l but not at 12 ug/l (Eaton, 1974a). Thus, in hard water, a criterion of 12 ug/l cadmium represents a demonstrated no-effect level for catfish and therefore was chosen to protect non-salmonid freshwater fish species. No data are available upon which to base an acceptable concentration for the chronic exposure of sensitive vertebrates or invertebrates in hard water, therefore a criterion is proposed which is reduced from the less sensitive aquatic life value by the same factor (0.1) as the criterion for soft water, i.e. 1.2 ug/l for salmonids and invertebrates.

Spehar (1974) reported on chronic toxicity tests with cadmium using a topminnow native to Florida in water with a hardness of 41 to 45 mg/l as $CaCO_3$, alkalinity of 38 to 43 mg/l, and a pH of 7.4. There was a significant reduction in the number of eggs produced per female at 8.1 ug/l cadmium, but fish in 4.1 ug/l were unaffected. A criterion of 4.0 ug/l cadmium is selected as offering protection to warm water fish species in soft water.

Three consecutive generations of brook trout, Salvelinus fontinalis, were exposed to cadmium concentrations ranging from 6.4 ug/l to 0.5 ug/l in a test water of similar hardness (Benoit, et al., In Press). Second generation fish exposed to 6.4 and 3.4 ug/l cadmium were smaller at three months than fish exposed to lower concentrations. Both first and second generation fish suffered extensive mortalities during spawning in 3.4 ug/l cadmium. Egg hatchability, survival, growth, and reproduction of fish exposed to 1.7 ug/l were equal to those of control fish.

When embryos and larvae-juveniles of brook trout, Salvelinus fontinalis; brown trout, Salmo trutta; lake trout, Salvelinus namaycush; northern pike, Esox lucius; white suckers, Catastomus commersoni; smallmouth bass, Micropterus dolomieui; and coho salmon, Oncorhynchus kisutch, were exposed to cadmium concentrations ranging from 0.4 to 100 ug/l in soft water, all suffered a reduction in standing crop at 12 ug/l. Standing crop survival times and weights were virtually equal among the four species when exposed to 4 ug/l; 1.2 ug/l was the highest cadmium concentration not causing a reduction among the other three salmonid species after 30 to 120 days of exposure (Eaton, 1974a).

Tests in water with 23 mg/l hardness, 18 mg/l alkalinity, and a pH of 7.3 in a flow-through system indicated a 96-hour LC50 of 2.0 ug/l for the initial feeding stage of chinook salmon, Oncorhynchus tshawytscha, and 0.92 ug/l for 5-month-old steelhead trout, Salmo gairdneri. A criterion of 0.4 ug/l for cadmium is believed to offer protection to the cladocerans and salmonids in soft water (Eaton, 1974a).

Anderson, et al., (1975) , determined the 10-day LC50 for the midge Tanytarsus dissimilis to be 3.4 ug/l in a flow-through system, soft water (48 mg/l) bioassay involving at least one molt. A concentration of 3.1 ug/l retarded growth but 1.9 ug/l elicited no obvious effect. Biesinger and Christensen (1972) measured the toxicity of cadmium to Daphnia magna during an entire life cycle in test water with a hardness of 45 mg/l, alkalinity of 42 mg/l, and a pH of 7.7. It was found that

50 percent of the daphnids exposed to cadmium concentrations of 5 ug/l were killed in three weeks. The production of young was reduced by 50 percent compared to the controls in a cadmium concentration of 0.7 ug/l. Several invertebrate species have been found much less sensitive to cadmium in acute tests than in the midge and cladoceran exposures cited above. (Rehwoldt, et al., 1973; Thorp and Lake, 1974; Warnick and Bell, 1969). These data support a criterion for some invertebrates in soft water that is identical to that for salmonids.

Steward and Pesche, as reported by Eisler (1974), stated that for grass shrimp, Palaemonetes pugio, which were subjected to cadmium chloride in flowing sea water, 69 percent died in 43 days in 500 ug/l cadmium and 10 percent died in a 250 ug/l cadmium solution. Hermit crabs, Pagurus longicarpus, all died in a 250 ug/l cadmium solution, and 30 percent died in 43 days in a 120 ug/l solution. In 63 days, 40 percent died in a 60 ug/l cadmium solution. All of the controls survived.

Eisler (1971) found that the 96-hour LC50 for three species of marine decapod crustaceans ranged between 320 and 420 ug/l at 20° C and 20 mg/l alkalinity. The 96-hour LC25 was 180 ug/l for the hermit crab, Pagurus longicarpus, and the grass shrimp, Palaemonetes vulgaris, and 80 ug/l for the sand shrimp, Crangon septemspinosa.

Zaroogian, as reported by Eisler (1974), states that adult oysters, Crassostrea virginica, exposed to 10 ug/l cadmium between April 1973 and August 1973 accumulated 18,000 ug/kg of cadmium in wet whole

meat, which exceeds the human emetic threshold of 13,000 to 15,000 ug/kg. Oysters retained virtually all of the accumulated cadmium for at least several months and some histopathology was evident. Under natural conditions, significantly greater numbers of larvae from cadmium-stressed oysters failed to develop when compared to controls after 48 or 72 hours. A criterion of one-half of the level at which oysters accumulate cadmium in excess of the human emetic threshold (i.e. 5 ug/l) is believed to provide protection for consumers of oysters.

Data by Page, et al. (In Press), show that the yield of beans, beets, and turnips was reduced about 25 percent by 0.10 mg/l cadmium in nutrient solutions; whereas cabbage and barley yields decreased 20 to 50 percent at 1.0 mg/l.

Yamagata and Shigematsu (1970) have demonstrated that foods cultured on cadmium-polluted soils irrigated with cadmium-polluted water can accumulate sufficient cadmium to be hazardous to humans who consume these foods. Chaney (1973) suggests that it is not the cadmium concentration per se in the soil that determines cadmium accumulation by plants and that as long as the ratio of zinc to cadmium is 100 or greater, foods will not accumulate hazardous concentrations of cadmium. The subject is complex and additional research is needed to resolve potential hazards associated with the cadmium-zinc-soil-plant system.

Fish and certain invertebrates have been found to be sensitive to low levels of cadmium in water. Salmonids and cladocerans appear to be the most sensitive among organisms tested. Increased hardness and/or alkalinity have been demonstrated to decrease the toxicity of cadmium in acute freshwater mortality tests, but may have less of an effect at low cadmium levels. Edible marine organisms can concentrate cadmium levels and become hazardous to be ultimate consumer. Lowman, et al. (1971) reported a concentration factor of 1,000 for cadmium in fish muscle. The criteria necessary to protect fish and other aquatic life are more stringent than those necessary to protect a public water supply or other uses. Data support a division of criteria for "hard" and "soft" water environments.

The data used to develop a criteria for cadmium were obtained over a range of hardness, alkalinity and pH values. Their usefullness in developing criteria will be limited to the range of physical parameters for which experimental data are available. Interpolation of the present data will be required to derive criteria for aquatic ecosystems which do not approximate the experimental conditions.

REFERENCES CITED:

Anderson, R.A., et al, 1975. Survival and growth of Tanytarsus dissimilis (Chironomidae) exposed to copper, cadmium, zinc and nickel. National Environmental Research Laboratory, Duluth, Minn., Quarterly Reports.

Benoit, D.A., et al., Chronic effects of cadmium on three generations of brook trout (Salvelinus fontinalis), EPA Ecological Research Series, In Press.

Biesinger, K.E. and G.M. Christensen, 1972. Effects of various metals on survival, growth, reproduction, and metabolism of Daphnia magna. Jour. Fish. Res. Bd. Can., 29:1691.

Chaney, R.L., 1973. Crop and food chain effects of toxic elements in sludges and effluents. U.S. Environmental Protection Agency, U.S. Department of Agriculture, Universities Workshop, Champaign, Ill. (July).

Chaube, S., et al., 1973. Zinc and cadmium in normal human embryos and fetuses. Arch. Environ. Health, 26:237.

Chernoff, N., 1973. Teratogenic effects of cadmium in rats. Teratology, 8(1):27-32.

Decker, L.E., et al., 1958. Chronic toxicity studies. I: Cadmium administered in drinking water to rats. A.M.A. Arch. Ind. Health, 18:228.

Eaton, J.G., 1974a. Testimony in the matter of proposed toxic pollutant effluent standards for Aldrin-Dieldrin et al. FWPCA (307), Docket No. 1.

Eaton, J.G., 1974b. Cadmium toxicity to the bluegill (Lepomis macrochirus Rafinesque). Trans. Amer. Fish. Soc., 103:729.

Eisler, R., 1971. Cadmium poisoning in Fundulus heteroclitus (Pisces: Cyprinodontidae) and other marine organisms. Jour. Fish. Res. Bd. Can., 28:1225.

Eisler, R., 1974. Testimony in the matter of proposed toxic pollutant effluent standards for Aldrin-Dieldrin et al. FWPCA (307) Docket No. 1.

EPA, 1975. Scientific summary document on cadmium. (In Press)

Ferm, V.H., et al., 1965. The permeability of the hamster placenta to radioactive cadmium. J. Embryol. Exp. Morph. 22:107.

Fleischer, M., et al., 1974. Environmental impact of cadmium: A review by the panel on hazardous trace substances. Env. Health Perspectives, U.S. Government Printing Office, Washington, D.C., 7:253.

Frant, S. and I. Kleeman, 1941. Cadmium "food poisoning." Jour. Amer. Med. Assn., 17:86.

Friberg, L., et al., 1974. Cadmium in the environment. 2nd E. CRC Press, Cleveland, pp 30-31.

Ginn, J.T. and J.F. Volker, 1944. Effect of Cd and F on rats. Proc. Soc. Exp. Biol. Med., 57:189.

Kobayashi, J., 1970. Relation between the "itai-itai" disease and the pollution of river water by cadmium from a mine. Fifth International Water Pollution Research Conference, I-25, 1-7.

Kopp, J.F. and R.C. Kroner, 1967. Trace metals in waters of the United States. U.S. Dept. of the Interior, Federal Water Pollution Control Admin., Cincinnati, Ohio.

Lieber, M. and W.E. Welsch, 1954. Contamination of ground water by cadmium. Jour. Amer. Water Works Assn., 46:51.

Lowman, F.G., et al., 1971. Accumulation and redistribution of radionuclides by marine organisms. In: Radioactivity in the Marine Environment, National Academy of Sciences, Washington, D. C., p. 161.

Page, A.L., et al. (In Press). Cadmium absorption and growth of various plant species as influenced by solution cadmium concentration. Jour. of Environmental Quality.

Pickering, Q.H. and M.H. Gast, 1972. Acute and chronic toxicity of cadmium to the fathead minnow (Pimephales promelas). Jour. Fish. Res. Bd. Can., 29:1099.

Rehwoldt, R., et. al., 1973. The acute toxicity of some heavy metal ions toward benthic organisms. Bull. Environ. Contam. and Tox., 10:291.

Schroeder, H.A., et al., 1961. Abnormal trace metals in man: Cadmium. Jour. Chron. Dis., 14:236.

Spehar, R., 1974. Cadmium and zinc toxicity to Jordanella floridae. M.S. Thesis, University of Minnesota, Duluth.

Thorpe, V.J. and P.S. Lake, 1974. Toxicity bioassays of cadmium on selected freshwater invertebrates and the interaction of cadmium and zinc on the freshwater shrimp, Paratya tasmaniensis, Riek. Aust. Jour. Mar. Freshwater Research, 25:97.

Warnick, S.L. and H.L. Bell, 1969. The acute toxicity of some heavy metals to different species of aquatic insects. Jour. Water Poll. Control Fed., 41:280.

Yamagata, N. and I. Shigematsu, 1970. Cadmium pollution in perspective. Bull. Inst. Public Health, 19:1.

CHLORINE

CRITERIA:

Total residual chlorine:

2.0 µg/l for salmonid fish;
10.0 µg/l for other freshwater and marine organisms

INTRODUCTION:

Elemental chlorine is a greenish-yellow gas that is highly soluble in water. It reacts readily with many inorganic substances and all animal and plant tissues. The denaturing effect of chlorine on animal and plant tissues forms the basis for its use as an effective water or wastewater disinfectant. When chlorine dissolves in water, it hydrolyzes according to the reaction: $Cl_2 + H_2O \rightarrow HOCl + H^+ + Cl^-$. Unless the concentration of the chlorine solution is above 1000 mg/l, all chlorine will be in the form of HOCl or its dissociated ions H^+ and OCL^-. The HOCl is a weak acid and is dissociated according to the equation, $HOCl \rightleftharpoons H^+ + OCl^-$.

The ratio between HOCl and OCl^- is a function of the pH, with 96 percent HOCl remaining at pH 6, 75 percent at pH 7, 22 percent at pH 8, and 3 percent at pH 9. The relationship of HOCl to pH is significant as the undissociated form appears to be the bactericidal agent in the use of chlorine for disinfection (Moore, 1951).

Chlorine is not a natural constituent of water. Free available chlorine (HOCl and OCl^-) and combined available chlorine (mono- and

di-chloramines) appear transiently in surface or ground waters as a result of disinfection of domestic sewage or from industrial processes that use chlorine for bleaching operations or to control organisms that grow in cooling water systems.

RATIONALE:

Chlorine, in the free available form reacts readily with nitrogenous organic materials to form chloramines. These compounds are toxic to fish. Chloramines have been shown to be slightly less toxic to fish than free chlorine, but their toxicity is considered to be close enough to free chlorine that differentiation is not warranted (Merkens, 1958). Since the addition of chlorine or hypochlorites to water containing nitrogenous materials rapidly forms chloramines, toxicity in most waters is related to the chloramine concentration. The toxicity to aquatic life of chlorine will depend upon the concentration of total residual chlorine, which is the amount of free chlorine plus chloramines. The persistence of chloramines is dependent on the availability of material with a lower oxidation-reduction potential.

In field studies in Maryland and Virginia, Tsai (1973) observed that downstream from plants discharging chlorinated sewage effluents, the total numbers of fish species were drastically reduced with the stream bottom clear of aquatic organisms characteristically present in unchlorinated wastewater discharges. No fish were found in water with a chlorine residual above 0.37 mg/l and the species diversity

index reached zero at 0.25 mg/l. A 50 percent reduction in the species diversity index occurred at 0.10 mg/l. Of the 45 species of fish observed in the study areas, the brook trout and brown trout were the most sensitive and were not found at residual chlorine levels above about 0.02 mg/l. In studies of caged fish placed in waters downstream from chlorinated wastewater discharges, the Michigan Department of Natural Resources (1971) reported that 50 percent of the rainbow trout died within 96 hours at residual chlorine concentrations of 0.014 to 0.029 mg/l. Some fish died as far as 0.8 miles (1.3 km) downstream from the outfall.

Studies described by Brungs (1973) indicate that salmonids are the most sensitive fish to chlorine. A residual chlorine concentration of 0.006 mg/l was lethal to trout fry in two days (Coventry, et al., 1935). The 7-day LC_{50} for rainbow trout was 0.08 mg/l with an estimated median period of survival of one year at 0.004 mg/l (Merkens, 1958). Rainbow trout were shown to avoid a concentration of 0.001 mg/l (Sprague and Drury, 1969). Dandy (1972) demonstrated that brook trout had a mean survival time of 9 hours at 0.35 mg/l, 18 hours at 0.08 mg/l and 48 hours at 0.04 mg/l, with mortality of 67 percent after 4 days at 0.01 mg/l. Pike (1971) observed a 50 percent brown trout mortality at 0.02 mg/l within 10.5 hours and 0.01 mg/l with 43.5 hours.

The range of acutely lethal residual chlorine concentrations is narrow for various species of warm water fish. Arthur (1972) determined

96-hour LC_{50} values for the walleye, black bullhead, white sucker, yellow perch, largemouth bass, and the fathead minnow. The observed concentration range was 0.09 to 0.30 mg/l.

Using fathead minnows in a continuous bioassay technique at Michigan treatment plants, Zillich (1972) found that an average concentration of 0.16 to 0.21 mg/l killed all of the test fish and that concentrations as low as 0.07 mg/l caused some mortalities. Pyle (1960) demonstrated a 50 percent mortality of smallmouth bass exposed to 0.5 mg/l within 15 hours. The mean 96-hour LC_{50} value for golden shiners was 0.19 mg/l (Esvelt, et al., 1971). Arthur and Eaton (1971), working with fathead minnows and the freshwater crustacean, Gammarus pseudolimnaeus, in dilute wastewater, found that the 96-hour LC_{50} of total residual chlorine for Gammarus was 0.22 mg/l and that all fathead minnows were dead after 72 hours at 0.15 mg/l. At concentrations of .09 mg/l, all fish survived until the seventh day when the first death occurred. In exposure to 0.05 mg/l residual chlorine, these investigators found reduced survival of Gammarus and at 0.0034 mg/l there was reduced reproduction. Growth and survival of fathead minnows after 21 weeks were not affected by continuous exposure to 0.043 mg/l residual chlorine. The highest level showing no significant effect was 0.016 mg/l. Working with secondary wastewater effluent, Arthur (1972) found that reproduction by Gammarus was reduced by residual concentrations above 0.012 mg/l residual chlorine.

In marine water, 0.05 mg/l was the critical chlorine level for young Pacific salmon exposed for 23 days (Holland, et al., 1960). The

lethal threshold for chinook salmon and coho salmon for a 72-hour exposure was noted by these investigators to be less than 0.01 mg/l chlorine. Studies on the effect of residual chlorine to marine phytoplankton indicate that continuous exposure to 0.10 mg/l reduced primary production by 70 percent while 0.2 mg/l for 1.5 hours reduced primary production by 25 percent (Carpenter, et al., 1972). Laboratory studies on ten species of marine phytoplankton indicate that a 50 percent reduction in growth rate occurred at chlorine concentrations of 0.075 to 0.250 mg/l during a 24-hour exposure period (Gentile, et al., 1973). Oysters are sensitive to chlorine concentrations of 0.01 to 0.05 mg/l and react by reducing pumping activity. At chlorine concentrations of 1.0 mg/l, effective pumping could not be maintained (Galtsoff, 1946).

Chlorine residuals of 10 ug/l have been shown to kill adult salmonid fish in a period of several days in fresh water and the fry of these species have been killed in chlorine residuals of 6 ug/l. The criterion of 2 ug/l chlorine should afford protection to this group of fish when exposed on a continuing basis. Considering the data presented above, a criterion of 10 ug/l should afford protection to other freshwater fish and marine aquatic life (Brungs, In Press). Brungs (1973) reported that aquatic organisms may tolerate short-term exposure to higher levels of residual chlorine than the concentrations which have adverse chronic effects. Basch and Thuchan (In Press) have shown that repeated daily exposure at these levels will have toxic effects on aquatic life.

REFERENCES CITED:

Arthur, J.W., 1972. Progress Report, Natl. Water Quality Lab., Environmental Protection Agency, Duluth, Minn.

Arthur, J.W., and L.G. Eaton, 1971. Chloramine toxicity to the amphipod, Gammarus pseudolimnaeus, and the fathead minnow, Pimephales promelas. Jour. Fish. Res. Bd. Can., 28:1841.

Basch, R.E. and J.G. Truchan, In Press. Toxicity of chlorinated power plant condenser cooling waters to fish. U.S. Environmental Protection Agency, Ecological Research Series. Grant R800700 Michigan Water Resources.

Brungs, W.A., 1973. Effects of residual chlorine on aquatic life. Jour. Water Poll. Cont. Fed., 45:2180.

Brungs, W.A., In Press. Effects of wastewater and cooling water chlorination on aquatic life. U.S. Environmental Protection Agency, Ecological Research Series.

Carpenter, E.J., et al., 1972. Cooling water chlorination and productivity of entrained phytoplankton. Marine Biology, 16:57.

Coventry, F.L., et al., 1935. The conditioning of a chloramine treated water supply for biological purposes. Ecology, 16: 60.

Dandy, J.W.T., 1972. Activity response to chlorine in brook trout, Salvelinus fontinalis. Can. Jour. Zool., 50:405.

Esvelt, L.A., et al., 1971. Toxicity removal from municipal wastewaters. Vol. IV, A study of toxicity and biostimulation in San Francisco Bay-Delta Water. SERL Rept. No. 71-7, San Eng. Res. Lab., Univ. of California, Berkeley.

Galtsoff, P.S., 1946. Reactions of oysters to chlorination. Fish and Wildlife Service, USDI Research Report 11.

Gentile, J.H., et al., 1973. The effects of chlorine on the growth and survival of selected species of estuarine phytoplankton and zooplankton. 36th Ann. Meeting Amer. Soc. Limnol. and Oceanog.

Holland, G.A., et al., 1960. Toxic effects of organic and inorganic pollutants on young salmon and trout. State of Wash., Dept. of Fisheries, Res. Bull. No. 5:198.

Merkens, J.C., 1958. Studies on the toxicity of chlorine and chloramines to the rainbow trout. Water Waste Trt. Jour., (C.B.), 7:150.

Michigan Department of Natural Resources, 1971. Chlorinated municipal waste toxicities to rainbow trout and fathead minnows. Water Poll. Cont. Res. Ser., 18050 GZZ, EPA, Washington, D. C.

Moore, E.W., 1951. Fundamentals of chlorination of sewage and waste. Water and Sewage Works, 98:130.

Pike, D.J., 1971. Toxicity of chlorine to brown trout. New Zealand Wildlife, No. 33.

Pyle, E.A., 1960. Neutralizing chlorine in city water for use in fish distribution tanks. Progressive Fish Cult., 22:30.

Sprague, J.B. and D.E. Drury, 1969. Avoidance reactions of salmonid fish to representative pollutants. In: Advances in water pollution research. Proc. 4th Intl. Conf. Water Poll. Res., Pergamon Press, London, England.

Tsai, C., 1973. Water quality and fish life below sewage outfalls. Trans. Amer. Fish. Soc., 102:281.

Zillich, V.A., 1972. Toxicity of combined chlorine residuals to freshwater fish. Jour. Water Poll. Cont. Fed., 44:212.

CHROMIUM

CRITERIA:

50 ug/l for domestic water supply (health);

100 ug/l for freshwater aquatic life

INTRODUCTION:

Chromium is the seventeenth most abundant nongaseous element in the earth's crust (Schroeder, 1970); its concentration range in the continental crust is 80 to 200 mg/kg, with an average of 125 mg/kg (NAS, 1974a). Although chromium has oxidation states ranging from Cr^{-2} to Cr^{+6}, the trivalent form most commonly is found in nature. Chromium is found rarely in natural waters, ranking twenty-seventh or lower among the elements in seawater and generally is well below 1 ug/l. Kopp (1969) reported that for 1,577 surface water samples collected at 130 sampling points in the United States, 386 samples contained from 1 to 112 ug/l; the mean was 9.7 ug/l chromium. Durum, et al.,(1971) in a similar survey of 700 samples, found that none contained over 50 ug/l of hexavalent chromium and 11 contained over 5 ug/l. Chromium is found in air, soil, some foods, and most biological systems; it is recognized as an essential trace element for humans (NAS, 1974a).

RATIONALE:

The earliest consequences of mild chromium deficiency in experimental animals is a reduced sensitivity of peripheral tissues to insulin; more severe deficiency in rats and mice results in fasting hyperglycemia,

glycosuria, and mild growth retardation that is probably due, at least in part, to reduced insulin activation. Glucose intolerance is a common human problem, and one of its many possible causes is chromium deficiency (NAS, 1974a).

The biological activity of chromium, i.e., its effect as an essentia metal, is restricted to its trivalent state, Cr^{3+}. The Cr^{3+} ion forms complexes that are stable at or below pH 4, but which readily hydrolyze at "high" pH values; resulting in the formation of polynucleate bridge complexes. At the normal pH of blood, Cr^{3+} exists in large, insoluble macro-molecules which precipitate and become biologically inert; Cr^{3+} must therefore be supplied as a complex of suitable stability in order to be utilized.

Because of the present inadequacy of knowledge about the forms and biological availability of chromium in foods, it is not possible to quantify human dietary requirements. It is known that adult urinary loss is 5 to 10 ug/day, and since this constitutes the major portion of the daily loss, at least this much must be replaced to maintain balance. Based on rat studies indicating that absorption of various Cr^{3+} compounds can range from below 1 percent to 25 percent of a given dose, and assuming that the human case is similar, a dietary intake of 20 to 500 ug/day would balance urinary losses. It is estimated that daily chromium intakes in the U.S. are marginal and vary from 5 to 100 ug (WHO, 1973); it seems unlikely that any Cr^{3+} ingested via public drinking water would be appreciably assimilated.

No harmful effects were observed when food or water containing moderate amounts of Cr^{3+} was administered to laboratory animals, e.g., cats that were fed chromic phosphate or oxydicarbonate at 50 to 1000 mg/day for 80 days, or rats drinking water containing 25 mg/l for a year or 5 mg/l throughout their lives (NAS, 1974a).

Hexavalent chromium, on the other hand, is irritating and corrosive to the mucous membranes; it is absorbed via ingestion, through the skin, and by inhalation, and is toxic when introduced into laboratory animals systemically (NAS, 1974a). Knowledge of the harmful human health effects of hexavalent chromium has been obtained almost entirely from occupational health effects. Lung cancer, ulceration and perforation of the nasal septum, and a variety of other respiratory complications and skin effects have been observed.

Symptoms of excessive dietary intake of chromium in man are unknown, and chromium deficiency is of greater nutritional concern than over-exposure. Chromium is the only element whose tissue concentration appears to decline with increasing age in the U.S. population; lung concentrations do not decline with age, suggesting that lung chromium is not in equilibrium with that in the rest of the body (NAS, 1974a).

Berg and Burbank (1972) compared concentrations of eight carcinogenic trace metals in water supplies (after Kopp and Kroner, 1967) with State cancer mortalities for major U.S. water basins, and no significant correlations were found for chromium. It should be emphasized that these data are not conclusive.

The U.S. Public Health Service Drinking Water Standard (USPHS, 1962) states that the presence of hexavalent chromium in excess of 0.05 mg/l shall constitute grounds for rejection of the supply, and no harmful human health effects have been reported at this level. The NAS/NAE Committee on Water Quality Criteria recommended (NAS, 1974) that public water supply sources for drinking water contain no more than 0.05 mg/l total chromium, largely on the basis that lifetime tolerable levels of chromate ion are not known for man. There are insufficient data on the effect of the defined treatment process on chromate chromium removal. Chromium as Cr^{3+} is not likely to be present in waters of pH 5 and above because the hydrated oxide is very sparingly soluble.

A family of four individuals is known to have drunk water for a period of 3 years at a chromium level of 0.45 mg/l without known effects on their health as determined by a single medical examination (Davids and Lieber, 1951). A study was designed by MacKenzie, et al. (1958) to determine the toxicity of hexavalent and trivalent chromium ions to rats at various drinking water levels. After one year at levels of 0.45 to 25 mg/l, this study showed no evidence of toxic response in body weight, food consumption, blood changes, or mortality. However, significant accumulation of chromium occurred in the tissues at concentrations greater than 5 mg/l. Recent studies (Naumova, 1965) demonstrated that 0.1 mg of $K_2Cr_2O_7$/kg enhances the secretory and motor activity of the dog intestine.

From these and other studies (Gross and Heller, 1946; Brard, 1935; Conn, et al., 1932; Schroeder, et al., 1963; Schroeder, et al., 1963a), it appears that a concentration of 50 ug/l of chromium in domestic water supply incorporates a reasonable safety factor to avoid any hazard to human health.

In addition, the possibility of dermal effects from bathing in water containing 50 ug/l chromium would likewise appear remote, although chromium is recognized as a potent skin sensitizer. Domestic water supplies should, therefore, contain no more than 50 ug/l total chromium.

Fish appear to be relatively tolerant of chromium, but some aquatic invertebrates are quite sensitive. Toxicity varies with species, chromium oxidation state, and pH. Pickering and Henderson (1966) conducted static bioassays with four warm water fish species; fathead minnows, Pimephales promelas; bluegill, Lepomis macrochirus; goldfish, Carassius auratus; and guppies, Poecilia reticulata. They obtained soft water (hardness - 20 mg/l; alkalinity - 18 mg/l; pH - 7.5) 96-hour LC_{50} values for hexavalent chromium ranging from 17.6 mg/l for fathead minnows to 118 mg/l for bluegill; hard water (hardness - 360 mg/l; alkalinity - 300 mg/l; pH - 8.2) 96-hour LC_{50} values for hexavalent chromium ranging from 27.3 mg/l for fathead minnows to 133 mg/l for bluegill. Their 96-hour LC_{50} trivalent chromium values (chromium potassium sulfate) ranged from 3.33 mg/l for guppies to 7.46 mg/l for bluegill in soft water. The LC_{50} for fathead minnows exposed to potassium chromate in soft water was 45.6 mg/l.

Pickering (NAS, 1974) found 96-hour LC_{50} and safe hexavalent chromium concentrations of 33 mg/l and 1.0 mg/l, respectively, for fathead minnows in hard water. Pickering's Cr^{3+} data for fathead minnows in hard water showed a 96-hour LC_{50} of 27 mg/l and a safe concentration of 1.0 mg/l. Benoit (In Press) obtained soft water (45 mg/l hardness) 96-hour LC_{50} and safe hexavalent chromium values of 59 mg/l and 0.2 mg/l, respectively, for brook trout, Salvelinus fontinalis, and 69 and 0.2 mg/l, respectively, for rainbow trout, Salmo gairdneri.

Olson (1958) found the growth and survival of chinook salmon, Oncorhynchus kisutch, alevins and juveniles to be significantly reduced at hexavalent chromium concentrations of 0.2 mg/l. He saw no detrimental effects on salmon alevins at trivalent chromium concentrations of 0.2 mg/l. Beisinger and Christensen (1972) reported a 16 percent reproductive impairment in Daphnia magna in soft water (45 mg/l as $CaCO_3$) at a concentration of 0.33 mg/l of trivalent chromium.

The data available indicate hexavalent chromium to be somewhat more toxic than trivalent chromium in the case of chinook salmon, and since significant effects were seen on fish at 0.2 mg/l of hexavalent chromium a recommended criterion of 0.10 mg/l should provide adequate protection for both freshwater invertebrates and fish.

Lowman, et al. (1971) reported marine chromium concentration factors of 1,600 in benthic algae, 2,300 in phytoplankton, 1,900 in zooplankton, 440 in molluscan soft parts, and 70 in fish muscle.

Raymont and Shields (1964) reported chromium threshold toxicity levels of 5 mg/l for small prawns, Leander squilla, 20 mg/l (as Na_2CrO_4) for the shore crab, Carcinas maenus, and 1 mg/l for the polychaete, Nereis virens. Doudoroff and Katz (1953) found that mummichogs, Fundulus heteroclitus, tolerated 200 mg/l $K_2Cr_2O_4$ in seawater for over a week.

Holland, et al. (1960) reported that 31.8 mg/l chromium (as K_2CrO_4) in sea water was 100 percent fatal to coho salmon, Oncorhynchus kisutch, and Gooding (1954) reported that 17.8 mg/l hexavalent chromium in sea-water was toxic to the same species.

Clendenning and North (1960) showed that 5.0 mg/l hexavalent chromium reduced photosynthesis by 50 percent in the giant kelp, Macrocystis pyrifera, during 4 days of exposure.

Based on the foregoing discussion of the marine toxicity of chromium, its accumulation at all trophic levels, and the sensitivity of lower aquatic forms to chromium, the freshwater criterion should protect marine populations.

REFERENCES CITED

Benoit, D.A., 1976. Toxic effects of hexavalent chromium on brook trout (Salvelinus fontinalis) and rainbow trout (Salmo gairdneri) Water Research, (In Press).

Berg, J.W. and F. Burbank, 1972. Correlations between carcinogenic trace metals in water supplies and cancer mortality. Ann. N.Y. Acad. Sci., 199: 249.

Biesinger, K.E. and G.M. Christensen, 1972. Effects of various metals on survival, growth, reproduction and metabolism of Daphnia magna. Jour. Fish Res. Bd. of Canada, 29: 1691.

Brard, D., 1935. Toxicological study of certain chromium derivatives. II. J. Pharm. Chem., 21: 5.

Clendenning, K.A. and W.J. North, 1960. Effect of wastes on the giant kelp, Macrocystis pyrifera. Proc. 1st Int'l. Conf. on Waste Disposal in the Marine Environment. Pergamon Press, New York, p. 82.

Conn, L.W., et al., 1932. Chromium toxicology. Absorption of chromium by the rat when milk containing chromium was fed. Amer. Jour. Hyg., 15:760.

Davids, H.W. and M. Lieber, 1951. Underground water contamination by chromium wastes. Water and Sewage Works, 98: 528.

Doudoroff, P. and M. Katz, 1953. Critical review of literature on the toxicity of industrial wastes and their components on fish. The Metals as Salts. Sew. and Ind. Wastes, 25:802.

Durum, W.H., et al., 1971. Reconnaissance of selected minor elements in surface waters of the United States, October 1970. U.S. Geological Survey Circular 643. Wash., D.C. U.S. Geological Survey.

Gooding, D., 1954. Pollution research, toxicity studies. Sixty-fourth Annual Report, Washington State Department of Fish. Olympia, Washington.

Gross, W.G. and V.G. Heller, 1946. Chromates in animal nutrition. Jour. Ind. Hyg. Toxicol., 28:52.

Holland, G.A., et al., 1960. Toxic effects of organic and inorganic pollutants on young salmon and trout. Washington Department of Fish Res. Bull. No. 5. Seattle, Washington.

Kopp, J.F., 1969. The occurrence of trace elements in water, In: D.D. Hemphill, Ed., Trace Substances in Environmental Health - III. Proc. Univ. of Missouri's 3rd Annual Conference on Trace Substances in Environmental Health. Univ. of Missouri, Columbia, p. 59.

Kopp, J.F. and R.C. Kroner, 1967. Trace metals in waters of the United States. U.S. Department of the Interior, Federal Water Pollution Control Administration, Cincinnati, Ohio.

Lowman, F.G., et al., 1971. Accumulation and redistribution of radionuclides by marine organisms, In: Radioactivity in the Marine Environment. National Academy of Sciences, Wash., D.C. p. 161.

MacKenzie, R.D., et al., 1958. Chronic toxicity studies. II: Hexavalent and trivalent chromium administered in drinking water to rats. A.M.A. Archives of Industrial Health, 18: 232.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

National Academy of Sciences, 1974a. Chromium. U.S. Government Printing Office, Washington, D.C.

Naumova, M.K., 1965. Changes in the secretory and motor functions of the intestine following administration of potassium dichromate. Gig. Tr. Prof. Zabol., 9: 52.

Olson, P.A., 1958. Comparative toxicity of Cr(VI) and Cr(III) in salmon. Hanford Biology Research Annual Report for 1957, January 10, 1958. HW-53500, p. 215.

Pickering, Q.H., and C. Henderson, 1966. The acute toxicity of some heavy metals to different species of warm water fishes. Int'l. Jour. Air-Water Pollution, 10: 453.

Raymont, J.E.G., and J. Shields, 1964. Toxicity of copper and chromium in the marine environment, In: E.A. Pearson, Ed., Advances in Water Pollution Research, Proc. 1st Int'l. Conf. Macmillan Co., New York, Vol. 3, p. 275.

Schroeder, H.A., et al., 1963. Effects of chromium, cadmium and other trace metals on the growth and survival of mice. Jour. Nutrition, 80:39.

Schroeder, H.A., et al., 1963a. Effects of chromium, cadmium and lead on the growth and survival of rats. Jour. Nutrition, 80:48.

Schroeder, H.A., 1970. Chromium. Air Quality Monograph #70-15. American Petroleum Institute, Wash., D.C.

USPHS, 1962. Public Health Service Drinking Water Standards (rev. 1962). PHS Publication 956. Wash., D.C.

WHO, 1973. Chromium, In: Trace Elements in Human Nutrition. World Health Organization Technical Report Series No. 532. Geneva, p. 20.

FECAL COLIFORM BACTERIA

CRITERION:

Bathing Waters

Based on a minimum of not less than five samples taken over a 30-day period, the fecal coliform bacterial level should not exceed a log mean of 200 per 100 ml, nor should more than 10 percent of the total samples taken during any 30 day period exceed 400 per 100 ml.

Shellfish Harvesting Waters

Not to exceed a median fecal coliform bacterial concentration of 14 MPN per 100 ml with not more than 10 percent of samples exceeding 43 MPN per 100 ml for the taking of shellfish.

INTRODUCTION:

It was recognized even before the microbial etiology of disease was known, that water can serve as a medium for the transfer of disease. The cause-effect relationship of disease transmission and specific fecally-associated microbes was initially defined by Von Fritsch in 1880, when he identified Klebsiella spp. in human feces. Further confirmation of the

relationship between fecally-associated microbes and potential disease was developed by Escherich when he described Bacillus coli (Escherichia coli) as an indicator of pollution (Wolf, 1972; Guarraia, 1972). Since these early observations, the role of biological indicator organisms in defining water quality has become essential and addresses three categories generally: to identify environmental changes; to quantify pollution levels; or to be used in laboratories to study under controlled conditions phenomena which could be extrapolated to the environment (Butler, et al., 1972).

Microbiological indicators have been used to determine or indicate the safety of water for drinking, swimming and shellfish harvesting. As our knowledge concerning microbiology has increased, so has our understanding of the complex interrelationship of the various organisms with disease. Viruses causing a number of diseases and non-fecally associated bacteria causing infections of the ear, eye, nose and throat all have been isolated from water (Bonde, 1974; Scarpino, 1974). The relationship between numbers of specific disease causing organisms in water and the potential for transmission of disease remains elusive since the number of organisms required to cause disease varies depending upon the organism, the host, and the manner in which the bacteria and host interact. For example, in some instances a single cell of Salmonella, or a single plaque-forming unit (PFU) may be all that is necessary to cause a

disease; however, in other instances the numbers of bacteria necessary to cause an illness may be up to 10^6 to 10^7 or even more viable organisms. The use to which water is put (i.e., swimming or drinking) the type of water, (marine or fresh), and the geographical location are all factors to be weighed in determining safe microbiological criteria.

Ideally, a microbiological indicator organism should fulfill all of the following criteria: (1) It should be applicable to all types of water, (2) it should be present whenever pathogens are present with a survival time equal to that of the hardiest enteric pathogen, and (3) the indicator should not reproduce in contaminated waters thus resulting in inflated values (Scarpino, 1974). Unfortunately, no such indicator organisms are known. Use has been made of coliform or fecal coliform bacteria as indicators of pollution.

Bacteria of the coliform group are considered the primary indicators of fecal contamination and are one of the most frequently applied indicators of water quality. The coliform group is made up of a number of bacteria including the genera Klebsiella, Escherichia, Serratia, Erwinia and Enterobacteria. Total coliform bacteria are all gram negative asporogenous rods and have been associated with feces of warmblooded animals.

and with soil. The fecal coliform bacteria, which comprise a portion of the total coliform group, are able to grow at 44.5° C and ferment lactose producing acid and gas. Use of fecal coliform bacteria has proven to be of more sanitary significance than the use of total coliform bacteria because they are restricted to the intestinal tract of warm-blooded animals and are now used to define water quality for swimming. Arguments have been advanced for the use of Escherichia coli as the indicator of choice for fresh fecal pollution (Bonde, 1966). However, the methods for its identification require time, complex biochemical reactions, and experienced microbiologists, and the information gained by the additional work and expense involved in its identification may not provide sufficient additional information above the elevated temperature test (for fecal coliform bacteria) to warrant its use.

Enterococci were recognized as early as 1890 as being indicators of recent fecal pollution from warm-blooded animals (Geldreich and Kenner, 1969). The enterococci possess many characteristics which make them an ideal indicator system since they do not multiply in the aquatic environment and are serologically characteristic. However, there are numerous biotypes, and no good standardized method is available for biochemical testing. Such methodology is essential since identification is based primarily upon biochemical characterization.

Additional microbial systems have also been proposed as potential indicators of fecal pollution. Clostridium perfringens, an anaerobic spore former, has been associated frequently with sewage pollution. However, in a suspension of fecal material the ratio of Cl. perfringens to E. coli may differ from that in the effluent from a sewage treatment plant, in seawater or in sludge. Also the spores of Cl. perfringens are resistant and will vegetate upon culturing, hence it would be difficult to associate their presence with recent pollution.

Presence of microorganisms other than fecal coliform bacteria may also be indicative of water quality; however, the strict correlation between a pollutant problem other than fecal pollution and the numbers of the indicator organism is not always clear. The pseudomonads that are comprised of numerous free living saprophytes found in both fresh and marine waters have only one of its members as a known pathogen, Pseudomonas aeruginosa, which may be an indicator of pollution from the presence of warm-blooded animals (Ringem and Drake, 1952; Taylor, 1968; Reitler and Seligaman, 1957). While the number of P. aeruginosa in sewage usually is quite low and occurs too sporadically to be of value as an indicator organism for fecal pollution (Bonde, 1974), this organism may be an indicator of human pollution other than fecal.

Bacterial pathogens, which occur in bathing waters and may cause disease even when not ingested are Klebsiella pneumoniae and Pseudomonas aeruginosa.

RATIONALE:

Bathing Waters

Pollution of aquatic systems by the excreta of warmblooded animals creates public health problems for man and animals and potential disease problems for aquatic life. It is known that enteric microbial pathogens may inhabit the gut of most warmblooded animals and are shed in feces. The presence of bacterial, viral, protozoan, and possibly fungal species which are either pathogens for or possess the potential to infect man and other organisms is indicated by the presence of the fecal coliform group of bacteria. Thus, the number of fecal coliforms present is indicative of the degree of health risk associated with using the water for drinking, swimming, or shellfish harvesting.

Arguments against the use of fecal coliform bacteria to define swimming quality in waters have noted the paucity of epidemiological evidence linking fecal coliform levels in bathing waters and the incidence of disease (Moore, 1959; 1971).

A problem of potential medical significance is the transfer of characteristics which will alter the resistance of pathogenic bacteria, i.e. the R-factor (RTF - resistance transfer factor), to certain antibiotics, heavy metals and ultraviolet light. The significance of this, while not fully known, suggests that human pathogens in water may become resistant to common antibiotic therapy once the bacteria infect man or animals. One example of this problem has been already reported. Recently an outbreak of typhoid fever was found to be caused by Salmonella typhi containing the chloramphenical and amphicillin resistant factor (Datta and Olearte, 1974; Olearte and Galindo, 1973).

Disease transmission via the aquatic route including drinking water, recreational water, and seafood from polluted water, has been and continues to be a problem. Presently, the indicator systems considered to be the most practical are the coliform and the fecal coliform groups. Additional microbiological problems, which can be anticipated by the presence of specific bacteria or viruses, are recognized. Correlation between human pollution sources and the numbers and significance of the microbial system in question remain elusive. Nevertheless, as the relationships become clear additional criteria for water quality will evolve. Berg (1974) has shown that Polio Virus I at a concentration of 2 plaque forming units (PFU), a standard means for measuring virus concentrations in tissue culture, will cause disease in 67 percent of the uninnoculated population.

However, the lack of epidemiological correlation between fecal coliform levels in coastal swimming waters and the incidence of disease may not have validity in fresh waters and it does not take into account non-reported diseases which may develop as an unrecognized result of swimming in polluted waters. Epidemiological evidence is but one consideration in setting microbiological criteria. The presence of fecal coliform bacteria indicates degradation of water quality and a relative risk of disease transmission.

In studies conducted in Lake Michigan at Chicago, Illinois (Smith, et al., 1951), in an inland river (Smith and Woolsey, 1952) and in tidal waters at Long Island, New York (Smith and Woolsey, 1961), a statistically significant increase in the incidence of illness was observed among swimmers who used the Lake Michigan beaches on selected days and the Ohio River beach of poorest water quality. The mean total coliform bacteria content of fresh waters was 2,300 per 100 ml and 2,700 per 100 ml, respectively. No relationship between the total coliform levels and swimming-related diseases was found at the ocean beach. These studies demonstrated that an appreciably higher overall illness incidence may be expected among swimmers when compared to non-swimmers, but the data are inconclusive. The data provide a positive correlation between total coliform numbers and the increased risk of disease associated with swimming in these waters. The diseases found were infections of the eye, ear,

nose, and throat, as well as intestinal ailments (Stevenson, 1953).

Outbreaks of typhoid fever (Salmonella typhi) have been associated with swimmers in "heavily" polluted beaches in western Australia (Kovacs, 1959; Snow, 1959). In this case, a sewer outfall pipe was located one mile from the bathing beach where the typhoid outbreaks occurred and the sewer was overloaded because of rapid population growth. Use of a log mean of 200 fecal coliform bacteria per 100 ml, with the provision that 10 percent of the total samples during a thirty-day period not exceed 400 fecal coliforms per 100 ml, allows for variations in environmental conditions such as shifts in wind direction, current flow, and tidal fluctuations. At levels above the 200 fecal coliforms per 100 ml the risk of exposure to pathogenic microbes increases. Correlation between the fecal coliform level and microbial pathogen levels is the important relationship, since the fecal coliforms themselves serve as an indicator of the quality of water in relation to fecally associated microbial pathogens. Direct demonstration of the microbial pathogens is not always feasible.

Detection of Salmonella has shown that in fecally polluted marine waters the level may vary between 1 and 1,000 per liter (McCoy, 1964). Occurrences of viruses in ocean waters at levels of 60 plaque-forming units (PFU) per liter have been found

(Shuval, et al., 1971). It is estimated that these values may represent only 10 to 50 percent of the total viral pathogens present because of the limited recovery efficiency of the methods used. Also, other viruses associated with feces may be present but may not grow in the tissue culture systems used.

Another important consideration in determining the safe microbiological criterion is the minimum dosage necessary to infect a bather. As few as 3 to 5 organisms of S. typhosa have been reported to cause infection, whereas, 1×10^5 to 1×10^7 cells for other Salmonella serotypes may be required to produce disease. Similarly, massive concentrations of cells of enteropathogenic E. coli have been reported necessary to produce infections in adult volunteers. Alteration of gastric function either by raising the pH in the stomach or by facilitating gastric emptying can reduce this dosage several orders of magnitude. Recent data from experiments with adult volunteers indicate that the infectious dose for Shigella flexneri 2a is less than 200 cells (Geldreich, 1974). Although enteric viruses are found in relatively small numbers in polluted waters, their occurrence could be hazardous since the minimum infective dose for humans has not been firmly established. For children, the minimum infective dose may be 1 or 2 PFU (Plotkin and Katz, 1967), while for adults the minimum infective dose may be the same or higher.

However, less is known concerning the minimal infective dose for pathogenic bacteria that cause ear, eye, nose, and throat ailments (Geldreich, 1974).

Use of fecal coliform bacteria as a single parameter for monitoring recreational water quality must ultimately relate to the probable occurrence of waterborne pathogens. Currently, the only relationship which has been developed between the fecal coliform indicator and waterborne pathogens is that of Salmonella to fecal coliform density Data which have been developed indicate a sharp increase in the frequency of Salmonella detection when fecal coliform densities are above 200 organisms per 100 ml of freshwater. When there are over 200 fecal coliforms per 100 ml, Salmonella isolation should approach 100 percent frequency. Data from estuarine waters were grouped to include the level of 1 to 70 fecal coliforms that is of interest to investigators of shellfish waters. For this range, below 70 fecal coliforms per 100 ml, only 6.7 percent of the 184 Salmonella examinations were positive. At the 200 fecal coliform level, the 28.4 percent occurrence of Salmonella in estuarine waters was essentially identical with data compiled from freshwater environments. In polluted estuarine waters containing fecal coliforms ranging from 201 to above 2,000, a recovery of

Salmonella was seen 60 percent of the time. The recovery rate of Salmonella in estuarine waters is lower than that in fresh waters. In fresh water, Salmonella were recovered 85 to 98 percent of the time, in the range of from 201 to 2,000 fecal coliforms. The lower value for isolation of Salmonella from estuarine waters may be related to limitations of Salmonella methodology (Geldreich, 1972). It must be noted that any projection of the qualitative recoveries of Salmonella into a comparison with fecal coliform quantification has recognized limitations. Such projections do nevertheless suggest that the 200 fecal coliform per 100 ml limit for recreational waters is a useful water quality value.

Evaluation of the microbiological suitability for marine and fresh waters should be based upon the fecal coliform levels. As determined by either the multiple-tube fermentation for marine water or the membrane filter method for fresh water, and based upon a minimum of not less than five samples taken over not more than a 30-day period, the fecal coliform bacterial level should not exceed a log mean of 200 per 100 ml, nor should more than 10 percent of total samples during any 30-day period exceed 400 per 100 ml.

Shellfish

Shellfish, being filter feeders, require a high quality of water in order to be microbiologically safe for human consumption

as either raw or partially cooked. Fecal coliform bacteria, other bacterial pathogens, and viruses found in water and sediments are concentrated by shellfish, depending upon temperature, density of pathogens, currents, depth, water chemistry, and shellfish feeding activity (Van Donsel and Geldreich, 1971; Metcalf and Stiles, 1968). Once concentration of pathogens occurs, flushing of microorganisms will not necessarily occur at the same rate (Janssen, 1974; Kelly and Arcisy, 1954). Because of the established relationship between coliform levels and enteric pathogens, shellfish waters historically have been classified on the basis of total coliform levels.

An attempt by the National Shellfish Program has been made to correlate fecal coliform bacteria to enteric pathogens. Ideally, any specific fecal coliform bacterial limit for shellfish should be based on a correlation with pathogenic occurrences in the aquatic environment and with epidemiological evidence of increased health risk among shellfish consumers. However, these data are not available and calculations have been based on health risks incurred through increased pathogen occurrence in waters of differing levels of fecal contamination. Recent data in shellfish growing waters have shown that Salmonella occurred in 4.7 percent of water samples having fecal coliform densities of 1 to 29 per 100 ml (Slanetz et al., 1974). Oysters growing in these waters accumulated from 33 to 2200 fecal coliform bacteria

per 100 grams of shellfish meat, with Salmonella occurrence at 6.1 percent.

Shellfish contamination is intensified further by the normal accumulation of waterborne organisms in bottom sediments through the action of sedimentation. Investigation of both the overlying water and bottom sediments from lakes and streams has indicated a 100- to 1000-fold increase in fecal coliform densities at the water-sediment interface (Van Donsel and Geldreich, 1971). Enteric viruses (Coxsackie B3) in the bottom sediment of shellfish growing waters along the New Hampshire estuary have been found when the fecal coliform densities were as low as 10 organisms per 100 ml in the overlying waters (Slanetz et al., 1965)

Indicators of fecal pollution more specific than the total coliforms in shellfish waters have been sought. Candidate organisms or groups of organisms include E. coli and the fecal coliform group. The fecal coliforms have a higher positive correlation with fecal contamination from all warm blooded animals than does E. coli (Geldreich, 1974). Usually E. coli is the most numerous bacterium of the fecal coliform group; however, under some conditions other fecal coliforms may predominate (Sears et al., 1950). Analysis of data comparing the correlation of fecal coliforms and E. coli to fecal coliform sources confirms the fact that the fecal coliforms

reflect sanitary quality of water. In comparing results of the fecal coliform test to those of the E. coli procedure in shellfish waters, E. coli was reported to range from 75 to 90 percent of the fecal coliform population with the fecal coliform bacteria giving a 96.6 percent correlation (Presnell, 1974). Therefore, the use of the fecal coliform test avoids the undesirable risk of excluding some fecal contamination.

The microbiological criterion for shellfish water quality has been accepted by international agreement to be 70 total coliforms per 100 ml, using a median MPN, with no more than 10 percent of the values exceeding 230 total coliforms. No evidence of epidemiological outbreak from consumption of raw shellfish which were grown in waters meeting this bacteriological criterion has been demonstrated. This standard has proven to be a practical limit when supported by sanitary surveys of the growing waters, acceptable quality in shellfish meats and good epidemiological evidence. However, evidence from field investigations suggests that not all total coliform occurrences can be associated with fecal pollution (Gallagher, et al., 1969). Thus, attention has been directed toward the adoption of the fecal coliform test to measure more precisely the occurrence and magnitude of fecal pollution in shellfish growing waters.

A series of studies was initiated by the National Shellfish Sanitation Program and data relating the occurrence of total

coliforms to numbers of fecal coliforms were compiled. Information was received from 15 States and 2 Canadian provinces and was arbitrarily divided into 4 geographical areas: northwest, southern states, mid-Atlantic, and northeast. A total of 3,695 coliform values and 3,574 fecal coliform values were included in the tabulations. The prime objective was to determine the correlation between the two indicator groups and secondarily, to determine whether or not coliform data could be used as a basis for evaluation of a potential fecal coliform standard.

The data show that a 70 coliform MPN per 100 ml at the 50th percentile was equivalent to a fecal coliform MPN of 14 per 100 ml. The data, therefore, indicate that a median value for a fecal coliform standard is 14 and the 90th percentile should not exceed 43 for a 5 tube, 3 dilution method (Hunt and Springer, 1973).

Evaluation of the microbiological suitability of waters for recreational taking of shellfish should be based upon the fecal coliform bacterial levels. When possible, samples should be collected under those conditions of tide and reasonable rainfall when pollution is most likely to be maximum in the area to be classified. The median fecal coliform value should not exceed an MPN of 14 per 100 ml and not more than 10 percent of the samples should exceed an MPN of 43.

REFERENCES CITED

Berg, G. 1974. Reassessment of the virus problem in sewage and in surface and renovated waters. Sixth International Water Poll. Res. California. Pergamon Press.

Bonde, G.J., 1966. Bacteriological methods for the estimation of water pollution. Hlth. Lab. Sci. 3:124.

Bonde, G.J., 1974. Bacterial indicators of sewage pollution. International Symposium on Discharge of Sewage from Sea Outfalls. Pergamon Press.

Butler, P., et al., 1972. Test, monitoring and indicator organisms. In: A Guide to Marine Pollution, E.D. Goldberg, ed. Gordon and Beach, N.Y.

Datta, N. and J. Olearte, 1974. R-Factors in strains of Salmonella typhi and Shigella dysenteri I isolated during epidemics in Mexico: Classification by compatibility. Antimicrobial Agents and Chemotherapy 5: 310.

Gallagher, T.P., et al., 1969. Pollution affecting shellfish harvesting in Mobile Bay, Alabama, Tech. Servs. F.W.P.C.A. Southeast Water Lab., Athens, Georgia.

Geldreich, E.E., et al., 1969. Fecal-coliform-organism medium for the membrane filter technique. Amer. Water Works Assn., 51:208.

Geldreich, E.E. and B.A. Kenner, 1969. Concepts of fecal streptococci in stream pollution. Jour. Water Poll. Contr. Fed. 41: R 336.

Geldreich, E.E., 1972. Buffalo Laboratory recreational water quality: A study in bacteriological data interpretation. Water Res. 6:912.

Geldreich, E.E., 1974. Microbiological criteria concepts for coastal bathing waters. Ocean Mgt. (In Press).

Geldreich, E.E., 1974. Monitoring marine waters for microbiological quality. WHO Conference - Scientific Aspects of Marine Pollution. Geneva Switzerland.

Guarraia, L. J. 1972. Brief literature review of Klebsiella as pathogens. In seminar on the significance of Fecal Coliforms in Industrial Waste. E.P.A. T. D. 3, National Field Investigations Center, Denver, Colo. p. 94.

Hunt, D.A. and J. Springer, 1974. Preliminary report on "A comparison of total coliform and fecal coliform values in

shellfish growing areas and a proposed fecal coliform growing area standard. Presented at 8th National Shellfish Sanitation Workshop. (FDA. Wash. D.C.).

Janssen, W.A., 1974. Oysters; Retention and excretion of three types of human water-borne disease bacteria. Health Lab. Sci. 11:20.

Kelly, C.B. and W. Arcisy, 1954. Survival of enteric organisms in shellfish. Pub. Health Repts. 69:1205.

Kovacs, N., 1959. Enteric fever in connection with pollution of seawater. Western Australia. Report of the Commissioner of Public Health for the year 1958.

Metcalf, T.G. and W.C. Stiles, 1968. Viral pollution of shellfish in estuary waters. Jour. San. Eng. Div. Proc. Amer. Soc. Civ. Eng. 94:595.

McCoy, J.H., 1964. Salmonella in crude sewage, sewage effluent and sewage polluted natural waters. Abs. in Water Poll. Res. pp 206.

Moore, B. 1959. Sewage contamination of coastal bathing waters in England and Wales. Jour. Hyg., 57:435.

Moore, B., 1971. The Health hazards of pollution in microbial aspects of pollution. Sykes and Skinner, Eds., Academ. Press. London: pp 11-32.

Olearte, J. and E. Galindo, 1973. Salmonella typhi resistance to chloramphenical, amphicillin and other antimicrobial agents: Strains isolated during an extensive typhoid fever epidemic in Mexico. Antimicrobial Agents and Chemotherapy 4:597.

Plotkin, S.A. and M. Katz, 1967. Minimal infective doses of viruses for man by the oral route. In: Transmission of Viruses by the Water Route. G. Berg, Ed., John Wiley Interscience, N.Y. p. 155.

Presnell, N, 1974. Discussion of fecal coliforms for shellfish growing waters. Proc. 7th National Shellfish Sanitation Workshop Oct. 21 - 22, 1971. Ratcliff and Wilt, Eds., FDA. Wash., D.C.

Reitler, R. and R. Seligaman, 1957. Pseudomonas aeruginosa in drinking water. Jour. Appl. Bact. 20:145.

Ringem, L.M. and C.H. Drake, 1952. A study of the incidence of Pseudomonas aeruginosa from various national sources. Jour. Bact. 64:841.

Scarpino, D., 1974. Human enteric viruses and bacteriophages as indicators of sewage pollution. International Symposium from Sea Outfalls. Pergamon Press.

Sears, H.J. et al., 1950. Persistence of individual strains of Escherchia coli in the intestinal tract of man. Jour. Bact. 59:293.

Shuval, H.J., et al., 1971. Natural inactivation processes of viruses in seawater. Proc. Natural Specialty Conf. on Disinfection. Amer. Soc. of Civil Engineers, N.Y.

Slanetz, L.W., et al., 1965. Correlation of coliform and fecal streptococci indices with the presence of Salmonella and enteric viruses in sea water and shellfish. Adv. in Water Pollution Res. 2nd International Confr., Tokyo. 3:17.

Smith, R.S., et al., 1951. Bathing water quality and health. I. Great Lakes (U.S. Public Health Service, Cincinnati, Ohio).

Smith, R.S. and T.D. Woolsey, 1952. Bathing water quality and health. II. Inland river and pool. (U.S. Public Health Service, Cincinnati, Ohio).

Smith, R.S. and T.D. Woolsey, 1961. Bathing water quality and Public Health. III. Coastal Waters (U. S. Public Health Service, Cincinnati, Ohio).

Snow, D.J.R., 1959. Typhoid and City Beach. Western Australia. Report of the Commissioner of Public Health for the Year 1958, p. 52.

Stevenson, A.H., 1953. Studies of bathing water quality and health. Amer. Jour. Public Health 43:529.

Taylor, E.W., 1968. Forty-second report of Director of Water Examination, 1965-66, Metropolitan Water Board, London, p. 117.

Van Donsel, D.J. and E.E. Geldreich, 1971. Relationships of Salmonellae to fecal coliforms in bottom sediments. Water Res. 5:1079.

Wolf, H.W., 1972. The coliform count as a measure of water quality. In Water Pollution Microbiology. R. Mitchell, Eds., Wiley-Interscience.

COLOR

CRITERIA:

Waters shall be virtually free from substances producing objectionable color for aesthetic purposes;

the source of supply should not exceed 75 color units on the platinum-cobalt scale for domestic water supplies; and

increased color (in combination with turbidity) should not reduce the depth of the compensation point for photosynthetic activity by more than 10 percent from the seasonally established norm for aquatic life.

INTRODUCTION:

Color in water principally results from degradation processes in the natural environment. Although colloidal forms of iron and manganese occasionally are the cause of color in water, the most common causes are complex organic compounds originating from the decomposition of naturally-occurring organic matter (AWWA, 1971). Sources of organic material include humic materials from the soil such as tannins, humic acid and humates; decaying plankton; and other decaying aquatic plants. Industrial discharges may contribute similar compounds, for example those from the pulp and paper and tanning industries. Other industrial discharges may contain brightly colored substances such as those from certain processes in textile and chemical industries.

Surface waters may appear colored because of suspended matter which comprises turbidity. Such color is referred to as apparent color and is differentiated from true color caused by colloidal humic materials (Sawyer, 1960). Natural color is reported in color "units" which generally are determined by use of the platinum-cobalt method (Standard Methods, 1971).

There is no general agreement as to the chemical composition of natural color, and in fact, the composition may vary chemically from place to place (AWWA, 1971). Black and Christman (1963a) characterized color-causing colloids examined as aromatic, polyhydroxy, methoxy carboxylic acids. Shapiro (1964) characterized color-causing constituents as being dialyzable and composed of aliphatic, polyhydroxy, carboxylic acids with molecular weights varying from less than 200 to approximately 400. The colloidal fraction of color exists in the 3.5 to 10 mμ diameter range (Black and Christman, 1963b). These same authors summarized other characteristics of color observed in laboratory studies of natural waters: color is caused by light scattering and fluorescence rather than absorption of light energy and pH affects both particle size of the color-causing colloids and the intensity of color itself.

RATIONALE:

Color in water is an important constituent in terms of aesthetic considerations. To be aesthetically pleasing, water should be virtually free from substances introduced by man's activities which produce objectionable color. "Objectionable color" is defined to be a significant increase over natural background levels. Non-natural colors such as dyes should not be perceptible by the human eye as such colors are especially objectionable to those who receive pleasure by viewing water in its natural state. Because of the extreme variations in the natural background amount of color, it is meaningless to attempt numerical limits. The aesthetic attributes of water depend on one's appreciation of the water setting.

The effects of color on public water supplies also are principally aesthetic. The 1962 Drinking Water Standards (PHS, 1962) recommended that color in finished waters should not exceed 15 units on the platinum-cobalt scale. Water consistently can be treated using standard coagulation, sedimentation and filtration processes to reduce color to substantially less than 15 color units when the source water does not exceed 75 color units (AWWA, 1971; NAS, 1974).

The effects of color in water on aquatic life principally are to reduce light penetration and thereby generally reduce photosynthesis by phytoplankton and to restrict the zone for aquatic vascular plant growth.

The light supply necessary to support plant life is dependent on both intensity and effective wave lengths (Welch, 1952). In general, the rate of photosynthesis increases with the intensity of the incident light. Photosynthetic rates are most affected in the red region and least affected in the blue-violet region of incident light (Welch, 1952). It has been found that in colored waters the red spectrum is not a region of high absorption so that the effective penetration, and therefore the intensity for photosynthesis, is not as restricted as are other wave lengths. It should be emphasized that transmission of all parts of the spectrum is affected by color, but the greatest effect is on the shortwave or blue end of the spectrum (Birge and Juday, 1930). In highly colored waters (45 to 132 color units) Birge and Juday (1930) measured the light transmission as a percentage of the incident level and found very little blue, 50 percent or less yellow, and 100 to 120 percent red.

The light intensity required for some aquatic vascular plants to photosynthetically balance the oxygen used in respiration may be 5 percent of full sunlight during maximum summer illumination periods (NTAC, 1968). As much as 10 percent of the incident light may be required for plankton to likewise photosynthetically produce sufficient oxygen to balance their respiration requirements (NTAC, 1968). The depth at which such a compensation point is reached, called the compensation depth, delineates the zone of effective photosynthetic oxygen production. To maintain satisfactory biological conditions, this depth cannot be substantially reduced.

Industrial requirements as related to water color have been summarized (NAS, 1974). Table 2 lists the maximum value used as a source of water for various industries and industrial uses. Through treatment, such waters can be made to meet almost any industrial requirement.

Table 2 .

Maximum color of surface waters that have been used as sources for industrial water supplies.

Industry or Industrial Use	Color, units
Boiler make-up	1,200
Cooling water	1,200
Pulp and paper	360
Chemical and allied products	500
Petroleum	25

REFERENCES CITED:

American Water Works Association, 1971. Water quality and treatment. 3rd edition, McGraw-Hill Book Co., New York.

Birge, E.A. and C. Juday, 1930. A second report on solar radiation and inland lakes. Trans. Wisc. Acad. Science, Arts, Let., 25: 285.

Black, A.P. and R.F. Christman, 1963a. Characteristics of colored surface waters. Jour. Amer. Water Works Assn., 55: 753.

Black, A.P. and R.F. Christman, 1963b. Chemical characteristics of fulvic acids. Jour. Amer. Water Works Assn., 55: 897.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

National Technical Advisory Committee to the Secretary of the Interior, 1968. Water quality criteria. U.S. Government Printing Office, Washington, D. C.

Public Health Service, 1962. Drinking water standards, 1962. PHS Publ. No. 956, U.S. Government Printing Office, Washington, D. C.

Sawyer, C.N., 1960. Chemistry for sanitary engineers, McGraw-Hill Book Co., New York.

Shapiro, J., 1964. Effect of yellow organic acids on iron and other metals in water. Jour. Amer. Water Works Assn., 56: 1062.

Standard Methods for the Examination of Water and Wastewater, 13th Edition, 1971. American Public Health Assn., Washington, D. C.

Welch, P.S., 1952. Limnology. McGraw-Hill Book Co., Inc., New York.

COPPER

CRITERIA:

1.0 mg/l for domestic water supplies (welfare).

For freshwater and marine aquatic life, 0.1 times a 96-hour LC50 as determined through nonaerated bioassay using a sensitive aquatic resident species.

INTRODUCTION:

Copper occurs as a natural or native metal and in various mineral forms such as cuprite and malachite. The most important copper ores are sulfides, oxides, and carbonates. Copper has been mined and used in a variety of products by man since prehistoric times. Uses for copper include electrical products, coins, and metal plating. Copper frequently is alloyed with other metals to form various brasses and bronzes. Oxides and sulfates of copper are used for pesticides, algicides, and fungicides. Copper frequently is incorporated into paints and wood preservatives to inhibit growth of algae and invertebrate organisms, such as the woodborer, Teredo, on vessels.

Copper is an essential trace element for the propagation of plants and performs vital functions in several enzymes and a major role in the synthesis of chlorophyll. A shortage of copper in soil may lead to chlorosis which is characterized by yellowing of plant leaves. In copper deficient soils it may be added as a trace nutrient supplement to other fertilizers.

Copper is required in animal metabolism. It is important in invertebrate blood chemistry and for the synthesis of hemoglobin. In some invertebrate organisms a protein, hemocyanin, contains copper and serves as the oxygen-carrying mechanism in the blood. An overdose of ingested copper in mammals acts as an emetic.

In examining over 1500 surface water samples from the United States, Kopp and Kroner (1967) found soluble copper in 74 percent of the samples with an average concentration of 15 µg/l and a maximum concentration of 280 µg/l of copper. The average concentration of copper in seawater approximately 3.0 µg/l (Mero, 1964).

RATIONALE:

Concentrations of copper found in natural waters are not known to have an adverse effect on humans. Prolonged oral administration of excessive quantities of copper may result in liver damage, but water supplies seldom have sufficient copper to effect such damages. Young children require approximately 0.1 mg/day of copper for normal growth and the daily requirement for adults was estimated to be about 2 mg/day (Sollman, 1957). Copper in excess of 1 mg/l may impart some taste to water. Because of a possible undesirable taste in drinking water at higher concentrations, a limit of 1 mg/l is recommended.

Testing precipitated copper in lake bottom muds resulting from copper sulfate application to control nuisance algae, Mackenthun and Cooley (1952) concluded that the toxic limit to a midge, Tendipides plumosus,

and a fingernail clam, Pisidium idahoense was about 9,000 mg/kg of copper in mud on a dry weight basis.

The toxicity of copper to aquatic life is dependent on the alkalinity of the water as the copper ion is complexed by anions present, which in turn affect toxicity. At lower alkalinity copper is generally more toxic to aquatic life. Other factors affecting toxicity include pH and organic compounds. Relatively high concentrations of copper may be tolerated by adult fish for short periods of time; the critical effect of copper appears to be its higher toxicity to young or juvenile fish.

Doudoroff and Katz (1953), in reviewing literature on the acute toxicity of copper, concluded that in most natural fresh waters in the United States copper concentrations below 25 µg/l as copper evidently are not rapidly fatal for most common fish species. In acute tests Jones (1964) reported that copper sulfate in soft water (12 mg/l $CaCO_3$) was toxic to rainbow trout at 60 µg/l copper. In very hard water (320 mg/l $CaCO_3$) the toxic concentration was 600 µg/l copper. A summary of acute toxicity data is given in Table 3. In checking this table the reader should consider the species tested, pH, alkalinity, and hardness if alkalinity is not given (in most natural waters alkalinity parallels hardness). In general the salmonids are very sensitive and the centrarchids are less sensitive to copper.

TABLE 3

The acute toxicity (24-, 48-, 96-hr TL50 values) of copper to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Rainbow trout (Salmo gairdneri)	4 month old (7g)	Copper	96	FT*	11.6-12.4	0.02 Cu	6.8-7.0	17-26	20-25	(6)
Rainbow trout	Juveniles	Copper sulfate	24 96	S** S	15 -15.6 15	1.25 Cu 0.89 Cu	7.3-7.4	200	290	(5)
Rainbow trout	—	Copper	48	S	15	0.4-0.5 Cu	7.6	200	320	(2)
Brook trout (Salvelinus fontinalis)	14 month old	Copper	96	FT	12±1	0.10 Cu	7.5	41.6	45	(9)
Atlantic salmon (Salmo salar)	Juveniles	Copper	96	S	18-21	.125 Cu	6.5-6.7	4	8-10	(22)
Atlantic salmon	Juveniles	Copper	24	FT	15	.028 Cu	7.1-7.5	—	20	(16)
Chinook salmon (Oncorhynchus tshawytscha)	At hatch 1 month old	Copper Copper	96 96	FT FT	11.1-12.0 10.8-12.5	0.031 Cu 0.018 Cu	6.8-7.0	17-26	20-25	(6)

*FT - flow-through bioassay
**S - static bioassay

The acute toxicity (24-, 48-, 96-hr TL50 values) of copper to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Chinook salmon	-	Cupric nitrate	<96	s	9.5	0.178 Cu^{++}	7.75-7.85	-	80	(8)
Silver salmon (Oncorhynchus kisutch)	-	Copper sulfate	96	s	10 10	0.45 Cu^{++}	7.75-7.85	-	40	(8)
Bluegills (Lepomis macrochirus)	1-2 g	$CuSO_4 \cdot 5H_2O$	24 48 96	s	25	0.86 $CuSO_4 \cdot 5H_2O$ 0.74 $CuSO_4 \cdot 5H_2O$ 0.66 $CuSO_4 \cdot 5H_2O$	7.5	18	20	(13)
Bluegills	Juveniles (35 g)	$CuSO_4$	96	FT	20±1	1.1 Cu	7-8	43	45	(1)
Bluegills	5-9 cm	Copper chloride	96	s	20	0.71 Cu^{++}	≤5.3	3-6	45-47	(18)
		Copper sulfate	96	s	20	0.77 Cu^{++}				
Bluegills	-	Copper sulfate	96	s	--	0.2 Copper sulfate	7.4	18	20	(17)
Bluegills	1-2 g	$CuSO_4 \cdot 5H_2O$	24 48 96	s	25	10.7 $CuSO_4 \cdot 5H_2O$ 10.2 $CuSO_4 \cdot 5H_2O$ 10.2 $CuSO_4 \cdot 5H_2O$	7.5	300	360	(13)
Bluegills	-	Copper sulfate	96	s	-	10.2 Copper sulfate	8.2	360	400	(17)

The acute toxicity (24-, 48-, 96-hr TL50 values) of copper to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Bluegills	-	Copper sulfate (synthetic water)	48	s	20	7.0 Cu	8.3	72	101.2	(19)
Fathead minnow (Pimephales promelas)	1-2 g	$CuSO_4 \cdot 5H_2O$	24 48 96	s	25	0.038 $CuSO_4 \cdot 5H_2O$ 0.028 $CuSO_4 \cdot 5H_2O$ 0.023 $CuSO_4 \cdot 5H_2O$	7.5	18	20	(13)
Fathead minnow	-	$CuSO_4$	96	FT s	25±1 25±1	0.075 Cu 0.084 Cu	6.9-7.2	30	31	(11)
Fathead minnow	-	Copper sulfate	96	s	-	0.05 Copper sulfate	7.4	18	20	(17)
Fathead minnow	1 g	Copper cyanide	24 48 96	s	19.5-20.5	1.8 Cu 1.6 Cu 1.2 Cu	6.5	10	14	(7)
Fathead minnow	1-2 g	$CuSO_4 \cdot 5H_2O$	24 48 96	s	25	2.15 $CuSO_4 \cdot 5H_2O$ 1.50 $CuSO_4 \cdot 5H_2O$ 1.45 $CuSO_4 \cdot 5H_2O$	7.5	300	360	(13)
Fathead minnow	20-69 mm	Copper	96	s	4-30	0.56-0.99 Cu	8-8.5	90-230	120-336	(4).

The acute toxicity (24-, 48-, 96-hr TL50 values) of copper to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Fathead minnow	10-15 mm	$CuSO_4$	96	S FT	16-25	0.430 Cu 0.470 Cu	7.9	161	200	(10)
Fathead minnow	56 mm 1.6 g	Copper	96	FT	24±1	0.44 Cu	-	-	200	(12)
Fathead minnow	-	Copper sulfate	96	S	-	1.4 Copper sulfate	8.2	360	400	(17)
Banded killifish (Fundulus diaphanus)	≤20 cm	$Cu(NO_3)_2$	24 48 96	S	17	1.50 Cu^{++} 0.92 Cu^{++} 0.86 Cu^{++}	7.8	-	5.3	(14)
Banded killifish	≤20 cm	$Cu(NO_3)_2$	24 48 96	S	28	1.30 Cu^{++} 0.98 Cu^{++} 0.84 Cu^{++}	8.0	-	55	(15)
Striped bass (Morone saxatilis)	≤20 cm	$Cu(NO_3)_2$	24 48 96	S	17	8.3 Cu^{++} 6.2 Cu^{++} 4.3 Cu^{++}	7.8	-	53	(14)
Striped bass	≤20 cm	$Cu(NO_3)_2$	24 48 96	S	28	8.4 Cu^{++} 6.6 Cu^{++} 4.0 Cu^{++}	8.0	-	55	(15)
Striped bass	Fingerlings (2.7 g)	Copper sulfate	24 48 96	S	21	1.5 Copper sulfate 1.15 Copper sulfate 0.62 Copper sulfate	8.2	64	35	(21)

The acute toxicity (24-, 48-, 96-hr TL50 values) of copper to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Pumpkin-seed (Lepomis gibbosus)	≤20 cm	$Cu(NO_3)_2$	24 48 96	s	17	3.8 Cu^{++} 2.9 Cu^{++} 2.4 Cu^{++}	7.8	-	53	(14)
Pumpkin-seed	≤20 cm	$Cu(NO_3)_2$	24 48 96	s	28	3.5 Cu^{++} 2.9 Cu^{++} 2.7 Cu^{++}	8.0	-	55	(15)
White perch (Morone) americana	≤20 cm	$Cu(NO_3)_2$	24 48 96	s	17	11.8 Cu^{++} 8.0 Cu^{++} 6.2 Cu^{++}	7.8	-	53	(14)
White perch	≤20 cm	$Cu(NO_3)_2$	24 48 96	s	28	11.5 Cu^{++} 7.9 Cu^{++} 6.4 Cu^{++}	8.0	-	55	(15)
Carp (Cyprinus carpio)	≤20 cm	$Cu(NO_3)_2$	24 48 96	s	17	2.10 Cu^{++} 1.00 Cu^{++} 0.81 Cu^{++}	7.8	-	53	(14)
Carp	≤20 cm	$Cu(NO_3)_2$	24 48 96	s	28	1.90 Cu^{++} 1.20 Cu^{++} 0.80 Cu^{++}	8.0	-	55	(15)

The acute toxicity (24-, 48-, 96-hr TL50 values) of copper to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Brown bullhead (Ictalurus nebulosus)	Juveniles	Copper sulfate	96	FT	23	0.18 Cu	7.2-8.2	156	200	(3)
Bluntnose minnow (Pimephales notatus)	84 mm 6.7 g	Copper	96	FT	24±1	0.29 Cu	7.9	154	200	(12)
Stoneroller (Campostoma anomalum)	60 mm	Copper	96	FT	24±1	0.30 Cu	7.9	154	200	(12)
Creek chub (Semotilus atromaculatus)	64 mm 4.0 g	Copper	96	FT	24±1	0.31 Cu	7.9	154	200	(12)
Blacknose dace (Rhinichthys atratulus)	47 mm 1.1 g	Copper	96	FT	24±1	0.33 Cu	7.9	154	200	(12)
Rainbow darter (Etheostoma caeruleum)	41 mm	Copper	96	FT	24±1	0.33 Cu	7.9	154	200	(12)

The acute toxicity (24-, 48-, 96-hr TL50 values) of copper to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Goldfish (Carassius auratus)	1-2 g	$CuSO_4 \cdot 5H_2O$	24 48 96	s	25	0.094 $CuSO_4 \cdot 5H_2O$ 0.043 $CuSO_4 \cdot 5H_2O$ 0.036 $CuSO_4 \cdot 5H_2O$	7.5	18	20	(13)
Guppy (Lebistes reticulatus)	0.1-0.2 g	$CuSO_4 \cdot 5H_2O$	24 48 96	s	25	0.130 $CuSO_4 \cdot 5H_2O$ 0.073 $CuSO_4 \cdot 5H_2O$ 0.036 $CuSO_4 \cdot 5H_2O$	7.5	18	20	(13)
Mosquito fish (Gambusia affinis	-	Copper sulfate	24 48 96	s	24-27	122 Copper sulfate 84 Copper sulfate 75 Copper sulfate	6.1-8.1	<100	-	(20)

References Cited in Table 3:

1. Benoit, D.A. (1975). Chronic effects of copper on survival, growth, and reproduction of the bluegill Lepomis macrochirus. Trans. Amer. Fish. Society, 104:353.

2. Brown, V.B., 1968. The calculation of the acute toxicity of mixtures of poisons to rainbow trout. Water Res. 2:723.

3. Brungs, W.A., et al., 1973. Acute and long-term accumulation of copper by the brown bullhead Ictalurus nebulosus. Jour. Fish. Res. Bd. Can., 30:583.

4. Brungs, W.A., et al., 1974. Acute and chronic toxicity of copper to the fathead minnow (Pimephales promelas Rafinesque) in a surface water of variable water quality. (In press).

5. Calamari, D., and R. Marchetti, 1973. The toxicity of mixtures of metals and surfactants to rainbow trout. Water Res. 7:1453.

6. Chapman, G.A. Unpublished data available at the National Water Quality Laboratory, Duluth, Minnesota.

7. Doudoroff, P., 1956. Some experiments on the toxicity of complex cyanides to fish. Sewage Ind. Wastes. 28:1020.

8. Holland, G.A., et al., 1960. Toxic effects of organic pollutants on young salmon and trout. State of Washington Dept. of Fisheries Res. Bull. No. 5. 264 p.

9. McKim, J.M., and D.A. Benoit, 1971. Effects of long-term exposures of copper on survival, growth, and reproduction of brook trout Salvelinus fontinalis. Jour. Fish. Res. Bd. Can. 28:655.

10. Mount, D.I., 1968. Chronic toxicity of copper to fathead minnows (Pimephales promelas Rafinesque). Water Res. 2:215.

11. Mount, D.I., and C.E. Stephan, 1969. Chronic toxicity of copper to the fathead minnow (Pimephales promelas) in soft water. Jour. Fish. Res. Bd. Can. 26:2449.

12. National Water Quality Laboratory (NWQL), 6201 Congdon Boulevard, Duluth, Minnesota (Unpublished data).

13. Pickering, Q.H., and C. Henderson, 1966. The acute toxicity of some heavy metals to different species of warm water fishes. Int. Jour. Air and Water Poll., 10:453.

14. Rehwoldt, R., et al., 1971. Acute toxicity of copper, nickel, and zinc ions to some Hudson River fish species. Bull. Environ. Contam. Toxicol. 6:445.

15. Rehwoldt, R., et al., 1972. The effect of increased temperature upon the acute toxicity of some heavy metal ions. Bull. Environ. Contam. Toxicol. 8:91.

16. Sprague, J.B., 1964. Lethal concentrations of copper and zinc for young Atlantic salmon. Jour. Fish. Res. Bd. Can. 21:17.

17. Tarzwell, C.M., and C. Henderson, 1960. Toxicity of less common metals to fishes. Ind. Wastes. 5:12.

18. Trama, F.B., 1954. The acute toxicity of some common salts of sodium, potassium, and calcium to the common bluegill (Lepomis macrochirus Rafinesque). Proc. Acad. Nat. Sci., Philadelphia. 106:185.

19. Turnbull, H., et al., 1954. Toxicity of various refinery materials to freshwater fish. Ind. Eng. Chem. 46:324.

20. Wallen, I.E., et al., 1957. Toxicity to Gambusia affinis of certain pure chemicals in turbid water. Sewage Ind. Wastes. 29:695.

21. Wellborn, Jr., T.L., 1969. The toxicity of nine therapeutic and herbicidal compounds to striped bass. The Prog. Fish. Cult. 31:27.

22. Wilson, R.C.H., 1972. Prediction of copper toxicity in receiving waters. Jour. Fish. Res. Bd. Can. 29:1500.

Sprague and Ramsay (1965) determined the level beyond which the organism can no longer survive for an indefinite period of time for juvenile Atlantic salmon, Salmo salar, at 17° C. For copper in a water with a total hardness of 14 mg/l as $CaCO_3$ this level was 32 µg/l. At 28 µg/l copper there were no salmon deaths in 168 hours.

Sprague (1964) found that Atlantic salmon, Salmo salar, tended to avoid a concentration of copper as low as 4.0 µg/l.

Mount and Stephan (1969) reported that in chronic tests with fathead minnows in water with a hardness of 200 mg/l as $CaCO_3$, 33 µg/l copper did not affect survival or the physical appearance of the fish but did prevent spawning. No effects were noted at 14.5 µg/l copper. In soft water with a hardness of 30 mg/l as $CaCO_3$, the no-effect level for the fathead minnow, Pimephales promelas, was about 10.6 ug/l copper. These investigators reported application factors of between 0.13 and 0.22 and between 0.03 and 0.08, respectively, for soft and hard water. The maximum concentration of copper having no detectable effect on the brown bullhead, Ictalurus nebulosus, in 600 days in water with a hardness of 202 mg/l as $CaCO_3$ was determined to be between 16 and 27 µg/l (Brungs et al., 1973). Benoit (1975) exposed bluegills (Lepomis macrochirus) to copper in soft water for 22 months and found the "no-effect" level to be 21 ug/l copper.

McKim and Benoit (1971) conducted chronic tests with brook trout, Salvelinus fontinalis, exposed to copper in water with a mean alkalinity of 41.6 mg/l as $CaCO_3$. A copper concentration of 17.5 µg/l did not adversely affect survival, growth, or spawning of adult brook trout or the hatchability of the eggs; however this concentration affected the survival and growth of juveniles. The "no-effect" level established

for the young brook trout was 9.5 µg/l copper. In a second generation exposure of brook trout to copper, McKim and Benoit (1974) found that exposure to "sublethal concentrations of copper from yearling through spawning to 3 month juveniles" was sufficient to establish a "no-effect" concentration (i.e., the "no-effect" level noted above caused no adverse effects on the second generation). These authors reported an application factor of between 0.17 and 0.10 of the 96-hour LC50 value for the brown trout only.

Biesinger and Christensen (1972) found a 16 percent reproductive impairment at 22 µg/l copper for Daphnia magna in chronic (3-week) tests in water with a total hardness of 45.3 mg/l as $CaCO_3$. The 3-week LC50 was 44 µg/l copper. The total copper concentration having no effect on Campeloma decisum, Physa integra, and Gammarus pseudolimnaeus in chronic studies was between 8.0 and 14.8 µg/l in water with a total hardness of 45.3 mg/l as $CaCO_3$ (Arthur and Leonard, 1970).

The concentration of copper that has been associated experimentally with no harmful effect for several aquatic species is about 5 to 15 µg/l. This is very close to the average ambient freshwater concentration now found where copper occurs in measurable quantity. In waters with high alkalinity and/or with much organic material many species will be able to tolerate higher ambient copper concentrations. In such cases, the criterion should not exceed 0.1 of the 96-hour LC50 (the approximate mean application factor from tests reported above) as determined through bioassays using sensitive resident species.

Copper is present in sea water at a concentration of approximately 3 µg/l but copper added to the marine environment is readily precipitated in the alkaline and saline environment. Toxicity of copper to fishes in

marine waters has not been studied, but for Nereis virens, a polychaete invertebrate, the toxic threshold for copper was 100 µg/l (Raymont and Shields, 1964). Copper is toxic to oysters at concentrations above 100 µg/l (Galtsoff, 1932). Clendenning and North (1960) found inhibition of photosynthesis in the giant kelp, Macrocystis pyrifera, at copper concentrations of 60 µg/l. This commercially important marine plant is used for several industrial processes and for important food additives. In areas where this plant is especially significant it may be prudent to establish a restrictive copper criterion.

Adult softshell clams, Mya arenaria, were the most sensitive marine macroorganisms tested in static copper toxicity bioassays. LC0, LC50 and LC100 values after 168 hours at 30 o/oo salinity and 22°C were 25, 35 and 50 ug/l, respectively. At 17°C, these values were 75, 86 and 100 ug/l, respectively, for the same time period (unpublished manuscript). Copper is selectively concentrated over zinc by adult softshell clams, Mya arenaria. Concentrations of greater then 20 ug/l are fatal after exposure of several weeks (Pringle, et al., 1968). The 9-day LC50 for newly hatched Fundulus heteroclitus larvae was 160 ug/l (Gentile, 1975).

These data are insufficient to derive a satisfactory criteria number, but it is apparent that copper does exhibit toxicity to the few species tested. Therefore, it is recommended that .1 of the 96-hour LC50 for a sensitive aquatic species present be adopted.

The only known industrial use of water affected by copper is the production of textiles which requires a minimum concentration (as low as 10 µg/l) for some select processes. If needed, the low concentrations found in natural water may be reduced readily by various forms of treatment including coagulation and precipitation or ion exchange resins. Thus, no water quality criterion for copper in industrial water supplies is proposed.

The minimum reported concentration of copper that begins to exhibit toxicity to some agricultural plants is 100 µg/l, which is considerably more than the average found in the Nation's waters. The adverse effect of copper on plants can be overcome readily by proper management through irrigation or by the addition of materials such as lime, phosphate. fertilizers, or iron salts to the soil (Reuther and Labanauskas, 1966). No criterion is proposed for copper in water used for agricultural purposes.

Copper sulfate has been used widely in the control of algae in water supply reservoirs and in recreational lakes. At Madison, Wisconsin, its use for such purposes began in 1918 (Mackenthun and Cooley, 1952). Copper sulfate was used first as a fish poison in 1914, and in 1953 it was used experimentally by the Massachusetts Division of Fisheries and Game in accelerating fish movements in ponds which were being fyke-trapped for the removal of overabundant pan and weed species. It has since been used as a standard accessory tool in netting operations (Tompkins and Bridges, 1958).

REFERENCES CITED:

Arthur, J.W. and E.N. Leonard, 1970. Effects of copper on Gammarus pseudolimnaeus, Physa integra and Campeloma in soft water. Jour. Fish. Res. Bd. Canada, 27:1277

Benoit, D.A., 1975. Chronic effects of copper on survival, growth, and reproduction of the bluegill Lepomis macrochirus. Trans. Amer. Fish. Society, 104:353.

√ Biesinger, K.E. and G.M. Christensen, 1972. Effects of various metals on survival, growth, reproduction, and metabolism of Daphnia magna. Jour. Fish. Res. Bd. Can., 29: 1691.

Brungs, W.A., et al., 1973. Acute and long-term accumulation of copper by the brown bullhead Ictalurus nebulosus. Jour. Fish. Res. Bd. Canada, 30:583.

Clendenning, K.A. and W.J. North, 1960. Effect of wastes on the giant kelp, Macrocystis pyrifera, In: Proceedings, 1st International Conference on Waste Disposal in the Marine Environment. Pergamon Press, N.Y.

Cohen, J.M., et al., 1960. Taste threshold concentrations of metals in drinking water. Jour. Amer. Water Works Assn., 52: 1299.

Doudoroff, R. and M. Katz, 1953. Critical review of literature on the toxicity of industrial wastes and their components to fish, II: The metals, as salts. Sew. and Ind. Wastes, 25: 802.

Eisler, R., Environmental Research Laboratory, Narragansett, R.I., Toxicities of salts of metals mixtures to marine bivalve molluscs. Unpublished manuscript.

Galtsoff, P.A., 1932. Life in the ocean from a biochemical point of view. Jour. Washington Academy Sciences, 22:246.

Gentile, J., 1975. Environmental Research Laboratory, Narragansett, R.I., Semi-Annual Report.

Jones, J.R.E., 1964. Fish and river pollution. Butterworths, London.

Kopp, J.F. and R.C. Kroner, 1967. Trace metals in waters of the United States, October 1, 1962 to September 30, 1967. U.S. Dept. of Interior, Fed. Water Poll. Control Admin., Cincinnati, Ohio.

Mackenthun, K.M. and H.L. Cooley, 1952. The biological effect of copper sulphate treatment on lake ecology. Wisc. Acad. Sci. Arts Lett., 41: 177.

McKim, J.M. and D.A. Benoit, 1971. Effects of long-term exposures to copper on survival, growth and reproduction of brook trout (Salvelinus fontinalis). Jour. Fish. Res. Bd. Can., 28: 655.

Mero, J.L., 1964. Mineral resources of the sea. American Elsevier Publishing Company.

Moore, G.T. and K.F. Kellerman, 1905. Copper as an algicide and disinfectant in water supplies. U.S. Dept. of Agriculture, Bureau of Plant Industry, Bull. No. 76, U.S. Govt. Print. Office, Washington, D. C.

Mount, D.I. and C.E. Stephan, 1969. Chronic toxicity of copper to the fathead minnow (Pimephales promelas) in soft water. Jour. Fish. Res. Bd. Can., 26: 2449.

Pringle, B.H., et al., 1968. Trace metal accumulation by estuarine mollusks. Jour. Sanit. Eng. Div., 94SA3:455.

Raymont, J.E.G. and J. Shields, 1964. Toxicity of copper and chromium in the marine environment, In: Pearson, E.A. (ed), advances in water pollution research, proceedings, 1st International Conference. Macmillan Co., N.Y.

Reuther, W. and C.K. Labanauskas, 1966. Copper, In: Chapman, H.D., ed., diagnostic criteria for plants and soils. Univ. Calif., Berkley.

Sollman, T.H., 1957. A manual of pharmacology, 8th edition. W.B. Saunders Company, Philadelphia, Pennsylvania.

Sprague, J.B., 1964. Avoidance of copper-zinc solutions by young salmon in the laboratory. Jour. Water Poll. Cont. Fed., 36: 990.

Sprague, J.B. and B.A. Ramsay, 1965. Lethal levels of mixed copper-zinc solutions for juvenile salmon. Jour. Fish. Res. Bd. Canada, 22: 425.

Tompkins, W.A. and C. Bridges, 1958. The use of copper sulfate to increase fykenet catches. Progressive Fish-Culturist , 20:16

Warnick, S.L. and H.L. Bell, 1969. The acute toxicity of some heavy metals to different species of aquatic insects. Jour. Fed. Water Poll. Cont. Fed., 41:(Part 1), 280.

CYANIDE

CRITERIA:

5.0 ug/l for freshwater and marine aquatic life and wildlife.

INTRODUCTION:

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

In addition to the simple hydrocyanic acid (HCN), the alkali metal salts such as potassium cyanide (KCN) and sodium cyanide (NaCN), are commonly occurring forms and sources of cyanide. The latter compounds are readily dissolved in water; the extent of HCN formation is pH-dependent. A significant fraction of the cyanide exists as HCN molecules up to a pH of approximately 8, and the fraction increases rapidly as the pH of the solution decreases. When these simple salts dissociate in aqueous solution, the cyanide ion combines with the hydrogen ion to form hydrocyanic acid, which is toxic to aquatic life. Chemically, the cyanide ion behaves similarly to the halide ions--chloride, fluoride, bromide and iodide.

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

A wide variety of organic compounds may contain cyanide functional groups. These compounds belong to a class of organic chemicals called nitriles, few of which dissociate to liberate cyanide ions or molecular HCN. In addition, there are also complex organic acids, alcohols, esters, and amides that contain the cyanide radicals. These organic compounds are used for numerous products or may be a waste by-product. Their toxicity, persistence, and chemistry in the aquatic environment are not well known except for a few specific compounds.

Cyanide toxicity is essentially an inhibition of oxygen metabolism, i.e., rendering the tissues incapable of exchanging oxygen. The cyanogen compounds are true noncumulative protoplasmic poisons (can be detoxified readily) since they arrest the activity of all forms of animal life. Cyanide shows a very specific type of toxic action. It

inhibits the cytochrome oxidase system which facilitates electron transfer from reduced metabolites to molecular oxygen. The ferric iron-porphyrin molecule responsible for the catalytic action of cytochrome oxidase is the reactive site where cyanide combines with ferric(+++) iron atoms to form a reversible complex. Other enzymes containing a metal porphyrin molecule, e.g., peroxidases and xanthine oxidase, are also strongly inhibited by cyanide. Only undissociated HCN inhibits the consumption of oxygen in the tissues, causing cellular asphyxia (histotoxic anoxia) by attaching itself to the iron of the prosthetic group of the enzyme cytochrome oxidase.

Hydrocyanic acid can be absorbed readily and carried in the plasma but does not combine with hemoglobin because its iron atom is divalent (ferrous). Instead, cyanide combines with methemoglobin, a mildly oxidized form of hemoglobin in which the iron atom is trivalent (ferric). Methemoglobin, which cannot carry oxygen, normally represents only a small fraction of the total hemoglobin. Since it forms an irreversible and innocuous complex with cyanide, it is an active cyanide detoxifying agent. Amyl nitrite and other agents can be used to increase the level of methemoglobin to counteract cyanide toxicity. A few of the ways in which cyanide can be metabolized within a pattern of normal physiology are by the production of thiocyanate, reaction with hydroxocobalamin to form the harmless cyanocobalamin, combination with amino acids, oxidation to carbon dioxide and formate, etc. The conversion of only free cyanide and not organically bound cyano groups

to thiocyanate (SCN^-) by action of the enzyme rhodanese is considered to be the primary method of detoxification of cyanide. Rhodanese is absent from blood and skeletal muscle, but is abundant in the liver. Thiocyanate is eliminated irregularly and slowly in the urine.

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

The persistence of cyanide in water is highly variable. This variability is dependent upon the chemical form of cyanide in the water, the concentration of cyanide, and the nature of other constituents. Cyanide may be destroyed by strong oxidizing agents such as permanganates and chlorine. Chlorine is commonly used to oxidize strong cyanide solutions to produce carbon dioxide and ammonia; if the reaction is not carried through to completion, cyanogen chloride may remain as a residual and this material is also toxic. If the pH of the receiving waterway is acid and the stream is well aerated,

gaseous hydrogen cyanide may evolve from the waterway to the atmosphere. At low concentrations or toxicity and with acclimated microflora, cyanide may be decomposed by microorganisms in both anaerobic and aerobic environments or waste treatment systems.

Hydrocyanic acid probably is the most toxic form of cyanide in water. The ratio of hydrocyanic acid to total cyanide is quite variable. Under natural conditions this variation is due to fluctuations in pH. Photochemical action can also affect this ratio. Fluctuation in pH is caused by acid wastewater discharges, and photosynthetic and respiration cycles of aquatic plant life affect the formation, stability and toxicity of HCN. Since such chemical and physical conditions will dictate the form of cyanide, the cyanide criteria must be based on the concentration of total cyanide present in the water.

RATIONALE:

Cyanide ingested by humans at quantities of 10 mg or less per day is not toxic and is biotransformed to the less toxic thiocyanate. Lethal toxic effects from the ingestion of water containing cyanide occur only when cyanide concentrations are high and overwhelm the detoxifying mechanisms of the human body. Continuous long-term consumption of up to nearly 5 mg per day has shown no injurious effects (Bodansky and Levy, 1923).

A review of the available pertinent data on the acute toxicity of simple cyanides to fish reveals the following minimum lethal (threshold) concentrations of free cyanide from data obtained from experiments ranging from 12 minutes to 10 days: brook trout, Salvelinus fontinalis (Karsten, 1934); rainbow trout, Salmo gairdneri (Herbert and Merkens, 1952); brown trout, Salmo trutta (Burdick, et al., 1958); smallmouth bass, Micropterus dolomieu (Burdick, et al., 1958); bluegills, Lepomis macrochirus (Doudoroff, et al., 1966); and fathead minnows, Pimephales promelas (Doudoroff, 1956), are reported to be 50, 70 (60 determined concentration), 70, 104, 150, and 180 ug/l as cyanide, respectively.

Research at the University of Minnesota has revealed that the minimum lethal (threshold) concentrations, as determined from continuous flow bioassays in which routine analyses for cyanide were performed, are generally lower than the above reported values. The threshold concentrations, expressed as mg/l cyanide, for the brook trout, Salvelinus fontinalis; bluegill, Lepomis macrochirus; and fathead minnow, Pimephales promelas, were determined to be 0.057 at 10°C, 0.104 at 25°C, and 0.120 at 25°C, respectively (Broderius, 1974).

In review it can be concluded that free cyanide concentrations in the range from 50 to 100 ug/l as cyanide have proven eventually

fatal to many sensitive fishes, and levels much above 200 ug/l probably are rapidly fatal to most fish species.

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

The tolerance of fish to cyanide solutions that are rapidly lethal has been reported to decrease with a rise in temperature. This increased toxicity may be explained in part by the increased metabolism of fish at higher temperatures.

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

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

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

The effects of cyanide on marine life have not been investigated adequately to determine separate water quality criteria, but based on the physiological mechanisms of cyanide, toxicity to marine life probably is similar to that of freshwater life. Since marine waters generally are alkaline, the toxicity of cyanide should be less than in fresh waters where pH fluctuations occur more readily and frequently. Thus, an additional factor exists to provide a margin of safety and compensation for a lack of specific data on which to base the criterion for marine aquatic life.

Cyanide has not been reported to have any direct effect on recreational uses of water other than its effects on aquatic life. No information is available on adverse effects of cyanide in agricultural

practices nor in industrial uses of water containing cyanide.

Since cyanide concentrations as low as 10 ug/l have been reported to cause adverse effects on fish, an ambient concentration of 5 ug/l is believed to provide protection with a reasonable margin of safety.

REFERENCES CITED

Bodansky, M., and M.D. Levy, 1923. I: Some factors influencing the detoxication of cyanides in health and disease. Arch. Int. Med. 31: 373.

Broderius, S.J., 1974. Testimony in the matter of proposed toxic pollutant effluent standards for Aldrin-Dieldrin, et al. Fed. Water Poll. Cont. Agency (307) Docket No. 1.

Burdick, G.E., et al., 1958. Toxicity of cyanide to brown trout and smallmouth bass. New York Fish and Game Jour., 5: 133.

Cairns, J., Jr. and A. Scheier, 1958. The effect of periodic low oxygen upon toxicity of various chemicals to aquatic organisms. Proc. 12th Industr. Waste Conf., Purdue Univ. Eng. Bull., 42: 165.

Costa, H.H., 1965. Responses of freshwater animals to sodium cyanide solutions. Ceylon Jour. Sic., Biol. Sic., 5: 41.

Doudoroff, P., 1956. Some experiments on the toxicity of complex cyanides to fish. Sew. and Ind. Wastes, 28: 1020.

Doudoroff, P., et al., 1966. Acute toxicity to fish of solutions containing complex metal cyanides, in relation to concentrations of molecular hydrocyanic acid. Trans. Amer. Fisheries Soc., 95: 6.

Downing, K.M., 1954. The influence of dissolved oxygen on the toxicity of potassium cyanide to rainbow trout. Jour. Exp. Biol., 31: 161.

Herbert, D.W.M. and J.C. Merkens, 1952. The toxicity of potassium cyanide to trout. Jour. Exp. Biol., 29: 632.

Karsten, A., 1934. Investigation of the effect of cyanide on Black Hills trout. South Dakota School of Mines Black Hills Engineer, 22:145.

Leduc, H., 1966. Some physiological and biochemical responses of fish to chronic poisoning by cyanide. Ph.D. Thesis, Oregon State University, Corvallis, Oregon.

Lipschuetz, M. and A.L. Cooper, 1955. Comparative toxicities of potassium cyanide and potassium cuprocyanide to the western black-nosed dace (Rhinichthys atratulus meleagris). New York Fish and Game Jour., 2:194.

Neil, J.H., 1957. Some effects of potassium cyanide on speckled trout, Salvelinus fontinalus. 4th Ontario Industrial Wastes Conference, Water Poll. Advisory Committee, Ontario Water Resources Commission, p. 74-96.

Turnbull, H., et al., 1954. Toxicity of various refinery materials to freshwater fish. Ind. and Eng. Chem., 46:24.

Washburn, G.H., 1948. The toxicity to warm water fishes of certain cyanide plating and carburizing salts before and after treatment by the alkali-chlorination method. Sew. Works Jour., 20:1074.

GASES, TOTAL DISSOLVED

CRITERION:

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

RATIONALE:

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

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

Total dissolved gas concentrations must be determined because analysis of individual gases may not determine with certainty that gas supersaturation exists. For example, water could be highly supersaturated with oxygen, but if nitrogen were at less than saturation, the saturation as measured by total gas pressure might not exceed one hundred percent. Also, if the water was highly supersaturated with dissolved oxygen, the oxygen alone might be sufficient to

create gas pressures or tensions greater than the criterion limits, but one would not know the total gas pressure or tension, or by how much the criterion was exceeded. The rare and inert gases such as argon, neon, and helium are not usually involved in causing gas bubble disease as their contribution to total gas presures is very low. Dissolved nitrogen (N_2), which comprises roughly 80 percent of the earth's atmosphere, is nearly inert biologically and is the most significant cause of gas bubble disease in aquatic animals. Dissolved oxygen, which is extremely bioactive, is consumed by the metabolic processes of the organism and is less important in causing serious gas bubble disease though it may be involved in initiating emboli formation in the blood (Nebeker, et al., 1976a.).

Percent saturation of water containing a given amount of gas varies with the absolute temperature and with the pressure. Because of the pressure changes, percent saturation with a given amount of gas changes with depth of the water. Gas supersaturation decreases by 10 percent per meter of increase in water depth due to hydrostatic pressure; a gas that is at 130 percent saturation at the

surface would be at 100 percent saturation at 3 meters' depth. Compensation for altitude may be needed because a reduction in atmospheric pressure changes the water/gas equilibria resulting in changes in solubility of dissolved gases.

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

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

(2) Lindroff (1957) reported that water spillage at hydropower dams caused supersaturation. When excess water is spilled over the face of a dam it entrains air as it plunges to the stilling or plunge pool at the base of the dam. The momentum of the fall carries the water and

entrained gases to great depths in the pool and, under increased hydrostatic pressure, the entrained gases are driven into solution causing supersaturation of dissolved gases.

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

In recent years, gas bubble disease has been identified as a major problem affecting valuable stocks of salmon and trout in the Columbia River system (Rulifson and Abel, 1971). The disease is caused by high concentrations of dissolved atmospheric gas which "enter" the river's water during heavy spilling at hydroelectric dams. A report by

Ebel, et al. (1975) presents results from field and laboratory studies on the lethal, sublethal, and physiological effects of gas on fish, depth distribution of fish in the river (fish can compensate for some high concentrations of gas by moving deeper into the water column), detection and avoidance of gas concentrations by fish, intermittent exposure of fish to gas concentrations, and bioassays of many species of fish exposed to different concentrations of gas. Several conclusions resulting from these studies are:

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

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

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

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

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

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

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

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

Rucker (1974), using juvenile coho salmon, Oncorhynchus kisutch, determined the effect of individual ratios of oxygen and nitrogen and established that a decrease in lethal effect occurred when the nitrogen content fell below 109 percent saturation even though total gas saturation remained at 119 percent saturation, indicating the importance of determining the concentration of the individual components (O_2 and N_2) of the atmospheric supersaturation. Nebeker, et al. (1976a), using juvenile sockeye salmon, Oncorhynchus nerka, also showed that there was a significant increase in fish mortality when the nitrogen concentration was increased while holding the total percent saturation constant. They also showed that there

was no significant difference in fish mortality at different CO_2 concentrations.

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

Daphnia magna exhibited a sensitivity to supersaturation similar to that of the salmonids (Nebeker, et al., 1975), with 115 percent saturation lethal within a few days; stoneflies exhibited an intermediate sensitivity similar to bass with mortality at 130 percent saturation; and crayfish

were very tolerant, with levels near 140 percent total gas saturation resulting in mortality.

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

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

operculum. The behavioral changes described were also observed at 115 percent, and clearly defined subcutaneous emphysema was observed in the fins and occasionally in the eye. At 120 percent and 130 percent nitrogen saturation, menhaden developed (within a few hours) classic symptoms of gas bubble disease. Externally, emboli were evident in all fins, the operculum, and within the oral cavity. Exophthalmia also occurred and emboli developed in internal organs. The bulbous arteriosis and swim bladder were severely distended, and emboli were found along the length of the gill arterioles resulting in hemostasis. At water temperatures of 30°C, menhaden did not survive, regardless of gas saturation level. At water temperatures of 15°, 22°, and 25°C, 100 percent of the menhaden died within 24 hours at 120 percent and 130 percent gas saturation. Fifty percent died after 96 hours at 115 percent (22°C). Menhaden survival after 96 hours at 110 percent nitrogen saturation ranged from 92 percent at 22° and 25° to 83 percent at 15°C. Observations on the relationship between the mortality rate of menhaden and gas saturation levels at Pilgrim Station during the April 1975 incident suggest that the fish may tolerate somewhat higher gas saturation levels in nature.

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

REFERENCES CITED:

Bouck, G.R., et al., 1975. Mortality, saltwater adaptation and reproduction of fish exposed to gas supersaturated water. Unpublished report. U.S. Environmental Protection Agency, Western Fish Toxicology Station, Corvallis, Oregon.

Clay, A., et al., 1975. Experimental induction of gas bubble disease in menhaden. Presented at the American Fisheries Society, September, 1975, Las Vegas, Nevada. New England Aquarium, Boston, Mass.

Dawley, E.M., and W.J. Ebel. 1975. Effects of various concentrations of dissolved atmospheric gas on juvenile chinook salmon, Oncorhynchus tshawytscha, and steelhead trout. Fish. Bull., U.S. (In Press).

Dawley, E., et al., 1975. Bioassays of total dissolved gas pressure. Unpublished report. National Marine Fisheries Service, Seattle, Washington.

DeMont, J.D. and R.W. Miller. 1972. First reported incidence of gas bubble disease in the heated effluent of a steam electric generating station. Proc. 25th Annual Meeting, Southeast Assoc. of Game and Fish Commissioners.

Ebel, W.J., et al., 1975. Effect of atmospheric gas supersaturation caused by dams on salmon and steelhead trout of the Snake and Columbia Rivers. Final Report. Northwest Fisheries Center, NMFS, Seattle, Washington.

Keup, L.E. 1975. Factors in fish kill investigations. Water and Sewage Works, 121:48.

Lindroth, A. 1957. Abiogenic gas supersaturation of river water. Arch. Hydrobiology, 53:589.

Malouf, R., 1972. Occurrence of gas bubble disease in three species of bivalve mollusks. Jour. Fish. Res. Bd. Can., 29:588.

Marcello, R.A., et al., 1975. Evaluation of alternative solutions to gas bubble disease mortality of menhaden at Pilgrim Nuclear Power Station. Yankee Atomic Electric Co., Westboro, Mass. YAEC-1087.

National Academy of Sciences, National Academy of Engineering. 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

Nebeker, A.V., et al., 1975. Effects of gas supersaturated water on freshwater invertebrates. Proc. Gas Bubble Disease Workshop. Battelle Northwest, ERDA Special Report (In Press).

Nebeker, A.V., et al., 1976a. Nitrogen, oxygen, and carbon dioxide as factors affecting fish survival in gas supersaturated water. Trans. Amer. Fish. Soc. (In Press).

Nebeker, A.V., et al., 1976b. Survival of coho and sockeye salmon molts in seawater after exposure to gas supersaturated water. Trans. Amer. Fish. Soc. (In Press).

Renfro, W.C. 1963. Gas bubble mortality of fishes in Galveston Bay, Texas. Trans. Amer. Fish. Soc. 92:320.

Rucker, R.R. 1974. Gas bubble disease: Mortalities of coho salmon, Oncorhynchus kisutch, in water with constant total gas pressure and different oxygen-nitrogen ratios. National Oceanic and Atmos. Admin., Natl. Mar. Fish. Serv., Northwest Fish Center, Seattle, Washington, unpublished manuscript.

Rulifson, R.L. and G. Abel. 1971. Nitrogen supersaturation in the Columbia and Snake Rivers. Tech Rept. TS-09-70-208-016.2, Environmental Protection Agency, Region X, Seattle, Washington.

Van Slyke, D.D., et al. 1934. Studies of gas and electrolyte equilibria in blood, XVIII. Solubility and physical state of atmospheric nitrogen in blood cells and plasma. Jour. Biol. Chem., 105:571.

HARDNESS

INTRODUCTION:

Water hardness is caused by the polyvalent metallic ions dissolved in water. In fresh waters these are principally calcium and magnesium although other metals such as iron, strontium, and manganese contribute to the extent that appreciable concentrations are present. Hardness commonly is reported as an equivalent concentration of calcium carbonate ($CaCO_3$).

The concept of hardness comes from water supply practice. It is measured by soap requirements for adequate lather formation and as an indicator of the rate of scale formation in hot water heaters and low pressure boilers. A commonly used classification is given in the following table (Sawyer, 1960).

Classification of Water by Hardness Content

Conc., mg/l $CaCO_3$	Description
0 - 75	soft
75 - 150	moderately hard
150 - 300	hard
300 and up	very hard

Natural sources of hardness principally are limestones which are dissolved by percolating rainwater made acid by dissolved carbon dioxide. Industrial and

industrially related sources include the inorganic chemical industry and discharges from operating and abandoned mines.

Hardness in fresh water frequently is distinguished in carbonate and non-carbonate fractions. The carbonate fraction is chemically equivalent to the bicarbonates present in water. Since bicarbonates generally are measured as alkalinity, the carbonate hardness usually is considered equal to the alkalinity.

RATIONALE:

The determination of hardness in raw waters subsequently treated and used for domestic water supplies is useful as a parameter to characterize the total dissolved solids present and for calculating chemical dosages where lime-soda softening is practiced. Because hardness concentrations in water have not been proven health related, the final level achieved principally is a function of economics. Since hardness in water can be removed with treatment by such processes as lime-soda softening and zeolite or ion exchange systems, a criterion for raw waters used for public water supply is not practical.

The effects of hardness on freshwater fish and other aquatic life appear to be related to the ions causing the hardness rather than hardness. Both the NTAC (NTAC, 1968) and NAS (NAS, 1974) panels have recommended against the use of the term hardness but suggest the inclusion of the concentrations of the specific ions. This procedure should avoid confusion in future studies but is not helpful in evaluating previous studies. For most existing data, it is difficult to determine whether toxicity of various metal ions is reduced because of the formation of metallic hydroxides and carbonates caused by the associated increases in alkalinity, or because of an antagonistic effect of one of the principal cations contributing to hardness, e.g., calcium, or a combination of both

effects. Stiff (1971) presented a theory (without proof) that if cupric ions were the toxic form of copper while copper carbonate complexes were relatively non-toxic, then the observed difference in toxicity of copper between hard and soft waters can be explained by the difference in alkalinity rather than hardness. Doudoroff and Katz (1953), in their review of the literature on toxicity, presented data showing that increasing calcium in particular reduced the toxicity of other heavy metals. Under usual conditions in fresh water and assuming that other bivalent metals behave similarly to copper, it is reasonable to assume that both effects occur simultaneously and explain the observed reduction of toxicity of metals in waters containing carbonate hardness. The amount of reduced toxicity related to hardness, as measured by a 40-hour LC_{50} for rainbow trout, has been estimated to be about four times for copper and zinc when the hardness was increased from 10 to 100 mg/l as $CaCO_3$ (NAS, 1974).

Limits on hardness for industrial uses are quite variable. Table 4 lists maximum values that have been accepted by various industries as a source of raw water (NAS, 1974). Subsequent treatment generally can reduce hardness to tolerable limits although costs of such treatment are an important factor in determining its desirability for a particular water source.

Hardness is not a determination of concern for irrigation use of water. The concentrations of the cations, calcium and magnesium, which comprise hardness, are important in determining the exchangeable sodium in a given water. This particular calculation will be discussed under total dissolved solids rather than hardness.

Table 4

Maximum Hardness Levels Accepted
by Industry as a Raw Water Source*

Industry	Maximum Concentration mg/l as $CaCO_3$
Electric Utilities	5,000
Textile	120
Pulp and Paper	475
Chemical	1,000
Petroleum	900
Primary Metals	1,000

* Requirements for final use within a process may be essentially zero which requires treatment for concentration reductions.

REFERENCES CITED:

Doudoroff, P. and Katz, M., 1953. Critical review of literature on the toxicity of industrial wastes and their components to fish. II. The metals as salts. Sew. and Ind. Wastes, 25, 7, p. 802.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

National Technical Advisory Committee to the Secretary of the Interior, 1968. Water quality criteria. U.S. Government Printing Office, Washington, D. C.

Sawyer, C.H., 1960. Chemistry for sanitary engineers. McGraw Hill Book Co., Inc., New York.

Stiff, M.J., 1971. Copper/bicarbonate equilibria in solutions of bicarbonate ion at concentrations similar to those found in natural water. Water Research, 5: 171.

IRON

CRITERIA:

0.3 mg/l for domestic water supplies (welfare).

1.0 mg/l for freshwater aquatic life.

INTRODUCTION:

Iron is the fourth most abundant, by weight, of the elements that make up the earth's crust. Common in many rocks it is an important component of many soils, especially the clay soils where usually it is a major constituent. Iron in water may be present in varying quantities dependent upon the geology of the area and other chemical components of the waterway.

Iron is an essential trace element required by both plants and animals. In some waters it may be a limiting factor for the growth of algae and other plants; especially this is true in some marl lakes where it is precipitated by the highly alkaline conditions. It is a vital oxygen transport mechanism in the blood of all vertebrate and some invertebrate animals.

The ferrous, or bivalent (Fe^{++}), and the ferric, or trivalent (Fe^{+++}) irons, are the primary forms of concern in the aquatic environment, although other forms may be in organic and inorganic wastewater streams. The ferrous (Fe^{++}) form can persist in waters void of dissolved oxygen and originates usually from groundwaters or mines when these are pumped or drained. For practical purposes the ferric (Fe^{+++}) form is insoluble. Iron can exist in natural organometallic or humic compounds and colloidal forms. Black or brown swamp waters may contain iron concentrations of several mg/l in the presence or absence of dissolved oxygen, but this iron form has litte effect on aquatic

life because it is complexed or relatively inactive chemically or physiologically.

In stratified lakes with anaerobic hypolimnia, soluble ferrous iron occurs in the deep, anaerobic waters. During the autumnal or vernal overturns and with aeration of these lakes, it is oxidized rapidly to the ferric ion that precipitates to the bottom sediments as a hydroxide, $Fe(OH)_3$, or with other anions. If hydrogen sulfide (H_2S) is present in anaerobic bottom waters or muds, ferrous sulfide (FeS) may be formed. Ferrous sulfide is a black compound and results in the production of black mineral muds.

Prime iron pollution sources are industrial wastes, mine drainage waters, and iron-bearing groundwaters. In the presence of dissolved oxygen, iron in water from mine drainage is precipitated as a hydroxide, $Fe(OH)_3$. These yellowish or ochre precipitates produce "yellow boy" deposits found in many streams draining coal mining regions of Appalachia. Occasionally ferric oxide (Fe_2O_3) is precipitated forming red waters. Both of these precipitates form as gels or flocs that may be detrimental, when suspended in water, to fishes and other aquatic life. They can settle to form flocculant materials that cover stream bottoms thereby destroying bottom-dwelling invertebrates, plants or incubating fish eggs. With time these flocs can consolidate to form cement-like materials, thus consolidating bottom gravels into pavement-like areas that are unsuitable as spawning sites for nest building fishes; particularly this is detrimental to trout and salmon populations whose eggs are protected in the interstices of gravel and incubated with oxygen-bearing waters passing through the gravel.

RATIONALE:

Iron is an objectionable constituent in water supplies for either domestic or industrial use. Iron appreciably affects the taste of beverages (Riddick, et al., 1958) and can stain laundered clothes and plumbing fixtures. A study by the Public Health Service (Cohen, et al., 1960) indicates that the taste of iron may be detected readily at 1.8 mg/l in spring water and at 3.4 mg/l in distilled water.

The daily nutritional requirement for iron is 1 to 2 mg, but intake of larger quantities is required as a result of poor absorption. Diets contain 7 to 35 mg per day and average 16 mg (Sollman, 1957). The iron criterion in water is to prevent objectionable tastes or laundry staining (0.3 mg/l) constitutes only a small fraction of the iron normally consumed and is of aesthetic rather than toxicological significance.

Warnick and Bell (1969) obtained 96-hour LC_{50} values of 0.32 mg/l iron for mayflies, stoneflies, and caddisflies; all are important fish food organisms. Brandt (1948) found iron toxic to carp, Cyprinus carpio, at concentrations of 0.9 mg/l when the pH of the water was 5.5. Pike, Esox lucius, and trout (species not known) died at iron concentrations of 1 to 2 mg/l (Doudoroff and Katz, 1953). In an iron polluted Colorado stream, neither trout nor other fish were found until the waters were diluted or the iron had precipitated to effect a concentration of less than 1.0 mg/l even though other water quality constituents measured were suitable for the presence of trout (FWPCA, 1967).

Ferric hydroxide flocs have been observed to coat the gills of white perch, Roccus americanus; minnows and silversides, Menidia sp. (Olsen, et al.,

1941). The smothering effects of settled iron precipitates may be particularly detrimental to fish eggs and bottom-dwelling fish food organisms. Iron deposits in the Brule River, Michigan and Wisconsin were found to have a residual long-term adverse effect on fish food organisms even after the pumping of iron-bearing waters from deep shaft iron mines had ceased (West, et al., 1963). Settling iron flocs have also been reported to trap and carry diatoms downward in waters (Olsen, et al., 1941).

Ellis (1937) found that in 69 of 75 study sites with good fish fauna, the iron concentration was less than 10.0 mg/l. The European Inland Fisheries Advisory Commission (1964) recommended that iron concentrations not exceed 1.0 mg/l in waters to be managed for aquatic life.

Based on field observations principally, a criterion of 1 mg/l iron for freshwater aquatic life is believed to be adequately protective. As noted, data obtained under laboratory conditions suggest a greater toxicity for iron than that obtained in natural ecosystems. Ambient natural waters will vary with respect to alkalinity, pH, hardness, temperature and the presence of ligands which change the valence state and solubility, and therefore the toxicity of the metal.

The effects of iron on marine life have not been investigated adequately to determine a water quality criterion. Dissolved iron readily precipitates in alkaline sea waters. Fears have been expressed that these settled iron flocs may have adverse effects on important benthic commercial mussels and other shellfish resources.

Iron has not been reported to have a direct effect on the recreational uses of water other than its effects on aquatic life. Suspended iron precipitates may interfere with swimming and be aesthetically objectionable. Deposits of yellow ochre or reddish iron oxides can be aesthetically objectionable.

Iron at exceedingly high concentrations has been reported to be toxic to livestock and interfere with the metabolism of phosphorus (NAS, 1974). Dietary supplements of phosphorus can be used to overcome this metabolic deficiency (McKee and Wolf, 1963). In aerated soils, iron in irrigation waters is not toxic. Precipitated iron may complex phosphorus and molybdenum making them less available as plant nutrients. In alkaline soils, iron may be so insoluble as to be deficient as a trace element and result in chlorosis, an objectionable plant nutrient deficiency disease. Rhoades (1971) reported a reduction in the quality of tobacco because of precipitated iron oxides on the leaves when the crop was spray irrigated with water containing 5 mg/l of soluble iron.

For some industries, iron concentrations in process waters lower than that prescribed above for public water supplies are required or desirable. Examples include high pressure boiler feed waters; scouring, bleaching, and dyeing of textiles; certain types of paper production; some chemicals; some food processing; and leather finishing industries.

LITERATURE CITED:

Brandt, H.H., 1948. Intensified injurious effects on fish, especially the increased toxic effect produced by a combination of sewage poisons. Beitr. Wass. Abwass., Fischereichemi. 15.

Cohen, J. M., et al., 1960. Taste threshold concentrations of metals in drinking water. Jour. Amer. Water Works Assn., 52: 660.

Doudoroff, P., and M. Katz, 1953. Critical review of literature on the toxicity of industrial wastes and their components to fish. II. The metals, as salts. Sew. Ind. Wastes, 25:802.

Ellis, M. M., 1937. Detection and measurement of stream pollution. Bulletin, U. S. Bur. Fisheries, 48: 365.

European Inland Fisheries Advisory Commission, 1964. Water quality criteria for European freshwater fish. Report on finely divided solids and inland fisheries. Tech. Paper 1.

FWPCA, 1967. Effects of pollution on the aquatic life resources of the South Platte River Basin. Two volumes. South Platte River Basin Project, Denver, Colorado and Technical Advisory and Investigations Branch, Cincinnati, Ohio. Fed. Water Pollution. Cont. Admin., U.S. Dept. of Interior.

McKee, J. E., and H. W. Wolf, 1963. Water quality criteria. State Water Quality Control Board, Sacramento, California, Pub. 3-A.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Olsen, R.A., et al., 1941. Studies of the effects of industrial pollution in the lower Patapsco River area. I: Curtis Bay Region, Chesapeake Biological Laboratory, Solomans Island, Maryland.

Rhoades, F.M., 1971. Relations between Fe in irrigation water and leaf quality of cigar wrapper tobacco. Agron. Jour., 63: 939.

Riddick, T.M., et al., 1958. Iron and manganese in water supplies. Jour. Amer. Water Works Assn., 50: 688.

Sollman, T.M., 1957. A manual of pharmacology, 8th ed. W.B. Saunders Co., Philadelphia, Pa.

Warnick, S.L. and H.L. Bell, 1969. The acute toxicity of some heavy metals to different species of aquatic insects. Jour. Water Poll. Cont. Fed., 41(Part 2): 280.

West, A.W., et al., 1963. Report on pollution of the interstate waters of the Menominee and Brule rivers, Michigan-Wisconsin. U.S. Dept. of Health, Education and Welfare, Public Health Service, R.A. Taft Sanitary Engineering Center, Cincinnati, Ohio.

LEAD

CRITERIA:

50 ug/l for domestic water supply (health);

0.01 times the 96-hour LC_{50} value, using the receiving or comparable water as the diluent and soluble lead measurements (non-filtrable lead using an 0.45 micron filter), for sensitive freshwater resident species.

INTRODUCTION:

In addition to their natural occurrence, lead and its compounds may enter and contaminate the global environment at any stage during mining, smelting, processing, and use. The annual increase in lead consumption in the U.S. during the 10-year period from 1962-1971 averaged 2.9 percent, largely due to increased demands for electrochemical batteries and gasoline additives (Ryan, 1971). In 1971 the total U.S. lead consumption was 1,431,514 short tons, of which 42 percent came from recycled lead (Ryan, 1971). Of the 1971 U.S. lead consumption, approximately 25 percent was as metallic lead or lead alloy (Ryan, 1971; NAS, 1972). Non-industrial sources that may contribute to the possibility of ingestion of lead by man include the indoor use of lead-bearing paints and plaster, improperly glazed earthenware, lead fumes on ashes produced in burning lead battery casings, and exhaust from internal combustion engines.

Most lead salts are of low solubility. Lead exists in nature mainly as lead sulfide (galena); other common natural forms are lead carbonate (cerussite), lead sulfate (anglesite), and lead chlorophosphate (pyromorphite). Stable complexes result also from the interaction of

lead with the sulfhydryl, carboxyl, and amine coordination sites characteristically found in living matter. The toxicity of lead in water, like that of other heavy metals, is affected by pH, hardness, organic materials and the presence of other metals. The aqueous solubility of lead ranges from 500 ug/l in soft water to 3 ug/l in hard water.

Lead enters the aquatic environment through precipitation, lead dust fallout, erosion and leaching of soil, municipal and industrial waste discharges, and the runoff of fallout deposits from streets and other surfaces. Extrapolations from recent studies (EPA, 1972; University of Illinois, 1972) indicate that nationally as much as 5,000 tons of lead per year may be added to the aquatic environment as a result of urban runoff.

Mediterranean and Pacific surface waters contain up to 0.20 and 0.35 mg/l of lead, respectively (NAS, 1972), which is about 10 times the estimated pre-industrial lead content of marine waters. The lead content of rivers and lakes also has increased in recent years (NAS, 1972). It may be inferred from available data that the mean natural lead content of the world's lakes and rivers ranges from 1 to 10 ug/l (Livingstone, 1963); the lead content of rural U.S. soils is 10 to 15 ug/g (Chow and Patterson, 1962), and the usual range of lead-in-soil concentrations is 2 to 200 ppm, exclusive of areas near lead ore deposits (Motto, et al., 1970), although many urban soil concentrations are much higher.

In the analyses of over 1,500 stream samples, Kopp and Kroner (1967) report that lead was observed at measurable levels with a

frequency of under 20 percent. The mean concentration of the positive occurrences was 23 ug/l. The highest incidence of occurrence of lead was observed in the Western Great Lakes Basin where the frequency was slightly above 40 percent. The highest recorded concentration was 140 ug/l in the Ohio River at Evansville, Indiana.

RATIONALE:

As far as is known, lead has no beneficial or desirable nutritional effects. Lead is a toxic metal that tends to accumulate in the tissues of man and other animals. Although seldom seen in the adult population, irreversible damage to the brain is a frequent result of lead intoxication in children. Such lead intoxication most commonly results from ingestion of lead-containing paint still found in older homes. The major toxic effects of lead include anemia, neurological dysfunction, and renal impairment. The most common symptoms of lead poisoning are anemia, severe intestinal cramps, paralysis of nerves (particularly of the arms and legs), loss of appetite, and fatigue; the symptoms usually develop slowly. High levels of exposure produce severe neurologic damage, often manifested by encephalopathy and convulsions; such cases frequently are fatal. Lead is strongly suspected of producing subtle effects (i.e., effects due to low level or long term exposures insufficient to produce overt symptoms) such as impaired neurologic and motor development and renal damage in children (EPA, 1973). Subclinical lead effects are distinct from those of residual damage following lead intoxication.

Biochemical effects of lead include inhibition of erythrocyte delta-aminolevulinic acid dehydrase (ALAD) activity, increased urinary

excretion of delta-aminolevulinic acid (ALA-U), and increased blood lead concentration. The increase in ALA-U is of particular interest because it rarely is produced by any substance other than lead; it is related directly to inhibition of ALAD, the enzyme that converts ALA-U to porphobilinogen. Recent work indicates that lead interferes with heme biosynthesis, and thus elevates ALA-U excretion, at levels well below 40 ug/100 ml whole blood (Secchi, et al., 1974; Haeger-Aronsen, et al., 1974). As a result of this work, the Center for Disease Control has recommended that the upper limit of normal for lead in the blood of children be revised downward from 40 ug to 30 ug/100 ml whole blood. An ad hoc committee, appointed by the U.S. Public Health Service to establish a daily permissible intake of lead without excessive body lead burden in children, concluded that the level of such intake is 300 ug from all sources (King, 1971). The gastrointestinal absorption and retention of lead is greater in children than in adults, 53 percent and 18 percent, respectively, as shown in recent studies (Alexander, et al., 1973). The average daily intake of lead from diet and air among young children probably amounts to up to 2/3 of the daily permissible intake, leaving a very narrow margin of safety (King, 1971; Lin-Fu, 1973). Compared to adults, food and air intake by children is proportionally greater than their weight, e.g., a 1-year-old child, with only about 1/7 of the body weight of an adult, has 1/4 to 1/3 of the daily adult air intake and 40 to 60 percent of the dietary intake of an adult (NAS, 1972), so that his lead intake is proportionally greater on a body weight basis. Alexander, et al. (1973) have suggested a daily limit of lead intake for 0-5 year-olds of 10 ug/kg.

Considerable evidence has been developed demonstrating that laboratory animals on high lead dosages show teratogenic effects; however, no such effects have been observed in cattle or sheep (NAS, 1972). Epidemiologic studies have demonstrated no relationship between lead exposure and cancer incidence in man, but it is known that lead at concentrations of one percent or more in the diet causes renal cancer in rats (NAS, 1972).

The lead content in public water supplies in the U.S. in 1962 ranged from traces to 62 ug/l (Dufor and Becker, 1964). Continuous monitoring of the U.S. water supplies since 1962 has demonstrated that their lead content has, in general, not exceeded the U.S. Public Health Service standard of 50 ug/l (USPHS, 1962). McCabe (1970) reported on 2,595 distribution samples and showed that 73 percent contained less than the USPHS limit. McCabe, et al. (1970) found that the range of lead concentrations in finished U.S. community water was from non-detectable to 0.64 mg/l. Of the 969 water supplies surveyed, 1.4 percent exceeded 0.05 mg/l of lead. Five of the water supplies in this sample contained sufficient lead to equal or exceed Kehoe's (1966) estimated maximum safe level of lead intake (600 ug/day) without considering the possible additional contributions to the total intake by other sources and routes of exposure. In drinking water lead should be kept to a minimum; a criterion of 50 ug/l is attainable and protective. Experience indicates that fewer than four percent of the water samples analyzed exceed the 50 ug/l limit and that the majority of these are due to corrosion problems and are not due to naturally occurring lead content in raw waters.

For the fathead minnow, Pimephales promelas, Pickering and Henderson (1966) have determined the 96-hour TLm values for lead (chloride) to be 5.6 to 7.3 mg/l in soft water (20 to 45 mg/l as $CaCO_3$) Brown (1968) reported a 96-hour LC_{50} of 1 mg/l for rainbow trout, Salmo gairdneri, in soft water (50 mg/l as $CaCO_3$). The 96-hour LC_{50} value for lead in hard water was 482 mg/l for fathead minnows (Pickering and Henderson, 1966). Other short-term fish toxicity data are in Table 5.

Preliminary information on 2- to 3-month exposures of rainbow and brook trout indicated detrimental effects at 0.10 mg/l of lead in soft water (20-45 mg/l as $CaCO_3$) (NAS, 1974). Growth of guppy species was affected by 1.24 mg/l of lead (Crandall and Goodnight, 1962); Jones (1939) and Hawksley (1967) found chronic or sublethal effects on three-spine stickelback species from lead concentrations of 0.1 and 0.3 mg/l, and the conditioned behavior of goldfish, Carassius auratus, in a light-dark shuttlebox was adversely affected by 0.07 mg/l of lead in soft water (50 ppm $CaCO_3$ added to deionized tap water) (Weir and Hine, 1970).

A concentration of 30 ug/l lead in a 3-week exposure produced 16 percent reproductive impairment in Daphnia magna in water with a hardness of 45 mg/l $CaCO_3$ (Biesinger and Christensen, 1972). The 96-hour LC_{50} for rainbow trout species in hard water (alkalinity 243 mg/l) for total lead was 471 mg/l and the highest mean continuous flow concentration that did not have an adverse effect on survival, growth, and reproduction was 0.12 and 0.36 mg/l (Davies and Everhart, 1973).

For dissolved lead, the 96-hour LC_{50} was 1.38 mg/l and the no-effect level was 18 to 32 ug/l. Total and free lead were considered to be the same in soft water. The 18-day LC_{50} in soft water (alkalinity 26.4 mg/l) was 140 ug/l and the highest mean continuous flow concentration that did not have an adverse effect on survival, growth and reproduction was 6.0 to 11.9 ug/l. The no-effect concentrations were determined on the occurrence of abnormal black tails caused by chronic lead exposure. Acute toxicity values for several species of fish in water of various qualities are presented in Table 5. When referring to this table, the reader should consider the species tested, pH, alkalinity, and hardness if alkalinity is not given (in most natural waters alkalinity parallels hardness). In general, the salmonids are most sensitive to lead in soft water, but the influence of pH, and other factors on the solubility and form of the lead preclude the recommendation of a freshwater criteria based on acute toxicities alone.

Table 5

The acute toxicity (24-, 48-, 96-hr. TL50 values) of lead to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alk.	Hd.	Reference
Rainbow trout (Salmo gairdneri)	~31.5 g/fish	$Pb(NO_3)_2$	24	F.T.*	14.7	3.75 Pb	7.15-7.5	41-42	43-45	1
Rainbow trout	~24 g/fish ~132 mm/fish	$Pb(NO_3)_2$	96	S*	7	1.38 free Pb	6.89-8.78	7-21 228 in control	300	3
Rainbow trout	~24 g/fish ~132 mm/fish	$Pb(NO_3)_2$	96	S	7	471 Pb	6.89-8.78	7-21 228 in control	300	3
Rainbow trout	~5.9 g/fish ~85.9 mm/fish	$Pb(NO_3)_2$	96	S	13.8-14.2	542 Pb	6.23-7.17	6-25 228 in control	385	3
Brook trout (Salvelinus fontinalis)	~ 102 g/fish	$Pb(NO_3)_2$	96	F.T.	12	4.5 Pb	6.9-7.1	43	45	1
Brook trout	~ 40 g/fish	$Pb(NO_3)_2$	96	F.T.	14	5.8 Pb	7.0-7.4	40-42	43-45	1
Coho Salmon (Oncorhyncus kisutch)	4-wk. post-hatch	$PbCl_2$	96	S	10	0.8 Pb	-	-	17-26	2
Coho Salmon	5-wk. post-hatch	$PbCl_2$	96	S	10	0.8 Pb	-	-	17-26	2
Coho Salmon	6-wk. post-hatch	$PbCl_2$	96	S	10	0.52 Pb	-	-	17-26	2
Coho Salmon	7-wk. post-hatch	$PbCl_2$	96	S	10	0.52 Pb	-	-	17-26	2

* F.T. = Flow Through
* S = Static

The acute toxicity (24-, 48-, 96-hr. TL50 values) of lead to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alk.	Hd.	Reference
Sticklebacks (Gasterosteus aculeatus)	-	-	96	S	-	0.1 Pb	7.0-8.5	-	-	4
Mosquito fish (Gambusia affinis)	Adult Females	$Pb(NO_3)_2$	24	S	22-24	240 $Pb(NO_3)_2$ (150 Pb)	7.7-8.3	<100	-	9
Mosquito fish	Adult Females	$Pb(NO_3)_2$	48	S	22-24	240 $Pb(NO_3)_2$ (150 Pb)	7.7-8.3	<100	-	9
Mosquito fish	Adult Females	$Pb(NO_3)_2$	96	S	22-24	240 $Pb(NO_3)_2$ (150 Pb)	7.7-8.3	<100	-	9
Mosquito fish	Adult Females	Lead oxide	24	S	18-20	>56,000 PbO	7.1-7.2	<100	-	9
Mosquito fish	Adult Females	Lead oxide	48	S	18-20	>56,000 PbO	7.1-7.2	<100	-	9
Mosquito fish	Adult Females	Lead oxide	96	S	18-20	>56,000 PbO	7.1-7.2	<100	-	9
Gold fish (Carassius auratus)	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	24	S	25	45.4 Pb	7.5	18	20	6
Gold fish	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	48	S	25	31.5 Pb	7.5	18	20	6
Gold fish	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	96	S	25	31.5 Pb	7.5	18	20	6

The acute toxicity (24-, 48-, 96-hr. TL50 values) of lead to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alk.	Hd.	Reference
Guppies (Poecilia reticulatus)	0.1-0.2 g/fish 0.75-1 in./fish	$PbCl_2$	24	S	25	24.5 Pb	7.5	18	20	6
Guppies	0.1-0.2 g/fish 0.75-1 in./fish	$PbCl_2$	48	S	25	24.5 Pb	7.5	18	20	6
Guppies	0.1-0.2 g/fish 0.75-1 in./fish	$PbCl_2$	96	S	25	20.6 Pb	7.5	18	20	6
Bluegills (Lepomis macrochirus)	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	24	S	25	25.9 Pb	7.5	18	20	6
Bluegills	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	48	S	25	24.5 Pb	7.5	18	20	6
Bluegills	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	96	S	25	23.8 Pb	7.5	18	20	6
Bluegills	~5 g/fish ~7 cm/fish	$(C_2H_5)_4Pb$	24	S	20	2.0 Pb	6.9-7.5	33-81	84-163	8
Bluegills	~5 g/fish ~7 cm/fish	$(C_2H_5)_4Pb$	48	S	20	1.4 Pb	6.9-7.5	33-81	84-163	8
Bluegills	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	24	S	25	482 Pb	8.2	300	360	6
Bluegills	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	48	S	25	468 Pb	8.2	300	360	6
Bluegills	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	96	S	25	442 Pb	8.2	300	360	6

The acute toxicity (24-, 48-, 96-hr. TL50 values) of lead to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alk.	Hd.	Reference
Fatheads (Pimephales promelas)	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	24	S	25	8.18-11.5 Pb	7.5	18	20	6
Fatheads	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	48	S	25	5.99-11.5 Pb	7.5	18	20	6
Fatheads	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	96	S	25	5.58-7.33 Pb	7.5	18	20	6
Fatheads	-	$PbCl_2$	48	F.T.	-	1.1 Pb	7.4	18	20	6
Fatheads	-	$PbCl_2$	96	F.T.	-	0.97 Pb	7.4	18	20	5
Fatheads	-	$PbCl_2$	96	S	-	5.6 Pb	7.4	18	20	5
Fatheads	-	$PbCl_2$	96	-	-	2.4 Pb	7.4	18	20	7
Fatheads	1-2 g/fish 1.5-2.5 in./fish	$Pb(C_2H_3O_2)_2 \cdot 3H_2O$	24	S	25	14.6 Pb	7.5	18	20	6
Fatheads	1-2 g/fish 1.5-2.5 in./fish	$Pb(C_2H_3O_2)_2 \cdot 3H_2O$	48	S	25	10.4 Pb	7.5	18	20	6
Fatheads	1-2 g/fish 1.5-2.5 in/fish	$Pb(C_2H_3O_2)_2 \cdot 3H_2O$	96	S	25	7.48 Pb	7.5	18	20	6
Fatheads	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	24	S	25	482 Pb	8.2	300	360	6
Fatheads	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	48	S	25	482 Pb	8.2	300	360	6
Fatheads	1-2 g/fish 1.5-2.5 in./fish	$PbCl_2$	96	S	25	482 Pb	8.2	300	360	6
Fatheads	-	$PbCl_2$	96	S	-	>75 Pb	8.2	360	400	7

References Cited in Table 5:

1. Benoit, D. and G. Holcombe. Unpublished data available from Water Quality Laboratory, Duluth, Minnesota.

2. Chapman, G. Unpublished data available from National Water Quality Laboratory, Duluth, Minnesota.

3. Davies, P. H. and W. H. Everhart, 1973. Effects of chemical variations in aquatic environments, Vol. III. Lead toxicity to rainbow trout and testing application factor concept. Ecological Research Series, EPA R3-73-011c.

4. Hawksley, R. A. 1967. Advanced water pollution analysis by a water laboratory. Analyzer, 8:13.

5. Henderson, C. Unpublished data available from National Water Quality Laboratory, Duluth, Minnesota.

6. Pickering, Q. H. and C. Henderson. 1965. The acute toxicity of some heavy metals to different species of warm water fishes. Purdue University. Proc. 19th Ind. Waste Conf., 578-591.

7. Tarzwell, C. M. and C. Henderson. 1960. Toxicity of less common metals to fishes. Industrial Wastes, 5:12.

8. Turnbull, H., et al. 1954. Toxicity of various refinery materials to freshwater fish. Symposium of waste disposal in the petroleum industry. Ind. Eng. Chem. 46:325.

9. Wallen, I. E., et al. 1957. Toxicity to Gambusia affinis of certain pure chemicals in turbid waters. Sewage and Industrial Wastes, 29:695.

There are few data from which to draw conclusions about the relationship of safe levels for a given species in hard and soft water. The differences in lethal levels between very soft and very hard water is 100 to 500 times because of insolubility and precipitation. The single source of information for chronic and acute effects in hard and soft water is for rainbow trout (Davies and Everhart, 1973) and shows these significant factors:

Hardwater data were reported in terms of both total and free lead. The static acute hard water bioassay (mean hardness, 353.5 mg/l; methyl orange alkalinity, 243.1 mg/l; and pH, 8.02) demonstrated 96-hour TL_{50} (50 percent tolerance limit) concentration of 471 mg/l total lead and 1.38 mg/l free lead.

In the chronic hard water tests, lead-attributed mortalities occurred at levels of 3240 ug/l total lead and 64 ug/l free lead, while the maximum acceptable toxicant concentrations, based on the occurrence of black tails, were found to be 120 ug/l to 360 ug/l total lead and 18 ug/l to 32 ug/l free lead.

Soft water data were reported in terms of free lead. The flow-through acute soft water bioassay (mean hardness, 27.2 mg/l; methyl orange alkalinity, 26.4 mg/l; and pH 6.88) demonstrated an 18-day TL_{50} (50 percent tolerance limit) of 140 ug/l free lead.

In the chronic soft water tests, lead-attributed mortalities occurred in fry and fingerling fish at a concentration of 95.2 ug/l, while three-year-old brood fish produced viable eggs and fry when exposed to any but the highest (27.9 ug/l) concentration. The maximum acceptable toxicant concentration, based on the occurrence of black tails, was determined to be between 11.9 and 6.0 ug/l.

A criterion involving an application factor of 0.01 multiplied by the acute 96-hour LC_{50} value expressed as dissolved lead is used to estimate the safe concentration for various fish species. Based upon the existing data, as well as upon the sensitivity of various species of fish to other metals, it is highly probable that salmon, trout, minnows and catfish will be especially sensitive to lead as compared to bass, sunfish and perch. Therefore, tests for acute toxicity should be performed on the more sensitive species when establishing standards for lead. This approach requires the experimental determination of LC_{50} values before a criterion can be determined, but the extreme effect of various water characteristics on lead solubility and toxicity warrants this additional effort.

Berry (1924) found that a concentration of lead nitrate of 25 mg/l was required to cause toxic effects to oats and tomato plants. At a concentration of 50 mg/l, plant death occurred. Hopper (1937) found that 30 mg/l of lead in nutrient solutions was toxic to bean plants.

Wilkins (1957) found that lead at 10 mg/l as lead nitrate reduced root growth. Since dissolved lead contents in soils were usually from 0.05 to 5.0 mg/kg (Brewer, 1966), little toxicity can be expected. It was shown that the principal entry of lead into plants was from aerial deposits rather than from absorption from soils (Page, et al., 1971), indicating that lead that falls into the soil is not available to plants.

There is no question that some marine organisms can concentrate the lead present in sea water. Wilder (1952) reported lobster dying in 6 to 20 days when held in lead-lined tanks. Calabrese, et al. (1973) found a 48-hour LC_{50} of 1730 ug/l and a 48-hour LC_{50} of 2450 ug/l for oyster, Crassostrea virginica, eggs. The remarkable ability of the eastern oyster, Crassostrea virginica, to concentrate lead was demonstrated (Pringle, et al., 1968) by exposing them to flowing sea water containing lead concentrations of 25 ug/l, 50 ug/l, 100 ug/l, and 200 ug/l; after 49 days, the total accumulation of lead amounted to 17, 35, 75, and 200 ppm (wet weight), respectively, and those oysters exposed to the two highest lead levels, upon gross examination, showed considerable atrophy and diffusion of the gonadal tissue, edema, and less distinction of hepatopancreas and mantle edge.

North and Clendenning (1958) reported that lead nitrate at 4.1 mg/l of lead showed no deleterious effect on the photosynthesis rate in kelp, Macrocystis pyrifera, exposed for four days. However, there are insufficient data upon which to base a marine criterion at this time.

REFERENCES CITED

Alexander, F.W., et al., 1973. The uptake and excretion by children of lead and other contaminants, In: Proc. of the int'l. symposium on environmental health aspects of lead. Amsterdam, October 2-6, 1972, Commission of the European Communities, Luxembourg, p. 319.

Berry, R.A., 1924. The manurial properties of lead nitrate. Jour. Agric. Sci. (London), 14: 58.

Biesinger, K.E., and G.M. Christensen, 1972. Effects of various metals on survival, growth, reproduction and metabolism of Daphnia magna. Jour. Fish. Res. Bd. of Canada, 29: 1691.

Brewer, R.F., 1966. Lead, In: H.D. Chapman, Ed., Diagnostic criteria for plants and soils. Berkeley, University of California, Division of Agric. Sci., p. 213.

Brown, V.M., 1968. The calculation of the acute toxicity of mixtures of poisons to rainbow trout. Water Res., 2: 723.

Calabrese, A., et al., 1973. The toxicity of heavy metals to embryos of the american oyster, Crassostrea virginica. Marine Biol., 18: 162.

Chow, T.J., and C.C. Patterson, 1962. The occurrences and significance of lead isotopes in pelagic sediments. Geochim. Cosmochim. Acta., 26: 263.

Crandall, C.A. and C.J. Goodnight, 1962. Effects of sublethal concentrations of several toxicants on growth of the common guppy, Lebistes reticulatus. Limnol. Oceanog., 1: 233.

Davies, P.H. and W.H. Everhart, 1973. Effects of chemical variations in aquatic environments: volume III. Lead toxicity to rainbow trout and testing application factor concept. Environmental Protection Agency, Ecol. Res. Series Report, EPA-R3-73-011.

Durfor, C. and E. Becker, 1964. Selected data on public water supplies of the 100 largest cities in the United States, 1962. Jour. Amer. Water Works Assn., 56: 237.

Environmental Protection Agency, 1972. Water pollution aspects of street surface contaminants. EPA-R2-72-081, U.S. Environmental Protection Agency, Washington, D. C.

Environmental Protection Agency, 1973. EPA's position on the health implications of airborne lead. U.S. Environmental Protection Agency, Washington, D. C.

Haeger-Aronsen, et al., 1974. Effect of lead on delta-aminolevulinic acid dehydratase activity in red blood cells. Arch. Environ. Health, 29:150.

Hawksley, R.A., 1967. Advanced water pollution analysis by a water laboratory. Analyzer, 8: 13.

Hopper, M.C., 1937. Effect of lead on plants. Ann. Appl. Biol., 24: 690.

Jones, J.R.E., 1939. The relation between the electrolytic solution pressures of the metals and their toxicity to the stickleback (Gasterosteus aculeatus L.). Jour. Exp. Biol., 16: 425.

Kehoe, R.A., 1966. Under what circumstances is ingestion of lead dangerous? In: Symposium on environmental lead contamination. U.S. Dept. of Health, Education, and Welfare, Public Health Service, Publication 1440. Washington, D.C., p. 51.

King, B.G., 1971. Maximum daily intake of lead without excessive body lead-burden in children. Amer. Jour. Dis. Children, 122:337.

Kopp, J.F., and R.C. Kroner, 1967. Trace metals in waters of the United States. U.S. Department of the Interior, Federal Water Pollution Control Administration, Cincinnati, Ohio.

Lin-Fu, J.S., 1973. Vulnerability of children to lead exposure and toxicity. New Eng. Jour. Med., 289:1229.

Livingstone, D.A., 1963. Chemical composition of rivers and lakes, pp. G1-G64. Geological Survey Professional Paper 440-G. In: M. Fleischer, Ed., Data of Geochemistry (6th ed.). U.S. Government Printing Office, Washington, D.C.

McCabe, L.J., et al., 1970. Survey of community water supply systems. Jour. Amer. Water Works Assn. 62:670.

McCabe, L.J., 1970. Metal levels found in distribution samples. Amer. Water Works Assn. Seminar on Corrosion by Soft Water, Washington, D.C. 9 pp.

Motto, H.L., et al., 1970. Lead in soils and plants: Its relationship to traffic volume and proximity to highways. Environmental Sci. Tech., 4:231.

National Academy of Sciences - Committee on Biologic Effects of Atmospheric Pollutants, 1972. Lead: Airborne lead in perspective. The National Academy of Sciences, Washington, D.C.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

North, W.J. and K.A. Clendenning, 1958. The effects of waste discharges on kelp. Annual Progress Report, Institute of Marine Resources (University of California, LaJolla, California). IMR Reference 58-11.

Page, A.L., et al., 1971. Lead quantities in plants, soil and air near some major highways in Southern California. Hilgardia, 41.

Pickering, Q.H., and C. Henderson, 1966. The acute toxicity of some heavy metals to different species of warm water fishes. Int. Jour. Air-Water Poll., 10:453.

Pringle, B.H., et al., 1968. Trace metal accumulation by estuarine mollusks. Jour. Sanit. Eng. Div.; Proc. Am. Soc. Civil Eng., 94:455.

Ryan, J.P., 1971. Minerals Yearbook, 1971. U.S. Department of the Interior, Washington, D.C.

Secchi, G.C., et al., 1974. Delta-aminolevulinic acid dehydratase activity of erythrocytes and liver tissue in man. Arch. Environ Health, 28:130.

University of Illinois, 1972. Environmental pollution by lead and other metals (NSF RANN Grant 6I-31605), Progress Report, May 1 - October 31, 1972, Chapter 6. University of Illinois at Urbana-Champaign.

USPHS, 1962. Public Health Service Drinking Water Standards, U.S. Department of Health, Education, and Welfare, Public Health Service Publication 956 (Revised), Washington, D.C.

Weir, P.A., and C.H. Hine, 1970. Effects of various metals on behavior of conditioned goldfish. Arch. Envir. Health., 20:45.

Wilder, D.C., 1952. The relative toxicity of certain metals to lobsters. Jour. Fish Res. Bd. Can., 8:486.

Wilkins, D.A., 1957. A technique for the measurement of lead tolerance in plants. Nature, 180:37.

MANGANESE

CRITERIA:

50 ug/l for domestic water supplies (welfare);

100 ug/l for protection of consumers of marine mollusks.

INTRODUCTION:

Manganese does not occur naturally as a metal but is found in various salts and minerals, frequently in association with iron compounds. The principal manganese-containing substances are manganese dioxide (MnO_2), pyrolusite, manganese carbonate (rhodocrosite) and manganese silicate (rhodonite). The oxides are the only important minerals mined. Manganese is not mined in the United States except when manganese is contained in iron ores that are deliberately used to form ferro-manganese alloys.

The primary uses of manganese are in metal alloys, dry cell batteries, micro-nutrient fertilizer additives, organic compounds used in paint driers and as chemical reagents. Permanganates are very strong oxidizing agents of organic materials.

Manganese is a vital micro-nutrient for both plants and animals. When manganese is not present in sufficient quantities, plants exhibit chlorosis (a yellowing of the leaves) or failure of the leaves to develop properly. Inadequate quantities of manganese in domestic animal food results in reduced reproductive capabilities and deformed or poorly maturing young. Livestock feeds usually have sufficient manganese, but beef cattle on a high corn diet may require a supplement.

RATIONALE:

Although inhaled manganese dusts have been reported to be toxic to humans, manganese normally is ingested as a trace nutrient in food. The average human intake is approximately 10 mg/day (Sollman, 1957). Very large doses of ingested manganese can cause some disease and liver damage but these are not known to occur in the United States. Only a few manganese toxicity problems have been found throughout the world and these have occurred under unique circumstances, i.e., a well in Japan near a deposit of buried batteries (McKee and Wolf, 1963).

It is possible to partially sequester manganese with special treatment but manganese is not removed in the conventional treatment of domestic waters (Riddick, et al., 1958; Illig, 1960). Consumer complaints arise when manganese exceeds a concentration of 150 ug/l in water supplies (Griffin, 1960). These complaints are concerned primarily with the brownish staining of laundry and objectionable tastes in beverages. It is possible that the presence of low concentrations of iron may intensify the adverse effects of manganese. Manganese at concentrations of about 10 to 20 ug/l is acceptable to most consumers. A criterion for domestic water supplies of 50 ug/l should minimize the objectionable qualities.

McKee and Wolf (1963) summarized data on toxicity of manganese to freshwater aquatic life. Ions of manganese are found rarely at concentrations above 1 mg/l. The tolerance values reported range from 1.5 mg/l to over 1000 mg/l. Thus, manganese is not considered to be a problem in fresh waters. Permanganates have been reported to kill fish in 8 to 18 hours at concentrations of 2.2 to 4.1 mg/l, but permanganates are not persistent because they rapidly oxidize organic materials and are

thereby reduced and rendered nontoxic.

Few data are available on the toxicity of manganese to marine organisms. The ambient concentration of manganese is about 2 ug/l (Fairbridge, 1966). The material is rapidly assimilated and bioconcentrated into nodules that are deposited on the sea floor. The major problem with manganese may be concentration in the edible portions of mollusks, as bioaccumulation factors as high as 12,000 have been reported (NAS, 1974). In order to protect against a possible health hazard to humans by manganese accumulation in shellfish, a criterion of 100 ug/l is recommended for marine water.

Manganese is not known to be a problem in water consumed by livestock. At concentrations of slightly less than 1 mg/l to a few milligrams per liter, manganese may be toxic to plants from irrigation water applied to soils with pH values lower than 6.0. The problem may be rectified by liming soils to increase the pH. Problems may develop with long-term (20 year) continuous irrigation on other soils with water containing about 10 mg/l of manganese (NAS, 1974). But as stated above, manganese rarely is found in surface waters at concentrations greater than 1 mg/l. Thus, no specific criterion for manganese in agricultural waters is proposed. In select areas, and where acidophilic crops are cultivated and irrigated, a criterion of 200 ug/l is suggested for consideration.

Most industrial users of water can operate successfully where the criterion proposed for public water supplies is observed. Examples of industrial

tolerance of manganese in water are summarized for industries such as dyeing, milk processing, paper, textiles, photography and plastics (McKee and Wolf, 1963). A more restrictive criterion may be needed to protect or ensure product quality.

REFERENCES CITED:

Fairbridge, R.W. (ed), 1966. The encyclopedia of oceanography. Reinhold, New York, New York.

Griffin, A.E., 1960. Significance and removal of manganese in water supplies. Jour. Amer. Water Works Assn., 52: 1326.

Illig, G.L. Jr., 1960. Use of sodium hexametaphosphate in manganese stabilization. Jour. Amer. Water Works Assn., 52: 867.

McKee, J.E. and H.W. Wolf, 1963. Water quality criteria. State Water Quality Control Board, Publ. , Sacramento, California, Pub. 3-A.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Riddick, J.M., et al., 1958. Iron and manganese in water supplies. Jour. Amer. Water Works Assn., 50: 688.

Sollman, T.H., 1957. A manual of pharmacology. W.B. Saunders, Philadelphia. Pennsylvania.

MERCURY

CRITERIA:

2.0 ug/l for domestic water supply (health);

0.05 ug/l for freshwater aquatic life and wildlife;

0.10 ug/l for marine aquatic life.

INTRODUCTION:

Mercury is a silver-white, liquid metal solidifying at -38.9° C to form a tin-white, ductile, malleable mass. It boils at 356.9° C, has a specific gravity of 13.6 and a vapor pressure of 1.2×10^{-3} mm of mercury. Mercury has three oxidation states: (1) zero (elemental mercury); (2) +1 (mercurous compounds); and (3) +2 (mercuric compounds). Mercury is widely distributed in the environment and biologically is a non-essential or non-beneficial element. Historically it was recognized to possess a high toxic potential and was used as a germicidal or fungicidal agent for medical and agricultural purposes.

Human poisoning by mercury or its compounds clinically has been recognized. Although its toxic properties are well known, dramatic instances of toxicosis in man and animals have occurred recently, e.g., the Minamata Bay poisonings (Irukayama, et al., 1962; Irukayama, 1967). In addition to the incidents in Japan, poisonings have also occurred in Iraq, Pakistan and Guatemala as a result of ingestion of flour and seed treated with methyl and ethylmercury compounds (Bakir, et al., 1973). Mercury intoxication may be acute or chronic and toxic effects vary with the form of mercury and its mode of entry into the organism. The mercurous salts are less soluble than the mercuric and consequently are less toxic. For man, the fatal oral dose of mercuric salts ranges from 20 mg to 3.0 g (Stokinger, 1963). Symptoms of acute, inorganic mercury poisoning include pharyngitis, gastroenteritis, vomiting followed by ulcerative hemorrhagic colitis, nephritis,

hepatitis, and circulatory collapse. Chronic mercury poisoning results from exposure to small amounts of mercury over extended time periods. Chronic poisoning from inorganic mercurials most often has been associated with industrial exposure, whereas poisoning from the organic derivatives has been the result of accidents or environmental contamination. Alkyl compounds are the derivatives of mercury most toxic to man, producing illness, irreversible neurological damage, or death from the ingestion of amounts in milligrams (Berglund and Berlin, 1969).

The mercury content of unpolluted U.S. rivers from 31 States where natural mercury deposits are unknown is less than 0.1 ug/l (Wershaw, 1970). Jenne (1972) found also that the majority of U.S. waters contained less than 0.1 ug/l of mercury. The lower limit of detection in these studies was 0.1 ug/l. Total mercury values of 0.045 ug/l recently were determined in Connecticut River water by Fitzgerald and Lyons (1973) using more sensitive methods. Marine waters have been shown to contain concentrations of mercury from a low of .03 to a high of 0.2 ug/l, but most marine waters fall within the range of .05 to .19 ug/l mercury (Robertson, et al., 1972). Mining, agriculture and waste discharges contribute to the natural levels found.

RATIONALE:

Several forms of mercury, ranging from elemental to dissolved inorganic and organic species, are expected to occur in the environment. The recent discovery that certain microorganisms have the ability to convert inorganic and organic forms of mercury to the highly toxic methyl or dimethyl mercury has made any form of mercury potentially hazardous to the environment

(Jensen and Jernelov, 1969). In studies on the biochemical kinetics of mercury methylation, Bisogni and Lawrence (1973) demonstrated that in water, under naturally occurring conditions of pH and temperature, inorganic mercury can be converted readily to methylmercury.

Wood (1974) has argued further that whenever mercury in any form is added to the aquatic environment, a combination of microbially catalyzed reactions and chemical equilibrium systems is capable of leading to steady state concentrations of dimethyl mercury, methylmercuric ion, metallic mercury, mercuric ion, and mercurous ion. Thus, it is evident that the total mercury level should be the basis for a mercury criterion instead of any particular form in which it may be found within a sample.

Hannerz (1968), using 0.1 mg/l of several mercury compounds in ponds, concluded that algae and other aquatic plants accumulate mercury primarily by surface adsorption. This study demonstrated that all of the mercury compounds used were taken up by fish both directly from the water and from food. The accumulation rate was shown to be fast, while the elimination rate was slow, leading to concentration factors of 3,000-fold and higher. According to McKim (1974), concentration factors by fish in excess of 10,000 times the amount of mercury in the surrounding water have been demonstrated.

In a test period of 20 to 48 weeks, several species of fish accumulated more than 0.5 ug/gm mercury in their tissues from a water habitat containing 0.018 to 0.030 ug/l methylmercury (McKim, et al., In Press) representing concentration factors of from 27,800 to 16,600.

Quantities of ingested mercury safe for man can be estimated from data presented in "Methyl Mercury in Fish" (1971). From epidemiological evidence, the lowest whole-blood concentration of methylmercury associated with toxic symptoms is 0.2 ug/g. This blood concentration can be compared to 60 ug/g mercury in hair. These values, in turn, correspond to prolonged continuous exposure at approximately 0.3 mg/70 kg body weight/day. By using a safety factor of 10, the maximum dietary intake from all sources, including air, water, and food, should not exceed 30 ug/person/day mercury. Although the safety factor is computed for adults, limiting ingestion by children to 30 ug/day of mercury is believed to afford some lesser degree of safety. If the exposure to mercury were from fish alone, the limit would allow for a maximum daily consumption of 60 grams (420 g/week) of fish containing 0.5 mg/kg mercury. A drinking water criterion of 2.0 ug/l would permit a daily intake of 4 ug mercury assuming an average consumption of 2 liters of water per day. If the mercury is not all in the alkyl form, a greater margin of safety will exist.

The levels of mercury in tissues of livestock consumed by humans should not exceed 0.5 mg/kg mercury. The tissue concentration of 0.5 mg/kg correlates approximately with a blood level of 0.1 ug/l. A mercury level of less than 1.0 ug/l in livestock water is considered compatible with these concentrations, thus a mercury concentration of 0.05 ug/l will provide an adequate safety factor.

Several chronic toxicity tests have been conducted to measure the adverse effects of organomercurials on survival, growth, and reproduction of several fish species. In a 3-year chronic toxicity study involving

three generations of brook trout, Salvelinus fontinalis, exposed to methylmercuric chloride, gross toxic symptoms were observed after six-months' exposure of yearling trout to 2.9 ug/l of mercury (McKim, et al., In Press). Spawning occurred at all lower mercury concentrations tested, but the offspring of parental fish exposed to 0.93 ug/l of mercury exhibited reduction in growth 90 days after hatching. After 24-months' exposure of second generation fish to 0.93 ug/l, behavioral symptoms were noted, there was no spawning, and mortality was 94 percent. No adverse effects were observed in the brook trout exposed to methylmercuric chloride concentrations of 0.29 ug/l mercury and below. A full life cycle chronic toxicity test was conducted with fathead minnows, Pimephales promelas, exposed to methylmercuric chloride (Mount, 1974). All died after three months' exposure to concentrations of 0.80 and 0.41 ug/l mercury. Ninety-two percent of the fish exposed to 0.23 ug/l mercury also died within the three-month period. Spawning was completely inhibited at 0.12 ug/l mercury with males not developing sexually. No toxic effects were noted on survival or growth of the offspring produced in 0.07 ug/l of mercury.

Two chronic toxicity tests were conducted with one invertebrate, Daphnia magna. Mercury as mercuric chloride and methylmercuric chloride caused significant reproductive impairment at concentrations of 2.7 ug/l and 0.04 ug/l mercury, respectively (Biesinger, 1974).

Matida, et al. (1971) found that the LC_{50} for phenylmercuric acetate, methylmercuric chloride, and mercuric chloride with rainbow trout fingerlings, Salmo gairdneri, were 8.5, 30, and 310 ug/l, respectively. Wobeser (1973) examined the toxicity of methylmercuric chloride to two life stages

of rainbow trout. The 96-hour LC_{50} for newly hatched sac fry was 24 ug/l of mercury, while rainbow trout fingerlings had a 96-hour LC_{50} value of 42 ug/l.

Eisler (1974) found that concentrations of 1.0 ug/l mercury represent a distinct threat to selected species of marine organisms and, based on a comparison with freshwater species, the accumulation of mercury is similar in fresh and marine water.

Because of methylation and bioconcentration of methylmercury, mercury limits must take into consideration the food chain transport path from aquatic organisms to man. Regardless of the mercury form present, the major portion of the mercury will ultimately reside in the bottom sediments where, through microbial action, mono- and dimethylmercury can be formed.

These forms of mercury are bioconcentrated many-fold in fish and other aquatic organisms because of the very rapid uptake and the relative inability of the fish to excrete methylmercury from their tissues. As a result, methylmercury in fish tissues may exceed the 0.5 mg/kg FDA guideline. This occurs in water concentrations that have no observed toxic effects on the fish. Methylation rates are highly dependent upon water quality conditions, but sufficient evidence exists to suggest that the process can occur in the pH range of 5 to 9 under aerobic or anaerobic conditions; hence, it is assumed that methylation can and will occur in natural waters.

Demethylation processes can deplete methylmercury concentrations in water. Methylmercury appears to persist for sufficient time periods, however to allow uptake by aquatic organisms. Hence, demethylation processes can have an effect on uptake rates of methylmercury, but do not terminate the transport path. It appears that the methylation process takes place at the water/sediment interface, particularly in the sediment area in which the benthic organisms are most active. The movement of benthos within the sediments contributes

to the methylation process by physically expanding the area of water/sediment interface. Through ingestion of the detritus in the sediments, benthos acquire a body burden of mercury that will in turn be transported to fish upon ingestion.

The FDA established guideline for mercury in edible fish is 0.5 mg/kg. Thus, the maximum level of mercury in receiving waters should be based on the premise that this level should not be exceeded. A mercury concentration factor for certain freshwater species has been shown to be in excess of 10,000.

Upon dividing the 0.5 mg/l FDA temporary tolerance by the 10,000-fold accumulation factor, a level of 0.05 ug/l for total mercury in fresh water results, which can be assumed to protect the human consumer of freshwater fish. It was pointed out above that 0.04 ug/l mercury as methylmercuric chloride caused significant reproductive impairment to Daphnia magna. Recognizing, however, that natural total mercury concentrations in fresh water are in the same range, and that a total mercury level of 0.05 ug/l would be divided among several chemical forms which differ markedly in their toxicity, it is believed that a criterion of 0.05 ug/l total mercury will offer a reasonable level of protection to freshwater aquatic life as well as to the human consumer.

Recognizing that seawater contains about 0.1 ug/l mercury and that this level is 1/10 of that found by Eisler (1974) to represent a threat to selected species of marine organisms, it is recommended that the criterion for the protection of marine aquatic life be 0.1 ug/l.

REFERENCES CITED

Bakir, F., S.F. Damluji, L. Amin-Zaki, M. Murtadha, A. Khalidi, N.Y. Al Rawl, S. Tikriti, H.I. Dhahir, T.W. Clarkson, J.C. Smith and R.A. Doherty, 1973. Methylmercury Poisoning in Iraq. Science 181:230-240.

Berglund, F. and M. Berlin, 1969. Risk of accumulation in men and mammals and the relation between body burden of methylmercury and toxic effects. In: Chemical Fallout, Thomas, Springfield, Ill. pp. 258-273.

Biesinger, K.E., 1974. Testimony in the matter of proposed toxic pollutant effluent standards for Aldrin-Dieldrin, et al. FWPCA (307) Docket No. 1, Exhibit No. 14.

Bisogni, J.J. and A.W. Lawrence, 1973. Methylation of mercury in aerobic and anaerobic environments. Technical Report 63, Cornell University Resources and Marine Sciences Center, Ithaca, New York.

Eisler, R., 1974. Testimony in the matter of proposed toxic pollutant effluent standards for Aldrin-Dieldrin, et al. FWPCA (307) Docket No. 1.

Fitzgerald, W.F., and W.B. Lyons, 1973. Organic mercury compounds in coastal waters. Nature, 242.

Food and Drug Administration, 1974. Poisonous or deleterious substances in peanuts, evaporated milk, fish and shellfish. Proposed Rules, Federal Register, Dec. 6, 1974, Washington, D.C.

Hannerz, L., 1968. Experimental investigations on accumulation of mercury in water organisms. Fishery Board of Sweden, Institute of Freshwater Research, Drottinghomm, Report 48.

Irukayama, K., et al., 1962. Studies on the origin of the causative agent of Minamata disease, III. Industrial wastes containing mercury compounds from Minamata factory. Kumamoto Medical Jour., 15(2):57.

Irukayama, K., 1967. The pollution of Minamata Bay and Minamata disease. Adv. Water Poll. Res., 3:153.

Jenne, E.A., 1972. Mercury in waters of the United States, 1970-71. Open file report, U.S. Geol. Surv., Menlo Park, California.

Jensen, S. and A. Jernelov, 1969. Biological methlyation of mercury in aquatic organisms. Nature, 223.

Matida, Y., et al., 1971. Toxicity of mercury compounds to aquatic organisms and accumulation of the compounds by the organisms. Bull. Freshwater Res. Lab., Vol. 21, No. 2.

McKim, J.M., 1974. Testimony in the matter of proposed toxic pollutant effluent standards for Aldrin-Dieldrin, et al. FWPCA (307) Docket No. 1.

McKim, J.M., et al., 1976. Long term effects of methylmercuric chloride on three generations of brook trout (Salvelinus fontinalis);)toxicity, accumulation, distribution, and elimination. Jour. Fish. Res. Bd. Canada, (In Press).

Methyl mercury in fish, 1971. A toxicologic-epidemiologic evaluation of risks. Report from an expert group. Nord. Hyg. Tidskr., Suppl. 4.

Mount, D.I., 1974. Chronic toxicity of methylmercuric chloride to fathead minnow. Testimony in the matter of proposed toxic effluent standards for Aldrin-Dieldrin, et al. FWPCA (307) Docket No. 1, Exhibit No. 4.

Robertson, E.E., et al., 1972. Battelle Northwest contribution to the IDOE Base-line Study, Battelle Northwest 1972 IDOE Workshop. p. 231.

Stokinger, H.E., 1963. Mercury Hg^{237}. In: Industrial Hygiene and Toxicology Vol. 2, 2nd ed. F.A. Patty, Ed., New York Interscience, p. 1090.

Wershaw, R.L., 1970. Mercury in the environment. Geological Survey Professional Paper #713., G.P.O.

Wobeser, G.A., 1973. Aquatic mercury pollution: Studies of its occurrence and pathologic effects on fish and mink. Thesis: Dept. of Vet. Pathology, U. of Saskatchewan, Saskatoon, Canada.

Wood, J.M., 1974. Biological cycles for toxit elements in the environment. Science, 183:1049.

MIXING ZONES

INTRODUCTION:

A mixing zone is an area contiguous to a discharge where receiving water quality may neither meet all quality criteria nor requirements otherwise applicable to the receiving water. It is obvious that any time an effluent is added to a receiving waterway, where the effluent is poorer in quality, there will be a zone of mixing. The mixing zone should be considered as a place where wastes and water mix and not as a place where effluents are treated.

RATIONALE:

Because damage to the aquatic resource can occur when quality standards are violated, the permissible size of a mixing zone is dependent upon the acceptable amount of damage. The permissible size depends in part on the size of the particular receiving water; the larger the water body, the larger the mixing zone may be without violating quality standards in more than a given percentage of the total area or volume of the receiving water. Likewise, the greater number of mixing zones within a reach of river or within a water body, the smaller each must be in order to maintain an appropriate mixing zone to water body ratio. Future industrial and population growths must be considered in designating such areas for wastes admixture.

As a guideline, the quality for life within a mixing zone should be such that the 96-hour LC_{50} for biota significant to the indigenous aquatic community is not exceeded; the mixing zone should be free from effluent substances that will settle to form objectionable deposits, free from effluent-associated materials that float to form unsightly masses, and free from effluent-associated substances that produce objectionable color, odor, or turbidity.

A prime purpose in designating the location, size, and area constraints of a mixing zone is to protect the aquatic life within the receiving waterway. Shallow water areas, generally, are the nursery areas for aquatic ecosystems. Designating offshore mixing areas or providing a larger available volume or area for mixing offshore as a viable alternative to a smaller shoreside area has a lesser potential for adverse biotic effects than a comparable discharge area in shallow water. Offshore, diffusion will tend to occur in all directions and not be constrained by a land barrier. Mixing zones may be less harmful biologically when located deep within the receiving water and, wherever possible, beneath the light-penetration area where photosynthesis occurs and algae and associated protozoa and other organisms provide the extensive base for the aquatic food web.

An axiom of environmental quality is that different areas vary in ecological importance, one from the other. Generally the highest importance, and therefore the greatest protection, must be placed on shallow-water shoreline areas of rivers, lakes and coastal zones and on the nation's wetlands. These are commonly the areas that protect the young and supply the food not only for the animals that live in open waters but also for those animals that depend upon water in some measure for their existence. Likewise, one local aquatic area may have a higher social or ecological value than another, and the higher that value the greater the protection from degradation that is warranted within a waste mixing area.

Mixing zones should be located in such a manner that they do not form a barrier to the migratory routes of aquatic species. On a given reach of a stream or river , it would be good practice to limit the total mixing zone area to one-third of the receiving water width. In the same fashion, the combined

areas of all mixing zones within a lake should not exceed ten percent of the lake surface area. In some cases, this maximum should be reduced depending on lake volume and other local conditions. Within an estuary, the maximal dimension of the mixing area should not exceed 10 percent of the cross-sectional area of the waterway. It is not the objective of this rationale to outline limits for effluents, but to provide the reader with some of the general biological and physical considerations necessary for the establishment of mixing zones.

In essence, the positioning of mixing zones should be accomplished in a manner that will provide the greatest protection to aquatic life and for the various uses of water. Generally, shoreline and surface areas for waste admixture should be discouraged in preference to deep water, offshore designations. The relative social and ecological values of the aquatic life that may inhabit a particular waterway area should be given due consideration in zone definition. (Fetterolf, 1973; NAS, 1974) The designation of particular mixing zones is a task that should follow the biological, physical and chemical appraisal of the receiving waterway.

References cited:

Fetterolf, C.M , Jr., 1973. Mixing zone concepts; Biological Methods for the Assessment of Water Quality, ASTM STP-528, American Society for Testing and Materials, pp. 31-45.

National Academy of Sciences, National Academy of Engineering, 1974. Water Quality Criteria, 1972. U. S. Government Printing Office, Washington, D.C.

NICKEL

CRITERION:

0.01 of the 96-hour LC50 for freshwater and marine aquatic life.

INTRODUCTION:

Nickel is a silver-white, metallic element seldom occurring in nature in the elemental form. Nickel salts are soluble and can occur as a leachate from nickel-bearing ores. Kopp and Kroner (1967) detected nickel in the Lake Erie Basin at a frequency of 53 percent and a mean concentration of 56 ug/l. At several selected stations, dissolved nickel ranged from 3 to 86 ug/l and suspended nickel from 5 to 900 ug/l. Nickel is present in sea water at 5 to 7 ug/l (NAS, 1974).

RATIONALE:

Nickel is considered to be relatively non-toxic to man (Schroeder, et al., 1961) and a limit for nickel is not included in the EPA National Interim Primary Drinking Water Regulations ((40 FR 59566, December 24, 1975) The toxicity of nickel to aquatic life, as reported by McKee and Wolf (1963), indicates tolerances that vary widely and that are influenced by species, pH, synergistic effects and other factors. The survival curves for sticklebacks in soft tap water indicate a lethal limit of 800 ug/l nickel (Jones, 1939). Pickering and Henderson (1964) reported that 96-hour LC_{50} values of nickel in soft water for four species of fish varied from 4.6 to 9.8 mg/l, and in hard

water for two fish species, the 96-hour LC_{50} varied from 39.2 to 42.4 mg/l. The 96-hour LC_{50} values for two species of aquatic insects were reported by Warnick and Bell (1969) to be 4.0 and 33.5 mg/l nickel. Biesinger and Christensen (1972) found that the three-week LC50 value for Daphnia magna in soft water was 130 ug/l nickel; 95 ug/l caused a 50 percent impairment in reproductivity, and 30 ug/l caused a 16 percent impairment.

In continuous bioassay tests designed to determine chronic effects, Pickering (1974) demonstrated that nickel concentrations of 380 ug/l and lower in hard water did not adversely affect survival, growth, or reproduction of the fathead minnow. Nickel concentrations of 730 ug/l caused a significant reduction both in the number of eggs per spawning and in the hatchability of the eggs.

Calabrese, et al.,(1973) reported a 48-hour LC50 of 1,180 ug/l for American oyster embryo, Crassostrea virginica, for larvae of the hard shell clam, Mercenaria mercenaria (Calabrese & Nelson, 1974). Jones (1939) reported a 96-hour LC50 of 800 ug/l for the euryhaline stickleback, Gasterosteus aculeatus. Gentile (1975) found that the 96-hour LC50 for the marine copepod, Acartia tonsa was 625 ug/l.

Nickel salts have been shown to be injurious to plants. In sand and nickel solution experiments, Vanselow (1966) demonstrated that at 0.5 to 1.0 mg/l, nickel is toxic to a number of plants. The toxicity exhibited to plants by nickel varied widely with the species. McKee and Wolf (1963) indicated that nickel was extremely toxic to citrus. Chang and Sherman (1953) found

that tomato seedlings were injured by 0.5 mg/l nickel. Hop plants were shown to be injured by nickel at 1.0 mg/l (Legg and Ormerod, 1958). Plants exhibiting less susceptibility to nickel were: oats, with toxic effects at 2.5 mg/l (Crooke, 1954); corn at 2 mg/l; and tobacco with no toxic effects at 3.0 mg/l (Soane and Saunders, 1959).

Data indicate that, (1) nickel in water is toxic to plant life at concentrations as low as 500 ug/l, (2) nickel adversely affects reproduction of a freshwater crustacean at concentrations as low as 95 ug/l, (3) marine clam larvae can be killed by concentrations of nickel as low as 310 ug/l, and (4) reproduction of the fathead minnow is detrimentally affected by nickel at concentrations as low as 730 ug/l. Concentrations of nickel at or below 100 ug/l should not be harmful to irrigated plants or marine and freshwater aquatic organisms.

REFERENCES CITED:

Biesinger, K.E. and G.M. Christensen, 1972. Effects of various metals on survival, growth, reproduction and metabolism of Daphnia magna. Jour. Fish. Res. Bd. Can., 29: 1691.

Calabrese, A., et al., 1973. The toxicity of heavy metals to embryos of the American oyster, Crassostrea virginica. Marine Biology, 18:162.

Calabrese, A. and D.A. Nelson, 1974. Inhibition of embryonic development of the hard clam, Mercenaria mercenaria, by heavy metals. Bull. Environ. Contam. Tox. 11:92

Chang, A.T. and G.D. Sherman, 1953. The nickel content of some Hawaiian soils and plants and the relation of nickel to plant growth. Hawaiian Agr. Exp. Sta. Bull., 9: 3.

Crooke, W.M., 1954. Effect of nickel versenate on oat plants. Nature 173: 403.

Gentile, J. 1975. Environmental Research Laboratory, Narragansett, R.I., Semi-Annual Report.

Jones, J.R.E., 1939. The relation between the electrolytic solution pressures of the metals and their toxicity to stickleback. Jour. Exper. Biol. 16:425.

Kopp, J.F. and R.C. Kroner, 1967. Trace metals in waters of the United States. U.S. Department of Interior, FWQA, Cincinnati, Ohio.

Legg, J.T. and P.J. Ormerod, 1958. The nickel content of hop plants with reference to nettlehead symptoms. Annual Report of the East Malling Research Station (England), 45: 129.

McKee, J.E. and H.W. Wolf, 1963. Water quality criteria. California State Water Resources Control Board, Pub. No. 3-A.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office,

Pickering, Q.H. and C. Henderson, 1964. The acute toxicity of some heavy metals to different species of warm water fishes. Purdue University Ext. Serv., 117: 578.

Pickering, Q.H., 1974. Chronic toxicity of nickel to the fathead minnow. Jour. Water Poll. Cont. Fed., 46: 760.

Schroeder, H.A., et al., 1961. Abnormal trace elements in man--nickel. Jour. Chron. Dis., 15: 51.

Soane, B.K. and D. H. Sanders, 1959. Nickel and chromium toxicity of serpentine soils in southern Rhodesia. Soil Science, 88: 322.

Vanselow, A.P., 1966. Diagnostic criteria for plants and soils. Univ. of Calif., Div. of Agr. Sci., Berkley, California, pp. 142-156.

Warnick, S.L. and H.L. Bell, 1969. The acute toxicity of some heavy metals to different species of aquatic insects. Jour. Water Poll. Cont. Fed., 41: 280.

NITRATES; NITRITES

CRITERION:

10 mg/l nitrate nitrogen (N) for
domestic water supply (health).

INTRODUCTION:

Two gases (molecular nitrogen and nitrous oxide) and five forms of nongaseous, combined nitrogen (amino and amide groups, ammonium, nitrite, and nitrate) are important in the nitrogen cycle. The amino and amide groups are found in soil organic matter and as constituents of plant and animal protein. The ammonium ion is either released from proteinaceous organic matter and urea, or is synthesized in industrial processes involving atmospheric nitrogen fixation. The nitrite ion is formed from the nitrate or the ammonium ions by certain microorganisms found in soil, water, sewage, and the digestive tract. The nitrate ion is formed by the complete oxidation of ammonium ions by soil or water microorganisms; nitrite is an intermediate product of this nitrification process. In oxygenated natural water systems nitrite is rapidly oxidized to nitrate. Growing plants assimilate nitrate or ammonium ions and convert them to protein. A process known as denitrification takes place when nitrate-containing soils become anaerobic and the conversion to nitrite, molecular nitrogen, or nitrous oxide occurs. Ammonium ions may also be produced in some circumstances.

Among the major point sources of nitrogen entry into water bodies are municipal and industrial wastewaters, septic tanks, and feedlot discharges. Diffuse sourcescof nitrogen include farm-site fertilizer and animal wastes, lawn fertilizer, leachate from waste disposal in dumps or sanitary landfills, atmospheric fallout, nitric oxide and nitrite

discharges from automobile exhausts and other combustion processes, and losses from natural sources such as mineralization of soil organic matter (NAS, 1972). Water reuse systems in some fish hatcheries employ a nitrification process for ammonia reduction; this may result in exposure of the hatchery fish to elevated levels of nitrite (Russo, et al., 1974).

RATIONALE:

In quantities normally found in food or feed, nitrates become toxic only under conditions in which they are, or may be, reduced to nitrites. Otherwise, at "reasonable" concentrations, nitrates are rapidly excreted in the urine. High intake of nitrates constitutes a hazard primarily to warm blooded animals under conditions that are favorable to their reduction to nitrite. Under certain circumstances, nitrate can be reduced to nitrite in the gastrointestinal tract which then reaches the bloodstream and reacts directly with hemoglobin to produce methemoglobin, with consequent impairment of oxygen transport.

The reaction of nitrite with hemoglobin can be hazardous in infants under three months of age. Serious and occasionally fatal poisonings in infants have occurred following ingestion of untreated well waters shown to contain nitrate at concentrations greater than 10 mg/l nitrate nitrogen (N) (NAS, 1974). High nitrate concentrations frequently are found in shallow farm and rural community wells, often as the result of inadequate protection from barnyard drainage or from septic tanks (USPHS, 1961; Stewart, et al., 1967). Increased concentrations of nitrates also have been found in streams from farm tile drainage in areas of intense fertilization and farm crop production (Harmeson, et al., 1971). Approximately 2000 cases of infant methemoglobinemia

have been reported in Europe and North America since 1945; 7 to 8 percent of the affected infants died (Walton, 1951; Sattelmacher, 1962). Many infants have drunk water in which the nitrate nitrogen content was greater than 10 mg/l without developing methemoglobinemia. Many public water supplies in the United States contain levels that routinely are in excess of this amount, but only one U.S. case of infant methemoglobinemia associated with a public water supply has ever been reported (Vigil, et al., 1965). The differences in susceptibility to methemoglobinemia are not yet understood but appear to be related to a combination of factors including nitrate concentration, enteric bacteria, and the lower acidity characteristic of the digestive systems of baby mammals. Methemoglobinemia symptoms and other toxic effects were observed when high nitrate well waters containing pathogenic bacteria were fed to laboratory mammals (Wolff, et al., 1972). Conventional water treatment has no significant effect on nitrate removal from water (NAS, 1974).

Because of the potential risk of methemoglobinemia to bottle-fed infants, and in view of the absence of substantiated physiological effects at nitrate concentrations below 10 mg/l nitrate nitrogen, this level is the criterion for domestic water supplies. Waters with nitrite nitrogen concentrations over 1 mg/l should not be used for infant feeding. Waters with a significant nitrite concentration usually would be heavily polluted and probably bacteriologically unacceptable.

Westin (1974) determined that the respective 96-hour and 7-day LC_{50} values for chinook salmon, Oncorhynchus tshawytscha, were 1310 and 1080 mg/l nitrate nitrogen in fresh water and 990 and 900 mg/l nitrate nitrogen in 15 o/oo saline water. For fingerling rainbow trout, Salmo gairdneri, the respective 96-hour and 7-day LC_{50} values were 1360 and 1060 mg/l nitrate nitrogen in fresh water, and 1050 and 900 mg/l nitrate nitrogen in 15 o/oo saline water. Trama (1954) reported that the 96-hour LC_{50} for bluegills, Lepomis macrochirus, at 20° C was 2000 mg/l nitrate nitrogen (sodium nitrate) and 420 mg/l

nitrate nitrogen (potassium nitrate). Knepp and Arkin (1973) observed that largemouth bass, Micropterus salmoides, and channel catfish, Ictalurus punctatus, could be maintained at concentrations up to 400 mg/l nitrate (90 mg/l nitrate nitrogen) without significant effect upon their growth and feeding activities.

The 96-hour and 7-day LC_{50} values for chinook salmon, Oncorhynchus tshawytscha, were found to be 0.9 and 0.7 mg/l nitrite nitrogen in fresh water (Westin, 1974). Smith and Williams (1974) tested the effects of nitrite nitrogen and observed that yearling rainbow trout, Salmo gairdneri, suffered a 55 percent mortality after 24 hours at 0.55 mg/l, fingerling rainbow trout suffered a 50 percent mortality after 24 hours of exposure at 1.6 mg/l, and chinook salmon, Oncorhynchus tshawytscha, suffered a 40 percent mortality within 24 hours at 0.5 mg/l. There were no mortalities among rainbow trout exposed to 0.15 mg/l nitrite nitrogen for 48 hours. These data indicate that salmonids are more sensitive to nitrite toxicity than are other fish species, e.g., minnows, Phoxinus laevis, that suffered a 50 percent mortality within 1.5 hours of exposure to 2030 mg/l nitrite nitrogen, but required 14 days of exposure for mortality to occur at 10 mg/l (Klingler, 1957), and carp, Cyprinus carpio, when raised in a water reuse system, tolerated up to 1.8 mg/l nitrite nitrogen (Saeki, 1965).

Gillette, et al. (1952) observed that the critical range for creek chub, Semotilus atromaculatus, was 80 to 400 mg/l nitrite nitrogen. Wallen, et al. (1957) reported a 24-hour LC_{50} of 1.6 mg/l nitrite nitrogen, and 48- and 96-hour LC_{50} values of 1.5 mg/l nitrite

nitrogen for mosquitofish, Gambusia affinis. McCoy (1972) tested the nitrite susceptibility of 13 fish species and found that logperch, Percina caprodes, were the most sensitive species tested (mortality at 5 mg/l nitrite nitrogen in less than 3 hours of exposure), whereas carp, Cyprinus carpio, and black bullheads, Ictalurus melas, survived 40 mg/l nitrite nitrogen for a 48-hour exposure period; the common white sucker, Catostomus commersoni, and the quillback, Carpiodes cyprinus, survived 100 mg/l for 48 and 36 hours, respectively.

Russo, et al. (1974) performed flow-through nitrite bioassays in hard water (hardness = 199 mg/l $CaCO_3$, alkalinity = 176 mg/l $CaCO_3$, pH = 7.9) on rainbow trout, Salmo gairdneri, of four different sizes, and obtained 96-hour LC_{50} values ranging from 0.19 to 0.39 mg/l nitrite nitrogen. Duplicate bioassays on 12-gram rainbow trout were continued long enough for their toxicity curves to level off, and asymptotic LC_{50} concentrations of 0.14 and 0.15 mg/l were reached in 8 days; on day 19, additional mortalities occurred. For 2-gram rainbow trout, the minimum tested level of nitrite nitrogen at which no mortalities were observed after 10 days was 0.14 mg/l; for the 235-gram trout, the minimum level with no mortality after 10 days was 0.06 mg/l.

It is concluded that: (1) levels of nitrate nitrogen at or below 90 mg/l would have no adverse effects on warm water fish (Knepp and Arkin, 1973); (2) nitrite nitrogen at or below 5 mg/l should be protective of most warm water fish (McCoy, 1972); and (3) nitrite nitrogen at or below 0.06 mg/l should be protective of salmonid fishes (Russo, et al., 1974; Russo and Thurston, 1975). These levels

either are not known to occur or would be unlikely to occur in natural surface waters.

Recognizing that concentrations of nitrate or nitrite that would exhibit toxic effects on warm or cold water fish could rarely occur in nature, restrictive criteria are not recommended.

REFERENCES CITED:

Gillette, L.A., et al., 1952. Appraisal of a chemical waste problem by fish toxicity tests. Sewage Ind. Wastes, 24:1397.

Harmeson, R.H., et al., 1971. The nitrate situation in Illinois. Jour. Amer. Water Works Assn., 63:303.

Klingler, K., 1957. Sodium nitrite, a slow acting fish poison. Schweiz. Z. Hydrol. 19(2):565.

Knepp, G.L. and G.F. Arkin, 1973. Ammonia toxicity levels and nitrate tolerance of channel catfish. The Progressive Fish-Culturist, 35:221.

McCoy, E.F., 1972. Role of bacteria in the nitrogen cycle in lakes. Environmental Protection Agency, Water Pollution Control Research Series, U.S. Government Printing Office (EP 2.10:16010 EHR 03/72), Washington, D. C.

National Academy of Sciences, 1972. Accumulation of nitrate. National Academy of Sciences, Washington, D. C.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Russo, R.C., et al., 1974. Acute toxicity of nitrite to rainbow trout (Salmo gairdneri). Jour. Fish. Res. Bd. Can., 31:1653.

Russo, R.C. and R.V. Thurston, 1975. Acute toxicity of nitrite to cutthroat trout (Salmo clarki). Fisheries Bioassay Laboratory Tech. Report No. 75-3, Montana State University.

Saeki, A., 1965. Studies on fish culture in filtered closed-circulating aquaria. II. On the carp culture experiments in the systems. Bull. Jap. Soc. Sci. Fish., 31:916.

Sattelmacher, P.G., 1962. Methemoglobinemia from nitrates in drinking water. SchrReihe. Ver. Wasser-, Boden-u. Lufthyg. No. 21, Fischer, Stuttgart.

Smith, C.E. and W.G. Williams, 1974. Experimental nitrite toxicity in rainbow trout and chinook salmon. Trans. Amer. Fish. Soc., 103:389.

Stewart, B.A., et al., 1967. Nitrate and other pollutants under fields and feedlots. Envir. Sci. Tech., 1:736.

Trama, F.B., 1954. The acute toxicity of some common salts of sodium, potassium and calcium to the common bluegill (Lepomis macrochirus Rafinesque). Proc. Acad. Nat. Sci., Philadelphia, Pennsylvania, 106:185.

United States Public Health Service, 1961. Groundwater contamination: Proceedings of 1961 symposium. Tech. Rpt. W61-5, R.A. Taft Sanitary Engineering Center, U.S. Public Health Service, Department of Health, Education and Welfare, Cincinnati, Ohio.

Vigil, J., et al., 1965. Nitrates in municipal water supplies cause methemoglobinemia in infants. Public Health Rept., 80:1119.

Wallen, I.E., et al., 1957. Toxicity to Gambusia affinis of certain pure chemicals in turbid waters. Sewage Ind. Wastes, 29:695.

Walton, G., 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Amer. Jour. Public Health, 41:986.

Westin, D.T., 1974. Nitrate and nitrite toxicity to salmonid fishes. The Progressive Fish-Culturist, 36:86.

Wolff, I.A. and Wasserman, 1972. Nitrates, nitrites, and nitrosamines Science, 177:15.

OIL AND GREASE

CRITERIA:

For domestic water supply: Virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products.

For aquatic life:

(1) 0.01 of the lowest continuous flow 96-hour LC50 to several important freshwater and marine species, each having a demonstrated high susceptibility to oils and petrochemicals.

(2) Levels of oils or petrochemicals in the sediment which cause deleterious effects to the biota should not be allowed.

(3) Surface waters shall be virtually free from floating nonpetroleum oils of vegetable or animal origin, as well as petroleum derived oils.

INTRODUCTION:

It has been estimated that between 5 and 10 million metric tons of oil enter the marine environment annually (Blumer, 1970). A major difficulty encountered in the setting of criteria for oils and grease is that these are not definitive chemical categories, but include thousands of organic compounds with varying physical, chemical, and toxicological properties. They may be volatile or non-volatile, soluble or insoluble, persistent or easily degraded.

RATIONALE:

Field and laboratory evidence have demonstrated both acute lethal toxicity and long-term sublethal toxicity of oils to aquatic organisms. Events such as the Tampico Maru wreck of 1957 in Baja, California, (Diaz-Piferrer, 1962), and the No. 2 fuel oil spill in West Falmouth,

Massachusetts,in 1969 (Hampson and Sanders, 1969), both of which caused immediate death to a wide variety of organisms, are illustrative of the lethal toxicity that may be attributed to oil pollution. Similarly, a gasoline spill in South Dakota in November 1969 (Bugbee and Walter, 1973), was reported to have caused immediate death to the majority of freshwater invertebrates and 2500 fish, 30 percent of which were native species of trout. Because of the wide range of compounds included in the category of oil, it is impossible to establish meaningful 96-hour LC values for oil and grease without specifying the product involved. However, as the data in Table 6 show, the most susceptible category of organisms, the marine larvae, appear to be intolerant of petroleum pollutants, particularly the water soluble compounds, at concentrations as low as 0.1 mg/l.

The long-term sublethal effects of oil pollution refer to interferences with cellular and physiological processes such as feeding and reproduction which do not lead to immediate death of the organism. Disruption of such behavior apparently can result from petroleum product concentrations as low as 10 to 100 ug/l (see Table 7).

Table 7 summarizes some of the sublethal toxicities for various petroleum pollutants and various aquatic species. In addition to sublethal effects reported at the 10 to 100 ug/l level, it has been shown that petroleum products can harm aquatic life at concentrations as low as 1 ug/l (Jacobson and Boylan, 1973).

Bioaccumulation of petroleum products presents two especially important public health problems: (1) the tainting of edible, aquatic

Table 6

Summary of lethal toxicities of various petroleum products to aquatic organisms (a thorough discussion of duration and test conditions is found in Moore, Dwyer & Katz, 1973)

TYPE ORGANISM	Soluble Hydrocarbons	Dispersants	#2 Fuel Oil/ Kerosene	Fresh Crude	Gasoline	Diesel Fuel	Refinery Effluents	Waste Oil	Lubricants	Residuals	Weathered Crude
Marine Flora	(29) (29) 10 ppm-650 ppm	(29) (31) 1.2 ppm-513 mg/l [ppm]	(31) <100 µl/l [ppm]	(31) Toxic to many salt marsh plants				(14) 10 ppm			(29) Costing more
Finfish	(29) (29) 5 ppm-50 ppm	(29) (29) 1 ppm->10,000 mg/l (ppm)	(40) 550 µg/ml (ppm)	(35) 88 mg/l (ppm) (31) 18 ml/l [x10³ ppm]	(29) 91 ppm	(29) 264-420 ppm		(14) 1700 ppm		(29) 2000-510,000 mg/l [ppm]	Significant than lethal toxicity
Larvae and eggs	(29) (29) 0.1 ppm-1 ppm	(29) (29) 1 ppm-42 ppm	(31) 0.1 µl/l [ppm] (40) 4 µg/ml [ppm]	(31) 0.1 µl/l [ppm] (27) (29) 100 ppm				(14) 1->25ppm		(29) No effect reported	
Pelagic Crustacea	(29) (29) 1 ppm-10 ppm	(29) (29) 5 ppm-100 ppm	(29) (29) 5 ppm-50ppm	(29) 0.1 ml/l [x10³ ppm]- (29) 40 ppt [x10³ ppm]				(14) 15->50ppm			
Benthic Crustacea	(29) (29) 1 ppm-10 ppm	(29) (29) 2 ppm-100 ppm	(29) (29) 5 ppm-50 ppm	(15) 0.56 mg/l [ppm]						(29) >10,000 ppm	
Gastropods	(29) (29)* 10 ppm-100 ppm	(29) (29) 5 ppm-2000 ppm	(29) (29) 50ppm-500ppm	(29) Toxicity reported for several species, but no concentration given.						(29) No effect reported	
Bivalves	(29) (29) 5 ppm-500 ppm	(29) (29) 0.5 ppm-100 ppm	(31) 30-40 ml/l [x10³ ppm]	(31) 1 ml/l [x10³ ppm]- (20) 10⁵ ppm						(29) Incorporation	
Other Benthic Invertebrates	(29) (29) 1 ppm-10 ppm	(29) (29) 5 ppm-100,000ppm	(29) (29) 5 ppm-50 ppm	(29) 100-(100) ppm						(31) 1,952-2,417 mg/l [ppm]	
Freshwater Finfish	(11) (34) 10 ppm-4,924 ppm	(31) 10 ppm	(31) 1,000-150,000 mg/l [ppm]	(32) 0.3-3 mg/l [ppm] (30) 500 mg/l [ppm]	(26) [ppm] (31) 180 mg/l [ppm]	(31) 160-4000 mg/l [ppm]	(13) ~39 ppm		(31) 5000-180,000 mg/l [ppm]		
Freshwater Flora		(31) 200 ppm									
Mammals & Birds	Most lethal (or sub-lethal) effects are caused by physical coating, entanglement, ingestion of fine oil droplets, or incorporation of hydrocarbons through food chains.										

Note: 1) Numbers in brackets represent reported values (volume to volume or weight to volume basis) converted to ppm.
2) Numbers in parentheses refer to references.
3) Asterisks indicate values estimated by Moore, Dwyer, & Katz, 1973 (However estimates are supported by examples given therein)
4) In some cases, the above values have been taken from summary type presentations (for example: Moore, Dwyer & Katz, 1973); the corresponding reference numbers therefore do not necessarily refer to the original publication.
5) The manner in which the above values are reported is only an attempt to exemplify the ranges in threshold lethal toxicities, based on some of the existing published data, and should therefore not be regarded as absolute limits of toxicity for a given category of toxicant or test organism. It must be understood that there exists numerous additional published data that fall within the ranges suggested above, and possibly data that may suggest further widening these ranges. It is not the author's intention to herein summarize all of the toxicity values reported thus far for petroleum and petroleum products.

TABLE 7

SUMMARY OF SOME SUBLETHAL EFFECTS OF PETROLEUM HYDROCARBONS ON MARINE LIFE

TYPE ORGANISM	SPECIES	REFERENCE	TYPE PETROLEUM PRODUCT	CONCENTRATION	SUBLETHAL EFFECTS
Marine Flora					
	phytoplankton (Chlorella vulgaris)	Kauss, et al, 1972[a2]	crude Naphthalene	1ppm 3ppm	Increase growth Reduction of bicarbonate uptake
	phytoplankton (diatoms and dinoflagellates)	Mironov, 1970[a1,2]	"oil"	10^{-1} 10^{-4} ppm	Inhibition or delay in cellular division
	phytoplankton (Asterionella japonica)	Aubert, et al., 1969[a1]	Kerosene	3ppm; 38ppm	Depression of growth rate
	phytoplankton (Phaeodactylum tricornutum)	Lacaze, 1967[a1]	Kuwait crude	"1 ppm"	Depression of growth rate
	phytoplankton (Monochrysis lutheri)	Strand, et al., 1971[a1]	Kuwait crude; dispersant emulsions	20-100 ppm	Inhibition of growth; reduction of bicarbonate uptake at 50 ppm
	phytoplankton (Phaeodactylum tricornutum, Skeletonema costatum, Chlorella sp., Chlorococcus sp.)	Guzzi, 1973[a1]	Extracts of outboard motor oils, No. 6 fuel oil, No. 2 fuel oil.		
	phytoplankton	Gordon and Prouse, 1973[a2]	Venezuelan crude, No. 2 and 6 Fuel oils.	10-200 mg/l (ppm)	Stimulation of photosynthesis at 10-30 mg/l decrease in photosynthesis at 100-200 mg/l No. 2 fuel oil.
	Kelp (Macrocystis angustifolia	Wilber, 1968[a2]	Toluene	10 ppm	75% reduction in photosynthesis within 90 hr
	Lichen (Lichen pyrenes)	Brown, 1972[a1]	Kuwait crude, HP_{1002}	0.1-100 ppm	1 ppm emulsifier increase total C^{14} fixation
Larvae and eggs	Pink Salmon fry (Oncorhynchus gorbuscha)	Rice, 1973	Prudhoe Bay crude	1.6 ppm	Avoidance effects; could have effect on migration behavior
	Black sea turbot (Psetta maeotica)	Mironov, 1967	"oil"	0.01 ppm	Irregularity and delay in hatching-resulting larvae deformed and inactive
	Plaice larvae (Pleuronectes platessa)	Wilson, 1970[a2]	HP 1002	0-10 ppm	Disruption of photokinesis and feeding behavior
	Cod fish larvae (Gadus morrhua)	Kuhnhold, 1970[a1]	Iranian Crude	Aqueous extracts from 10^{-3} ppm, 10^{-4} ppm	Adverse effect on behavior leading to death
	Lobster larvae (Homarus americanus)	Wells, 1972[a1]	Venezuelan crude	6 ppm	Delay molt to 4th stage
	Sea Urchin larvae (Strongylocentrotus purpuratus)	Allen, 1971[a2]	extracts of Bunker C	0.1-1 ppm	Interference with fertilized egg development
	Barnacle larvae (Balanus)	Mironov, 1970	"oil"	0.0-100 ml/l (ppm)	Abnormal development
	Crab larvae (Hemigrapsus nudus crenulatus)	Mironov, 1970	"oil"	0.0-100 ml/l (ppm)	Initial increase in respiration

SUMMARY OF SOME SUBLETHAL EFFECTS OF PETROLEUM PRODUCTS ON MARINE LIFE

TYPE ORGANISM	SPECIES	REFERENCE	TYPE PETROLEUM PRODUCT	CONCENTRATION	SUBLETHAL RESPONSE
FISH	Chinook salmon (Oncorhynchus tshawytscha) Striped bass (Morone saxatilis)	Kappel & Bailey, 1973*1	Benzene	5, 10 ppm	Initial increase in respiration.
	Menidia menidia	Gardner, et al,1975	Crude (whole fractions (water-soluble (water-insoluble	140 ppm (v/v) 12 ppm estimated 588 ppm (v/v)	Histological damage to chemoreceptors.
CRUSTACEANS	Lobster, Homarus americanus)	Blumer, et al,1973	Crude, kerosene	10 ppm	Effects of chemoreception, feeding times, stress behavior, aggression, growth
	Pollicipes polymerus	Straughan, 1971	Crude-Santa Barbara	Field study after blowout.	Apparent decrease in adult breeding; no recruitment in oiled areas.
	Lobster (H. americanus)	Atema and Stein, 1970	La Rosa Crude	Extracts	Delay in feeding.
	Pachycrassus crassipes	Kittredge, 1971*1	Crude	Dilutions of diethyl ether extracts (1:100)	Inhibition of feeding.
	Uca pugnax	Krebs, 1973*1	No. 2 Fuel oil	Field observations after W.Falmouth spill.	Adverse effects on sexual behavior.
MOLLUSCS	Mussel (Mytilus edulis)	Gilfillan,1973*2	Crude	1 ppm	Reduction in carbon budget (increase in respiration; decrease in feeding.)
	Snail (Nassarius obsoletus)	Blumer, et al,1973	Kerosene	Saturated extract diluted 10^{10}	40% reductin in chemotactic perception of food.
	Snail (Nassarius obsoletus)	Jacobson & Boylan 1973	Kerosene	0.001 - 0.004 ppm	Reductin in chemotactic perception of food
	Clam (Mya arenaria)	Harry and Yevich, 1974	No. 2 fuel oil	collected from field	Gonadal tumors
	Oyster (Crassostrea virginica)	Mackin and Hopkins, 1962	Bleedwater		Reduced growth and glycogen content
	Snail (Littorina littorea)	Perkins, 1970*1	BP 1002	30 ppm	significant inhibition to growth
	Oyster (Crassostrea virginica)	Menzel, 1948; in Nelson-Smith, 1973	"oil"	0.01 ppm	market tainting
	Mussel (Mytilus edulis)	Blumer, et al, 1971*1	No. 2 fuel oil	collected from field after spill	Inhibition in development of eggs
OTHER BENTHIC INVERTEBRATES	Polychaete (Capitella capitata)	Bellan, et al, 1972*1	Detergent	0.01-10 ppm	Decrease in survival, fecundity

Note: *1 taken from National Academy of Sciences, 1975
*2 taken from Moore, Dwyer, and Katz. 1973.

REFERENCES CITED IN TABLES 6 and 7

1 Allen, H., 1971. Effects of petroleum fractions on the early development of a sea urchin. Marine Pollution Bulletin, 2:138.

2 ATEMA, J. and L. Stein. 1972. Sublethal effects of crude oil on the behavior of the American lobster. Technical Report Woods Hole Oceanographic Inst., No. 72-72.

3 Aubert, M.R. et al., 1969. Etude de la toxicite de produits chemiques vis-a-vis de la chaine bibliogique marine. Rev. Int. Oceanogr. Med., 13/14:45.

4 BAKER, J. M. 1971. Several papers in E. B. Cowell (ed.), The ecological effects of oil pollution on littoral communities. Institute of petroleum, London. 250 p.

5 BARRY, M. and P. Yevich, 1974. Incidence of cancer in the soft-shell clam, Mya arenaria. Final report of State of Maine (Dept. Sea and Shore Fisheries) to U. S. Air Force. Contract No. F. 33600-72-C-0540. 32p.

6 Bellan, G., et al., 1972. The sublethal effects of a detergent on the reproduction, development, and settlement in the polychaetous annelid Capitella capitata. Marine Biology, 14:183.

7 Blumer, M., et al., 1971. A small oil spill. Environment, 13:2.

8 BLUMER, M., J. M. Hunt, J. Atema, and L. Stein, 1973. Interaction between marine organisms and oil pollution. EPA-R3-73-042. 97 p.

9 Brockson, R. W. and H. T. Bailey, 1973. Respiratory response of juvenile Chinook salmon and striped bass exposed to benzene, a water-soluble component of crude oil. In: Proceedings, Joint Conference on Prevention and Control of Oil Spills. Amer. Pet. Inst., Washington, D. C.

10 Brown, D. H. 1972. The effect of Kuwait crude oil and a solvent emulsifier on the metabolism of the marine lichen, Lichina pygmaea. Marine Biology, 12:309.

11 CAIRNS AND SCHEIER, 1959.

12 CHIPMAN, W. A. and P. S. Galtsoff. 1949. Effects of oil mixed with carbonized sand on aquatic animals. Spec. Scient. Rep. U. S. Fish Wildl. Serv. 1. 52 p.

13 DORRIS, T. C., W. Gould, and C. R. Jenkins. 1959. Toxicity bioassay of oil refinery effluents in Oklahoma. In Trans. 2nd Sem. Biol. Prob. Water Poll., R. A. Taft San. Eng. Center, Cincinnati, Ohio, Tech. Rep. WGO-3: 276-285.

14 ENVIRONMENTAL PROTECTION AGENCY. 1974. Waste Oil Study. Report to Congress. 401 p.

15 Gardner, G.R., et al., 1973. Analytical approach in the evaluation of biological effects. Jour. Fish. Res. Bd. Canada, 35:3185.

16 Gilfillan, E.C., 1973. Effects of seawater exracts of crude oil on carbon budgets in two species of mussels. In: Proceedings, Joint Conference on Prevention and Control of Oil Spills. Amer. Pet. Inst., Washington, D.C.

17 Gordon, D. C. and N. J. Prouse, 1973 ~~(in press)~~. The effects of three different oils on marine phytoplankton photosynthesis. Marine Biol. 22:329-333

18 Jacobson, S.M. and Boylan, 1973. Effect of seawater soluble fraction of kerosene on chemotaxis in a marine snail, Nassarius obsoletus. Nature. 241:213.

19 KARINEN, J. F. and S. D. Rice. 1974. Effects of Prudhoe Bay crude oil on molting tanner crabs, Chionoecetes bairdi. Marine Fisheries Review. 36:31.

20 Kauss, et al., 1972. Field and laboratory studies of the effects of crude oil spills on phytoplankton. In: Proceedings, 18th Annual Technical Conference, Environmental Progress in Science and Education.

21 Kittredge, J.S., 1973. Effects of water-soluble component of oil pollution on chemoreception by crabs. Fisheries Bulletin.

22 Krebs, C.T., 1973. Qualitative observations of the marsh fiddler (Uca Pugnax) populations in Wild Harbor Marsh following the September, 1969 oil spill. National Academy of Sciences, Washington, D.C., Unpublished manuscript.

23 Kuhnhold, W. W., 1970. The influence of crude oils on fish fry. In: Proceedings, FAO conference, Rome, Italy.

24 Lacaze, J. C., 1967. Etude de la croissance d'une algue planctonique en presence d'un detergent utilize pour la destruction des nappes de petrole en mer. C.R. Acad. Sci. (Paris) 265 (Ser. D):489.

25 MACKIN, J. M. and S. H. Hopkins. 1961. Studies on oyster mortality in relation to natural environments and to oil fields in Louisiana. Publs. Inst. Mar. Sci. Univ. Tex. 7:1-131.

26 MEINCK, F. et. al. 1956. Industrie-Abwasser. 2nd Edit. (Gustov Fischer Verlag, Stuttgart). p. 536, 48 D. M.

27 Mironov, O. G., 1967. Effects of low concentrations of petroleum and its products on the development of roe of the Black Sea flatfish. Vop. Ikhtiol. 7(3):557.

28 Mironov, O. G., 1970. The effect of oil pollution on flora and fauna of the Black Sea. In: Proceedings, FAO Conference on Marine Pollution and its Effects on Living Resources and Fish. Rome, December, 1970. Food and Agriculture Organization of the United Nations, Rome.

29 MOORE, S. F., R. L. Dwyer and S. N. Katz. 1973. A preliminary assessment of the environmental vulnerability of Machias Bay, Maine to oil supertankers. Report No. MITSG 73-6. 162 p.

30 MORROW, J. E. 1974. Effects of crude oil and some of its components on young coho and sockeye salmon. Publication EPA-660/3-73-018. U. S. E. P. A.

31 NELSON-SMITH, A. 1973. Oil pollution and marine ecology. Plenum press. New York. 260 p.

32 Nuzzi, R., 1973. Effects of water soluble extracts of oil on phytoplankton. In: Proceedings, Joint Conference on Prevention and Control of Oil Spills. Amer. Pet. Inst., Washington, D.C.

33 Perkins, E.J., 1970. Some effects of "detergents" in the marine environment. Chem. Ind., 1:14.

34 PICKERING, Q. H. and C. Henderson. 1966. Acute toxicity of some important petrochemicals to fish. Water Poll. Contr. Fed. J. 38 (9):1419-1429.

35 Rice, S. D., 1973. Toxicity and avoidance tests with Prudhoe Bay oil and pink salmon fry. Proceedings of joint conference on prevention of oil spills, Wash., D. C., pp. 667-670.

36 Steel, D.L. and B.J. Copeland, 1967. Metabolic responses of some estuarine organisms to an industrial effluent control. Mar. Sci. Univ. Texas 12:143-159

37 Strand, J. W., et al., 1971. Development of toxicity test procedure for marine phytoplankton, p. 279-286. In: American Petroleum Institute. Proceedings of a joint conference on prevention and control of oil spills, Washington, D. C.

38 STRAUGHAN, D. 1971. Biological and Oceanographical survey of the Santa Barbara channel oil spill. Vol. 1. Biology and bacteriology. Allan Hancock Foundation, Univ. South, Calif. 426 p.

39 Ted, J.H. 1972. An introduction to environmental ethology Woods Hole Oceanographic Institution. Ref. 72-42. Woods Hole Mass. Unpublished manuscript.

40 Vaughan, B.E. 1973. Effects of oil and chemically dispersed oil on selected marine biota - a laboratory study. Richland, Washington, Battelle Pacific Northwest Laboratories 120 p.

41 Wells, P.G., 1972. Influence of Venezuelan crude oil on lobster larvae. Marine Pollution Bull., 3:105.

42 Wilber, C.G., 1968. Biological aspects of water pollution.

43 Wilson, K.W., 1970. The toxicity of oil-spill dispersants to the embryos and larvae of some marine fish. In: Proceedings, FAO Conference, Rome, Italy.

44 Wohlschlog, D.E. and J.N. Cameron. 1967. Assessment of low level. Stress on the respiratory metabolism of the pinfish (Lagodon rhomboides). Inst. Mar. Sci. Univ. Texas 12:160-171

species, and (2) the possibility of edible marine organisms incorporating the high boiling, carcinogenic polycyclic aromatics in their tissues. Nelson-Smith (1971) reported that 0.01 mg/l of crude oil caused tainting in oysters. Moore, et al. (1973) reported that concentrations as low as 1 to 10 ug/l could lead to tainting within very short periods of time. It has been shown that chemicals responsible for cancer in animals and man (such as 3, 4-benzopyrene) occur in crude oil (Blumer, 1970). It has also been shown that marine organisms are capable of incorporating potentially carcinogenic compounds into their body fat where the compounds remain unchanged (Blumer, 1970).

Oil pollutants may also be incorporated into sediments. There is evidence that once this occurs in the sediments below the aerobic surface layer, petroleum oil can remain unchanged and toxic for long periods, since its rate of bacterial degradation is slow. For example, Blumer (1970) reported that No. 2 fuel oil incorporated into the sediments after the West Falmouth spill persisted for over a year, and even began spreading in the form of oil-laden sediments to more distant areas that had remained unpolluted immediately after the spill. The persistence of unweathered oil within the sediment could have a long-term effect on the structure of the benthic community or cause the demise of specific sensitive important species. Moore, et al. (1973) reported concentrations of 5 mg/l for the carcinogen, 3, 4-benzopyrene in marine sediments.

Mironov (1967) reported that 0.01 mg/l oil produced deformed and inactive flatfish larvae. Mironov (1970) also reported inhibition or delay of cellular division in algae by oil concentrations of 10^{-4} to 10^{-1} mg/l. Jacobsen and Boylan (1973) reported a reduction in the chemotactic perception of food by the snail, Nassarius obsoletus, at kerosene concentrations of 0.001 to 0.004 mg/l. Bellen, et al. (1972) reported decreased survival and fecundity in worms at concentrations of 0.01 to 10 mg/l of detergent.

Because of the great variability in the toxic properties of oil, it is difficult to establish a numerical criterion which would be applicable to all types of oil. Thus, an application factor of 0.01 of the 96-hour LC50 as determined by using continuous flow with a sensitive resident species should be employed for individual petrochemical components.

There is a paucity of toxicological data on the ingestion of the components of refinery wastewaters by humans or by test animals. It is apparent that any tolerable health concentrations for petroleum derived substances far exceed the limits of taste and odor. Since petroleum derivatives become organoleptically objectionable at concentrations far below the human chronic toxicity, it appears that hazards to humans will not arise from drinking oil-polluted waters (Johns Hopkins University, 1956; Mckee and Wolf, 1963). Oils of animal or vegetable origin generally are non-toxic to humans and aquatic life.

In view of the problem of petroleum oil incorporation in sediments, its persistence and chronic toxic potential, and the present lack of sufficient toxicity data to support specific criteria, concentrations of oils in sediments should not approach levels that cause deleterious effects to important species or the bottom community as a whole.

Petroleum and nonpetroleum oils share some similar physical and chemical properties. Because they share common properties, they may cause similar harmful effects in the aquatic environment by forming a sheen, film or discoloration on the surface of the water. Like petroleum oils, nonpetroleum oils may occur at four levels of the aquatic environment: (a) floating on the surface, (b) emulsified in the water column, (c) solubilized, and (d) settled on the bottom as a sludge. Analogous to the grease balls from vegetable oil and animal fats are the tar balls of petroleum origin which have been found in the marine environment or washed ashore on beaches.

Oils of any kind can cause: (a) drowning of waterfowl because of loss of buoyancy, exposure because of loss of insulating capacity of feathers, and starvation and vulnerability to predators due to lack of mobility, (b) lethal effects on fish by coating epithelial surfaces of gills, thus preventing respiration, (c) potential fish kills due to biochemical oxygen demand, (d) asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom, and (e) adverse aesthetic effects of fouled shore-lines and beaches. These and other effects have been documented in the U.S. Department of Health, Education and Welfare report on "Oil Spills Affecting the Minnesota and Mississippi Rivers" and the 1975 "Proceedings of the Joint Conference on Prevention and Control of Oil Spills."

Oils of animal or vegetable origin generally are chemically non-toxic to humans or aquatic life; however, floating sheens of such oils result in deleterious environmental effects described in this criterion.

Thus, it is recommended that surface waters shall be virtually free from floating non-petroleum oils of vegetable or animal origin. This same recommendation applies to floating oils of petroleum origin since they too may produce the above effects.

REFERENCES CITED:

Bellan, et al., 1972. The sublethal effects of a detergent on the reproduction, development, and settlement in the polychaetous annelid Capitella capitata. Marine Biology, 14:183.

Blumer, M., 1970. Oil contamination and the living resources of the sea. F.A.O. Tech. Conf. Rome. FIR:MP/70/R-1,11P.

Bugbee, S. L. and C. M. Walter, 1973. The response of macro-invertebrates to gasoline pollution in a mountain stream. In: Prevention and control of oil spills, proceedings of symposium March 13-17, Washington, D. C., p. 725.

Diaz-Piferrer, 1962. The effects of an oil spill on the shore of Guanica, Puerto Rico (abstract) Ass. Isl. Mar. Labs, 4th Mtg. Curacao, 12-13.

Hampson, G. R. and H. L. Sanders, 1969. Local oil spill. Oceanus, 15:8.

Jacobson, S. M. and D. B. Boylan, 1973. Effect of seawater soluble fraction of kerosene on chemotaxis in a marine snail, Nassarius obsoletus. Nature, 241:213.

Johns Hopkins University, 1956. Final report to the water quality subcommittee of the American Petroleum Institute, Project PG 49.41.

McKee and Wolf, 1963. Water quality criteria. California State Water Resources Control Board, Pub. No. 3-A.

Mironov, O.G., 1967. Effects of low concentrations of petroleum and its products on the development of roe of the Black Sea flatfish. Vop Ikhtiol., 7:557.

Mironov, O. G., 1970. The effect of oil pollution on flora and fauna of the Black Sea. In Proceedings: FAO Conference on Marine Pollution and its effects on living resources and fish. Rome, Dec., 1970, E-92. Food and Agriculture Organization of the United Nations.

Moore, S. F., et al. 1973. A preliminary assessment of the environmental vulnerability of Machias Bay, Maine to oil supertankers.

Nelson-Smith, A., 1971. Effects of oil on marine plants and animals, p. 273-380. In: P. Hepple (ed.), Water Pollution by Oil. I.P. London.

DISSOLVED OXYGEN

CRITERIA:

Aesthetics: Water should contain sufficient dissolved oxygen to maintain aerobic conditions in the water column and, except as affected by natural phenomena, at the sediment-water interface.

Freshwater aquatic life: A minimum concentration of dissolved oxygen to maintain good fish populations is 5.0 mg/liter. The criterion for salmonid spawning beds is a minimum of 5.0 mg/liter in the interstitial water of the gravel.

INTRODUCTION:

Dissolved oxygen historically has been a major constituent of interest in water quality investigations. It generally has been considered as significant in the protection of aesthetic qualities of water as well as for the maintenance of fish and other aquatic life. Traditionally, the design of waste treatment requirements was based on the removal of oxygen demanding materials so as to maintain the dissolved oxygen concentration in receiving waters at prescribed levels. Sophisticated techniques have been developed to predict the dissolved oxygen concentration under various hydrologic, hydrographic, and waste loading conditions (Velz, 1970). Dissolved oxygen concentrations are an important gage of existing water quality and the ability of a water body to support a well balanced aquatic fauna.

RATIONALE:

The aesthetic qualities of water require sufficient dissolved oxygen present to avoid the onset of septic conditions with its attendant malodorous emissions. Insufficient dissolved oxygen in the water column causes the anaerobic decomposition of any organic materials present. Such decomposition tends to cause the formation of noxious gases such as hydrogen sulfide and the development of carbon dioxide and methane in the sediments which bubble to the surface or which tend to float settled sludge as mats which are composed of various organic materials.

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

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

The disadvantage of substantial quantities of dissolved oxygen in water used as a source of municipal water supply is the increased rates of corrosion of metal surfaces in both the water treatment facilities and in the distribution system (NAS, 1974). Such corrosion, in addition to the direct damage, can increase the concentration of iron (and other metals) which may cause taste in the water, as well as staining.

A discussion of oxygen criteria for freshwater fish must take into account these facts: (1) fish vary in their oxygen requirements according to species, age, activity, temperature, and nutritional state; (2) they are found from time to time, and can survive for a while, at oxygen concentrations considerably below that considered suitable for a thriving population; and (3) although there is much literature on the oxygen consumption of fish and the effects of varying oxygen concentrations on behavior and survival, few investigators have employed methods or sought endpoints that can be related with confidence to maintaining a good fish population.

To allow for the differences among requirements affected by species and other variables, the dissolved oxygen criteria are based on the concentration that will support a well-rounded population of fish (Ellis, 1937) as it would occur under natural conditions. A population of fish is composed of a number of different but more or less interdependent species, of different feeding and reproductive habits, but which will include game and pan fish (bass, pike, trout, perch, sunfish, crappie, depending upon the location), some so-called rough or coarse fish (carp, buffalo, bullhead, sucker, chub), and large numbers of smaller 'forage' fish (e.g., minnows). Theoretically it should be possible to base oxygen criteria on the needs of the most sensitive component of such a population, but there is not enough information for this at present; that is why the criteria must be based on oxygen concentrations known to permit the maintenance and well-being of the population as a whole.

The requirement that the data be applicable to naturally occurring populations imposes limits on the types of research that can be used as a basis for the criterion. Aside from a few papers on feeding, growth, and survival in relation to oxygen concentration, very little of the laboratory-based literature has a direct bearing; field data are in general more useful. Field studies have the disadvantage that the numbers of variables encountered in the natural environment (temperature, pH, dissolved solids, food supply, and the like, as well as dissolved oxygen) make it necessary to be conservative in relating fish abundance and distribution to oxygen concentration alone, but enough observations have been made under a variety of conditions that the importance of oxygen concentration seems clear.

Field studies, in which fish catches have been related to dissolved oxygen concentrations measured at the same time, indicate that a dissolved oxygen concentration of 3 mg/liter is too low to maintain a good fish population (Thompson, 1925; Ellis, 1937; Brinley, 1944), and this finding is supported by laboratory observations that in the vicinity of 3 mg/liter and below feeding is diminished or stopped (Lindroth, 1949; Mount, 1960; Herrmann, et al., 1962), and growth is reduced (Hamdorf, 1961; Itazawa, 1971), even when the lowered oxygen concentration occurs for only part of the day (Stewart, et al., 1967).

A dissolved oxygen concentration of 4 mg/liter seems to be about the lowest that will support a varied fish population (Ellis, 1937), even in the winter (Thompson, 1925), and for a well-rounded population including game fish it should be above that. Both Ellis (1937) and Brinley (1944) set the minimum for a well rounded population at 5 mg/liter. It should be pointed out, however, that Thompson found the greatest variety of species at 9 mg/liter, Ellis found good populations more frequently at 6 than at 5 mg/liter, and Brinley reported the best concentrations for game fish populations to be above 5 mg/liter. The belief that 5 mg/liter is adequate is supported by the fact that the introduced rainbow trout thrives in Lake Titicaca (Everett, 1973) where, because of the altitude, the oxygen concentration in fully saturated water is not over 5 mg/liter.

Fish embryonic and larval stages are especially vulnerable to reduced oxygen concentrations because their ability to extract oxygen from the water is not fully developed and they cannot move away from adverse conditions. Although many species can develop at oxygen concentrations as low as 2.5 to 3 mg/liter, the effects of a reduced oxygen concentration even as high as 5 or 6 mg/liter can cause a partial mortality or at the least retard development (Brungs, 1971; Siefert et al., 1973, 1974, 1975; Carlson et al., 1974; Carlson and Siefert, 1974; Garside, 1966; Gulidov, 1969; Hamdorf, 1961). Unless it is extreme, however, the retardation need not be permanent or detrimental to the species (Brannon, 1965; Eddy, 1972). For most fish, maintaining a minimum of 5 mg/liter in the water mass in the vicinity of the embryos and larvae should suffice.

Special treatment is required for species, such as the salmonids, that bury their fertilized eggs in gravel. The flow through gravel is often slow, especially if siltation has occurred, and if it is slow enough the developing fish and other organisms can easily deplete the oxygen supply enough to cause damage, especially if the concentration in the water is relatively low before it enters the gravel (Cooper, 1965; Coble, 1961; Brannon, 1965). With a permeable gravel and abundant flow, 5 mg/liter in the overlying water should be enough. This concentration could well be inadequate, however, with a less porous gravel and a slower flow. Since the permeability and flow have so important a bearing on the initial oxygen concentration required to maintain the intragravel concentration, and since these characteristics vary with location, it is proposed that the criterion for salmonid spawning beds be stated as not less than 5 mg/liter in the gravel. This would require that the concentration in the water entering the gravel be 5 mg/liter or more, increasing as the intragravel flow rate decreased.

Decreased dissolved oxygen levels, if sufficiently severe, can adversely affect aquatic insects and other animals upon which fish feed. Sprague (1963) has evaluated such effects on several crustaceans while others have evaluated caddisfly larvae and stonefly nymphs (Doudoroff and Shumway, 1970). However, many other invertebrates are less sensitive to lowered dissolved oxygen concentrations and may be equally suitable fish food. Doudoroff and Shumway (1970) concluded that as long as dissolved oxygen concentrations remain entirely satisfactory for fish, no material impairment of the food resources for fish ascribable to dissolved oxygen insufficiency will occur.

LITERATURE CITED:

Brannon, E. L. 1965. The influence of physical factors on the development and weight of sockeye salmon embryos and alevins. International Pacific Salmon Fisheries Commission, Progress Report No. 12, pp. 1-26. mimeo.

Brinley, F. J. 1944. Biological Studies. House Document 266, 78th Congress, 1st session; Part II, Supplement F. pp. 1275-1353.

Brungs, W. A. 1971. Chronic effects of low dissolved oxygen concentrations on fathead minnow (Pimephales promelas). J. Fish. Res. Bd. Canada. 28: 1119-1123.

Carlson, A. R., et al., 1974. Effects of lowered dissolved oxygen concentrations on channel catfish (Ictalurus punctatus) embryo and larvae. Trans. Amer. Fish. Soc. 103: 623-626.

Carlson, A. R. and R. E. Siefert. 1974. Effects of reduced oxygen on the embryos and larvae of lake trout (Salvelinus namaycush) and largemouth bass (Micropterus salmoides). J. Fish. Res. Bd. Canada. 31: 1393-1396.

Coble, D. W. 1961. Influence of water exchange and dissolved oxygen in redds on survival of steelhead trout embryos. Trans. Amer. Fish. Soc. 90: 469-474.

Cooper, A. C. 1965. The effect of transported stream sediments on the survival of sockeye and pink salmon eggs and alevin. International Pacific Salmon Fisheries Commission, Bulletin 18: 1-71.

Doudoroff, P. and D. L. Shumway. 1970. Dissolved oxygen requirements of fresh water fishes. FAO Fish. Tech. Paper No. 86.

Eddy, R. M. 1972. The influence of dissolved oxygen concentration and temperature on the survival and growth of chinook salmon embryos and fry. M.S. Thesis, Oregon State Univ., June 1972.

Ellis, M. M. 1937. Detection and measurement of stream pollution. Bull. U.S. Bureau of Sport Fisheries, and Wildlife. 48(22): 365-437.

Environmental Protection Agency. 1973. The control of pollution from hydrographic modifications. EPA 430/9-73-017, U.S. Government Printing Office, Washington, D.C.

Everett, G. V. 1973. Rainbow trout, Salmo gairdneri (Rich.), fishery of Lake Titicaca. J. Fish. Biol. 5: 429-440.

Garside, E. T. 1966. Effect of oxygen in relation to temperature on the development of embryos of brook trout and rainbow trout. J. Fish. Res. Bd. Canada. 23: 1121-1134.

Gulidov, M. V. 1969. Embryonic development of the pike (Esox lucius L.) when incubated under different oxygen conditions. Probs. of Ichthyol. 9: 841-851.

Hamdorf, K. 1961. Die Beeinflussung der Embryonal- und Larvalentqicklung der Regenbogenforelle (Salmo irideus Gibb.) durch die Umwelgfactoren 02-Partialdruck und Temperatur. Z. vergl. Physiol. 44: 451-462.

Herrmann, R. B., et al., 1962. Influence of dissolved oxygen concentrations on the growth of juvenile coho salmon. Trans. Amer. Fish. Soc. 91: 155-167.

Itazawa, Y. 1971. An estimation of the minimum level of dissolved oxygen in water required for normal life of fish. Bull. Jap. Soc. Sci. Fish. 37: 273-276.

Lindroth, A. 1949. Vitality of salmon parr at low oxygen pressure. Inst. Freshwater Res. Drottningholm, Report #29. Fish. Bd. of Sweden (Annual report for 1948): 49-50.

Mount, D. I. 1960. Effects of various dissolved oxygen levels on fish activity. Ohio State Univ. Natur. Resources Inst., Ann. Fisheries Res. Rept. pp. 13-33.

National Academy of Sciences, National Academy of Engineering. 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

Siefert, R. E., et al., 1973. Effects of reduced oxygen concentrations on northern pike (Esox lucius) embryos and larvae. J. Fish. Res. Bd. Canada. 30: 849-852.

Siefert, R. E. and W. A. Spoor. 1974. Effects of reduced oxygen concentrations on embryos and larvae of white sucker, coho salmon, brook trout, and walleye. Proceedings of an International Symposium on the Early Life History of Fish. Oban, Scotland, May 17-23, 1973, Edited by J. H. S. Blaxter, Springer-Verlag Berlin Heidelberg New York. pp. 487-495.

Siefert, R. E., et al., 1975. Effects of reduced oxygen concentrations on the early life stages of mountain whitefish, smallmouth bass, and white bass. Accepted for publication in the Prog. Fish. Cult.

Sprague, J. B. 1963. Resistance of four fresh water crustaceans to lethal high temperature and low oxygen. J. Fish. Res. Bd. Canada. 20: 387.

Stewart, N. E., et al, 1967. Influence of oxygen concentration on the growth of juvenile largemouth bass. J. Fish. Res. Bd. Canada. 24: 475-494.

Thompson, D. H. 1925. Some observations on the oxygen requirements of fishes in the Illinois River. Ill. Nat. Hist. Surv. Bull. 15: 423-437.

Velz, C. J. 1970. Applied stream sanitation. Wiley-Interscience, New York.

ALDRIN-DIELDRIN

CRITERIA:

.003 ug/l for freshwater and marine aquatic life.

The persistence, bioaccumulation potential and carcinogenicity of aldrin-dieldrin cautions human exposure to a minimum.

RATIONALE:

Since dieldrin is a highly persistent chemical which bioaccumulates in aquatic organisms used for human food and is also considered a potential human carcinogen, levels of dieldrin in waterways should be kept as low as feasible. Action by the Environmental Protection Agency in suspending the production and use of dieldrin should result in a gradual decrease in concentrations in the environment. Such additions should not be permitted without substantial documentation that alternatives are either infeasible or potentially more hazardous. The persistence, bioaccumulative properties and carcinogenic potential of dieldrin should be taken into account when determining the uses of water with measurable amounts of dieldrin.

Aldrin is metabolically converted to dieldrin by aquatic organisms. Residues in goldfish, Carassius auratus, exposed to aldrin for 32 days were found to consist of 93.9 percent or more dieldrin except for visceral fat where residues were 100 percent dieldrin after 31.5 to 92.4 days' exposure (Gakstatter, 1968). Epoxidation of aldrin to dieldrin was found to occur also at lower trophic levels. The relative rates of this conversion were low for algae and high for the protozoa (Kahn, et al., 1972). Because of this metabolic conversion and because of evidence that dieldrin is as toxic or slightly more toxic than aldrin to aquatic organisms

(Jensen and Gaufin, 1966; Henderson, et al., 1959), an acceptable water concentration is based on the presence of either aldrin or dieldrin or the sum of both.

In two studies comprising five long-term oral studies feeding dieldrin to CF-1 mice at various concentrations, liver enlargements and tumors were detectable. Appearance of tumors was dose responsive since tumors occurred 9 months following treatment with 10 ppm; 19 months with 5 ppm; and 23 months with 2.5 ppm. Further, the group all experienced a decrease in survival rates. At intake rates of 1.25 ppm and 1 ppm dieldrin, no liver enlargements were detected clinically and survival was not affected (Walker, et al., 1972).

The best evidence that aldrin-dieldrin poses a cancer hazard to man is provided by mouse laboratory data. Although the liver was the principal organ affected in the mouse and was a major site of action in rats, there was an increase of tumors in the lungs and other organs (Keller, et al., 1974).

Ninety-six-hour LC50 values of 16, 7.9, and 8.5 ug/l dieldrin have been reported for the fathead minnow, Pimephales promelas; bluegill, Lepomis macrochirus; and green sunfish, Lepomis cyanellus, respectively (Tarzwell and Henderson, 1956).

The LC50 for the stonefly naiad, Acroneuria pacifica, exposed to dieldrin in a continuous flow bioassay system for 20 days was 0.2 ug/l (Jensen and Gaufin, 1966). The 48-hour EC50 (immobilization value at 15°C) for daphnids, Simocephalus serrulatus and Daphnia pulex, to dieldrin was 0.24 mg/l and 0.25 mg/l, respectively (Sanders and Cope, 1966).

The sailfin molly, Poecilia latipinna, appears to be the most sensitive freshwater fish species tested for chronic effects; growth rates and reproductive performance were adversely affected during a 34-week exposure to dieldrin at 0.75 ug/l (Lane and Livingston, 1970). Guppy, Poecilia reticulata, populations were affected by levels of dieldrin at 1.8 ug/l water concentration during a 14-month exposure. Exposed populations developed greater total numbers of individuals than did controls. This phenomenon may have been caused by slight change in the feeding behavior of the adults induced by dieldrin, consisting of inhibition of the normal predation by the adults upon the fry (Cairns, et al., 1967).

Residue accumulation of dieldrin and aldrin is well documented. Levels of dieldrin in fish tissue from Lake Michigan have been as much as 100,000 times (wet weight basis) the dieldrin levels occurring in the water (Reinert, 1970). Lake water concentrations in Lake Michigan were in the 1 to 3 nanogram/liter range, and whole-fish concentrations ranged from a low of 0.03 ppm for lake whitefish to 0.20 ppm for lake herring, 0.23 ppm for bloater, and 0.28 ppm for kiyi. Laboratory exposures of fish, invertebrates, and algae have indicated that residue accumulation

of aldrin and dieldrin is significant. The Reticulate sculpin, Cottus perplexus, exposed to 8.6, 1.7, 0.86, 0.17, 0.086, and 0.017 ug/l dieldrin in water for 32 days were found to have tissue concentrations (wet weight basis) often exceeding 50,000 times the water exposure level (Chadwick and Brockson, 1969). The sailfin molly, Poecilia latipinna, exposed for 34 weeks at 12, 6, 3, 1.5, and 0.75 ug/l dieldrin in water concentrated dieldrin in all tissues (wet weight basis) at least 10,000 times (Lane and Livingston, 1970). At the termination of a 64-week exposure of the ostracod, Chlamydotheca arcuata, to water concentrations of aldrin at 0.01 and 0.10 ug/l and dieldrin at 0.01 and 0.10 ug/l, dieldrin recovery from the tissue (dry weight basis) was 12,000 to 260,000 times the initial theoretical water concentrations (Kawatski and Schmulbach, 1971). In a model ecosystem study, residue accumulation factors (wet weight basis) for dieldrin were determined to be 114,935 times water concentration for the snail, 7,480 times water concentration for algae, 6,145 times water concentration for fish, 2,145 times water concentration for Daphnia, 1,280 times water concentration for Elodea, 247 times water concentration for the crab, and 1,015 times water concentration for the clam (Sanborn and Yu, 1973). In continuous flow exposure to less than 0.1 ug/l aldrin in water for a three-day period, residue accumulation factors (dry weight basis) were determined for cladocera, Daphnia magna, to be

141,000 times water concentration; ephemeroptera, Hexagenia billineata, 31,400 times water concentration; and diptera, Chironomus sp., 22,800 times water concentration (Johnson, et al., 1971); for the alga, Scenedesmus obliquus, 1,282 times water concentration after 1.5 days; 13,954 times water concentration for Daphnia magna after 3 to 4 days, and an estimated 49,307 times water concentration for the guppy, Poecilia reticulata, after 18 days' exposure (Reinert, 1972).

In relating accumulation factors to the acceptable level of aldrin and dieldrin allowable in water, it is necessary to know the significance of tissue residue levels. Data on the toxicity of ingested levels of aldrin and dieldrin in aquatic organisms are few. In rainbow trout, Salmo gairdneri, fed dietary dieldrin dosages of 0.36, 1.08, 3.6, and 10.8 ug dieldrin per gram of food (ppm), brain concentrations of three amino acids associated with ammonia detoxifying mechanisms - glutamate, aspartate, and alanine - were significantly altered. In the two highest dosages the brain ammonia concentration increased (Mehrle and Bloomfield, 1974). The implication is that brain ammonia detoxifying mechanisms play an important role in maintaining ammonia values within physiological limits, and that fish carrying body burdens of dieldrin would be less tolerant to increased concentrations of ammonia in water.

Some data available on terrestrial vertebrates indicate that aldrin-dieldrin dietary levels as low as 1 ppm may produce observable effects. In long-term feeding studies 1 ppm dieldrin affected reproduction in the

Hungarian partridge (Neill, et al., 1969). Slight eggshell thinning was noted in mallard ducks fed 3 ppm dieldrin (Lehner and Egbert, 1969). Deer were affected by long-term feeding at 2 ppm dieldrin (Murphy and Korschgen, 1970). A guideline of 0.3 ppm has been set by the Food and Drug Administration as an upper limit on food for human consumption.

Considering the 100,000-fold bioaccumulation of dieldrin in fish tissue from Lake Michigan cited above (Reinert, 1970) and the FDA tissue residue administrative guideline of 0.3 ppm as a maximum level for human consumption, the resulting maximum water level is 0.003 ug/l. This level is lower than the lowest measured 96-hour LC_{50} (7.9 ug/l for bluegill) by a factor of 0.0004, and lower than the 20 day LC_{50} for the stonefly, Acroneuria pacifica, by a factor of 0.02. It is therefore believed to provide an adequate level of protection for freshwater aquatic life.

In bioassays performed on the mullet, Mugilidae sp., death occurred for 15 percent of the test organisms at 0.5 ug/l dieldrin. When exposed to dieldrin at 1.35 ug/l for four days, fish were found to have degenerative changes in the gills and visceral tissue (Parrish, et al., 1973). A sensitive marine crab, Leptodius floridanus, exhibited delay in development at concentrations of 1 and 0.5 ug/l dieldrin (Epifanio, 1971). At 1 ug/l dieldrin there was a 70 percent mortality of pink shrimp, Penaeus duorarum, within 24 hours and the 96-hour LC_{50} was 0.7 ug/l for the same organism (Parrish, et al., 1973).

A residue accumulation factor of 800 times water concentration (wet weight basis) was found for the estuarine mollusc, Rangia cuneata, for a

36-hour exposure with a maximum accumulation factor of 2,000 times water concentration in a 72-hour exposure to dieldrin. The clam showed "no evidence for a cessation of the uptake mechanism over a period of time up to 72 hours" (Petrocelli, et al., 1973). At a water concentration of 0.5 ug/l it was determined that the rate of dieldrin uptake from water by crab larvae, Leptodius floridanus, was 0.191 ppm per day from water (Epifanio, 1973). Bioconcentration factors in estuarine organisms exposed for 96-hours to dieldrin ranged from 2,400 to 21,500 for oysters; 280 to 420 for pink shrimp; 470 to 750 for grass shrimp; and 3,500 to 7,300 for sheepshead minnows (Parrish, et al., 1973). Concentration factors in spot exposed to dieldrin for 35 days were as great as 113,000 in liver, 11,000 in muscle and 6,000 in whole fish. Spot lost all detectable dieldrin after 13 days in dieldrin-free sea water (Parrish, et al., 1973). Marine phytoplankton exposed for two hours have been shown to accumulate dieldrin (Rice and Sikka, 1973). In a study designed to investigate the accumulation and metabolism of dieldrin by species representing different taxonomic divisions of marine phytoplankton, concentration factors observed were: Skeletonema costatum (Bacillariophyta), 15,882; Cyclotella nana (Bacillariophyta), 4,810; Tetraselmis chuii (Euglenophyta), 8,588; Isochrysis galbana (Chrysophyta), 8,238; Olisthodiscus luteus (Xanthophyta), 4,900; and Amphidinium carteri (Pyrrophyta), 982.

No data on bioaccumulation from the environment are available for marine fish. In the absence of such data, there is no reason to assume that the accumulation factor for marine fish would be less than that observed for Lake Michigan fish. Therefore, the freshwater criterion of 0.003 ug/l is also recommended for marine aquatic life.

REFERENCES CITED:

Cairns, J., et al., 1967. Effects of sublethal concentrations of dieldrin on laboratory populations of guppies, Poecilia reticulata Peters. Proc. Acad. Nat. Sci., Philadelphia, Pennsylvania, 119:75.

Chadwick, G.G. and R.W. Brockson, 1969. Accumulations of dieldrin by by fish and selected fish-food organisms. Jour. Wildlife Management, 33:693.

Epifanio, C.E., 1971. Effects of dieldrin in seawater on the development of two species of crab larvae, Leptodius floridanus and Panopeus herbsti. Marine Biology, 11:366.

Epifanio, C.E., 1973. Dieldrin uptake by larvae of the crab Leptodius floridanus. Marine Biology, 19:320.

Gakstatter, J.H., 1968. Rates of accumulation of ^{14}C - dieldrin residues in tissues of goldfish exposed to a single sublethal dose of ^{14}C - aldrin. Jour. Fish. Res. Bd. Canada, 25:1797.

Henderson, C., et al., 1959. Relative toxicity of ten chlorinated hydrocarbon insecticides to four species of fish. Trans. Amer. Fish. Soc., 88:23.

Jensen, L.D. and A.R. Gaufin, 1966. Acute and long-term effects of organic insecticides on two species of stonefly naiads. Jour. Water Poll. Control Fed., 38:1273.

Johnson, B.T., et al. 1971 Biological magnification and degradation of DDT and aldrin by freshwater invertebrates. Jour. Fish. Res. Bd. Can. 28:705.

Kawatski, J.J. and J.C. Schmulbach, 1971. Accumulation of insecticide in freshwater ostracods exposed continuously to sublethal concentrations of aldrin or dieldrin. Trans. Amer. Fish. Soc., 100:565.

Keller, A.M., et al., 1974. Respondents brief, proposed findings and conclusions on suspension of aldrin-dieldrin. FIFRA Docket No. 145.

Kahn, M.A.Q., et al., 1972. In vivo and in vitro epoxidation of aldrin by aquatic food chain organisms. Bull. Environ. Contam. and Toxicol., 8:219.

Lane, C.E. and R.J. Livingston, 1970. Some acute and chronic effects of dieldrin on the sailfin molly, Poecilia latipinna. Trans. Amer. Fish. Soc., 3:489.

Lehner, P.N. and A. Egbert, 1969. Dieldrin and eggshell thickness in ducks. Nature, 224:1218.

Mehrle, P.M. and R.A. Bloomfield, 1974. Ammonia detoxifying mechanisms of rainbow trout altered by dietary dieldrin. Toxicology and Applied Pharmacology, 27:335.

Murphy, D.A. and L.J. Korschgen, 1970. Reproduction, growth, and tissue residues of deer fed dieldrin. Jour. Wildlife Management, 34:887.

Neill, D.D., et al., 1969. 58th Annual Meeting of the Poultry Science Association. 76.

Parrish, P.R., et al., 1973. Dieldrin effects on several estuarine organisms. Proc. Assoc. S.E. Game Fish. Comm., pp. 427-434, December, 1972.

Petrocelli, S.R., et al., 1973. Uptake and accumulation of an organochlorine insecticide (dieldrin) by an estuarine mollusk Rangia cuneata. Bull. Environ. Contam. and Toxicol., 10:315.

Reinert, R.E., 1970. Pesticide concentrations in Great Lakes fish. Pesticides Monitoring Jour., 3:233.

Reinert, R.E., 1972. Accumulation of dieldrin in an alga (Scenedesmus obliquus), Daphnia magna, and the guppy (Poecilia reticulata). Jour. Fish. Res. Bd. Canada, 29:1413.

Rice, C.P. and H.C. Sikka, 1973. Fate of dieldrin in selected species of marine algae. Bull. Environ. Contam. and Toxicol., 9:116.

Sanders, H.O. and O.B. Cope. 1966. Toxicities of several pesticides to two species of Cladocerans. Trans. Amer. Fish Soc. 95:165.

Sanborn, J.R. and C. Yu, 1973. The fate of dieldrin in a model ecosystem. Bull. Environ. Contam. and Toxicol., 10:340.

Tarzwell, C.M. and C. Henderson, 1956. Toxicity of dieldrin to fish. Trans. Amer. Fish. Soc., 86:245.

Walker, A.I.T., et al., 1972. The toxicity of dieldrin (HEOD). I. Long-term toxicity studies in mice. Food Cosmet. Toxicol., 11:415.

CHLORDANE

CRITERIA:

0.01 ug/l for freshwater aquatic life.
0.004 ug/l for marine aquatic life.

The persistence, bioaccumulation potential and carcinogenicity of chlordane cautions human exposure to a minimum.

RATIONALE:

Since chlordane is a highly persistent chemical which bioaccumulates in aquatic organisms used for human food and also is considered a potential human carcinogen (Train, 1974), levels of chlordane in waterways should be kept as low as feasible. The December 24, 1975 action by the Environmental Protection Agency in suspending the production and use of chlordane should result in a gradual decrease in concentrations in the environment. Such additions should not be permitted without substantial documentation that alternatives are either infeasible or potentially more hazardous. The persistence, bioaccumulative properties and carcinogenic potential of chlordane should be taken into account when determining the uses of water with measurable amounts of chlordane.

Literature references indicate the existence of an extremely wide range for the acute toxicity of chlordane to various species of freshwater fishes. As recorded, these values for 24- to 96-hour exposures range from 5 to 3,000 ug/l (Cardwell, et al., 1975; Katz, 1961; Lawrence, 1950; National Technical Advisory Committee, 1968; Clemens and Sneed, 1959; Macek, et al., 1969). Physical conditions varied among the tests reported and, in addition, most were conducted under

static conditions and actual water concentrations were not measured. Although individual acute toxicity values may be questionable, some tests using several species under uniform conditions indicate that variability of experimental techniques alone cannot account for the wide range of values published. A recent study by Cardwell, et al., (1975) utilizing flow-through systems and measured concentrations of chlordane yielded 96-hour LC50 values of 37, 47, and 59 ug/l for fathead minnows, brook trout, and bluegills, respectively. These values fall within the range of the majority of acute toxicity values reported in the literature. The most sensitive fish reported was the pike, which suffered distress after 24 hours at 5 ug/l and 100 percent mortality within this period at 50 ug/l (Ludemann and Neumann, 1962).

Published acute toxicity values for U.S. freshwater invertebrates are similar to those for fishes and range from 4 to 10,000 ug/l (Cardwell, et al., 1975; Naqvi and Ferguson, 1969; National Technical Advisory Committee, 1968; Sanders 1969, 1972). Daphnia magna and Hyallela azteca tested under flow-through conditions yielded 96-hour LC50 values of 28 and 97 ug/l (Cardwell, et al., 1975), falling within the range of the majority of values reported for acute toxicity to invertebrates. The most sensitive species reported was the glass shrimp, Palaemonetes kadiakensis, with 96-hour LC50 values of 10 and 4 ug/l, respectively, in static versus flow-through bioassays (Sanders, 1972). Ludemann and Neumann (1962) found that Chironomus larvae had a 24-hour static test LC50 value of 10 ug/l.

Cardwell, et al. (1975), conducted long-term flow-through studies which included reproduction and survival of progeny with several aquatic species. Statistically significant detrimental effects could not be measured at concentrations of 0.5 ug/l (bluegills), 0.7 ug/l (Chironomus No. 51, a midge), 5 ug/l (Hyallela azteca), and 12 ug/l (Daphnia magna). Survival of brook trout larvae to 12 days was reduced by 35 percent at 0.3 ug/l (the lowest concentration tested) but overall effects were decreasing at this level.

Available acute toxicity values indicate that individual species of both fishes and invertebrates vary greatly in sensitivity to chlordane but both groups are within the same general range. Most of these acute toxicity values are also nearer to the lower limits of this sensitivity range. Data for the application factors derived using acute and sub-chronic toxicity concentrations are limited to the study by Cardwell, et al. (1975). These factors were 0.009 and less than 0.008 for the fishes and 0.381 and 0.055 for the invertebrates.

Nominal pesticide concentrations in the water were reported for these tests. The true concentrations of chlordane may have been somewhat lower as Cardwell, et al. (1975), found that concentrations could not be maintained above half of nominal even when the test water was continually replaced. Therefore, the water quality criterion should be set lower than the calculated value using the experimentally determined application factor (0.009 X 5.0 ug/l = 0.045 ug/l). For this reason the calculated value for chlordane is lowered to a recommended concentration in freshwater that should not exceed 0.01 ug/l. If the proposed freshwater water quality criterion of 0.01 is not exceeded it is estimated that levels reached in freshwater

fishes should seldom be greater than 1.0 mg/kg. Based on present knowledge, it is not anticipated that a concentration of this magnitude would be detrimental to piscivorous birds.

Michael, et al. (1956), found that the time required to kill one-half of the brine shrimp, Artemia salina, nauplii exposed to 10 ug/l was 2-3 hours. Butler, et al. (1960), determined that 24 hours' exposure of the oyster, Crassostrea virginica, to 10 ug/l chlordane produced growth inhibition.

Korn and Earnest(1974) found the 96-hour LC50 of chlordane to be 11.8 ug/l for the striped bass, Morone saxatilis. Butler (1963) reported the following 48-hour LC50 values: brown shrimp, Penaeus aztecus -- 4.4 ug/l; juvenile blue crabs, Callinectes sapidus -- 480 ug/l; and juvenile white mullet, Mugil curema -- 5.5 ug/l. Parrish, et al. (In Press), reported the following 96-hour LC50 values: pink shrimp, Penaeus duorarum -- 0.4 ug/l; grass shrimp, Palaemonetes pugio -- 4.8 ug/l; sheepshead minnow, Cyprinodon variegatus -- 24.5 ug/l; and the pinfish, Lagodon rhomboides -- 6.4 ug/l. The EC50 for shell deposition by the eastern oyster, Crassostrea virginica, was found to be 6.2 ug/l. The most sensitive marine species tested was the pink shrimp, Penaeus duorarum, for which a 96-hour LC50 of 0.4 ug/l was reported.

It appears that reported concentrations of "total chlordane" in fishes have been calculated from measurements of one or two of its major components under the assumption that the ratio of components was similar in both the parent compound and the tissue residues. More

sophisticated analytical techniques currently available indicate that this assumption is in error and, in addition, the components in tissue residues may have been incorrectly quantified. Fishes can concentrate chlordane directly from water by a factor of 1000 to 3000 times and invertebrates may concentrate to twice this magnitude (Cardwell, et al., 1975). Data on the bioaccumulation of chlordane by estuarine organisms have been reported by Parrish, et al. (In Press). In the 96-hour test, chlordane concentration factors were 3,200 to 8,300 in oysters, Crassostrea virginica ; 4,000 to 6,000 in pink shrimp, Penaeus duorarum ; 1,900 to 2,300 in grass shrimp, Palaemonetes pugio ; 12,600 to 18,700 in pinfish, Lagodon rhomboides . Schimmel, et al., (In Press), exposed estuarine fishes to trans-chlordane for 96 hours and reported concentration factors ranging from 3,700 to 6,300 in edible portions. Reported concentrations of 10 to 100 ug/kg were common in fish samples obtained throughout the U.S. and a few residues above 1000 ug/kg were observed (Henderson, et al., 1969 and 1971). Corresponding water concentrations are not available, but measurements above nanogram/liter levels have seldom been found in natural waters except near the discharges of manufacturing and formulating operations (Barthel, et al., 1969; Casper, 1967; Godsil and Johnson, 1968). Therefore, allowing for errors in residue measurements and lack of direct correlation, the possibility exists that fishes can concentrate chlordane up to 100,000 times the ambient water concentration. High rates of accumulation could occur in higher trophic level organisms through multiple steps in the food chain although evidence directly related to chlordane is not available to support this hypothesis.

Based on the persistence, bioaccumulation potential and carcinogenicity of chlordane, an application factor of .01 is applied to the most sensitive marine aquatic species, the pink shrimp. This results in a marine criterion of .004 ug/l.

REFERENCES CITED:

Barthel, W.F., et al., 1969. Pesticide residues in sediments of the lower Mississippi River and its tributaries. Pest. Mon. Jour. 3:8.

Butler, P.A., et al., 1960. Effect of pesticides on oysters. Proceedings of the National Shell Fisheries Association. 51:23.

Butler, P.A., 1963. Commercial Fisheries Investigations. In: Pesticide Wildlife Studies. U. S. Dept. of the Interior. Washington, D. C. Circular 167.

Cardwell, R. D., et al., 1975. Acute and chronic toxicity of chlordane to fish and invertebrates. EPA Ecological Research Series. In preparation.

Casper, V. L., 1967. Galveston Bay pesticide study - water and oyster samples analyzed for pesticide residues following mosquito control program. Pest. Mon. Jour. 1:13.

Clemens, H. P. and K. E. Sneed, 1959. Lethal doses of several commercial chemicals for fingerling channel catfish. U. S. Fish. Wildl. Serv. Spec. Sct. Rep. Fish. No. 316.

Godsil, R. S. and W. C. Johnson, 1968. Residues in fish, wildlife, and estuaries--pesticide monitoring of the aquatic biota at the Tule Lake National Wildlife Refuge. Pest. Mont. Jour. 1:21.

Henderson, D., et al., 1969. Organochlorine insecticide residues in fish. Pest. Mon. Jour. 3:145.

Henderson, C., et al., 1971. Organochlorine insecticide residues in fish - fall, 1969, National Pesticide Monitoring Program. Pest. Mont. Jour. 5:1.

Katz, M., 1961. Acute toxicity of some organic insecticides to three species of salmonids and to the threespine stickleback. Trans. Amer. Fish. Soc. 90:264.

Korn, S. and R. Earnest, 1974. Acute toxicity of twenty insecticides to striped bass, Morone saxatilis. Calif. Fish and Game, 60:128.

Lawrence, J. M., 1950. Toxicity of some new insecticides to several species of pondfish. Prog. Fish. Cult. 12:141.

Ludemann, D., and H. Neumann, 1962. Uber die wirkung der neuzeitlichen kontakinsektizide auf die tiere des susswassers. Anzeiger fur Schadlingskunde und Pflanzenschutz, 35:5.

Macek, K. B., et al., 1969. Effects of temperature on the susceptibility of bluegills and rainbow trout to selected pesticides. Bull. Environ. Contam. Toxicol., 4:174.

Michael, A.S., et al., 1956. Artemia salina as a test organism for bioassay (of insecticides). Science 123:464.

Naqvi, S. M. and D. E. Ferguson. 1969. Pesticide tolerances of selected freshwater invertebrates. Jour. Miss. Acad. Sci. 14:121.

National Technical Advisory Committee, 1968. Water Quality Criteria. Fed. Water Poll. Control Admin. U.S. G.P.O. Washington, D.C.

Parrish, P. R., et al. (In Press), Chlordane: Effects on several estuarine organisms. Jour. Toxicol. Environ. Health.

Sanders, H. O., 1969. Toxicity of pesticides to the crustacean, Gammarus lacustris. Bur. Sport Fish. and Wildl. Tech. Paper No. 25.

Sanders, H. O., 1972. Toxicity of some pesticides to four species of malocostracan crustacea. Bur. Sport Fish. and Wildl. Tech. Paper No. 66.

Schimmel, S. C., et al., (In Press). Heptachlor: Toxicity to and uptake by several estuarine animals. Jour. Toxicol. Environ. Health.

Train, R. E., 1974. Pesticide products containing heptachlor or chlordane: Intent to cancel registrations, Federal Register, Vol. 39, No. 229, Tues, Nov. 26, 1974. P.41299.

Chlorophenoxy Herbicides
2, 4-D; 2, 4, 5-TP

CRITERIA:

2, 4-D	100 ug/l	for domestic water supply (health)
2,4,5-TP	10 ug/l	for domestic water supply (health)

RATIONALE:

Two widely used herbicides are 2, 4-D (2, 4-dichlorophenoxyacetic acid) and 2, 4, 5-TP (silvex) [2-(2, 4, 5-trichlorophenoxy) propionic acid]. Each of these compounds is formulated in a variety of salts and esters that may have a marked difference in herbicidal properties, but all are hydrolyzed rapidly to the corresponding acid in the body.

The subacute oral toxicity of chlorophenoxy herbicides has been investigated in a number of species of experimental animals (Palmer and Radeleff, 1964; Lehman, 1965). The dog was found to be sensitive and often displayed mild injury in response to doses of 10 mg/kg/day for 90 days, and serious effects from a dose of 20 mg/kg/day for 90 days. Lehman (1965) reported that the no-effect level of 2, 4-D is 0.5 mg/kg/day in the rat, and 8.0 mg/kg/day in the dog.

Data are available on the toxicity of 2, 4-D to man. A daily dosage of 500 mg (about 7 mg/kg) produced no apparent ill effects in a volunteer over a 21-day period (Kraus, 1946). When 2, 4-D was investigated as a possible treatment for disseminated coccidioidomycosis, the patient had no side effects from 18 intravenous doses during 33 days; each of the last 12 doses in the series was 800 mg (about 15 mg/kg) or more, the last being 2000 mg (about 37 mg/kg) (Seabury, 1963). A nineteeth and final dose of 3600 mg (67 mg/kg) produced mild symptoms.

Table 8 illustrates the derivation of the criteria for the two chlorophenoxy herbicides most widely used. The long-term no-effects levels (mg/kg/day) are listed for the rat and the dog. These values are adjusted by a factor of 1/500 for 2, 4-D and 2, 4, 5-TP. The safe levels are then readjusted to reflect total allowable intake per person. Since little 2, 4-D or 2, 4, 5-TP is expected to occur in foods, 20 percent of the safe exposure level can reasonably be allocated to water without jeopardizing the health of the consumer.

TABLE 8. DERIVATION OF APPROVAL LIMITS (AL) FOR CHLOROPHENOXY HERBICIDES

Compound	Lowest Long-Term Levels with Minimal or No Effects		Calculated Maximum Safe Levels From all Sources of Exposure			Water	
	Species	mg/kg/day[a]	Safety Factor (X)	mg/kg/day	mg/man/day[b]	% of Safe Level	AL mg/l[c]
2,4-D	Rat	0.5**	1/500	0.1	7.0	20	0.1
	Dog	8.0 **	1/500	0.016	1.12[d]		
2,4,5-TP	Rat	2.6 *	1/500	0.005	0.35	20	0.01
	Dog	0.9 *	1/500	0.002	0.14[d]		

252

[a] Assume weight of rat = 0.3 kg and of dog = 10 kg; assume average daily food consumption of rat = 0.05 kg and of dog = 0.2 kg.

[b] Assume average weight of human adult = 70 kg.

[c] Assume average daily intake of water for man = 2 liters.

[d] Chosen as basis on which to derive AL.

* Kraus, as cited by Mitchess, J.W., R.E. Hogson, and C.R. Gaetjens, 1946.

** Lehman, A.J., 1965.

REFERENCES CITED:

Kraus, as cited by Mitchess, J.W., R.E. Hogson, and C.R. Gaetjens, 1946. Tolerance of farm animals to feed containing 2, 4-dichlorophenoxyacetic acid. Jour. Animal. Sci., 5: 226.

Lehman, A.J., 1965. Summaries of pesticide toxicity. Association of Food and Drug Officials of the U.S., Topeka, Kansas, pp. 1-40.

Palmer, J.S. and R.D. Radeleff, 1964. The toxicologic effects of certain fungicides and herbicides on sheep and cattle. Ann. N.Y. Acad. Sci., 111: 729.

Seabury, J.H., 1963. Toxicity of 2, 4-dichlorophenoxyacetic acid for man and dog. Arch. Envir. Health, 7: 202.

DDT

CRITERION:

0.001 ug/l for freshwater and marine aquatic life.

The persistence, bioaccumulation potential and carcinogenicity of DDT cautions human exposure to a minimum.

RATIONALE:

In general, DDT (1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane) refers to DDT and its metabolites. Acute toxicity to mammals generally is low; however, aquatic organisms exhibit sensitivity to this pesticide at levels in micrograms per liter. Such levels range from a 96-hour LC_{50} of 0.24 ug/l for the crayfish, Orconectes nais (Sanders, 1972), to a 96-hour LC_{50} of 2 ug/l for the largemouth bass, Micropterus salmoides (Macek and McAllister, 1970), to a 96-hour LC_{50} of 27 ug/l for the goldfish, Carassius auratus (Henderson, et al., 1959).

Since DDT is a highly persistent chemical which bioaccumulates in aquatic organisms used for human food and also is considered a potential human carcinogen (Train, 1975), levels of DDT in waterways should be kept as low as feasible. Action by the Environmental Protection Agency in suspending the production and use of DDT should result in a gradual decrease in concentrations in the environment. Such additions should not be permitted without substantial documentation that alternatives are either infeasible or potentially more hazardous. The persistence, bioaccumulative properties and carcinogenic potential of

DDT should be taken into account when determining the uses of water containing measurable amounts of DDT.

Two- to 4-day LC50's for freshwater aquatic invertebrates and fish exposed to DDT in water generally have ranged in the low microgram per liter levels with the invertebrates being somewhat more sensitive (Sanders, 1969; Sanders, 1972; Sanders and Cope, 1966 and 1968 Henderson, et al, 1959; Macek and McAllister, 1970). The most sensitive freshwater organism for which there are data is the crayfish, Orconectes nais, which had a 96-hour LC50 of 0.24 ug/l (Sanders, 1972). Of marine organisms the most sensitive are the shrimp, Penaeus duorarum and P. setiferus, for which no survival was observed at 0.12 ug/l after 28 days' exposure and 30 percent mortality observed at 0.05 ug/l after 56 days (Nimmo, et al., 1970)

DDT will accumulate in the food chain. Field data where pesticide levels in both the water and aquatic organisms have been measured depict the quantity attributable to this bioaccumulation. A residue accumulation of up to two million times was calculated for fish from the field data of Reinert (1970). The measured DDT water concentration in his study of Lake Michigan was in the nanogram per liter range. This accumulation is 20 times greater than that observed in the National Water Quality Laboratory, Duluth, Minnesota, where residue accumulation of 100,000 times was observed. If one uses a two million times residue accumulation factor and a DDT water concentration of 0.002 ug/l an expected DDT body burden of 4 mg/kg (ppm) in fish would result.

Other studies show that measurable quantities of DDT can be found in natural waters of North America. DDT averaged 0.02 ug/l in the San Diego River, California, in the spring-fall dry weather flow of 1973 (Young and Beeson, 1974); DDT in ocean water along the west coast has ranged from 0.0023 to 0.0056 ug/l (Cox, 1971). Rivers flowing

into estuaries (Brazos and Colorado in Texas) each have a two-year average of 0.03 ug/l (Manigold and Schulze, 1969)..

In laboratory studies, Hansen and Wilson (1970) found that the bio-accumulation factor from water to pinfish, Lagodon rhomboides, was 10,000 times and to Atlantic croaker, Micropogon undulatus, 38,000 times, or an average of about 25,000 times for these fishes. The discrepancy between laboratory and field data may be due to the many additional trophic levels involved in field exposures.

In controlled studies, Heath, et al. (1969) determined that DDE, in concentrations of 10 and 40 ppm DDE in dry feed, impaired reproductive success of penned mallards, Anas platyrhynchos. Eggshells of birds exposed to these concentrations were 13 percent thinner, with 25 percent of the eggs showing cracking after one month. DDT induced thinning of shells at a concentration of 25 ppm with an 18 percent cracking of the shells. Since DDT metabolizes to DDE, eggshell thinning may be partly due to DDE. The association of DDE residues with eggshell thinning was shown for the brown pelican, Pelecanus occidentalis (Blus, et al., 1972). The level of DDE in the eggs which did not produce a significant effect, i.e., thinning, was estimated to be 0.5 mg/kg. However, from the data presented, 2.0 mg/kg represents a conservative estimate of the no-effect level in the eggs.

Feeding studies have shown that black ducks fed DDT in food produced eggs containing residues of about ten-fold the DDT in the diet. Furthermore, a continuous diet of 3.0 mg/kg (wet weight) in natural food

adversely affected reproduction (Longcore, et al., 1971). Extrapolating the egg concentration data in ducks to pelicans, an estimate can be made that the diet of pelicans should contain no more than 0.1 the estimated (2.0 mg/kg) no-effect level of DDT in eggs, or about 0.2 mg/kg.

Based on the considerations of bioaccumulation potential in fish, likelihood of conversion to DDE, and dosage levels known to adversely affect birds, it is recommended that DDT concentrations in water should not exceed 0.001 ug/l.

REFERENCES CITED:

Blus, L.J., et al., 1972. Further analysis of the logarithmic relationship of DDE residues to nest success. Nature, 240:164.

Cox, J.L., 1971. DDT residues in sea water and particulate matter in the California current system. Fishery Bulletin, 69:443.

Hansen, D.J. and A.J. Wilson, Jr., 1970. Residues in fish, wildlife and estuaries. Pesticides Monit. Jour., 4:51.

Heath, R.G., et al., 1969. Marked DDE impairment of mallard reproduction in controlled studies. Nature, 224:47.

Henderson, C., et al., 1959. Toxicity of organic phosphorus and chlorinated hydrocarbon insecticides to fish. Biol. Probl. Water Poll. Trans., Seminar, Robert A. Taft San. Eng. Center, Tech. Report W60-3:76.

Longcore, J.R., et al., 1971. DDE thins eggshells and lowers reproductive success of captive black ducks. Bull. Environ. Contam. Toxicol., 6:485.

Macek, K.J. and W.A. McAllister, 1970. Insecticide susceptibility of some common fish family representatives. Trans. Amer. Fish. Soc., 99:20.

Manigold, D.E. and J.A. Schulze, 1969. Pesticides in water. Pesticides Monit. Jour., 3:124.

Nimmo, D.R., et al., 1970. Localization of DDT in the body organs of pink and white shrimp. Bull. Environ. Contam. Toxicol., 5:333.

Reinert, R.E., 1970. Pesticide concentrations in Great Lakes fish. Pesticides Monit. Jour., 3:233.

Sanders, H.O., 1969. Toxicity of pesticides to the crustacean Gammarus lacustris. U.S. Bureau of Sport Fisheries and Wildlife, Tech. Paper No. 25.

Sanders, H.O., 1972. The toxicities of some insecticides to four species of malacostracan crustacea. U.S. Bureau of Sport Fisheries and Wildlife, Tech. Paper No. 66.

Sanders, H.O. and O.B. Cope, 1966. The toxicities of several pesticides to two species of cladocerans. Trans. Amer. Fish. Soc., 85:165.

Sanders, H.O. and O.B. Cope, 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. Oceanog., 13:112.

Train, R., 1975. Order and determination of the administration that reconsideration of the Agency's prior order of cancellation of DDT for use on cotton is not warranted, Federal Register, 40:15934.

Young, D.R. and T.C. Beeson, 1974. Inputs and distributions of chlorinated hydrocarbons in three Southern California harbors. Southern California Coastal Water Res. Project, Tech. Paper TM-214.

DEMETON

CRITERION:

0.1 ug/l for freshwater and marine aquatic life

RATIONALE:

Static LC_{50} bioassays yielded toxicity values for the organophosphorus pesticide, demeton, for carp, goldfish, fathead minnow, channel catfish, guppy, rainbow trout, and bluegill, ranging from 70 ug/l to 15,000 ug/l (Henderson and Pickering, 1958; Ludemann and Neumann, 1962; Macek and McAllister, 1970; McCann and Jasper, 1972; Pickering, et al., 1962). Results of these tests demonstrate an apparent sharp division in species sensitivity, with bluegill, Lepomis macrochirus; rainbow trout, Salmo gairdneri; and guppy, Poecilia reticulata, being susceptible to lower concentrations while the remaining species were comparatively resistant. In the 96-hour exposures toxicity did not increase significantly with time, indicating that concentrations close to nominal may not have been maintained for more than a few hours. Bluegills with a 24-hour LC_{50} of 70 ug/l were the most sensitive fish (McCann and Jasper, 1972).

When fish were exposed to acutely toxic levels of demeton for 12 hours by Weiss (1959 and 1961) the maximum inhibition of brain acetylcholinesterase (AChE) was not reached. The lowest levels of AChE occurred after 24 to 48 hours. It was demonstrated that maximum inhibition could last as long as two weeks after exposure, and subsequent recovery to

levels approaching normal took many more weeks. Weiss (1958) reported a significant increase in mortality of fathead minnows exposed for a second time to the organophosphate, Sarin, before the fish had recovered normal brain AChE levels. The resistance of fully recovered fish was equal to that of previously unexposed controls. Weiss and Gakstatter (1964a) reported no significant inhibition of brain AChE in bluegills, goldfish, and shiners, Notemigonus crysoleucas, following 15-day exposures to demeton at continuously replenished, nominal concentrations of 1 ug/l.

Acute toxicity values reported for invertebrates range from 10 to 100,000 ug/l (Ludemann and Neumann, 1962; Sanders, 1972). In general, molluscs and tubifex worms were very resistant while the smaller crustaceans and insect larvae were susceptible. Ludemann and Neumann (1962) reported that Chironomus plumosus larvae were the most sensitive species they tested. A 24-hour exposure at 10 ug/l produced undefined effects while 100 percent were killed at 1000 ug/l. Calculated LC_{50} data for invertebrates apparently are limited to a single, nominal concentration static exposure of Gammarus fasciatus (Sanders, 1972). These 24- and 96-hour LC_{50} values are reported as 500 and 27 ug/l, indicating a time-related effect not observed in the bioassays with fishes. As only a few of the sensitive species have been tested and great variance in response can result with different test methods, caution must be exercised in estimating the sub-acute concentration for aquatic fauna in general. It appears that no study has been made of possible residual effects, other than AChE inhibition, which might result from short exposures to sub-acute concentrations of organophosphates.

There are few data on the toxicity of demeton to marine organisms. Butler (1964) reported a 48-hour EC_{50} of 63 ug/l for the pink shrimp, Penaeus duorarum, and a 24-hour LC_{50} of 550 ug/l for the spot, Leiostomus xanthurus.

Chronic demeton toxicity data for freshwater organisms are not currently available. Since there are no data available at this time to indicate long-term no-effect levels for aquatic organisms, a criterion must be derived based partly on the fact that all organophosphates inhibit the production of the AChE enzyme. Demeton is unique, however, in that the persistence of its AChE inhibiting ability is greater than that of ten other common organophosphates, even though its acute toxicity is apparently less. The effective "half-life" of AChE inhibition for demeton is greater than one year (Weiss and Gakstatter, 1964b). Because such inhibition may be additive with repeated exposures and may be compounded by any of the organophosphates, it is recommended that a criterion for demeton be based primarily on its enzyme-inhibiting potential. A criterion of 0.1 ug/l demeton for freshwater and marine aquatic life is recommended since it will not be expected to significantly inhibit AChE over a prolonged period of time. In addition, the criteria recommendation is in close agreement with the criteria for the other organophosphates.

REFERENCES CITED:

Butler, P.A., 1964. Commercial fishery investigations. In: Pesticide Wildlife Studies, 1963. p. 5-28. U.S. Fish and Wildlife Service Circular 149. Washington, D.C.

Henderson, C. and Q.H. Pickering, 1958. Toxicity of organic phosphorus insecticides to fish. Trans. Amer. Fish. Soc., 87:39.

Ludemann, D. and H. Neumann, 1962. Uber die wirkung der neuzeitlichen kontakinseckitizide auf die tiere des susswassers. Anzeiger fur Schadlingskunde und Pflanzenschutz, 35:5.

Macek, K.J. and W.A. McAllister, 1970. Insecticide susceptibility of some common fish family representatives. Trans. Amer. Fish. Soc., 99:20.

McCann, J.A. and R.L. Jasper, 1972. Vertebral damage to bluegills exposed to acutely toxic levels of pesticides. Trans. Amer. Fish. Soc., 101:317.

Pickering, Q.H., et al., 1962. The toxicity of organic phosphorus insecticides to different species of warmwater fishes. Trans. Amer. Fish. Soc., 91:175.

Sanders, H.O., 1972. Toxicity of some insecticides to four species of malacostracan crustaceans. U.S. Dept. of the Interior, Washington, D.C., Bureau of Sport Fisheries and Wildlife, Tech. Paper No. 66. pp. 3-19.

Weiss, C.M., 1958. The determination of cholinesterase in the brain tissue of three species of freshwater fish and its inactiviation in vivo. Ecology, 39:194.

Weiss, C.M., 1959. Response of fish to sub-lethal exposures of organic phosphorus insecticides. Sew. and Ind. Wastes, 31:580.

Weiss, C.M., 1961. Physiological effect of organic phosphorous insecticides on several species of fish. Trans. Amer. Fish. Soc., 90:143.

Weiss, C.M. and J.H. Gakstatter, 1964a. Detection of pesticides in water by biochemical assay. Jour. Water Poll. Cont. Fed., 36:240.

Weiss, C.M. and J.H. Gakstatter, 1964b. The decay of anticholinesterase activity of organic phosphorus insecticides on storage in waters of different pH. Advances in Water Poll. Research, 1:83-95.

ENDOSULFAN

CRITERIA:

0.003 ug/l for freshwater aquatic life;

0.001 ug/l for marine aquatic life.

RATIONALE:

The acute toxicity of endosulfan (also known as thiodan) to different fish species varies widely. Macek, et al. (1969) exposed rainbow trout, Salmo gairdneri, to endosulfan at three temperatures and computed 24-hour and 96-hour LC50s. At 1.6° C, 7.2° C, and 12.7° C the 24-hour LC50's were 13, 6.1, and 3.2 ug/l, respectively. The corresponding 96-hour LC_{50} values were 2.6, 1.7, and 1.5 ug/l. Schoettger (1970), however, reports the 96-hour LC_{50} for rainbow trout to be 0.8 ug/l at 1.5° C and 0.3 ug/l at 10° C. He also determined the 96-hour LC_{50} for the western white sucker, Castostomus commersoni, to be 3.5 ug/l at 10° C and 3.0 ug/l at 19° C.

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

The 24-, 48-, and 96-hour LC_{50s} for the amphipod, Gammarus lacustris, were found to be 9.2, 6.4, and 5.8 ug/l (Sanders, 1969). Sanders and Cope (1968) determined the 24-, 48-, and 96-hour LC50's for naiads of the stonefly, Pteronarcys

californica, to be 24, 5.6, and 2.3 ug/l, respectively. The 96-hour LC_{50} for Gammarus fasciatus was found to be 6.0 ug/l (Sanders, 1972).

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

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

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

Butler (1963) reported a 48-hour EC_{50} death or loss of equilibrium of 0.2 ug/l for the brown shrimp, Penaeus aztecus, a 48-hour EC_{50} for juvenile blue crabs, Callinectes sapidus, of 35 ug/l; and a 48-hour EC_{50} of 0.6 ug/l for juvenile white mullet, Mugil curema. A concentration of 65 ug/l resulted in a 50 percent decrease in shell growth of the American oyster, Crassostrea virginica, at 28° C and a salinity

of 22 o/oo. Korn and Earnest (1974) report a 96-hour LC_{50} of 0.1 ug/l for the striped bass, Morone saxatilis.

Use of an application factor of 0.01 times the 96-hour LC_{50} of the most sensitive marine organism tested, the striped bass, results in a marine criterion of 0.001 ug/l.

REFERENCES CITED:

Butler, P.A., 1963. Pesticide wildlife studies. A review of Fish and Wildlife Service investigations during 1961 and 1962. USDI, Fish and Wildlife Service Circ. No. 167.

Greve, P.A. and S.L. Wit, 1971. Endosulfan in the Rhine River. Jour. Water Poll. Cont. Fed., 43:2338.

Heath, R.G., et al., 1972. Comparative dietary toxicities of pesticides to birds. U.S. Dept. of the Interior, Bureau of Sport Fisheries and Wildlife. Special Scientific Report - Wildlife No. 152. Washington D.C.. 57 p.

Herzel, E. and D. Ludemann, 1971. Behavior and toxicity of endosulfan in water under various experimental conditions. Z. Angew. Zool., 58:57.

Lindquist, D. and P.A. Dahm, 1957. Some chemical and biological experiments with thiodan. Jour. Econ. Entomol., 50:483.

Korn, S. and R. Earnest, 1974. Acute toxicity of twenty insecticides to striped bass, Morone saxatilis. Calif. Fish and Game, 60:128.

Macek, K.J., et al., 1969. Effects of temperature on the susceptibility of bluegills and rainbow trout to selected pesticides. Bull. Environ. Contam. Toxicol., 4:174.

Portman, J.E. and K.W. Wilson, 1971. The toxicity of 140 substances to the brown shrimp and other marine animals. Ministry of Agriculture, Fisheries and Food, Fisheries Laboratory, Burnham-on-Couch, Essex, England, Shellfish Information Leaflet No. 22, 12 pp.

Sanders, H.O., 1969. Toxicity of pesticides to the crustacean, Gammarus lacustris. U.S. Dept. of the Interior, Bur. Sport Fish. Wildl., Tech. Paper 25.

Sanders, H.O., 1972. Toxicity of some insecticides to four species of malacostracan crustaceans. U.S. Dept. of the Interior, Bur. of Sport Fish. and Wildl., Washington, D.C., Tech. Paper 66.

Sanders, H.O. and O.B. Cope, 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. Oceanog., 13:112.

Schoettger, R.A., 1970. Toxicology of thiodan in several fish and aquatic invertebrates. Bur. Sport Fish. Wildl., Investigations in Fish Control, 35. U.S. Government Printing Office, Washington,D.C.

ENDRIN

CRITERIA:

0.2 ug/l for domestic water supply (health);

0.004 ug/l for freshwater and marine aquatic life.

RATIONALE:

The highest level of endrin found to have minimal or no long-term effects in the most sensitive animal tested, the dog, is 1.0 mg/kg in the diet or 0.02 mg/kg of body weight/day (Treon, et al., 1955). Where adequate human data are not available for corroboration of the animal results, the total "safe" intake level is assumed to be 1/500 of the "no effect" or "minimal effect" level reported for the most sensitive animal tested.

Applying the available data and based upon the assumptions that 20 percent of the total intake of endrin is from drinking water, that the average person weighs 70 kg and consumes 2 liters of water per day, the formula for calculating a criterion is .02 mg/kg x 0.2 x 70 kg x 1/500 x 1/2 = .00028 mg/l thus deriving the criterion level for domestic water supply of 0.2 ug/l.

Toxicity data for the flagfish, Jordanella floridae, indicate that the "safe" concentration as determined in a long-term exposure involving reproduction is about 0.30 of the 96-hour LC_{50} (Hermanutz, 1974). Ninety-six-hour LC50's for some of the sensitive freshwater fish tested are as follows: bluegills, 0.6 ug/l (Henderson, et al., 1959); rainbow trout, 0.6 ug/l and coho salmon, 0.5 ug/l (Katz, 1961); juvenile striped bass (freshwater and estuarine life-phase), 0.094 ug/l (Korn and Earnest, 1974);

cutthroat trout, 0.113 ug/l (Post and Schroeder, 1971). Therefore, the estimated "safe" water concentrations for these sensitive fish, based on the flagfish application factor is approximately 0.03 to 0.18 ug/l.

Stonefly naiads appear to be the most sensitive invertebrates tested. Jensen and Gaufin (1966) found the 30-day LC_{50} for the naiad, Acroneuria pacifica, to be 0.035 ug/l. Based on these data, the safe water concentration should be less than 0.035 ug/l to fully protect stoneflies throughout their entire life cycle, as well as organisms more sensitive than those that have been tested.

Fathead minnows, Pimephales promelas, exposed to 0.015 ug/l in the ambient water had total body residues 10,000 times greater than the water concentrations (Mount and Putnicki, 1966). Residue accumulation up to 10,000-fold was observed in flagfish exposed for 60 days to several concentrations between 0.05 and 0.3 ug/l (Hermanutz, 1974). Concentration factors for estuarine organisms exposed to endrin for 96-hours were a maximum of 1,600 in oysters, 1,100 in pink shrimp, 860 in grass shrimp, 4,500 in sheepshead minnows and 2,500 in the sailfin molly (Schimmel, et al., 1975). Johnson (1967) calculated the concentration in adult medeka tissue to be 17,000 to 26,000 times the water concentration. It is quite possible that some fish would accumulate endrin to 30,000 times water concentration. This degree of accumulation is based only on observations of uptake directly from the water and does not allow for accumulation via the food chain or significantly higher accumulation rates possible in other, untested fish species.

Endrin has been found to be eliminated quickly after termination of exposure. Channel catfish tissue residues were reduced

from 0.41 to 0.02 ug/g (about 95 percent reduction) within 13 days after the addition of endrin to the water was stopped (Argyle, et al., 1973). Marine spot tissue residues of 78.0 ppb were reduced below detection levels within 13 days (Lowe, 1966). Recent unpublished data showing that flagfish eliminated about 95 percent in five days support this observation. This apparent ability to excrete endrin readily may reduce the threat of extremely high residue accumulations.

Levels of 0.1 ug/l have been shown to be 100 percent lethal to the marine spot, Leiostomus xanthuras, within five days, while approximately half the population survived at a level of 0.075 ug/l after 19 days (Lowe, 1966).

Davis and Hidu (1969) reported the 48-hour TLm of American oyster eggs to be 0.79 mg/l and the 14-day TLm for larvae to be greater than 10 mg/l.

Eisler (1969) determined the following 96-hour LC_{50} values: Sand shrimp, Crangon septemspinosa - 1.7 ug/l; grass shrimp, Palaemonetes pugio - 1.8 ug/l; and hermit crab, Pagurus longicarpus - 12 ug/l. The following 96-hour LC_{50} values were reported by Eisler (1970): Atlantic silverside, Menidia menidia - 0.05 ug/l; blue head, Thalassoma bifasciatum - 0.1 ug/l; striped killifish, Fundulus majalis - 0.3 ug/l; striped mullet, Mugil cephalus - 0.3 ug/l; American eel, Anguilla rostrata - 0.6 ug/l; mummichog, Fundulus heteroclitus - 0.6 ug/l; and Northern puffer, Sphoeroides maculatus - 3.1 ug/l. The 96-hour LC50 for striped bass was 0.094 ug/l

(Korn and Earnest, 1974), shiner perch, Cymatogastes aggregata, 0.12 ug/l and dwarf perch, Micrometrus minimus, 0.13 ug/l (Earnest and Benville, 1972). Ninety-six hour LC50's based on measured concentrations were 0.63 ug/l for sailfin mollies, Poecilia latipinna, 0.38 ug/l for sheepshead minnow, Cyprinodon variegatus, 0.63 ug/l for grass shrimp, Palaemonetes pugio, and 0.037 ug/l for pink shrimp, Penaeus duorarum (Schimmel, et al., 1975). Data on the effects of endrin in an exposure throughout the entire life cycle of the sheepshead minnow indicate that a "safe" concentration to protect for reproductive effects would be about 0.3 of the 96-hour LC50 (Hansen, In Press). An application factor of 0.01 of the 96-hour LC50 may be over protective. Therefore, for endrin this factor should be 0.1. Based on this, the criterion for salt water should be 0.1 of the 96-hour LC50 for pink shrimp: 0.004 ug/l. This criterion should also be protective of freshwater aquatic life.

REFERENCES CITED:

Argyle, R.L., et al., 1973. Endrin uptake and release by fingerling channel catfish (Ictalurus punctatus). Jour. Fish. Res. Bd. Can., 30:1743.

Davis, H.C. and H. Hidu, 1969. Effects of pesticides on embryonic development of clams and oysters and on survival and growth of the larvae. U.S. Dept. of the Interior, Fish. and Wildl. Serv, Fishery Bull. 67:393.

Earnest, R. and P.E. Benville, Jr. 1972. Acute toxicities of four organochlorine insecticides to two species of surf perch. Calif. Fish Game 58(2):127-132.

Eisler, R., 1969. Acute toxicities of insecticides to marine decapod crustaceans. Crustaceana, 16:302.

Eisler, R., 1970. Acute toxicities of organochlorine and organophosphorus insecticides to estuarine fishes. U.S. Dept. of the Interior, Bur. Sport Fish. and Wildl., Tech. Paper 46.

Hansen, D.J. In Press. Techniques to assess the effects of toxic organics on marine organisms. EPA Ecol. Res. Ser.

Henderson, C., et al., 1959. Relative toxicity of ten chlorinated hydrocarbon insecticides to four species of fish. Trans. Amer. Fish. Soc., 88:23.

Hermanutz, R., 1974. Northwest water quality report. National Water Quality Laboratory, Duluth, Minnesota.

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

Johnson, H.E., 1967. The effects of endrin on the reproduction of a freshwater fish (Oryzias latipes). Ph.D. Thesis, University of Washington, Seattle, 135 p.

Katz, M., 1961. Acute toxicity of some organic insecticides to three species of salmonids and to the threespine stickleback. Trans. Amer. Fish. Soc., 90:264.

Korn, S. and R. Earnest, 1974. Acute toxicity of twenty insecticides to striped bass, Morone saxatilis. Calif. Fish and Game, 60:128.

Lowe, J.I., 1966. Some effects of endrin on estuarine fishes. Proc. 19th Ann. Conf. S.E. Assn. Game and Fish. Commissioners, pp. 271-276.

Mount, D.I. and G.J. Putnicki, 1966. Summary report of the 1963 Mississippi fish kill. Trans. 31st No. Amer. Wildl. and Nat. Res. Conf., p. 177.

Post, G. and T.R. Schroeder, 1971. The toxicity of four insecticides to four salmonid species. Bull. Environ. Contam. Toxicol., 6:144.

Schimmel, S.C., et al. 1975 Endrin: Effects on several estuarine organisms. Proc. 29th Ann. Conf. S.E. Assn. Game and Fish. Commissioners.

Treon, et al., 1955. Toxicity of endrin for laboratory animals. Jour. Agric. and Food Chem., 3:842.

GUTHION

CRITERION:

.01 ug/l for freshwater and marine aquatic life.

RATIONALE:

Ninety-six-hour LC_{50} values for fish exposed to the organophosphorus pesticide, guthion, range from 4 to 4,270 ug/l (Katz, 1961; Pickering, et al., 1962; Lahav and Sarig, 1969; Macek, et al., 1969; Macek and McAllister, 1970). The only long-term fish exposure data available are those obtained recently by Adelman and Smith (unpublished data). Decreased spawning (eggs produced per female) was observed in fathead minnows, Pimephales promelas, exposed during a complete life cycle. An estimated "safe" long-term exposure concentration for fathead minnows lies between 0.3 and 0.5 ug/l. Survival of larvae was reduced at approximately 0.7 ug/l.

An investigation of the persistence of guthion in fish revealed that 50 percent of the chemical was lost in less than 1 week (Meyer, 1965). Analysis of plankton and pondwater in the same study indicated a 50 percent loss of guthion in about 48 hours. Flint, et al. (1970) determined the half-life of guthion at 30° C in pondwater and in a phosphate buffer protected from light in the laboratory. The half-life in pondwater was 1.2 days whereas that in the laboratory solution was 10 days. The more rapid degradation in pondwater was attributed to the effect of sunlight and microorganisms.

Organophosphate pesticides are toxic because they inhibit the enzyme acetylcholinesterase (AChE) which is essential to nerve impulse conduction

and transmission (Holland, et al., 1967). Weiss (1958, 1959, 1961) demonstrated that a 40 to 70 percent inhibition of fish brain AChE usually is lethal. Centrarchids generally are considered one of the more sensitive groups of fish to guthion (Pickering, et al., 1962; Weiss and Gakstatter, 1964; Meyer, 1965). Weiss and Gakstatter (1964) found that over a 15-day period bluegills, Lepomis macrochirus, exhibited AChE inhibition at 1.0 ug/l guthion but not at 0.1 ug/l. Exposure at 0.05 ug/l for 30 days also failed to produce inhibition below the range of normal variation, but the authors stated that it appeared there was a downward trend in brain enzyme activity and that if exposure was continued a definite reduction might develop. Weiss (1961) found that about 30 days were required for fathead minnow and bluegill brain AChE levels to recover after 8 to 24 hours exposure to 10 ug/l guthion.

Benke and Murphy (1974) showed that repetitive injection of fish with guthion caused cumulative inhibition of brain AChE and mortality. After substantial inhibition by guthion exposure, it takes several weeks for brain AChE of fishes to return to normal even though exposure is discontinued (Weiss, 1959 and 1961; Carter, 1971). Inhibition of brain AChE of fishes by 46 percent or more has been associated with harmful effects in exposures to other organophosphate pesticides for a life cycle (Eaton, 1970) and for shorter periods (Carter, 1971; Coppage and Duke, 1971; Coppage, 1972; Coppage and Matthews, 1974; Post and Leasure, 1974; Coppage, et al., In Press). In static tests, similar inhibition of AChE and mortality were caused in the sheepshead minnow, Cyprinodon variegatus, in 2, 24, 48, and 72 hours at concentrations of 50, 7, 3.5, and 3 ug/l, respectively (Coppage, 1972). These data indicate that reduction of brain AChE activity of marine fishes by 70 to 80 percent or more in short-term exposures to guthion may be associated with some deaths.

There is no evidence to indicate that guthion would cause adverse effects through the food chain. Tissue residue accumulation for whole fish calculated from the data of Meyer (1965) indicate no more than a 20-fold accumulation. LD_{50} toxicity values for birds are relatively high and range from 70 to 2,000 mg/kg (Tucker and Crabtree, 1970).

Ninety-six-hour LC_{50} values for aquatic invertebrates range from 0.10 to 22.0 ug/l (Nebeker and Gaufin, 1964; Gaufin, et al., 1965; Jensen and Gaufin, 1966; Sanders and Cope, 1968; Sanders, 1969 and 1972). Sanders (1972) exposed the grass shrimp, Paleomonetes kadiakensis, to guthion in a continuous flow bioassay for up to 20 days and found that the 5-and 20-day LC_{50} values were 1.2 and 0.16 ug/l, respectively. He found that the amphipod, Gammarus fasciatus, was the most sensitive aquatic organism tested, with a 96-hour LC50 of 0.10 ug/l. Jensen and Gaufin (1966), also using a continuous flow system, exposed two species of stonefly naiads in 4- and 30-day studies. They observed 96-hour and 30-day LC_{50} values for Acroneuria pacifica of 2.0 and 0.24 ug/l, respectively, whereas for Pteronarcys californica the values were 4.6 and 1.3 ug/l, respectively.

Results of other toxicity studies on marine organisms have been reported. The 24-hour LC_{50} for the white mullet, Mugil curema, was found to be 5.5 ug/l guthion (Butler, 1963). The 96-hour LC_{50} for the striped mullet, Mugil cephalus, was determined by Lahav and Sarig (1969) to be 8 ug/l guthion. Portman (1972) reported the 48-hour LC_{50} for the

fish, Pleuronectes limanda, to be 10 to 30 ug/l. The 48-hour LC_{50} for the European shrimp, Crangon crangon, was found to be 0.33 ug/l guthion (Portman, 1972). Butler (1963) found that the 24-hour EC_{50} for blue crab, Callinectes sapidus, was 550 ug/l and the 48-hour EC_{50} for pink shrimp, Penaeus duorarum, as 4.4 ug/l guthion. The 48-hour TLm was estimated to be 620 ug/l for fertilized oyster eggs, Crassostrea virginica, and 860 ug/l for fertilized clam eggs, Mercenaria mercenaria (Davis and Hidu, 1969).

A criterion level of 0.01 ug/l for guthion is based upon use of an 0.1 application factor applied to the 96-hour LC50 of 0.1 ug/l for Gammarus and a similar value of 0.3 ug/l for the European shrimp.

REFERENCES CITED:

Adelman, I.R. and L.L. Smith. (Unpublished data.) Department of Entomology, Fisheries and Wildlife, University of Minnesota, St. Paul.

Benke, G.M. and S.D. Murphy, 1974. Anticholinesterase action of methyl parathion, parathion and azinphosmethyl in mice and fish: Onset and recovery of inhibition. Bull. Environ. Contam. Toxicol., 12:117.

Butler, P.A., 1963. Commercial fisheries investigations. In: pesticide-wildlife studies during 1961 and 1962. U.S. Fish. Wildl. Serv. Circ. 167, Washington, D.C.

Carter, F.L., 1971. In vivo studies of brain acetylcholinesterase inhibition by organophosphate and carbamate insecticides in fish. Ph.D. Dissertation, Louisiana State University, Baton Rouge, Louisiana.

Coppage, D.L., 1972. Organophosphate pesticides: Specific level of brain ACHE inhibition related to death in sheepshead minnows. Trans. Amer. Fish. Soc., 101:534.

Coppage, D.L. and T.W. Duke, 1971. Effects of pesticides in estuaries along the gulf and southeast Atlantic coasts. In: Proceedings of the 2nd Gulf Coast Conference on Mosquito Suppression and Wildlife Management. (C.H. Schmidt, Ed.) National Mosquito Control - Fish and Wildlife Management Coordinating Committee, Washington, D.C.

Coppage, D.L. and E. Matthews, 1974. Short-term effects of organophosphate pesticides on cholinesterases of estuarine fishes and pink shrimp. Bull. Environ. Contam. Toxicol., 11:483.

Coppage, D.L., et al. (In Press) Brain acetylcholinesterase inhibition in fish as a diagnosis of environmental poisoning by malathion, O,O-dimethyl S-(1,1-dicarbethoxyethyl) phosphorodithioate. Pesticide Biochemistry and Physiology.

Davis, H.C. and H. Hidu, 1969. Effects of pesticides on embryonic development of clams and oysters and on survival and growth of the larvae. U.S. Fish and Wildlife Service, Fishery Bulletin, 67:393.

Eaton, J.G., 1970. Chronic malathion toxicity to the bluegill (Lepomis macrochirus Rafinesque). Water Research, 4:673.

Flint, D.R., et al., 1970. Soil runoff, leaching, and adsorption and water stability studies with guthion. Chemagro Rept. No. 28936.

Gaufin, A.R., et al., 1965. The toxicity of ten organic insecticides to various aquatic invertebrates. Water and Sew. Works, 112:276.

Holland, H.T., et al., 1967. Use of fish brain acetylcholinesterase to monitor pollution by organophosphorus pesticides. Bull. Environ. Contam. Toxicol., 2:156.

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

Katz, M., 1961. Acute toxicity of some organic insecticides to three species of salmonids and to the threespine stickleback. Trans. Amer. Fish. Soc., 90:264.

Lahav, M. and S. Sarig, 1969. Sensitivity of pond fish to cotnion (azinphos methyl) and parathion. Bamidgeh, 21:67.

Macek, K.J., et al., 1969. The effects of temperature on the susceptibility of the bluegills and rainbow trout to selected pesticides. Bull. Environ. Contam. Toxicol., 3:174.

Macek, K.J. and W.A. McAllister, 1970. Insecticide susceptibility of some common fish family representatives. Trans. Amer. Fish. Soc., 99:20.

Meyer, F.P., 1965. The experimental use of guthion as a selective fish eradicator. Trans. Amer. Fish. Soc., 94:203.

Nebeker, A.V. and A.R. Gaufin, 1964. Bioassays to determine pesticide toxicity to the amphipod crustacean, Gammarus lacustris. Proc. Utah Acad. Sci. Arts and Letters, 41:64.

Pickering, Q.H., et al., 1962. The toxicity of organic phosphorus insecticides to different species of warmwater fishes. Trans. Amer. Fish. Soc., 91:175.

Portman, J., 1972. Results of acute tests with marine organisms, using standard methods. In: Marine pollution and sea life (Ruivo, Ed.). Fishing News (Books) Ltd, London, England, pp. 212-217.

Post, G. and R.A. Leasure, 1974. Sublethal effect of malathion to three salmonid species. Bull. Environ. Contam. Toxicol., 12:312.

Sanders, H.O., 1969. Toxicity of pesticides to the crustacean, Gammarus lacustris. U.S. Dept. of the Interior, Bur. Sport Fish. and Wildl., Technical Paper No. 25, Washington, D.C.

Sanders, H.O., 1972. Toxicity of some insecticides to four species of malacostracan crustaceans. U.S. Dept. of the Interior, Bur. Sport Fish. and Wildl. Technical Paper No. 66, Washington, D.C.

Sanders, H.O. and O.B. Cope, 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. and Oceanog., 13:112.

Tucker, R.K. and D.G. Crabtree, 1970. Handbook of toxicity of pesticides to wildlife. U.S. Dept. of the Interior, Bur. Sport Fish. and Wildl., Resource Publ. No. 84, Washington, D.C.

Weiss, C.M., 1958. The determination of cholinesterase in the brain tissue of three species of freshwater fish and its activation in vivo. Ecology, 39:194.

Weiss, C.M., 1959. Response of fish to sub-lethal exposures of organic phosphorus insecticides. Sew. and Ind. Wastes, 31:580.

Weiss, C.M., 1961. Physiological effects of organic phosphorus insecticides on several species of fish. Trans. Amer. Fish. Soc., 90:143.

Weiss, C.M. and J.H. Gakstatter, 1964. Detection of pesticides in water by biochemical assay. Jour. Water Poll. Cont. Fed., 36:240.

HEPTACHLOR

CRITERION:

.001 ug/l for freshwater and marine aquatic life.

The persistence, bioaccumulation potential and carcinogenicity of heptachlor cautions human exposure to a minimum.

RATIONALE:

The acute toxicity of heptachlor to mammals is generally low; however, aquatic organisms exhibit sensitivity to this pesticide at microgram per liter levels. Such levels range from a 96-hour LC_{50} of 3 ug/l for the juvenile striped bass, Morone saxatilis (Korn and Earnest, 1974) to a 96-hour LC_{50} of 111.9 ug/l for the threespine stickleback, Gasterosteus aculeatus (Katz, 1961). Sanders and Cope (1968) reported even lower 96-hour LC_{50} values for three species of stoneflies; Pteronarcys californica, 1.1 ug/l; Pteronarcella badia, 0.9 ug/l and Claassenia sabulosa, 2.8 ug/l.

Anderson (1960) found that Daphnia magna were immobilized after 50 hours exposure to 57.77 ug/l. A 48-hour LC_{50} of 42 ug/l heptachlor for Daphnia pulex was reported by Cope (1966). Sanders and Cope (1968) determined the 96-hour LC_{50} values for the stoneflies, Pteronarcys californica, Pteronarcella badia and Claassenia sabulosa to be 1.1, 0.9 and 2.8 ug/l, respectively.

Sanders (1972) found the grass shrimp, Palaemonetes kadiakensis, with a 96-hour LC_{50} of 1.8 ug/l, to be the most sensitive among three crustaceans tested in static bioassays. Stoneflies, therefore, appear to be the most sensitive group of freshwater organisms among those tested.

Cope (1966) found the 48-hour LC_{50} for heptachlor to the bluegill, Lepomis macrochirus, and rainbow trout, Salmo gairdneri, to be 26 ug/l and 9 ug/l, respectively. The 96-hour LC_{50} of heptachlor for the bluegill was determined by Weiss (1964) to be 19 ug/l. Katz (1961) found that the 96-hour LC50's of heptachlor for the coho salmon, Oncorhynchus kisutch; chinook salmon, Oncorhynchus tschawytscha; rainbow trout, Salmo gairdneri; and threespine stickleback, Gasterosteus aculeatus, were 59, 17.3, 19.4, and 111.9 ug/l, respectively. The 96-hour LC_{50} of heptachlor to the fathead minnow, Pimephales promelas; bluegill, Lepomis macrochirus; goldfish, Carassius auratus; and guppy, Poecilia reticulata, were determined to be 94, 230 and 170 ug/l, respectively (Henderson, et al., 1959).

Data are available on marine animals exposed to heptachlor in 96-hour flow-through bioassays. The 96-hour LC50 of the marine bluehead, Thalassoma bifasciatum, was determined to be 0.8 ug/l (Eisler, 1970). Korn and Earnest (1974) determined the 96-hour LC50 using juvenile striped bass, Morone saxatilis, to be 3.0 ug/l. Schimmel et al., (In Press) reported the following 96-hour LC50's in flow-through bioassays: pink shrimp, Penaeus duorarum, 0.11 ug/l; grass shrimp, Palaemonetes vulgaris, 1.06 ug/l; sheepshead minnow, Cyprinodon variegatus, 3.68 ug/l; pinfish, Lagodon rhomboides, 3.77 ug/l; spot, Leiostomus xanthurus, 0.85 ug/l. Shell deposition of the American oyster, Crassostrea virginica, was inhibited by 50 percent (EC50) at 1.5 ug/l heptachlor.

Heptachlor will accumulate in the food chain. Wilson (1965) demonstrated that oysters can concentrate heptachlor almost 18,000 times (wet weight basis). Andrews et al. (1966) reported concentration factors as high as 1,840 in field tests with bluegill, Lepomis macrochirus. Since the effective (as opposed to the applied) concentration was apparently lower than the initial levels because of sorption and biological uptake, true concentration factors must have been higher than reported. For example, 24 hours after treatment with 24 ug/l the pond water contained only 2 ug/l. Using this concentration and the 46.0 ppm residue found in the fish, a concentration factor of 23,000 can be calculated. The involvement of several trophic levels might also increase the degree of accumulation. Schimmel, et al., (In Press) reported that heptachlor concentration factors ranged from 2,800 to 21,300 in estuarine fishes exposed for 96 hours; oyster bioconcentration factors ranged from 4,500 to 8,500, and factors of 206 to 700 occurred in similar tests with crustaceans.

Birds are sensitive to low dosages of heptachlor in their diet. Heath, et al. (1972) found the 5-day LC_{50} for the young of four species to range from 24 to 54 ppm. All woodcock died from a dietary dosage of 0.72 ppm and some died from 0.22 ppm (Stickel and Stickel, 1965). Residues of this magnitude (0.2 ppm) would result in fish or other aquatic life if they were exposed to 0.01 ug/l of heptachlor and accumulated it at 20,000 times water

concentrations. Such residues would pose a hazard to birds as sensitive as woodcock that fed on these fish. Residues of this magnitude fall just under the 0.3 ppm guideline established by the U.S. Food and Drug Administration as the limit allowed in edible fish tissue (U.S. Food and Drug Administration, 1974).

Since heptachlor is a highly persistent chemical which bioaccumulates in aquatic organims used for human food and also is considered a potential human carcinogen (Train, 1974) levels of heptachlor in waterways should be kept as low as feasible. The July 1975 action by the Environmental Protection Agency in suspending the production and use of heptachlor should result in a gradual decrease in concentrations in the environment. Any addition of heptachlor to water should be considered potentially hazardous to humans. Such additions should not be permitted without substantial documentation that alternatives are either infeasible or potentially more hazardous. The persistence, bioaccumulative properties and carcinogenic potential of heptachlor should be taken into account when determining the uses of water containing measurable amounts of heptachlor.

Using an 0.01 application factor to the 96-hour LC50 of 0.1 ug/l for the pink shrimp, a marine criterion of 0.001 ug/l is obtained. Toxicity and demonstrated bioaccumulation potential of heptachlor in freshwater biota reflects the applicability of the marine criterion to freshwater.

REFERENCES CITED:

Anderson, B. G., 1960. The toxicity of organic insecticides to Daphnia. In: Trans. 1959 Seminar Biol. Probs. Water Pollution. Robt. A. Taft San. Center, Tech. Dept. W60-3, p. 94.

Andrews, A.K., et al., 1966. Some effects of heptachlor on bluegills (Lepomis macrochirus). Trans. Amer. Fish. Soc., 95:297.

Cope, O.B., 1966. Contamination of the freshwater ecosystem by pesticides. Jour. Appl. Ecol. (Suppl.), 33.

Eisler, R., 1970. Acute toxicity of organochlorine and organophosphorus insecticides to estuarine fishes. Tech. Papers, Bur. Sport Fish. Wildl., 46.

Food and Drug Administration, 1974. Poisonous or deleterious substances in peanuts, evaporated milk, fish and shellfish. Proposed Rules. Federal Register, December 6, 1974. Washington, D. C.

Heath, R. G., et al., 1972. Comparative dietary toxicities of pesticides to birds. Bureau of Sport Fisheries and Wildlife, Wildlife Report No. 152, U.S. Dept. of the Interior, Washington, D. C.

Henderson, D., et al., 1959. Relative toxicity of ten chlorinated hydrocarbon insecticides to four species of fish. Trans. Amer. Fish. Soc., 90: 264.

Katz, M., 1961. Acute toxicity of some organic insecticides to three species of salmonids and to the threespine stickleback. Trans. Amer. Fish. Soc., 90: 264.

Korn, S., and R. Earnest, 1974. Acute toxicity of twenty insecticides to striped bass, Morone saxatilis. Calif. Fish and Game, 60:128.

Sanders, H.O., 1972. Toxicity of some pesticides to four species of malocostracan crustacea. U.S. Bureau of Sport Fisheries and Wildlife Technical Paper No. 66.

Sanders, H.O. and O.B. Cope, 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. and Oceanog., 13:112.

Schimmel, S.C., et al., 1976. Heptachlor: Toxicity to and uptake by several estuarine organisms. Jour. Toxicol. & Environ. Health (In Press).

Stickel, W.H. and L.F. Stickel. 1965. Effects of heptachlor-contaminated earthworms on woodcocks. Jour. Wildlife Management, 21:132.

Train, R. E., 1974. Pesticide products containing heptachlor or chlordane: Intent to cancel registrations, Federal Register, Vol. 39, No. 229, Tues., Nov. 26, 1974, p 41299.

Weiss, C.M., 1964. Organic pesticides and water pollution. Public Works 95:84.

Wilson, A.J., 1965. Chemical assays. In: Annual Report of the Bureau of Commerical Fisheries Biological Laboratory. Gulf Breeze, Florida, Fiscal Year 1965. U. S. Bur. Comm. Fish. Circ. 247.

LINDANE

CRITERIA:

4.0 ug/l for domestic water supply (health);

0.01 ug/l for freshwater aquatic life;

0.004 ug/l for marine aquatic life

RATIONALE:

The highest level of lindane found to have minimal or no long-term effects in the most sensitive mammal tested, the dog, is 15.0 mg/kg in the diet or 0.3 mg/kg of body weight/day (Lehman, 1965). Where adequate human data are not available for corroboration of the animal results, the total "safe" drinking intake level is assumed to be 1/500 of the "no effect" or "minimal effect" level reported for the most sensitive animal tested.

Applying the available data and based upon the assumption that 20 percent of the total intake of lindane is from drinking water, that the average person weighs 70 kg and consumes 2 liters of water per day, the formula for calculating a criterion is 0.3 mg/kg x 0.2 x 70 kg x 1/500 x 1/2 = .004 mg, thus deriving the criterion level for domestic water supply of 4 ug/l.

The brown trout, Salmo trutta, apparently is the fish most sensitive to lindane among those species on which aquatic bioassays have been performed, with a 96-hour LC_{50} of 2 ug/l (Macek and McAllister, 1970). Several authors (Boyd and Ferguson, 1964; Naqvi, et al., 1969; Minchew and Ferguson, 1970) have documented increased

resistance to lindane toxicity among fish and

invertebrates experiencing previous exposure to the chemical. The most sensitive invertebrates tested appear to be only slightly more sensitive than fish. Two investigators (Snow, 1958; Cope, 1965) reported TLm values of 1 ug/l for stoneflies. Sanders and Cope (1968) reported a TLm for stoneflies of 4.5 ug/l lindane. Macek, et al. (1974) determined the acute and chronic toxicities of lindane to Daphnia magna; the midge, Chironomus tentans; and the scud, Gammarus fasciatus. The midge was the most sensitive of these species, with 2.2 ug/l being the highest concentration producing no observable adverse effect. A concentration of 11 ug/l was determined to be "safe" for Daphnia, the least sensitive, over three consecutive generations of exposure.

A criterion of 0.01 ug/l for fresh waters is derived by applying an application factor of 0.01 to the TLM for the stonefly, 1.0 ug/l. The brown trout, a sensitive human food organism, was shown to have a 96-hour LC_{50} of 2.0 ug/l. Hence the criterion level would provide for a margin of safety for both recreationally important organisms as well as trophic levels in the aquatic environment which are significant in the food chain.

Only limited information is available on accumulation of lindane in fish tissues. However, Macek, et al. (1974) observed whole-body (eviscerated) levels (wet weight) of 500 times the corresponding water concentrations in fathead minnows, Pimephales promelas, that had been exposed for several months. Butler (1967) observed accumulations of up to 250 times exposure concentrations in marine mollusks (wet weight basis).

The recommended guideline of the U.S. Food and Drug Administration for lindane in edible fish tissue is 0.3 mg/kg (FDA, 1974). Thus, if the observed 500-fold accumulation were to occur, a lindane criterion of 0.01 ug/l in water would result in a tissue concentration in freshwater fish of .005 mg/kg, which is well below the FDA guideline.

Eisler (1969) reported 96-hour LC_{50} values of 5.0 ug/l for both the hermit crab, Pagurus longicarpus, and the sand shrimp, Crangon septemspinosa, and 10 ug/l for the grass shrimp, Palaemonetes pugio. Butler (1963) found the following 48-hour LC_{50} values: brown shrimp, Penaeus aztecus - 0.4 ug/l; juvenile white mullet, Mugil curema - 30 ug/l; and the longnose killifish, Fundulus similis - 240 ug/l. A 96-hour LC_{50} of 7.3 ug/l for the striped bass, Morone saxatilis was reported by Korn and Earnest (1974). Schimmel (unpublished data) found the pink shrimp, Penaeus duorarum, 96-hour LC50 to be 0.17 ug/l, grass shrimp Palaemonetes pugio, 4.4 ug/l and pinfish, Lagodon rhomboides, 30.6 ug/l in flow-through bioassays.

Until additional data on the effect of lindane to marine organisms are available, it is recommended that a marine criterion be based on 0.01 of the 0.4 ug/l 96-hour LC_{50} value for the brown shrimp. The marine criterion is, therefore, 0.004 ug/l.

Unpublished data suggest that the pink shrimp is a more sensitive species than the brown shrimp; however for purposes of achieving a criterion the published data were used. A level of 0.004 ug/l should provide a margin of safety for both sensitive species.

REFERENCES CITED:

Boyd, C.E. and D.E. Ferguson, 1964. Susceptibility and resistance of mosquitofish to several insecticides. Econ. Entomol., 57:430.

Butler, P.A., 1963. Commercial fisheries investigations. In: Pesticide wildlife studies. U.S. Dept. of Interior, Fish and Wildlife Service, Washington, D. C., Circ. 167, p. 11-25.

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

Cope, O.B., 1965. 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 Service, Circ. 226. pp. 51-63.

Eisler, R., 1969. Acute toxicities of insecticides to marine decapod crustaceans. Crustaceana, 16:302.

Food and Drug Administration, 1974. Poisonous or deleterious substances in peanuts, evaporated milk, fish and shellfish. Proposed Rules. Federal Register, December 6, 1974, Washington, D. C.

Korn, S. and R. Earnest, 1974. Acute toxicity of twenty insecticides to striped bass, Morone saxatilis. Calif. Fish and Game, 60:128.

Lehman, A.J., 1965. Summaries of pesticide toxicity. Assoc. of Food and Drug Officials of the U.S., Topeka, Kansas, pp 1-40.

Macek, K.J. and McAllister, 1970. Insecticide susceptibility of some common fish family representatives. Trans. Amer. Fish Soc., 99:20.

Macek, K.J., et al., 1974. Chronic toxicity of lindane to selected aquatic invertebrates and fishes. Environmental Protection Agency Research Contract Report, Environmental Protection Agency Ecol. Res. Series, (In preparation).

Minchew, G.D. and D.E. Ferguson, 1970. Toxicities of six insecticides to resistant and susceptible green sunfish and golden shiners in static bioassays. Jour. Miss. Acad. Sci., 15:29.

Naqvi, S.M. and D.E. Ferguson, 1969. Pesticide tolerances of selected freshwater invertebrates. Jour. Miss. Acad. Sci., 14:121.

Sanders, H.O., and O.B. Cope, 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. and Oceanog., 13:112.

Schimmel, S.C., 1976. Semi-Annual Report. Gulf Breeze Environmental Research Laboratory (In Press).

Snow, J.R., 1958. A preliminary report on the comparative testing of some of the newer herbicides. Proc. 11th Ann. Conf., Southeast Assoc. Game Fish Comm., pp. 125-132.

MALATHION

CRITERION:

0.1 ug/l for freshwater and marine aquatic life.

RATIONALE:

The freshwater fish most sensitive to malathion, an organophosphorus pesticide, appear to be the salmonids and centrarchids. Post and Schroeder (1971) report a 96-hour LC_{50} between 120 and 265 ug/l for 4 species of salmonids. Macek and McAllister (1970) found a 96-hour LC_{50} range between 101 and 285 ug/l for 3 species of centrarchids and 3 species of salmonids. Other 96-hour LC50's are: rainbow trout, Salmo gairdneri, 68 ug/l (Cope, 1965); largemouth bass, Micropterus salmoides, 50 ug/l (Pickering, et al., 1962) and chinook salmon, Oncorhynchus tshawytscha, 23 ug/l (Katz, 1961). All of the above tests were in static systems. Eaton (1970) determined a 96-hour LC_{50} for bluegill, Lepomis macrochirus, in a flow-through system at 110 ug/l. Macek and McAllister (1970) reported a similar 96-hour LC_{50} for the bluegill in a static exposure. Static 96-hour LC50s of 120 and 160 ug/l were reported by Post and Schroeder (1971) for brook trout, Salvelinus fontinalis. Bender (1969) indicated that the acute toxicity to fathead minnows, Pimephales promelas, is slightly greater (about 2.0 times) in a static system than in a flow-through system. The flow-through acute toxicity to fathead minnows reported by Mount and Stephan (1967) approximated the static acute toxicity reported by Henderson and Pickering (1958) and Bender (1969).

Many aquatic invertebrates appear to be more sensitive than fish to malathion. The 96-hour LC_{50} for Gammarus lacustris was 1.0 ug/l (Sanders, 1969); for Pteronarcella badia, 1.1 ug/l (Sanders and Cope, 1968); and for Gammarus fasciatus, 0.76 ug/l (Sanders, 1972). The 48-hour LC_{50} for Simocephalus serrulatus was 3.5 ug/l and for Daphnia pulex, 1.8 ug/l (Sanders and Cope, 1966). Daphnia were immobilized in 50 hours in 0.9 ug/l (Anderson, 1960). The 24-hour LC50s for two species of midge larvae were 2.1 ug/l (Mulla and Khasawinah, 1969) and 2.0 ug/l (Karnak and Collins, 1974).

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

The stability of malathion in water is dependent on the chemical and biological conditions of the water (Paris, et al., 1975). Weiss and Gakstatter (1964) have shown that the half-life of malathion was reduced from about 5 months at pH 6 to one to two weeks at pH 8. Eichelberger and Lichtenberg (1971) found that only 10 percent remained in the Little Miami River (pH

7.3-8.0) after 2 weeks. Bender (1969) states that one of the malathion breakdown products may be more toxic than the parent compound.

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

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

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

Malathion enters the aquatic environment primarily as a result of its application as an insecticide. Because it degrades quite rapidly in most waters depending on pH, its occurrence is sporadic rather than continuous. Because the toxicity

is exerted through inhibition of the enzyme acetylcholinesterase (AChE) and because such inhibition may be additive with repeated exposures and may be caused by any of the organo-phosphorus insecticides, inhibition of AChE by more than 35 percent may be expected to result in damage to aquatic organisms.

An application factor of 0.1 is applied to the 96-hour LC50 data for Gammarus lacustris, G. fasciatis and Daphnia , which are all approximately 1.0 ug/l, yielding a criterion of 0.1 ug/l.

REFERENCES CITED:

Anderson, B.G., 1960. The toxicity of organic insecticides to Daphnia. Second Seminar on Biol. Problems in Water Pollution. Robert A. Taft Sanitary Engineering Center Technical Report W60-3, Cincinnati, Ohio.

Bender, M.E., 1969. The toxicity of the hydrolysis and breakdown products of malathion to the fathead minnow (Pimephales promelas, Rafinesque). Water Res., 3:571.

Butler, P.A., 1963. Commercial fisheries investigations. In: Pesticide Wildlife Studies during 1961 and 1962. U.S. Fish Wildl. Ser. Circ. 167, Washington, D.C.

Coppage, D.L. and T.W. Duke, 1971. Effects of pesticides in estuaries along the Gulf and Southeast Atlantic Coasts. In: Proceedings of the 2nd Gulf Coast Conference on Mosquito Suppression and Wildlife Management. (C.H. Schmidt, ed.) National Mosquito Control - Fish and Wildlife Management Coordinating Committee, Washington, D.C.

Coppage, D.L. and E. Matthews, 1974. Short-term effects of organophosphate pesticides on cholinesterases of estuarine fishes and pink shrimp. Bull. Environ. Contam. Toxicol. 11: 483.

Coppage, D.L., et al., 1975. Brain acetylcholinesterase inhibition in fish as a diagnosis of environmental poisoning by malathion, O, O-dimethyl S-(1, 1-dicarbethoxy-ethyl) phosphorodithioate. Pesticide Biochemistry and Physiology (in press).

Cope, O.B., 1965. Sport fishery investigations. In: The Effects of Pesticides on Fish and Wildlife. U.S. Department of the Interior, Washington, D.C. Fish and Wildlife Service Circular 266.

Davis, H.C. and H. Hidu, 1969. Effects of pesticides on embryonic development of clams and oysters and on survival and growth of the larvae. U.S. Fish and Wildlife Service, Fishery Bulletin. 67: 393.

Eaton, J.G., 1970. Chronic malathion toxicity to the bluegill (Lepomis macrochirus Rafinesque). Water Res., 4: 673.

Eichelberger, J.W. and J.J. Lichtenberg, 1971. Persistence of pesticides in river water. Environ. Sci. & Technol., 5: 541.

Eisler, R., 1969. Acute toxicities of insecticides to marine decapod crustaceans. Crustaceana. 16: 302.

Eisler, R., 1970. Acute toxicities of organochlorine and organophosphorus insecticides to estuarine fishes. U.S. Bureau of Sport Fisheries and Wildlife, Technical Paper 46.

Henderson, C. and Q.H. Pickering, 1958. Toxicity of organic phosphorus insecticides to fish. Trans. Amer. Fish. Soc., 87: 39.

Karnak, R.E. and W.J. Collins, 1974. The susceptibility to selected insecticides and acetylchlolinesterase activity in a laboratory colony of midge larvae, Chironomus tentans (Diptera: chironomidae). Bull. Environ. Contam. Toxicol., 12: 62.

Katz, M., 1961. Acute toxicity of some organic insecticides to three species of salmonids and to the threespine stickleback. Trans. Amer. Fish Soc. 90: 264.

Macek, K.J. and W.A. McAllister, 1970. Insecticides susceptibility of some common fish family representatives. Trans. Amer. Fish. Soc., 99: 20.

Mount, D.T. and C.E. Stephan, 1967. A method for establishing acceptable toxicant limits for fish-malathion and the butoxyethanol ester of 2,4-D. Trans. Amer. Fish. Soc., 21: 185.

Mulla, M.S. and A.M. Khasawina, 1969. Laboratory and field evaluations of larvicides against chironomid midges. Jour. Econ. Entomol., 62: 37.

Paris, D.F., et al 1975. Rates of degradation of malathion by bacteria isolated from aquatic systems. Environ. Sci. & Technol., 9: 135.

Pickering, Q.H., et al., 1962. The toxicity of organic phosphorus insecticides to different species of warmwater fishes. Trans. Amer. Fish. Soc., 91: 175.

Post, G. and T.R. Schroeder, 1971. The toxicity of four insecticides to four salmonid species. Bull. Environ. contam. Toxicol., 6: 144.

Sanders, H.O., 1969. Toxicity of pesticides to the crustacean, Gammarus lacustris. U.S. Department of the Interior, Washington, D.C., Bureau of Sport Fisheries and Wildlife Technical Paper No. 25.

Sanders, H.O., 1972. Toxicity of some insecticides to four species of malacostracan crustaceans. U.S. Department of the Interior, Washington, D.C. Bureau of Sport Fisheries and Wildlife Technical Paper No. 66.

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

Sanders, H.O. and O.B. Cope, 1968. The relative toxicities of several pesticides to naiads of three species of stonefly. Limnol. & Oceanog., 13: 112.

U.S. Bureau of Sport Fisheries and Wildlife, 1970. Resource Publication 106. Washington, D.C.

Weiss, C.M. and J.H. Gakstatter, 1964. The decay of anticholinesterase of organic phosphorus insecticides on storage in waters of different pH. Advances in Water Pollution Research, 1: 83.

METHOXYCHLOR

CRITERIA:

100 ug/l for domestic water supply (health);

0.03 ug/l for freshwater and marine aquatic life.

RATIONALE:

The highest level of methoxychlor found to have minimal or no long-term effects in man is 2.0 mg/kg of body weight/day (Lehman, 1965). Where adequate human data are available for corroboration of the animal results, the total "safe" drinking water intake level is assumed to be 1/100 of the "no effect" or "minimal effect" level reported for the most sensitive animal tested, in this case, man.

Applying the available data and based upon the assumptions that 20 percent of the total intake of methoxychlor is from drinking water, that the average person weighs 70 kg and consumes 2 liters of water per day, the formula for calculating a criterion is 2.0 mg/kg x 0.2 x 70 kg x 1/100 x 1/2 = 0.14 mg/l. A criterion level for domestic water supply of 100 ug/l is recommended.

Few data are available on acute and chronic effects of methoxychlor on freshwater fish. Merna and Eisele (1973) observed reduced hatchability of fathead minnow, Pimephales promelas, embryos at 0.125 ug/l and lack of spawning at 2.0 ug/l. Yellow perch, Perca flavescens, exposed to 0.6 ug/l for eight months exhibited reduced growth. The 96-hour LC_{50} concentration was 7.5 and 22 ug/l for the fathead minnow and yellow perch, respectively. Korn and Earnest (1974) obtained a 96-hour LC_{50} of 3.3 ug/l with juvenile striped bass, Morone saxatilis, exposed to methoxychlor in a flowing-water bioassay.

Sanders (1972) determined a 96-hour LC_{50} value of 0.5 ug/l for the crayfish, Orconectes nais. Merna and Eisele (1973) obtained a 96-hour LC_{50} value of 0.61 ug/l for the scud, Gammarus pseudolimnaeus and 96-hour LC50s ranging from 1.59 to 7.05 ug/l for the crayfish, Orconectes nais, and three aquatic insect larvae. In 28-day exposures, reduction in emergence of mayflies, Stenonema sp., and in pupation of caddisflies, Cheumatopsyche sp., were observed at 0.5 and 0.25 ug/l concentrations, respectively. They also found methoxychlor to be degraded in a few weeks or less in natural waters.

Eisele (1974) conducted a study in which a section of a natural stream was dosed at 0.2 ug/l methoxychlor for one year. The near extinction of one species of scud, Hyallella azteca, and

reductions in populations of other sensitive species, as well as biomass were observed. Residue accumulation of up to 1,000 times the level in the stream was observed in first-year crayfish, Orconectes nais. Metcalf, et al. (1971) traced the rapid conversion of methoxychlor to water soluble compounds and elimination from the tissues of snails, mosquito larvae, and mosquitofish. Thus, methoxychlor appears to be considerably less bioaccumlative in aquatic organisms than some of the other chlorinated pesticides.

Methoxychlor has a very low accumulation rate in birds and mammals (Stickel, 1973), and relatively low avian (Heath, et al., 1972), and mammalian (Hodge, et al., 1950) toxicities. No administrative guidelines for acceptable levels in edible fish tissues have been established by the U.S. Food and Drug Administration.

The above data indicate that 0.1 ug/l methoxychlor would be just below chronic effect level for the fathead minnow and one-fifth the acute toxicity level in a crayfish species. Therefore, a criterion level of 0.03 ug/l is recommended. This criterion should protect fish as sensitive as striped bass and is ten times lower than the level causing effects on some invertebrate populations in a one-year dosing of a natural stream.

Bahner and Nimmo (1974) found the 96-hour LC_{50} of methoxychlor for the pink shrimp, Penaeus duorarum, to be 3.5 ug/l and the 30-day LC_{50} to be 1.3 ug/l. Using an application factor of 0.01 with the pink shrimp acute toxicity of 3.5 ug/l, the recommended criterion for the marine environment is 0.03 ug/l.

Butler (1971) found accumulation factors of 470 and 1,500 for the molluscs, Mercenaria mercenaria, and Mya arenaria, respectively, when exposed to 1 ug/l methoxychlor for 5 days. Using the 1,500 accumulation factor as a basis, a water concentration of 0.2 ug/l would be required to meet the U.S. Food and Drug Administration's guideline for methoxychlor in meat products. Thus, the recommended marine criterion of 0.03 ug/l is an order of magnitude lower than this concentration.

REFERENCES CITED:

Bahner, L.H. and D.R. Nimmo, 1974. Methods to assess effects of combinations of toxicants, salinity, and temperature on estuarine animals. Proc. 9th Am. Confr. on Trace Substances in Env. Health, Univ. Miss. Columbia, Mo.

Butler, P.A. 1971. Influence of pesticides on marine ecosystems. Proc. Royal Soc. Lond., 177:321.

Eisele, P.J., 1974. The effects of methoxychlor on aquatic invertebrate populations and communities. Ph.D. Thesis, Univ. of Michigan, Ann Arbor, 151 p.

Heath, R.G., et al., 1972. Comparative dietary toxicities of pesticides to birds. Bureau of Sport Fisheries and Wildlife, Wildlife Report No. 152, U.S. Dept. of the Interior, Washington, D.C., 57 p.

Hodge, H.C., et al., 1950. Short-term oral toxicity tests of methoxychlor in rats and dogs. Jour. Pharmacol. Exp. Therapy, 99:140.

Korn, S. and R. Earnest, 1974. Acute toxicity of twenty insectioides to striped bass, Morone saxatilis. Calif. Fish and Game, 60:128.

Lehman, A.J., 1965. Summaries of pesticide toxicity. Assoc. of Food and Drug Officials of the U.S., Topeka, Kansas, pp. 1-40.

Merna, J.W. and P.J. Eisele, 1973. The effects of methoxychlor on aquatic biota. U.S. EPA Ecological Res. Series, No. EPA- R3-73-046. U.S. Government Printing Office, Washington, D.C.

Metcalf, R.L., et al., 1971. Biodegradable analogs of DDT. Bull. World Health Organization, 44:363.

Sanders, H.O., 1972. Toxicity of some insecticides to four species of malacostracan crustaceans. Tech. Paper No. 66, Bureau of Sport Fisheries and Wildlife. U.S. Government Printing Office, Washington, D.C.

Stickel, L.F., 1973. Pesticide residues in birds and mammals. In: Environmental pollution by pesticides. (C.A. Edwards, Ed.) Plenum Press, New York, pp. 254-312.

MIREX

CRITERION:

0.001 ug/l for freshwater and marine aquatic life.

RATIONALE:

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

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

Procambarus blandingi juveniles were exposed to 1 or 5 ug/l for 6 to 144 hours, transferred to clean water and observed for 10 days. After 5 days in clean water, 95 percent of the animals exposed to 1 ug/l for 144 hours were dead. Exposure to 5 ug/l for 6, 24, and 58 hours resulted in 26, 50, and 98 percent mortality 10 days after transfer to clean water.

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

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

Mirex residues in the fish at termination of the experiment ranged from 0.015 ug/g (ppm) in the fillet to 0.255 ug/g in the fat.

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

In laboratory and field test systems reported concentrations of mirex usually are between 0.5 and 1.0 ug/l (Van Valin, et al., 1968; Ludke, et al., 1971). Although mirex seldom is found above 1 ug/l in the aquatic environment, several field studies have shown that the insecticide is accumulated through the food chain. Borthwick, et al. (1973) reported the accumulation of mirex in South Carolina estuaries. Their data revealed that mirex was transported from treated land and marsh to the estuary animals and that accumulation, especially in predators, occurred. In the test area, water samples consistently were less than 0.01 ug/l. Residues in fish varied from non-detectable to 0.8 ug/g with 15 percent of the samples containing residues. The amount of mirex and the percent of samples containing mirex increased at higher trophic levels. Fifty-four percent of the raccoons sampled contained mirex residues up to 4.4 ug/g

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

Mirex does not appear to be greatly toxic to birds, with LC50's for the young of four species ranging from 547 to greater than 1667 ug/g (Heath, et al., 1972). Long-term dietary dosages caused no adverse effect at 3 ug/g with mallards and 13 ug/g with pheasants (Heath and Spann, 1973). However, it has been reported (Stickel, et al., 1973) that the persistence of mirex in bird tissue exceeds that of all organochlorine compounds tested except for DDE. Delayed mortality occurred among birds subjected to doses above expected environmental concentration.

A summary examination of the data available at this time shows a mosaic of effects. Crayfish and channel catfish survival is affected by mirex in the water or by ingestion of the bait particles. Bioaccumulation is well established for a wide variety of organisms but the effect of this bioaccumulation on the aquatic ecosystem is unknown. There is evidence that mirex is very persistent in bird tissue. Considering the extreme toxicity and potential for bioaccumulation, every effort should be made to keep mirex bait particles out of water containing aquatic organisms and

water concentrations should not exceed 0.001 ug/l mirex. This value is based upon an application factor of 0.01 applied to the lowest levels at which effects on crayfish have been observed.

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

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

REFERENCES CITED:

Borthwick, P.W., et al., 1973. Accumulation and movement of mirex in selected estuaries of South Carolina, 1969-71. Pesticides Monit. Jour., 7:6.

Bookhout, C.G., et al., 1972. Effects ofmirex on the larval development of two crabs. Water, Air, and Soil Pollution, 1:165.

Heath, R.G. and J.W. Spann, 1973. Pesticides in the environment: A continuing controversy. Intercont. Med. Book Corp., N.Y., pp.421-435.

Heath, R.G., et al., 1972. Comparative dietary toxicities of pesticides to birds. Bureau of Sport Fisheries and Wildlife, Wildlife Report No. 152, U.S. Dept. of the Interior, Washington, D. C., 57 p.

Hyde, K.M., et al., 1974. The effect of mirex on channel catfish production. Trans. Amer. Fish Soc., 103:366.

Kaiser, K.L.E., 1974. Mirex: an unrecognized contaminant of fishes from Lake Ontario. Science 185:523.

Lowe, J.I., et al., 1971. Effects of mirex on selected estuarine organisms. In: Transactions of the 36th North American Wildlife Resources Conference. pp. 171-186.

Ludke, J.L., et al., 1971. Toxicity of mirex to crayfish, Procambarus blandingi. Bull. Environ. Contam. Toxicol., 6:89.

Naqvi, S.M. and A. de la Cruz, 1973. Mirex incorporation in the environment: Residues in non-target organisms,1972. Pesticides Monit. Jour., 7:104.

Stickel, W.H., et al., 1973. Pesticides and the environment: A continuing controversy. Intercont. Med. Book Corp., N.Y., pp. 437-467.

Tagatz, M.E., et al. (1975). Seasonal effects of leached mirex on selected estuarine animals. Arch. Environ. Contam. Toxicol. 3:371.

Van Valin, C., et al., 1968. Some effects of mirex on two warm water fishes. Trans. Amer. Fish. Soc., 97:185.

Wolfe, J.L. and B.R. Norment, 1973. Accumulation of mirex residues in selected organisms after an aerial treatment, Mississippi, 1971-72. Pesticides Monit. Jour., 7:112.

PARATHION

CRITERION:

0.04 ug/l for freshwater and marine aquatic life.

RATIONALE:

Acute static LC_{50} values of the organophosphorus pesticide, parathion, for freshwater fish have ranged generally from about 50 ug/l for more sensitive species such as bluegills, Lepomis macrochirus, to about 2.5 mg/l for the more resistant species such as minnows (U.S. Environmental Protection Agency, 1975). In flowing water exposures, Spacie (1975) obtained 96-hour LC_{50} values of 0.5 mg/l, 1.6 mg/l, and 1.76 mg/l for bluegills, Lepomis macrochirus, fathead minnows, Pimephales promelas, and brook trout, Salvelinus fontinalis, respectively. Korn and Earnest (1974) found a 96-hour LC_{50} of 18 ug/l for juvenile freshwater and estuarine striped bass Morone saxatilis, in a flowing water system.

Few chronic exposure data are available for aquatic organisms. Brown bullheads, Ictalurus nebulosus, exposed to 30 ug/l parathion for 30 days exhibited tremors; at 60 ug/l they convulsed and were found to have developed a deformed vertebral column (Mount and Boyle, 1969). In a 23-month exposure of bluegills, Spacie (1975) observed deformities (scoliosis and a characteristic protrusion in the throat region) at 0.34 ug/l, but not at 0.16 ug/l. Tremors, convulsions, hypersensitivity, and hemorrhages also were evident at higher concentrations.

Reproductive impairment and deformities were observed in fathead minnows exposed to 4.0 ug/l for 8 1/2 months. Development of brook trout, S. fontinalis embryos exposed to 32 ug/l was abnormal and mortalities associated with premature hatching were observed. Embryos at 10 ug/l appeared normal. No adverse effect on juveniles and adults was evident during 9 months exposure to 7 ug/l.

Inhibition of cholinesterase enzymes is the well established mode of physiological action of parathion and other organic phosphorous pesticides (Weiss, 1958). The degree of inhibition of brain acetylcholinesterase (AChE) activity has been the most frequently used measure of effect of these pesticides. Various studies (Weiss, 1958, 1959, 1961; Murphy et al., 1968; Gibson et al., 1969) have shown the degree of inhibition to be dependent upon toxicant concentration, length of exposure, and species sensitivity. The results of these studies have also indicated that death results from AChE inhibition ranging from 25 to 90 percent of normal. Weiss (1959) also showed that susceptibility depended upon the extent of recovery of AChE activity following prior exposure and that the recovery period for fish exposed to parathion was relatively long. In bluegills, AChE activity was only 50 percent recovered 30 days after exposure to 1 mg/l for 6 to 7 hours (Weiss, 1961).

Some of the other physiological effects observed to result from exposure of fish to parathion have been inhibition of spermatogenesis in guppies Poecilia reticulata, at 10 ug/l (Billard and deKinkelin, 1970), alternation of oxygen consumption rate in bluegills, Lepomis macrochirus at 100 ug/l (Dowden, 1966), and liver enlargement associated with increased pesticide-hydrolizing capability in mosquitofish, Gambusia affinis, (Ludke, 1970).

Parathion has been found acutely toxic to aquatic invertebrates at under one microgram per liter, e.g., a 50-hour LC_{50} of 0.8 ug/l for Daphnia magna, 48-hour LC_{50} of 0.6 ug/l for Daphnia pulex, and 48-hour LC_{50} of 0.37 for Simocephalus serrulatus (a daphnid) (Sanders and Cope, 1966); a 5-day LC_{50} of 0.93 ug/l for the larval stonefly, Acroneuria pacifica (Jensen and Gaufin, 1964); and a 96-hour LC_{50} of 0.43 ug/l for the larval caddisfly, Hydropsyche californica (Gaufin et al., 1965). Mulla and Khasawinah (1969) obtained a 24-hour LC_{50} of 0.5 ug/l for 4th instar larvae of the midge Tanypus grodhausi. Spacie (1975) obtained 96-hour LC_{50}'s in flow-through bioassays of 0.62 ug/l for Daphnia magna, 0.40 ug/l for the scud, Gammarus fasciatus, and 31.0 ug/l for 4th instar of Chironomous tentans, a midge. Other invertebrates have been found acutely sensitive to parathion in concentrations of from 1 to 30 ug/l in water (U. S. Environmental Protection Agency, 1975).

Few longer exposures have been conducted. Jensen and Gaufin (1964) obtained 30-day LC_{50}'s for Pteronarcys californica and Acroneuria pacifica of 2.2 and 0.44 ug/l, respectively. Spacie (1975) found the three-week LC_{50} for Daphnia magna to be 0.14 ug/l. Statistically significant reproductive impairment occurred at concentrations above 0.08 ug/l. A 43-day LC_{50} of 0.07 ug/l was reported for Gammarus fasciatus and a concentration of 0.04 ug/l produced significantly greater mortality than among controls.

Limited information is available on persistence of parathion in water. Eichelberger and Lichtenberg (1971) determined the half-life in river water (pH 7.3 - 8.0) to be one week. Using AChE inhibitory capacity as the indicator, Weiss and Gakstatter (1964) found the half-life of parathion

or its active breakdown products to be 40, 35, and 20 days in "natural" waters having a pH of 5.1, 7.0, and 8.4, respectively. The possibility of breakdown resulting in compounds more toxic than parathion was suggested by Burke and Ferguson (1969) who determined that the toxicity of this pesticide to mosquitofish, Gambusia affinis, was greater in static than in flowing water test systems. Sanders (1972), in 96-hour bioassays with the scud, Gammarus fasciatus, and glass shrimp, Palaemonetes kadiakensis, also observed greater toxicity under static than in flow-through conditions.

Tissue accumulations of parathion by exposed aquatic organisms are not great and do not appear to be very persistent. Mount and Boyle (1969) observed concentrations in the blood of bullhead, Ictalurus melas, up to about 50 times water concentrations. Spacie (1975) found muscle concentrations in chronically exposed brook trout, S. fontinalis to be several hundred times water concentrations; bluegills, Lepomis macrochirus, had about 25 times water concentrations in their bodies. Leland (1968) demonstrated a biological half-life of parathion in rainbow trout, Salmo gairdneri, exposed and then placed in fresh water to be only 30 to 40 hours. It is not expected that parathion residues in aquatic organisms exposed to the recommended criterion concentrations will be a hazard to consumer organisms.

Weiss and Gakstatter (1964) have shown that 15-day continuous exposure to parathion (1.0 ug/l) can produce progressively greater (i.e., cumulative) brain AChE inhibition in a fish species. After substantial inhibition by parathion exposure, it takes several weeks for brain AChE of exposed fishes to return to normal even though exposure is discontinued (Weiss 1959, 1961). Inhibition of brain AChE of fishes by 46 percent or

more has been associated with harmful effects in exposures to organophosphate pesticides for one life cycle (Eaton, 1970) and for short periods (Carter, 1971; Coppage and Duke, 1971; Coppage, 1972; Coppage and Matthews, 1974; Post and Leasure, 1974; Coppage et al., 1975). It has been shown that a concentration of 10 ug parathion/l of flowing sea water kills 40 to 60 percent of the marine fishes Lagodon rhomboides (pinfish) and Leostomus xanthurus (spot) in 24 hours and causes about 87 to 92 percent brain AChE inhibition (Coppage and Matthews, 1974). Similar inhibition of AChE and mortality were caused in sheepshead minnows, Cyprinodon variegatus, in 2, 24, 48, and 72 hours at concentrations of 5000, 2000, 100, and 10 ug/l, respectively in static tests (Coppage, 1972). These data indicate that reductions of brain AChE activity of marine fishes by 70 to 80 percent or more in short-term exposures to parathion may be associated with some deaths.

Other estimates of parathion toxicity to marine organisms follow. The 48-hour EC_{50} for parathion to Penaeus duorarum was found to be 0.2 ug/l (Lowe et al., 1970). Lahav and Sarig (1969) reported the 96-hour LC_{50} for mullet, Mugil cephalus, to be 125 ug/l. The shell growth of the oyster, Crassostrea virginica, was found by Lowe et al. (1970) to be decreased by 22 percent after 96 hours in 1.0 mg/l.

An application factor of 0.1 is applied to the 96-hour LC50 data for invertebrates which range upward from 0.4 ug/l. A criteria of 0.04ug/l is recommended for marine and freshwater aquatic life.

REFERENCES CITED:

Billard, R. and deKinkelin, 1970. Sterilization of the testicles of guppies by means of non-lethal doses of parathion. Annales D Hydrobiologie, 1(1):91.

Burke, W.D. and D. E. Ferguson, 1969. Toxicities of four insecticides to resistant and susceptible mosquitofish in static and flowing solutions. Mosquito News, 29(1):96.

Carter, F. L., 1971. In vivo studies of brain acetylcholinesterase inhibition by organophosphate and carbamate insecticides in fish. Ph.D. dissertation, Louisiana State University, Baton Rouge, La.

Coppage, D. L., 1972. Organophosphate pesticides: specific level of brain ACHE inhibition related to death in sheepshead minnows. Trans. Amer. Fish. Soc., 101:534.

Coppage, D. L. and T. W. Duke, 1971. Effects of pesticides in estuaries along the Gulf and Southeast Atlantic Coasts. In: Proceedings of the 2nd Gulf Coast Conference on Mosquito Suppression and Wildlife Management (C. H. Schmidt, ed.) National Mosquito Control - Fish and Wildlife Management Coordinating Committee, Washington, D. C.

Coppage, D. L. and E. Matthews, 1974. Short-term effects of organophosphate pesticides on cholinesterases of estuarine fishes and pink shrimp. Bull. Environ. Contam. Toxicol., 11:483.

Coppage, D. L. et al., 1975. Brain acetylcholinesterase inhibition in fish as a diagnosis of environmental poisoning by malathion, O, O-dimethyl S-(1, 1-dicarbethoxy-ethyl) phosphorodithioate. Pesticide Biochemistry and Physiology (in press).

Dowden, B. F., 1966. Effects of five insecticides on the oxygen consumption of the bluegill sunfish, Lepomis macrochirus. Ph.D. Thesis, Louisiana State University, Baton Rouge, La.

Eaton, J. G., 1970. Chronic malathion toxicity to the bluegill, Lepomis macrochirus, Rafinesque). Water Research. 4:673.

Eichelberger, J. W. and J. J. Lichtenberg, 1971. Persistence of pesticides in river water. Environ. Sci. and Technol., 5:541.

Gaufin, A. R., et al., 1965. Toxicity of ten organic insecticides to various aquatic invertebrates. Water and Sew. Works, 112:276.

Gibson, J. R., et al., 1969. Sources of error in the use of fish-brain acetylcholinesterase activity as a monitor for pollution. Bull. Environ. Contam. Toxicol., 4:17.

Jensen, L. D. and A. R. Gaufin, 1964. Long-term effects of organic insecticides on two species of stonefly naiads. Trans. Amer. Fish. Soc., 93:357.

Korn, S. and R. Earnest, 1974. Acute toxicity of twenty insecticides to striped bass, Morone saxatilis. Calif. Fish and Game., 60:128.

Lahav, M. and S. Sarig, 1969. Sensitivity of pond fish to cotnion (azinphosmethyl) and parathion. Bamidgeh, Bull. Fish. Cult. Israel, 21:67.

Leland, H. V. II, 1968. Biochemical factors affecting toxicity of parathion and selected analogs to fishes. University of Michigan, Ann Arbor, Michigan.

Lowe, J. I., et al., 1970. Laboratory bioassays. In: Progress Report for Fiscal Year 1969, Pesticide Field Station, Gulf Breeze, Florida. U. S. Fish. Wildl. Serv. Circ. 335.

Ludke, J. L., 1970. Mechanism of resistance to parathion in mosquitofish, Gambusia affinis. Ph.D. Thesis, Mississippi State University, University, Mississippi.

Mount, D. I. and H. W. Boyle, 1969. Parathion - Use of blood concentration to diagnose mortality of fish. Environ. Sci. and Technol., 3:1183.

Mulla, M. S. and A. M. Khasawinah, 1969. Laboratory and field evaluation of larvicides against Chironomic midges. Jour. Econ. Entomol., 62:37.

Murphy, S. D., et al., 1968. Comparative anticholinesterase action of organophosphorus insecticides in invertebrates. Toxicol. Appl. Pharmacol., 12:22.

Post, G. and R. A. Leasure, 1974. Sublethal effect of malathion to three salmonid species. Bull. Environ. Contam. Toxicol., 12:312.

Sanders, H. O., 1972. Toxicity of some inecticides to four species of malacostracan crustaceans. U. S. Department of the Interior, Washington, D. C., Bureau of Sport Fisheries and Wildlife, Technical Paper No. 66.

Sanders, H. O. and O. B. Cope, 1966. Toxicities of several pesticides to two species of Cladocerans. Trans. Amer. Fish. Soc., 95:165.

Spacie, A., 1975. Acute and chronic parathion toxicity to fish and invertebrates. U. S. Environmental Protection Agency, EPA Ecological Research Series. (In preparation).

U. S. Environmental Protection Agency, 1975. Initial scientific and minieconomic review of parathion. U. S. Environmental Protection Agency, Office of Pesticides Programs, Report. No. EPA-540/1-75-001. National Technical Information Service, Springfield, Virginia.

Weiss, C. M., 1958. The determination of cholinesterase in the brain tissue of three species of freshwater fish and its inactivation in vivo. Ecology, 39:194.

Weiss, C. M., 1959. Response of fish to sublethal exposures of organic phosphorus insecticides. Sew. and Ind. Wastes, 31:580.

Weiss, C. M., 1961. Physiological effect of organic phosphorus insecticides on several species of fish. Trans. Amer. Fish. Soc., 90:143.

Weiss, C. M. and J. H. Gakstatter, 1964. The decay of anticholinesterase activity of organic phosphorus insecticides on storage in waters of different pH. Advances in Water Poll. Res., 1:83.

TOXAPHENE

CRITERIA:

5 ug/l for domestic water supply (health);

0.005 ug/l for freshwater and marine aquatic life.

RATIONALE:

The highest level of toxaphene found to have minimal or no long-term effects in the most sensitive mammal tested, the dog, is 10.0 mg/kg in the diet or 1.7 mg/kg of body weight/day (Lehman, 1965). Where adequate human data are not available for corroboration of the animal results, the total "safe" intake level is assumed to be 1/500 of the "no effect" or "minimal effect" level reported for the most sensitive animal tested.

Applying the available data and based upon the assumption that 20 percent of the total intake of toxaphene is from drinking water, that the average person weighs 70 kg and consumes 2 liters of water per day, the formula for calculating a criterion is 1.7 mg/kg X 0.2 X 70 kg X 1/500 X 1/2 =0.024 mg/l. However, at 0.024 mg/l there is an organoleptic effect which has been shown to occur at the level of 0.005 mg/l (Cohen, et al., 1961). Thus the criterion is set at 5 ug/l.

Macek and McAllister (1970) reported 96-hour LC50 values for 12 fish species ranging from 2 ug/l for largemouth bass, Micropterus

salmoides, to 18 ug/l for bluegill, Lepomis macrochirus. Mahdi (1966) reported 96-hour LC50 values as low as 1.8 ug/l for black bullhead, Ictalurus melas, and as high as 50 ug/l for goldfish, Carrassius auratus, and Henderson, et al. (1959) reported 96-hour TLm values from 3.5 ug/l for bluegill to 20 ug/l for guppies, Poecilia reticulata. The 96-hour LC_{50} values for three stonefly species, Pteronarcys californica, Pteronarcella badia, and Claassenia sabulosa, were reported by Sanders and Cope (1968) to range from 1.3 to 3.0 ug/l.

In a chronic continuous flow bioassay with brook trout, Salvelinus fontinalis, Mayer, et al. (1975) found that toxaphene in water at a level of 0.039 ug/l adversely affected the growth and development of brook trout fry. The mortality of fish at this level was significantly greater than the controls, indicating a no-effect level of less than 0.039 ug/L. The 96-hour LC_{50} of toxaphene for 16-month-old trout was 10.8 ug/l. Mehrle and Mayer (1975) conducted a series of long-term studies on the fathead minnow, Pimephales promelas, exposing the organisms for 150 days to concentrations of toxaphene as low as 0.055 ug/l in a flow-through system. Their results confirmed the Mayer, et al. (1975) work, showing that growth of all fish exposed to concentrations of 0.055 ug/l was significantly reduced. A no-effect level for this freshwater fish would be less than 0.055 ug/l.

Hughes (1968) reported that lakes treated with toxaphene concentrations ranging from 40 to 150 ug/l remained toxic to fish for periods of a few months to five years. Terriere, et al. (1966) reported that a lake treated with toxaphene as a piscicide remained toxic to fish for at least five years. Bioconcentration factors of toxaphene were 500 for aquatic plants, 1,000 to 2,000 for aquatic animals other than fish, and 10,000 to 20,000 for rainbow trout in the lake. Mayer, et al. (1975) observed accumulations of 5,000 to 21,000 times water concentrations in brook trout exposed only through the water. Accumulation factors of 3,400 to 17,000 from aqueous solution have been reported for bacteria, algae and fungi (Paris, et al., 1975).

Heath, et al. (1972) reported the 5-day dietary LC_{50} to be 96 to 142 ppm for young birds of four species. No reproductive impairment occurred in mallards resulting from a long-term dietary dosage of 7 ppm and starlings tolerated 45 ppm for long periods (Patuxent Wildlife Research Center, Laurel, Maryland, unpublished data). At water levels known to affect fish (0.04 ug/l, Mayer, et al., 1975) and with accumulation factors similar to those cited above (20,000 times), resulting residue levels (0.04 x 20,000= 0.8 ppm) would not be expected to approach dosage levels known to be a hazard to birds.

No guideline for toxaphene has been set by the U.S. Food and Drug Administration as a residue limit for edible tissues of fish for human consumption.

Lowe (1964) reported a 24-hour LC_{50} of 3.2 ug/l, a 48-hour LC_{50} of 1.0 ug/l, and a 144-hour LC_{50} of 0.5 ug/l for the spot, Leiostomus xanthurus. Butler (1963) reported a 90.8 percent decrease in productivity of natural phytoplankton communities during a 4-hour exposure to a concentration of 1,000 ug/l of toxaphene. Lowe also reported a 48-hour EC_{50} of 4.9 ug/l for the brown shrimp, Penaeus aztecus; and a 48-hour EC_{50} of 330 ug/l for juvenile blue crabs, Callinectes sapidus. A concentration of 57 ug/l resulted in a 50 percent decrease in oyster, Crassostrea virginica, shell growth after 96 hours of exposure at a water temperature of 31° C and 24 o/oo salinity, whereas 63 ug/l produced the same effect at 19° C and 19 o/oo salinity. The 48-hour LC_{50} for the juvenile white mullet, Mugil curema, was 5.5 ug/l.

Korn and Earnest (1974) report a 96-hour LC_{50} for the striped bass, Morone saxatilis of 4.4 ug/l. The 96-hour LC_{50} for the pinfish, Lagodon rhomboides, an organism of wide geographic distribution and ecological importance (Caldwell, 1957), has been reported as 0.5 ug/l (Schimmel, et al., in preparation). While the use of an application factor of 0.01 has been recommended by the NAS-NAE (NAS, 1974) its use is especially appropriate in the case of toxaphene because long-term studies with fathead minnows, Pimephales promelas (Mehrle and Mayer, 1975), and brook trout, Salvelinus fontinalis (Mayer, et al., 1975), have failed to establish a no-effect level. Application of the 0.01 factor to the 96-hour LC_{50} for the pinfish yields a marine criterion of 0.005 ug/l.

A no-effect level was not achieved in the studies using freshwater organisms. Mortality and adverse physiological and physical effects were detected at the lowest concentration used, 0.039 ug/l. Hence, for toxaphene, the use of the same criterion for both marine and freshwater is recommended.

REFERENCES CITED:

Butler, P.A., 1963. Pesticide-wildlife studies. A review of Fish and Wildlife Service Investigations during 1961 and 1962. U.S. Dept. of the Interior, Fish and Wildl. Ser., Circ. 167.

Caldwell, D.K. 1957. The biology and systematics of the pinfish, Lagodon rhomboides (Dinmaeus). Bull. Florida State Mus. 2:1.

Cohen, J.M., et al., 1961. Effects of fish poisons on water supplies. Jour. Amer. Water Works Ass., 53:49.

Heath, R.G., et al., 1972. Comparative dietary toxicity of pesticides to birds. U.S. Dept. of the Interior, Bur. of Sport Fish. and Wildl., Washington, D.C., Wildlife Rept. No. 152.

Henderson, D., et al., 1959. Relative toxicity of ten chlorinated hydrocarbon insecticides to four species of fish. Trans. Amer. Fish. Soc., 88:23.

Hughes, R.A., 1968. Persistence of toxaphene in natural waters. M.S. Thesis, University of Washington, Seattle.

Korn, S. and R. Earnest, 1974. Acute toxicity of twenty insecticides to striped bass, Morone saxatilis. Calif. Fish and Game, 60:128.

Lehman, A.J., 1965. Summaries of pesticide toxicity. Assn. of Food and Drug Officials of the U.S., Topeka, Kansas, pp. 1-40.

Lowe, J.I., 1964. Chronic exposure of the spot, Leiostomus xanthurus, to sublethal concentrations of toxaphene in seawater. Tran. Amer. Fish Soc. 93:396.

Macek, K.J. and W.A. McAllister, 1970. Insecticide susceptibility of some common fish family representatives. Trans. Amer. Fish. Soc., 1:20.

Mahdi, M.A., 1966. Mortality of some species of fish to toxaphene at three temperatures. U.S. Dept. of the Interior. Bur. of Sport Fish. and Wildl., Investigation in Fish Control, 6:1.

Mayer, F.L., Jr., et al., 1975. Toxaphene effects on reproduction, growth and mortality of brook trout. Fish Pesticide Research Laboratory, Columbia, Mo., U.S. Environmental Protection Agency Contract No. EPA-IAG-0153.

Mehrle, P.M. and F.L. Mayer, 1975. Toxaphene effects on growth and bone composition of fathead minnow Pimephales promelas Jour. Fish. Res. Bd. Canada. 32:593.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

Paris,D.F., et al., 1975. Microbial degradation and accumulation of pesticides in aquatic systems. U.S. Environmental Protection Agency, Washington D.C., EPA-660/3-75-007.

Patuxent Wildlife Research Center, 1974. Toxaphene research data. U.S. Dept of Interior, Bur. of Sport Fish. and Wildlife, (Unpublished).

Sanders, H.O. and O.B. Cope, 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. Oceanog., 13:112.

Schimmel, S.C., et al, (in preparation). Uptake and toxicity of toxaphene in several estuarine organisms. Gulf Breeze Enviromental Research Laboratory, contribution No. 269.

Terriere, L.C., et al., 1966. The persistence of toxaphene in lake water and its uptake by aquatic plants and animals. Jour. Agr. Food Chem., 14:66.

pH

CRITERIA:

Range	
5 - 9	Domestic water supplies (welfare);
6.5 - 9.0	Freshwater aquatic life;
6.5 - 8.5*	Marine aquatic life.

INTRODUCTION:

"pH" is a measure of the hydrogen ion activity in a water sample. It is mathematically related to hydrogen ion activity according to the expression: $pH = -\log 10 (H^+)$, where (H^+) is the hydrogen ion activity.

The pH of natural waters is a measure of acid-base equilibrium achieved by the various dissolved compounds, salts and gases. The principal system regulating pH in natural waters is the carbonate system which is composed of carbon dioxide (CO_2), carbonic acid (H_2CO_3), bicarbonate ion (HCO_3^-), and carbonate ions ($CO_3^=$). The interactions and kinetics of this system have been described by Stumm and Morgan (1970).

pH is an important factor in the chemical and biological systems of natural waters. The degree of dissociation of weak acids or bases is affected by changes in pH. This effect is important because the toxicity of many compounds is affected by the degree of dissociation. One such

---

*. . . but not more than 0.2 units outside of normally occurring range.

example is hydrogen cyanide (HCN). Cyanide toxicity to fish increases as the pH is lowered because the chemical equilibrium is shifted toward an increased concentration of HCN. Similar results have been shown for hydrogen sulfide (H_2S) (Jones, 1964).

The solubility of metal compounds contained in bottom sediments or as suspended material also is affected by pH. For example, laboratory equilibrium studies under anaerobic conditions indicated that pH was an important parameter involved in releasing manganese from bottom sediments (Delfino and Lee, 1971).

The pH of a water does not indicate ability to neutralize additions of acids or bases without appreciable change. This characteristic, termed "buffering capacity," is controlled by the amounts of alkalinity and acidity present.

RATIONALE:

Knowledge of pH in the raw water used for public water supplies is important because without adjustment to a suitable level, such waters may be corrosive and adversely affect treatment processes including coagulation and chlorination.

Coagulation for removal of colloidal color by use of aluminum or iron salts generally has an optimum pH range of 5.0 to 6.5 (Sawyer, 1960). Such optima are predicated upon the availability of sufficient alkalinity to complete the chemical reactions.

The effect of pH on chlorine in water principally is on the equilibrium between hypochlorous acid (HOCl) and the hypochlorite ion (OCl^-) according to the reaction:

$$HOCl = H^+ + OCl^-$$

Butterfield (1948) has shown that chlorine disinfection is more effective at values less than pH 7. Another study (Reid and Carlson, 1974) has indicated, however, that in natural waters no significant difference in the kill rate for Escherichia coli was observed between pH 6 and pH 8.

Corrosion of plant equipment and piping in the distribution system can lead to expensive replacement as well as the introduction of metal ions such as copper, lead, zinc and cadmium. Langelier (1936) developed a method to calculate and control water corrosive activity that employs calcium carbonate saturation theory and predicts whether the water would tend to dissolve or deposit calcium carbonate. By maintaining the pH at the proper level, the distribution system can be provided with a protective calcium carbonate lining which prevents metal pipe corrosion. Generally, this level is above pH 7 and frequently approaches pH 8.3, the point of maximum bicarbonate/carbonate buffering.

Since pH is relatively easily adjusted prior to and during water treatment, a rather wide range is acceptable for waters serving as a source of public water supply. A range of pH from 5.0 to 9.0 would provide a water treatable by typical (coagulation, sedimentation, filtration and chlorination) treatment plant processes. As the range is extended, the cost of neutralizing chemicals increases.

A review of the effects of pH on freshwater fish has been published by the European Inland Fisheries Advisory Commission (EIFAC, 1969). The Commission concluded: "There is no definite pH range within which a fishery is unharmed and outside which it is damaged, but rather, there is a gradual deterioration as the pH values are further removed from the normal range. The pH range which is not directly lethal to fish is 5 - 9; however, the toxicity of several common pollutants is markedly affected by pH changes within this range, and increasing acidity or alkalinity may make these poisons more toxic. Also, an acid discharge may liberate sufficient CO_2 from bicarbonate in the water either to be directly toxic, or to cause the pH range 5 - 6 to become lethal."

Mount (1973) performed bioassays on the fathead minnow, Pimephales promelas, for a 13-month, one generation time period to determine chronic pH effects. Tests were run at pH levels of 4.5, 5.2,

pH Range	Effect on Fish*
5.0 - 6.0	Unlikely to be harmful to any species unless either the concentration of free CO_2 is greater than 20 ppm, or the water contains iron salts which are precipitated as ferric hydroxide, the toxicity of which is not known.
6.0 - 6.5	Unlikely to be harmful to fish unless free carbon dioxide is present in excess of 100 ppm.
6.5 - 9.0	Harmless to fish, although the toxicity of other poisons may be affected by changes within this range.

* EIFAC, 1969.

5.9, 6.6, and a control of 7.5. At the two lowest pH values (4.5 and 5.2) behavior was abnormal and the fish were deformed. At pH values less than 6.6, egg production and egg hatchability were reduced when compared with the control. It was concluded that a pH of 6.6 was marginal for vital life functions.

Bell (1971) performed bioassays with nymphs of caddisflies (two species), and stoneflies (four species), dragonflies (two species), and mayflies (one species). All are important fish food organisms. The 30-day TL_{50} values ranged from 2.45 to 5.38 with the caddisflies being the most tolerant and the mayflies the least tolerant. The pH values at which 50 percent of the organisms emerged ranged from 4.0 to 6.6 with increasing percentage emergence occurring with the increasing pH values.

Based on present evidence, a pH range of 6.5 to 9.0 appears to provide adequate protection for the life of freshwater fish and bottom dwelling invertebrate fish food organisms. Outside of this range, fish suffer adverse physiological effects increasing in severity as the degree of deviation increases until lethal levels are reached.

Conversely, rapid increases in pH can cause increased NH_3 concentrations which are also toxic. Ammonia has been shown to be ten times as toxic at pH 8.0 as at pH 7.0 (EIFAC, 1969).

The chemistry of marine waters differs from that of fresh water because of the large concentration of salts present. In addition to alkalinity based on the carbonate system, there is also alkalinity from other weak acid salts such as borate. Because of the buffering system present in sea water, the naturally occurring variability of pH is less than in fresh water. Some marine communities are more sensitive to pH change than others (NAS, 1974). Normal pH values in sea water are 8.0 to 8.2 at the surface decreasing to 7.7 to 7.8 with increasing depth (Capurro, 1970). The NAS Committee's review (NAS, 1974) indicated that plankton and benthic invertebrates are probably more sensitive than fish to changes in pH and that mature forms and larvae of oysters are adversely affected at the extremes of the pH range of 6.5 to 9.0. However, in the shallow, biologically active waters in tropical or subtropical areas, large diurnal pH changes occur naturally because of photosynthesis. pH values may range from 9.5 in the daytime to 7.3 in the early morning before dawn. Apparently these communities are adapted to such variations or intolerant species are able to avoid extremes by moving out of the area.

For open ocean waters where the depth is substantially greater than the euphotic zone, the pH should not be changed more than 0.2 units

outside of the naturally occurring variation or in any case outside the range of 6.5 to 8.5. For shallow, highly productive coastal and estuarine areas where naturally occurring pH variations approach the lethal limits for some species, changes in pH should be avoided but in any case not exceed the limits established for fresh water, i.e., pH of 6.5 to 9.0. As with the freshwater criteria, rapid pH fluctuations that are due to waste discharges should be avoided. Additional support for these limits is provided by Zirino and Yamamoto (1972). These investigators developed a model which illustrates the effects of variable pH on copper, zinc, cadmium, and lead; small changes in pH cause large shifts in these metallic complexes. Such changes may affect toxicity of these metals.

For the industrial classifications considered, the NAS report (NAS, 1974) tabulated the range of pH values used by industry for various process and cooling purposes. In general, process waters used varied from pH 3.0 to 11.7, while cooling waters used varied from 5.0 to 8.9. Desirable pH values are undoubtedly closer to neutral to avoid corrosion and other deleterious chemical reactions. Waters with pH values outside these ranges are considered unusable for industrial purposes.

The pH of water applied for irrigation purposes is not normally a critical parameter. Compared with the large buffering capacity of the soil matrix, the pH of applied water is rapidly changed to approximately that of the soil. The greatest danger in acid soils is that metallic ions such as iron, manganese or aluminum may be dissolved

in concentrations which are subsequently directly toxic to plants. Under alkaline conditions, the danger to plants is the toxicity of sodium carbonates and bicarbonates either directly or indirectly (NAS, 1974).

To avoid undesirable effects in irrigation waters, the pH should not exceed a range of 4.5 to 9.0.

REFERENCES CITED:

Bell, H.L., 1971. Effect of low pH on the survival and emergence of aquatic insects. Water Res., 5:313.

Butterfield, C.T., 1948. Bacterial properties of free and combined available chlorine. Jour. Amer. Water Works Assn., 40:1305.

Capurro, L.R.A., 1970. Oceanography for Practicing Engineers. Barnes and Noble, Inc., New York.

Delfino, J.J. and G.F. Lee, 1971. Variation of manganese, dissolved oxygen and related chemical parameters in the bottom waters of Lake Mendota, Wisconsin. Water Res., 5:1207.

European Inland Fisheries Advisory Commission, 1969. Water quality criteria for European freshwater fish--extreme pH values and inland fisheries, prepared by EIFAC Working Party on Water Quality Criteria for European Freshwater Fish. Water Research, 3:593.

Jones, J.R.E., 1964. Fish and River Pollution. Butterworth, London.

Langelier, W.F., 1936. The analytical control of anti-corrosion water treatment. Jour. Amer. Water Works Assn., 28:1500.

Mount, D.I., 1973. Chronic effect of low pH on fathead minnow survival, growth and reproduction. Water Res., 7:987.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Reid, L.C. and D.A. Carlson, 1974. Chlorine disinfection of low temperature waters. Jour. Enviro. Engineering Div., ASCE, 100: No. EE2, Proc. Papers 10443, 339.

Sawyer, C.N., 1960. Chemistry for sanitary engineers. McGraw-Hill, New York.

Stumm, W. and J.J. Morgan, 1970. Aquatic Chemistry. John Wiley and Sons, Inc., New York, Chapter 4.

Zirino, A. and S. Yamamoto, 1972. A pH-dependent model for the chemical specialization of copper, zinc, cadmium and lead in seawater. Limn. and Oceanog., 17(5).

PHENOL

CRITERION:

1 ug/l for domestic water supply (welfare), and
to protect against fish flesh tainting.

INTRODUCTION:

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

RATIONALE:

Phenolic compounds can affect freshwater fishes adversely by direct toxicity to fish and fish-food organisms; by lowering the amount of available oxygen because of the high oxygen demand of the compounds and by tainting of fish flesh (EIFAC, 1973). Shelford (1917) observed

that a concentration of 1 cc per liter (purity of compound and concentration are unknown) was rapidly fatal to fish but solutions of one half to three quarters of this amount (i.e., .5 to .75 cc) would require up to one hour to kill fish. Subsequent studies have confirmed the toxicity of phenol to both adult and immature organisms (EIFAC, 1973). Decreased egg development in the oyster, Crassostrea virginica, has been found to occur at levels of 2 mg/l phenol (Davis and Hidu, 1969).

Various environmental conditions will increase the toxicity of phenol. Lower dissolved oxygen concentrations, increased salinity and increased temperature all enhance the toxicity of phenol (EIFAC, 1973). It has been shown that phenol and o-cresol have 24-hour LC50's of 5 and 2 mg/l respectively for trout embryos (Albersmeyer and von Erichsen, 1959). Rainbow trout were killed in 7.3 mg/l phenol in 2 hours and in 6.5 mg/l phenol in 12 hours; at these concentrations there was rapid damage to gills and severe pathology of other tissues (Mitrovic, et al., 1968). Pathologic changes in gills and in fish tissues were found at concentrations in the range of 20 to 70 ug/l phenol (Reichenback-Klinke, 1965).

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

A major aesthetic problem associated with phenolic compounds is their organoleptic properties in water and fish flesh. Threshold odor levels range from 55 ug/l for p-cresol (Rosen, et al., 1962) to

2 ug/l for 2-chlorophenol (Burttschell, et al., 1959). The chlorinated phenols present problems in drinking water supplies because phenol is not removed efficiently by conventional water treatment and can be chlorinated during the final water treatment process to form persistent odor-producing compounds. Thus, odor problems are created in the distribution system. Boetius (1954), Fetterolf (1962), Schulze (1961), and Shumway (1966) estimated threshold fish flesh tainting concentrations for o-chlorophenol, p-chlorophenol, and 2, 4-dichlorophenol to range from 0.1 ug/l to 15 ug/l. The O-chlorophenol produced tainting at the lower concentration.

A criterion of 1 ug/l phenol, which is about half of the chlorophenol odor effect level for a water supply and near the threshold fish flesh tainting concentration, should protect the freshwater environment for such users.

REFERENCES CITED:

Albersmeyer, W. and L. von Erichsen, 1959. Untersuchungen zur wirung von Teerbestandteilen in abwassern. Mitteilungen, Z. Fisch., 3:29.

Boetius, J., 1954. Foul taste of fish and oysters caused by chlorophenol. Medd. Dan. Fisk. Havunders. 1:1.

Burttschell, et al., 1959. Chlorine derivatives of phenol causing taste and odor. Jour. Amer. Water Works Assn., 51:205.

Davis, H.C. and H. Hidu, 1969. Effects of pesticides on embryonic development of clams and oysters and on survival and growth of the larvae. Fish. Bull., 67:393.

European Inland Fisheries Advisory Commission, 1973. Water quality criteria for European freshwater fish. Report on mono-hydric phenols and inland fisheries. Water Res., 7:929.

Fetterolf, C.M., 1962. Investigation of fish off flavor, Muskegon lake. Bur. of Water Management, Michigan Department of Natural Resources, Lansing, Michigan.

McKee, J.E. and H.W. Wolf, 1963. Water quality criteria. State Water Quality Control Board, Sacramento, California. Pub. 3-A.

Mitrovic, V.A., et al., 1968. Some pathologic effects of sub-acute and acute poisoning of rainbow trout by phenol in hard water. Water Res., 2:249.

Reichenbach-Klinke, H.H., 1965. Der phenolgeholt des wasser in seiner auswerkung auf den fischorgaismus. Arch. Fischwiss., 16:1.

Rosen, A.A., et al., 1962. Odor thresholds of mixed organic chemicals, Jour. Water Poll. Cont. Fed., 34:7.

Schulze, E., 1961. The influence of phenol-containing effluents on the taste of fish. Int. Rev. ges. Hydrobiol. 46:84.

Shelford, V.E., 1917. An experimental study of the effects of gas wastes upon fishes, with especial reference to stream pollution. Bull. of the Illinois State Lab. of Nat. Hist., XI:381.

Shumway, D.I., 1966. Effects of effluents on flavor of salmon flesh. Agricultural Experiment Station, Oregon State University, Corvallis, 17 p.

PHOSPHORUS

CRITERION:

0.10 ug/l yellow (elemental) phosphorus for marine or estuarine waters.

INTRODUCTION

Phosphorus in the elemental form is particularly toxic and is subject to bioaccumulation in much the same way as mercury. Phosphorus as phosphate is one of the major nutrients required for plant nutrition and is essential for life. In excess of a critical concentration, phosphates stimulate plant growths. During the past 30 years, a formidable case has developed for the belief that increasing standing crops of aquatic plants, which often interfere with water uses and are nuisances to man, frequently are caused by increasing supplies of phosphorus. Such phenomena are associated with a condition of accelerated eutrophication or aging of waters. Generally, it is recognized that phosphorus is not the sole cause of eutrophication but there is substantiating evidence that frequently it is the key element of all of the elements required by freshwater plants, and generally, it is present in the least amount relative to need. Therefore, an increase in phosphorus allows use of other already present nutrients for plant growth. Further, of all of the elements required for plant growth in the water environment, phosphorus is the most easily controlled by man.

Large deposits of phosphate rock are found near the western shore of Central Florida, as well as in a number of other States. Deposits in Florida are found in the form of pebbles which vary in size from fine sand to about the size of a human foot. These pebbles are embedded in a matrix of clay and

and sand. The phosphate rock beds lie within a few feet of the surface and mining is accomplished by use of hydraulic water jets and a washing operation that separates the phosphate from waste materials. The process is similar to that of strip-mining. Florida, Idaho, Montana, North Carolina, South Carolina, Tennessee, Utah, Virginia, and Wyoming share phosphate mining activities.

Phosphates enter waterways from several different sources. The human body excretes about one pound per year of phosphorus expressed as "P." The use of phosphate detergents and other domestic phosphates increases the per capita contribution to about 3-1/2 pounds per year of phosphorus as P. Some industries, such as potato processing, have wastewaters high in phosphates. Crop, forest, idle, and urban land contribute varying amounts of phosphorus diffused sources in drainage to watercourses. This drainage may be surface runoff of rainfall, effluent from tile lines, or return flow from irrigation. Cattle feedlots, concentrations of domestic duck or wild duck populations, tree leaves, and fallout from the atmosphere all are contributing sources.

Evidence indicates that: (1) high phosphorus concentrations are associated with accelerated eutrophication of waters, when other growth-promoting factors are present; (2) aquatic plant problems develop in reservoirs and other standing waters at phosphorus values lower than those critical in flowing streams; (3) reservoirs and lakes collect phosphates from influent streams and store a portion of them within consolidated sediments, thus serving as a phosphate sink; and, (4) phosphorus concentrations critical to noxious plant growth vary and nuisance growths may result from a particular concentration of phosphate in one geographical area but not in another. The amount or percentage of inflowing nutrients that may be retained by a lake or reservoir is variable and will depend upon: (1) the nutrient loading to the lake or reservoir; (2) the volume of the euphotic zone; (3) the extent of biological activities;

(4) the detention time within the lake basin or the time available for biological activities; and, (5) the level of discharge from the lake or of the penstock from the reservoir.

Once nutrients are combined within the aquatic ecosystem, their removal is tedious and expensive. Phosphates are used by algae and higher aquatic plants and may be stored in excess of use within the plant cell. With decomposition of the plant cell, some phosphorus may be released immediately through bacterial action for recycling within the biotic community, while the remainder may be deposited with sediments. Much of the material that becomes combined with the consolidated sediments within the lake bottom is bound permanently and will not be recycled into the system.

RATIONALE

Elemental Phosphorus

Isom (1960) reported an LC50 of 0.105 mg/l at 48 hours and 0.025 mg/l at 160 hours for bluegill sunfish, Lepomis macrochirus, exposed to yellow phosphorus in distilled water at 26° C and pH 7. The 125- and 195-hour LC50's of yellow phosphorus to Atlantic cod, Gadus morhua, and Atlantic salmon, Salmo salar smolts in continuous-exposure experiments was 1.89 and 0.79 ug/l, respectively.(Fletcher and Hoyle, 1972). No evidence of an incipient lethal level was observed since the lowest concentration of P_4 tested was 0.79 ug/l. Salmon that were exposed to elemental phosphorus concentrations of 40 ug/l or less developed a distinct external red color and showed signs of extensive hemolysis. The predominant features of P_4 poisoning in salmon were external redness, hemolysis, and reduced hematocrits.

Following the opening of an elemental phosphorus production plant in Long Harbour, Placentia Bay, Newfoundland, divers observed dead fish upon the bottom throughout the Harbour (Peer, 1972). Mortalities were confined to a water depth of less than 18 meters. There was visual evidence of selective mortality among benthos. Live mussels were found within 300 meters of the effluent pipe, while all scallops within this area were dead.

Fish will concentrate elemental phosphorus from water containing as little as 1 ug/l (Idler, 1969). In one set of experiments, a cod swimming in water containing 1 ug/l elemental phosphorus for 18 hours concentrated phosphorus to 50 ug/kg in muscle, 150 ug/kg in fatty tissue, and 25,000 ug/kg in the liver (Idler, 1969 ; Jangaard, 1970). The experimental findings showed that phosphorus is quite stable in the fish tissues.

The criterion of 0.10 ug/l elemental phosphorus for marine or estuarine waters is 1/10 of demonstrated lethal levels to important marine organisms and of levels that have been found to result in significant bioaccumulation.

Phosphate Phosphorus

Although a total phosphorus criterion to control nuisance aquatic growths is not presented, it is believed that the following rationale to support such a criterion, which currently is evolving, should be considered.

Total phosphate phosphorus concentrations in excess of 100 ug/l P may interfere with coagulation in water treatment plants. When such concentrations exceed 25 ug/l at the time of the spring turnover on a volume-weighted basis in lakes or reservoirs, they may occasionally stimulate excessive or nuisance

growths of algae and other aquatic plants. Algal growths impart undesirable tastes and odors to water, interfere with water treatment, become aesthetically unpleasant and alter the chemistry of the water supply. They contribute to the phenomenon of cultural eutrophication.

To prevent the development of biological nuisances and to control accelerated or cultural eutrophication, total phosphates as phosphorus (P) should not exceed 50 ug/l in any stream at the point where it enters any lake or reservoir, nor 25 ug/l within the lake or reservoir. A desired goal for the prevention of plant nuisances in streams or other flowing waters not discharging directly to lakes or impoundments is 100 ug/l total P (Mackenthun, 1973). Most relatively uncontaminated lake districts are known to have surface waters that contain from 10 to 30 ug/l total phosphorus as P (Hutchinson, 1957).

The majority of the Nation's eutrophication problems are associated with lakes or reservoirs and currently there are more data to support the establishment of a limiting phosphorus level in those waters than in streams or rivers that do not directly impact such water. There are natural conditions, also, that would dictate the consideration of either

a more or less stringent phosphorus level. Eutrophication problems may occur in waters where the phosphorus concentration is less than that indicated above and, obviously, there would be a need in such waters to have nutrient limits that are more stringent. Likewise, there are those waters within the Nation where phosphorus is not now a limiting nutrient and where the need for phosphorus limits is substantially diminished. Such conditions are described in the last paragraph of this rationale.

There are two basic needs in establishing a phosphorus criterion for flowing waters: one is to control the development of plant nuisances within the flowing water and, in turn, to control and prevent animal pests that may become associated with such plants; the other is to protect the downstream receiving waterway, regardless of its proximity in linear distance. It is evident that a portion of that phosphorus that enters a stream or other flowing waterway eventually will reach a receiving lake or estuary either as a component of the fluid mass, as bed load sediments that are carried downstream, or as floating organic materials that may drift just above the stream's bed or float on its water's surface. Superimposed on the loading from the inflowing waterway, a lake or estuary may receive additional phosphorus as fallout from the air shed or as a direct introduction from shoreline areas.

Another method to control the inflow of nutrients, particularly phosphates, into a lake is that of prescribing an annual loading to the receiving water. Vollenweider (1973) suggests total phosphorus (P) loadings in grams per square meter of surface area per year that will be a critical level for eutrophic conditions within the receiving waterway for a particular water volume where the mean depth of the lake in meters is divided by the hydraulic detention time in years. Vollenweider's data suggest a range of loading values that should result in oligotrophic lake water quality.

Mean Depth/Hydraulic Detention Time (meters/year)	Oligotrophic or Permissible Loading (grams/meter^2/year)	Eutrophic or Critical Loading (grams/meter^2/year)
0.5	0.07	0.14
1.0	0.10	0.20
2.5	0.16	0.32
5.0	0.22	0.45
7.5	0.27	0.55
10.0	0.32	0.63
25.0	0.50	1.00
50.0	0.71	1.41
75.0	0.87	1.73
100.0	1.00	2.00

There may be waterways wherein higher concentrations or loadings of total phosphorus do not produce eutrophy, as well as those waterways wherein lower concentrations or loadings of total phosphorus may be associated with popula-

tions of nuisance organisms. Waters now containing less than the specified amounts of phosphorus should not be degraded by the introduction of additional phosphates.

It should be recognized that a number of specific exceptions can occur to reduce the threat of phosphorus as a contributor to lake eutrophy. Often, naturally occurring phenomena limit the development of plant nuisances; often there are technological or cost-effective limitations to the control of introduced pollutants. Exceptions to the threat of phosphorus in eutrophication occur in waters highly laden with natural silts or colors which reduce the penetration of sunlight needed for plant photosynthesis; in those waters whose morphometric features of steep banks, great depth, and substantial flows contribute to a history of no plant problems; in those waters that are managed primarily for waterfowl or other wildlife; in those waters where an identified nutrient other than phosphorus is limiting to plant growth and the level and nature of such limiting nutrient would not be expected to increase to an extent that would influence eutrophication; and in those waters where phosphorus control cannot be sufficiently effective under present technology to make phosphorus the limiting nutrient. No national criterion is presented for phosphate phosphorus for the control of eutrophication.

REFERENCES CITED

Fletcher, G.L., and R.J. Hoyle, 1972. Acute toxicity of yellow phosphorus to Atlantic cod (Gadus morhua) and Atlantic salmon (Salmo salar) smolts. Jour. Fish. Res. Bd. of Canada, 29:1295.

Hutchinson, G.E., 1957. A treatise on limnology. John Wiley & Sons, New York.

Idler, D.R., 1969. Coexistence of a fishery and a major industry in Placentia Bay. Chemistry in Canada, 21(11):16.

Isom, B.G., 1960. Toxicity of elementary phosphorus. Jour. Water Poll. Control Fed., 32:1312.

Jangaard, P.M., 1970. The role played by the Fisheries Research Board of Canada in the "red" herring phosphorus pollution crisis in Placentia Bay, Newfoundland. Fisheries Research Board, Atlantic Regional Office (Circular No. 1) Halifax, Nova Scotia.

Mackenthun, K.M., 1973. Toward a cleaner aquatic environment. Environmental Protection Agency, Washington, D.C.

Peer, D.L., 1972. Observations on mortalities of benthic organisms after contamination of the bottom of Long Harbour, Placentia Bay, Newfoundland with elemental phosphorus. In: Effects of Elemental Phosphorus on Marine Life, Fish. Res. Bd. of Canada, Circular 2, pp. 181-186.

Vollenweider, R.A., 1973. Input output models. Schweiz Z. Hydrol. (In Press).

PHTHALATE ESTERS

CRITERION:

3 ug/l for freshwater aquatic life.

INTRODUCTION:

Phthalate esters are organic compounds extensively used as plasticizers, particularly in polyvinyl chloride plastics. The function of a plasticizer is to change the characteristics of the plastic resin by making them more flexible or to improve their workability. Some plastic formulations may contain up to 60 parts per hundred of a phthalate ester. Several phthalate esters are synthesized and vary in the side chain length and structure attached to the parent benzene ring. Certain esters, the di-2-ethylhexyl and di-n-butyl phthalates, are used as an orchard acaricide and insect repellent, respectively.

Occurrence of the phthalate residues has been demonstrated in fresh (Mayer, et al., 1971), marine (Morris, 1970), industrial and heavily populated waters (Stalling, 1972). No well documented information exists on the fate of the phthalate compounds in aquatic environments (Mayer, et al., 1972).

RATIONALE:

Acute toxicity of the phthalate esters tested has been shown to be quite low (Sanders et al., 1973). Mayer and Sanders (1973) determined the 96-hour LC_{50} of di-n-butyl phthalate to be 1.3 mg/l for the fathead minnow, Pimephales promelas; 0.73 mg/l for the bluegill, Lepomis macrochirus; 2.91 mg/l for channel catfish, Ictalurus punctatus; 6.47 mg/l for rainbow trout, Salmo gairdneri; 2.10 mg/l for the scud, Gammarus pseudolimnaeus; and >10 mg/l for the crayfish, Orconectes nais. Daphnia magna, when exposed to the di-2-ethylhexyl phthalate at

levels of from 3 ug/l to 30 ug/l for 21 days, exhibited a decrease in the numbe of young produced of 60 to 83 percent, respectively.

Ability of aquatic organisms to accumulate various phthalate residues depend upon the ester, concentration and time of exposure. Concentration factors up to 6,600 have been reported for di-n-butyl phthalate in midge larva, Chironomus plumosus, after seven days exposure to 0.18 ug/l. Amphipods, Gammarus pseudolimnaeus, were found to concentrate the di-2-ethylhexyl phthalate 13,400 times in water containing 0.1 ug/l after 14 days exposure (Sanders et al., 1973). Daphnia magna were exposed to 0.1 ug/l of radioactively labeled phthalate for 7 days and then transferred to fresh flowing water to determine the time required for elimination of phthalate residues (Mayer and Sanders, 1973). After 3 days, 50 percent of the total radioactivity remained; 25 percent of the activity was still present after 7 days in fresh water.

Phthalate esters can be detrimental to aquatic organisms at low water concentrations. Ability to concentrate high levels from water and reproductive impairment in certain species are suggestive of potential environmental damage. While the fate of these compounds remains obscure, their presence in water affects successful growth and reproduction essential for maintenance of animal populations. A freshwater criterion of 3 ug/l is recommended even though some reproductive impairment was seen in daphnids, since all other species tested were so much more resistant. Until additional effect data become available this criterion should be a goal for marine waters.

REFERENCES CITED

Mayer, Jr., F.L., et al., 1971. Phthalate esters: an environmental contaminant. Presented at Midwest Regional A.C.S. meeting, Oct. 28-29, 1971, St. Louis.

Mayer, Jr., F.L., et al., 1972. Phthalate esters as environmental contaminants. Nature 238: 411.

Mayer, Jr., F.L. and H.O. Sanders, 1973. Toxicology of phthalate acid esters in aquatic organisms. Environ. Health Perspec. 3:153.

Morris, R.J., 1970. Phthalate acid in the deep sea jellyfish, Atolla. Nature 227: 1264.

Sanders, H.O., et al., 1973. Toxicity, residue dynamics and reproductive effects of phthalate esters in aquatic invertebrates. Environ. Res. 6: 84.

Stalling, D.C., 1972. Analysis of organochlorine residues in fish: Current research at the Fish-Pesticide Research Laboratory. Pesticide Chemistry, Vol. 4, Methods in Residue Analysis, A.S. Tahori, ed. (Gordon & Breach Publ., N.Y.), pp. 413.

POLYCHLORINATED BIPHENYLS

CRITERION:

.001 ug/l for freshwater and marine aquatic life and for consumers thereof.

Every reasonable effort should be made to minimize human exposure.

INTRODUCTION:

Polychlorinated biphenyls (PCB's) are a class of compounds produced by the chlorination of biphenyls and are registered in the United States under the trade name, Aroclor (R) . The degree of chlorination determines their chemical properties, and generally their composition can be identified by the numerical nomenclature, e.g., Aroclor 1242, Aroclor 1254, etc. The first two digits represent the molecular type and the last two digits the average percentage by weight of chlorine (NTIS, 1972).

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

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

PCB compounds are slightly soluble in water (25-200 ug/l at 25°C), soluble in lipids, oils, organic solvents, and resistant to both heat and biological degradation (NTIS, 1972; Nisbet, et al., 1972). Typically, the specific gravity, boiling point, and melting point of PCB's increase with their chlorine content. PCB's are relatively non-flammable, have useful heat exchange and dielectric properties, and now are used principally in the electrical industry in capacitors and transformers.

RATIONALE:

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

Ninety-six-hour LC50 values for newly hatched fathead minnows, Pimephales promelas, were 15 ug/l for Aroclor 1242 and 7.7 ug/l for Aroclor 1254. In 60-day continuous flow bioassays 50 percent of the fathead minnows were killed in 8.8 ug/l Aroclor 1242 and in 4.6 ug/l Aroclor 1254 (Nebeker, et al., 1974). Nine-month continuous flow bioassay tests were conducted in the same series of experiments reported by Nebeker, et al. (1974). The spawning of fathead minnows was significantly affected at concentrations of 1.8 ug/l Aroclor 1254; concentrations of Aroclor 1242 above 10 ug/l were lethal to newly hatched fry. Defoe, et al. (In Press) conducted similar flow-through acute and chronic studies with fathead minnows using Aroclor 1248 and 1260. The calculated 30-day TL50 for newly hatched fathead minnows was 4.7 ug/l for Aroclor 1248 and 3.3 ug/l for Aroclor 1260. Fathead minnows were able to reproduce successfully at PCB concentrations which were acutely lethal to the larvae.

Stalling and Mayer (1972) determined 96-hour LC50 values ranging from 1,170 to 50,000 ug/l for cutthroat trout, Salmo clarki, using Aroclors 1221-1268. With rainbow trout, Salmo gairdneri, and Aroclors 1242-1260 the acute toxicity was

greater than 1500 mg/l. Fifteen-day intermittent-flow bioassays carried out with bluegills, Lepomis macrochirus, and Arcclors 1242, 1248 and 1254 resulted in LC50 values of 54, 76 and 204 ug/l, respectively.

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

Nebeker and Puglisi (1974) conducted bioassays with Daphnia magna exposed to concentrations of Aroclors 1221-1268. In continuous flow tests Aroclor 1254 was the most toxic with a 3-week LC50 of 1.3 ug/l; 100 percent mortality occurred at 3.5 ug/l and 100 percent reproductive impairment occurred at 3.8 ug/l. Stalling and Mayer (1972) and Mayer et al. (1975) conducted flow-through and static bioassay tests on freshwater invertebrates which likewise indicated that these organisms are generally more susceptible to acute toxic effects of PCB's than fish.

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

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

On the basis of the FDA action level of 5 ug/g in fish tissue, and both laboratory and field derived bioaccumulation levels for fathead minnows, Pimephales promelas, and goldfish, Carassius auratus, in the range of 0.7×10^3 to 2.3×10^5 , an ambient water concentration of no more than 0.022 ug/l would be permissible. However, lake trout from the Great Lakes larger than 12 inches in length generally contain more than 5 ug/g of PCB's and chub from the Great Lakes generally contain PCB's in amounts approaching or slightly exceeding 5 ug/g. Since monitoring data on the Great Lakes' waters consistently indicate concentrations equal to or less than .01 ug/l, a criterion of less than 0.01 ug/l for all fishes appears necessary.

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

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

Surveys of Escambia Bay (Florida) during the period September 1969 to December 1971 produced data on the

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

From the Escambia Bay data, which include flow-through bioassays with residue analyses where possible, the following conclusions were reached: (1) all of the Aroclors tested are acutely toxic to certain estuarine organisms; (2) bioassays lasting longer than 96 hours demonstrated that Aroclor 1254 is toxic to commercial shrimp at less than 1 ug/l; (3) fish, particularly sheepshead minnows, Cyprinodon variegatus, are extremely sensitive to Aroclor 1254 with 0.1 ug/l lethal to fry; and, (4) acute toxicity of Aroclor 1016 to estuarine organisms is similar to that of other Aroclors but it appears less toxic to marine fish in long-term exposures than does Aroclor 1254 (Duke, 1974; Schimmel, et al., 1974).

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

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

Blue crabs, Callinectes sapidus, apparently were not affected by 20 days' exposure to 5.0 ug/l of Aroclor 1254. Pink shrimp exposed under similar conditions experienced a 72 percent mortality. In subsequent flow-through bioassays, 51 percent of the juvenile shrimp were killed by Aroclor 1254 in 15 days and 50 percent of the adult shrimp were killed at 3.0 ug/l in 35 days. From pathological examinations of the exposed pink shrimp, it appears that Aroclor 1254 facilitates or enhances the susceptibility to latent viral infections. Aroclor 1254 was lethal to grass shrimp, Paleomonetes pugio, at 4.0 ug/l in 16 days, to

amphipods at 10 ug/l in 30 days, and to juvenile spot, Leiostomus xanthurus, at 5.0 ug/l after 20 to 45 days. Sheepshead minnows, Cyprinodon variegatus, were the most sensitive estuarine organisms to Aroclor 1254 with 0.9 ug/l lethal to the fry within 2 weeks. Aroclor 1016, in two different 42-day flow-through bioassays, caused significant mortalities of pinfish at 32 ug/l and 21 ug/l. Pathological examination of those exposed to 32 ug/l revealed several liver and pancreatic alterations. Sheepshead minnows in 28-day Aroclor 1016 flow-through bioassays were not affected by concentrations of 10 ug/l or less, but died at 32 and 100 ug/l (Duke, 1974). The bioaccumulation factors determined by the flow-through bioassays are:

Aroclor	Organism	Time	Accumulation Factors (as a multiplier of test water concentrations)
1254	Oyster (Crassostrea virginica)	30 days	1.01×10^5
1254	Blue crab (Callinectes sapidus)	20 days	4×10^3
1254	Grass shrimp (Paleomonetes pugio)	7 days	3.2×10^3 to 11×10^3
1254	Spot (Leiostomus xanthurus)	14-28 days	37×10^3

1254	Pinfish (Lagodon rhomboides)	35 days	21.8×10^3
1016	Pinfish (Lagodon rhomboides)	42 days	11×10^3 to 24×10^3
1016	Sheepshead minnow (Cyprinodon variegatus)		2.5×10^3 to 8.1×10^3

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

Evidence is accumulating that PCB's do not contribute to shell thinning of bird eggs (NAS, 1974). Dietary PCB's produced no shell thinning in eggs of mallard ducks (Heath, et al., 1972). PCB's may increase susceptibility to

infectious agents such as viral diseases (Friend and Trainer, 1970), and increase the activity of liver enzymes that degrade steroids, including sex hormones (Risebrough, et al., 1968; Street, et al., 1968). Laboratory studies have indicated that PCB's with their derivatives or metabolites, cause embryonic death of birds (Voss and Koeman, 1970). Feeding PCB's to White Leghorn pullets at a level of 20 ppm caused a significant decrease in hatchability of the eggs and viability of the surviving progeny (Lillie, et al., 1974; Lillie, et al., 1975); in many cases, the cause of embryo mortality was attributed to gross abnormalities which ranged from edema to malformed appendages (Cecil, et al., 1974).

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

dibenzofurans are a by-product of PCB production, it is not known whether they are also produced by the degradation of PCB's (NAS, 1974).

REFERENCES CITED:

Armour, J.A. and J.A. Burke, 1970. Method for separating polychlorinated biphenyls from DDT and its analogs. Assoc. Official Anal. Chem., 53:761.

Bauer, H., et al., 1961. Occupational poisonings in the production of chlorophenol compounds. Arch. Gewerbepath, 18:583.

Cecil, H.C., et al., 1974. Embryotoxic and teratogenic effects in unhatched fertile eggs from hens fed polychlorinated biphenyls (PCB's). Bull. Envir. Contam. and Tox., 11:489.

DeFoe, D.L., et al., 1976. Effects of Aroclor 1248 and 1260 on the fathead minnow. J. Fish. Res. Board Canada (In Press).

Duke, T.W., 1974. Testimony in the matter of proposed toxic pollutant effluent standards for Aldrin-Dieldrin et al. FWPCA (307) Docket #1.

Food and Drug Administation, 1971. U.S. Dept. of H.E.W. Pesticide Analytical Manual, Volumes I and II.

Friend, H. and D.O. Trainer, 1970. Polychlorinated biphenyl interaction with duck hepatitis virus. Science, 170:1314.

Hays, H. and R.W. Risebrough, 1972. Pollutant concentrations in abnormal spring tern from Long Island Sound. AUK, 89:19.

Heath, R.G., et al., 1972. Effects of polychlorinated biphenyls on birds. Proceedings IV International Ornithological Congress (In Press).

Johnson, W., 1973. Unpublished report on toxicity of Aroclor 1016. Fish-Pesticide Laboratory, USDI, Fish and Wildlife Service, Columbia, MO 65201.

Lillie, R.J., et al., 1974. Differences in response of caged White Leghorn layers to various polychlorinated biphenyls (PCB's) in the diet. Poultry Science, 53:726.

Lillie, R.J., et al., 1975. Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens. Poultry Science, 54:1550.

Mayer, F.L., et al., 1975. Residue dynamics and biological effects of PCB's in aquatic organisms. Arch. Env. Cont. and Toxic. (In Press).

Mayer, F.L. 1975. Toxicity of Aroclor 1016 to freshwater fishes. Unpublished data. Fish-Pesticide Laboratory, USDI, Fish and Wildlife Service, Columbia, MO 65201.

Michigan Water Resource Commission, 1973. Monitoring for polychlorinated biphenyls in the aquatic environment.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

National Technical Information Service, 1972. Polychlorinated biphenyls and the environment. NTIS Interdepartmental Task Force, Washington, D.C.

Nebeker, A.V., and F.A. Puglisi. 1974. Effect of polychlorinated biphenyls (PCB's) on survival and reproduction of Daphnia, Gammarus, and Tanytarsus. Trans. Amer. Fish. Soc. 103(4): 722.

Nebeker, A.V., et al., 1974. Effect of polychlorinated biphenyl compounds on survival and reproduction of the fathead minnow and flagfish. Trans. Amer. Fish. Soc. 103(3): 562.

Nisbet et al., 1972. Rates and routes of transport of PCB's in the environment. Environ. Health Perspectives 4:21.

Platonow, N.S. and L.H. Kalstad, 1973. Dietary effects of polychlorinated biphenyls on mink. Can. Jour. Comp. Med., 37:391.

Ringer, R.K., et al., 1972. Effect of dietary polychlorinated biphenyls on growth and reproduction of

mink. Amer. Chem. Soc., National Meeting Reprints of Papers, 12:149.

Risebrough, R.W., et al., 1968. Polychlorinated biphenyls in the global ecosystem. Nature, 220:1098.

Risebrough, R.W., 1969. Chlorinated hydrocarbons in marine ecosystems. IN: Chemical Fallout, M.W. Miller and G.G. Berg, eds., C.C. Thomas, Springfield.

Risebrough, R.W., 1970. More letters in the world. Environ., 12:16.

Schimmel, S.C., et al., 1974. Effects of Aroclor 1254 on laboratory-reared embryos and fry of sheepshead minnows (Cyprinodon variegatus). Trans. Amer. Fish. Soc., 103:582.

Schultz, K.H., 1968. Clinical picture and etiology of chloracne. Arbeitsmed. Sozialmed. Arbeitshyg, 3:25. Reprinted in translation in U.S., Congress. Senate, Committee on Commerce, Effects of 2, 4, 5-T on man and

the environment: Hearings, 91st Cong., 2 sess., pp. 336-341.

Schwartz, L. and S.M. Peck, 1943. Occupational acne. New York State Med., 43:1711.

Stalling, D.L. and F.L. Mayer, Jr., 1972. Toxicities of PCB's to fish and environmental residues. Env. Health Perspectives, 1:54.

Street, J.C., et al., 1968. Comparative effects of polychlorinated biphenyls and organochlorine pesticides in induction of hepatic microsomal enzymes. Amer. Chem. Soc., 158th National Meeting. Sept. 8-12, 1968.

Varrett, J., 1970. Statement in U.S. Congress, Senate, Committee on Commerce, Effects of 2, 4, 5-T on man and the environment: Hearings, 91st Cong., 2nd sess., pp. 190-360.

Veith, G.D., 1973. Chlorinated hydrocarbons in fish and lake fish. Final report, EPA Grant 1602 PBE.

Veith, G.D. and G.F. Lee, 1971. Chlorobiphenyls (PCB's) in the Milwaukee River, Water Res., 5:1107.

Veith, G.D. 1975. Personal Communication. PCB test data, National Water Quality Labcratory, Duluth, MN, 55804.

Voss, J.G. and J.H. Koeman, 1970. Comparative toxicologic study with polychlorinated biphenyls in chickens with special reference to porphyria, edema formation, liver nephrosis, and tissue residues. Toxicol. Apl. Pharmacol., 17:656.

Wasserman, M., et al., 1970. The effect of organochlorine insecticides on serum cholesterol level in people occupationally exposed. Bull. Environ. Contam. Toxicol., 5:368.

SELENIUM

CRITERIA:

10 ug/l for domestic water supply (health);

For marine and freshwater aquatic life; 0.01 of the 96-hour LC_{50} as determined through bioassay using a sensitive resident species.

INTRODUCTION:

Biologically, selenium is an essential, beneficial element recognized as a metabolic requirement in trace amounts for animals but toxic to them when ingested in amounts ranging from about 0.1 to 10 mg/kg of food. The national levels of selenium in water are proportional to the selenium in the soil. In low selenium areas, the content of water may be well below 1 ug/l (Lindberg, 1968). In water from seleniferous areas, levels of selenium of 50 to 300 ug/l have been reported (WHO, 1972). Selenium appears in the soil as basic ferric selenite, calcium selenate, and as elemental selenium. Elemental selenium must be oxidized to selenite or selenate before it has appreciable solubility in water.

RATIONALE:

Selenium is considered toxic to man. Symptoms appear similar to those of arsenic poisoning (Keboe, et al., 1944; Fairhill, 1941). Any consideration of the toxicity of selenium to man must take into consideration the dietary requirement for the element in amounts estimated to be 0.04 to .10 mg/kg of food. Considering this requirement in conjunction with evidence that ingestion of selenium in amounts as low

as .07 mg per day has been shown to give rise to signs of selenium toxicity, selenium concentrations above 10 ug/l should not be permitted in drinking water (Smith, et al., 1936; Smith and West, 1937). The USPHS drinking water standards recommend that drinking water supplies contain no more than 0.01 mg/l of selenium (USPHS, 1962).

As sodium selenite, 2.0 mg/l of selenium has been demonstrated to be lethal to goldfish, Carassius auratus, in 18 to 46 days (Ohio River Valley Water Sanitation Commission, 1950). Bringmann and Kuhn (1959) demonstrated the threshold effect of selenium as sodium selenite on a freshwater crustacean, an alga and a bacterium. In two days the median threshold effect occurred at 2.5 mg/l with Daphnia; in 4 days the median threshold effect was 2.5 mg/l with Scenedesmus, at 90 mg/l with Escherichia coli:, and 183 mg/l for the protozoan, Microregma.

Selenium in water apparently is toxic at concentrations of 2.5 mg/l or less to those few species tested. Animals can beneficially metabolize ingested selenium in amounts of 0.01 to 0.10 mg/kg of food.

Based on the data available, freshwater fish should not be exposed to water containing more than 0.01 of the 96-hour LC50 as determined through bioassay using a sensitive resident species.

REFERENCES CITED:

Bringmann, G. and R. Kuhn, 1959. The toxic effect of wastewater on aquatic bacteria, algae and small crustaceans. Gesundeits-Ing., 80:115.

Fairhill, L.T., 1941. Toxic contaminants of drinking water. Jour. North East Water Works Assn., 55:400.

Keboe, R.H., et al., 1944. The hygienic significance of the contamination of water with certain mineral constituents. Jour. Amer. Water Works Assn., 36:645.

Lindberg, P., 1968. Selenium determination in plant and animal material in water. Acta Vet Scand., Suppl. 32, Stockholm.

Ohio River Valley Water Sanitation Commission, 1950. Subcommittee on toxicities-metal finishing. Industries Action Committee, Report No. 3.

Smith, M.I., et al., 1936. The selenium problem in relation to public health. Public Health Report 51, (U.S.) 51:1496.

Smith, M.I. and B.B. West, 1937. Further field studies on the selenium problem in relation to public health. Public Health Report, 52:1375.

U.S. Public Health Service, 1962. Drinking water standards, 27 F.R. 2152.

World Health Organization, 1972. Health hazards of the human environment. Geneva.

SILVER

CRITERIA:

50 ug/l for domestic water supply (health).

For marine and fresh water aquatic life 0.01 of the 96-hour LC_{50} as determined through bioassay using a sensitive resident species.

INTRODUCTION:

Biologically, silver is a non-essential, non-beneficial element recognized as causing localized skin discoloration in humans, and as being systemically toxic to aquatic life. Ingestion of silver or silver salts by humans results in deposition of silver in skin, eyes and mucous membranes that causes a blue-gray discoloration without apparent systemic reaction (Hill, 1957). Because of its strong bactericidal action, silver has been considered for use as a water disinfectant. Dosages of 0.001 to 500 ug/l of silver have been reported sufficient to sterilize water (McKee and Wolf, 1963). At these concentrations, the ingestion of silver has no obvious detrimental effect on humans.

RATIONALE:

The 1962 USPHS Drinking Water Standards contained a limit for silver of 0.05 mg/l. This limit was established because of the evidence that silver, once absorbed, is held indefinitely in tissues, particularly the skin, without evident loss through usual channels of elimination or reduction by transmigration to other body sites, and because of the probable high absorbability of silver bound to sulfur components of food cooked in silver-containing water (Aub and Fairhall, 1942). A study of the toxic

effects of silver added to drinking water of rats showed pathologic changes in kidneys, liver and spleen at concentrations of 400, 700, and 1,000 ug/l (Just and Szniolis, 1936).

Using silver nitrate, Coleman and Clearly (1974) demonstrated that juvenile largemouth bass, Micropterus salmoides, and bluegill, Lepomis macrochirus exposed to silver in concentrations of 0.3 to 70 ug/l accumulated the metal, especially in the internal organs and gills. The quantity of accumulated silver increased as exposure concentrations increased with a subsequent equilibrium developing between the water and tissue concentrations. After two months' exposure to 7 ug/l silver, the concentration of silver in gills exceeded that in the gills of the control fish by 200-fold. The 70 ug/l concentration of silver was lethally toxic to bass.

Data compiled by Doudoroff and Katz (1953) show that sticklebacks were killed by a 20 ug/l concentration of silver nitrate in two days. Anderson (1948) reported that the toxic threshold of silver nitrate for sticklebacks, Gasterosteus aculeatus, was 3.0 ug/l as silver. He also found that Daphnia magna were immobilized by 3.2 ug/l as silver. Jones (1939) found the lethal concentration of silver nitrate for sticklebacks was 3.0 ug/l as silver. In differing concentrations the average survival times of the fish were; one week at 4.0 ug/l, four days at 10.0 ug/l, and only one day at 100.0 ug/l.

In a 10-month bioassay on rainbow trout, Salmo gairdneri, exposed to silver nitrate, Geottl, et al. (1974) determined that no significant number of test fish died when exposed to silver concentrations of .09 and 0.17 ug/l as silver. The results did not reflect possible effects of silver on spawning behavior or reproduction.

Using silver thiosulfate, silver nitrate, silver carbonate and silver chloride, Terhaar, et al. (1972) reported that all of a test group of 20 fathead minnows, Pimephales promelas, survived exposure for 96 hours to 5,000 ug/l silver as silver thiosulfate; at 250,000 ug/l 15 of 20 fish died. Silver nitrate in concentrations of 1,000 and 100.0 ug/l as silver killed 16 out of 20 and 12 out of 20 fish, respectively. Silver carbonate killed all 20 test fish at concentrations of 1,000 ug/l.

In marine waters a concentration of 400 ug/l as silver killed 90 percent of test barnacles, Balanus balanoides (Clarke, 1947). Silver nitrate effects on the development of sea urchin, Arbacia, have been reported at approximately 0.5 ug/l (Wilber, 1969). Calabrese, et al. (1973) reported an LC_{50} (48-hour) of 5.8 ug/l as silver for oyster larvae, Crassostrea virginica, and an LC_{50} (48-hour) of 21.0 ug/l as silver for larvae of the hard-shell clam, Mercenaria mercenaria. Jackim, et al. (1970) reported a sublethal enzyme effect at a concentration of 20.0 ug/l as silver for Fundulus heteroclitus.

It is apparent that there is a wide variation in the toxicity of silver compounds to aquatic life and that the degree of dissociation characteristic of these compounds affects toxicity. Since little information is available on the movement and chemical stability of these compounds in the aquatic environment, a silver criterion must be based on the total silver concentration.

The silver criterion should be established at 0.01 of the 96-hour LC_{50} as determined through bioassay using a sensitive resident species. This application factor has been recommended (NAS, 1974) for persistent or cumulative toxicants.

REFERENCES CITED:

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

Aub, J. C. and L. T. Fairhall, 1942. Excretion of silver in urine. Jour. Amer. Medical Assn., 118-316.

Clarke, G. L., 1947. Poisoning and recovery in barnacles and mussels. Biol. Bull., 92:73.

Calabrese, A., et al., 1973. The toxicity of heavy metals to embryos of the American oyster Crassostrea virginica. Marine Biology, 18:162.

Coleman, R. L. and J. E. Clearly, 1974. Silver toxicity and accumulation in largemouth bass and bluegill. Bull. of Environmental Contamination and Toxicology, Vol. 12 No. 1.

Doudoroff, P. and M. Katz, 1953. Critical review of literature of toxicity of industrial wastes and their components to fish. Sew. and Ind. Wastes, 25:802.

Geottl, J. P., et al., 1974. Water Pollution studies. Job Progress Report, Colorado Department of Natural Resources, Federal Aid Project F-33-R-9 41-44.

Hill, W. B., et al., 1957. Argyria investigation--toxicological properties of silver. Amer. Silver Producers. Res. Proj. Rept., Appendix II.

Jackim, E., et al., 1970. Effects of metal poisoning of five liver enzymes in killifish (Fundulus heteroclitus). Jour. Fish. Res. Bd. Can., 27:388.

Jones, J.R.E., 1939. The relation between the electrolytic solution pressures of the metals and their toxicity to the stickleback. Jour. Exp. Biol., 16:425.

Just, J. and A. Szniolis, 1936. Germicidal properties of silver in water. Jour. Amer. Water Works Assn., 28:492.

McKee, J.E. and H.W. Wolf, 1963. Water quality criteria. California State Water Resources Control Board, Publ. No. 3-A.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Terhaar, C.J., et al., 1972. Toxicity of photographic processing chemicals to fish. Photo. Sci. and Eng., 16:370.

Wilber, C.G., 1969. The biological aspects of water pollution. Charles Thomas, Springfield, Illinois, p. 296.

SOLIDS (DISSOLVED) AND SALINITY

CRITERION:

250 mg/l for chlorides and sulfates
in domestic water supplies (welfare).

INTRODUCTION:

Dissolved solids and total dissolved solids are terms generally associated with freshwater systems and consist of inorganic salts, small amounts of organic matter and dissolved materials (Sawyer, 1960). The equivalent terminology in Standard Methods is filtrable residue (Standard Methods, 1971). Salinity is an oceanographic term, and although not precisely equivalent to the total dissolved salt content it is related to it (Capurro, 1970). For most purposes, the terms total dissolved salt content and salinity are equivalent. The principal inorganic anions dissolved in water include the carbonates, chlorides, sulfates and nitrates (principally in ground waters); the principal cations are sodium, potassium, calcium, and magnesium.

RATIONALE:

Excess dissolved solids are objectionable in drinking water because of possible physiological effects, unpalatable mineral tastes and higher costs because of corrosion or the necessity for additional treatment.

The physiological effects directly related to dissolved solids include laxative effects principally from sodium sulfate and magnesium sulfate and the adverse effect of sodium on certain patients afflicted with cardiac disease and women with toxemia associated with pregnancy. One study was made

using data collected from wells in North Dakota. Results from a questionnaire showed that with wells in which sulfates ranged from 1,000 to 1,500 mg/l, 62 percent of the respondents indicated laxative effects associated with consumption of the water. However, nearly one-quarter of the respondents to the questionnaire reported difficulties when concentrations ranged from 200 to 500 mg/l (Moore, 1952). To protect transients to an area, a sulfate level of 250 mg/l should afford reasonable protection from laxative effects.

As indicated, sodium frequently is the principal component of dissolved solids. Persons on restricted sodium diets may have an intake restricted from 500 to 1,000 mg/day (Nat. Res. Coun., 1954). That portion ingested in water must be compensated by reduced levels in food ingested so that the total does not exceed the allowable intake. Using certain assumptions of water intake (e.g., 2 liters of water consumed per day) and sodium content of food, it has been calculated that for very restricted sodium diets, 20 mg/l in water would be the maximum, while for moderately restricted diets, 270 mg/l would be maximum. Specific sodium levels for entire water supplies have not been recommended but various restricted sodium intakes are recommended because: (1) the general population is not adversely affected by sodium, but various restricted sodium intakes are recommended by physicians for a significant portion of the population, and; (2) 270 mg/l of sodium is representative of mineralized waters that may be aesthetically unacceptable, but many domestic water supplies exceed this level. Treatment for removal of sodium in water supplies is costly (NAS, 1974).

A study based on consumer surveys in 29 California water systems was made to measure the taste threshold of dissolved salts in water (Bruvold et al., 1969) Systems were selected to eliminate possible interferences from other taste-causing substances than dissolved salts. The study revealed that consumers rated waters with 319 to 397 mg/l dissolved solids as "excellent" while those with 1283 to

1333 mg/l dissolved solids were "unacceptable" depending on the rating system used. A "good" rating was registered for dissolved solids less than 658 to 755 mg/l. These results should be interpreted with consideration of consumer acclimation to such waters. The PHS Drinking Water Standards (1962) recommended a maximum dissolved solids concentration of 500 mg/l unless more suitable supplies were unavailable.

Specific constituents included in the dissolved solids in water may cause mineral taste at lower concentrations than other constituents. Chloride ions have frequently been cited as having a low taste threshold in water. Data from Ricter and MacLean (1939) on a taste panel of 53 adults indicated that 61 mg/l NaCl was the median level for detecting a difference from distilled water. At a median concentration of 395 mg/l chloride a salty taste was distinguishable, although the range was from 120 to 1215 mg/l. Lockhart, et al. (1955) evaluated the effect of chlorides on water used for brewing coffee. Threshold concentrations for chloride ranged from 210 mg/l to 310 mg/l depending on the associated cation. These data indicate that a level of 250 mg/l chlorides is a reasonable maximum level to protect consumers of drinking water.

The causation of corrosion and encrustation of metallic surfaces by water containing dissolved solids is well known. In water distribution systems corrosion is controlled by insulating dissimilar metal connections by non-metallic materials, use of pH control and corrosion inhibitors, or some form of galvanic or impressed electrical current systems (Lehmann, 1964). Damage in household systems occurs to water piping, wastewater piping, water heaters, faucets, toilet flushing mechanisms, garbage grinders and both clothes and dishwashing machines.

By use of water with 1,750 mg/l dissolved solids as compared with 250 mg/l, service life reductions ranged from 70 percent for toilet flushing mechanisms to 30 percent for washing equipment. Such increased corrosion was calculated in 1968 to cost the consumer an additional $0.50 per 1,000 gallons used (Patterson and Banker, 1968).

All species of fish and other aquatic life must tolerate a range of dissolved solids concentrations in order to survive under natural conditions. Based on studies in Saskatchewan it has been indicated that several common freshwater species survived 10,000 mg/l dissolved solids, that whitefish and pike-perch survived 15,000 mg/l, but only the stickleback survived 20,000 mg/l dissolved solids. It was concluded that lakes with dissolved solids in excess of 15,000 mg/l were unsuitable for most freshwater fishes (Rawson and Moore, 1944). The NTAC Report (1968) also recommended maintaining osmotic pressure levels of less than that caused by a 15,000 mg/l solution of sodium chloride.

Marine fishes also exhibit variance in ability to tolerate salinity changes. However, fishkills in Laguna Madre off the Texas coast have occurred with salinities in the range of 75 to 100 o/oo. Such concentrated sea water is caused by evaporation and lack of exchange with the Gulf of Mexico (Rounsefell and Everhart, 1953).

Estuarine species of fish are tolerant of salinity changes ranging from fresh to brackish to sea water. Anadromous species likewise are tolerant although evidence indicates that the young cannot tolerate the change until the normal time of migration (Rounsefell and Everhart, 1953). Other aquatic species are more dependent on salinity for protection from predators or require

certain minimal salinities for successful hatching of eggs. The oyster drill cannot tolerate salinities less than 12.5 o/oo. Therefore, estuarine segments containing salinities below about 12.5 o/oo produce most of the seed oysters for planting (Rounsefell and Everhart, 1953). Based on similar examples, the NTAC Report (1968) recommended that to protect fish and other marine animals no changes in hydrography or stream flow should be allowed that permanently change isohaline patterns in the estuary by more than 10 percent from natural variation.

Many of the recommended game bird levels for dissolved solids concentrations in drinking water have been extrapolated from data collected on domestic species such as chickens. However, young ducklings were reported poisoned in Suisan Marsh by salt when maximum summer salinities varied from 0.55 to 1.74 o/oo with means as high as 1.26 o/oo (Griffith, 1963).

Indirect effects of excess dissolved solids are primarily the elimination of desirable food plants and other habitat forming plants. Rapid salinity changes cause plasmolysis of tender leaves and stems because of changes in osmotic pressure. The NTAC Report (1968) recommended the following limits in salinity variation from natural to protect wildlife habitats:

Natural Salinity (o/oo)	Variation Permitted (o/oo)
0 to 3.5	1
3.5 to 13.5	2
13.5 to 35	4

Agricultural uses of water are also limited by excessive dissolved solids concentrations. Studies have indicated that chickens, swine, cattle and sheep can survive on saline waters up to 15,000 mg/l of salts of sodium and calcium combined with bicarbonates, chlorides and sulfates but only 10,000 mg/l of corresponding salts of potassium and magnesium. The approximate limit for highly alkaline waters containing sodium and calcium carbonates is 5,000 mg/l (NTAC, 1968).

Irrigation use of water is not only dependent upon the osmotic effect of dissolved solids, but also on the ratio of the various cations present. In arid and semiarid areas general classification of salinity hazards has been prepared (NTAC, 1968) (see Table 9).

Table 9 Dissolved Solids Hazard for Irrigation Water (mg/l)

Water from which no detrimental effects will usually be noticed	500
Water which can have detrimental effects on sensitive crops	500-1,000
Water that may have adverse effects on many crops and requires careful management practices	1,000-2,000
Water that can be used for tolerant plants on permeable soils with careful management practices	2,000-5,000

The amount of sodium and the percentage of sodium in relation to other cations are often important. In addition to contributing to osmotic pressure, sodium is toxic to certain plants, especially fruits, and frequently causes problems in soil structure, infiltration and permeability rates (Agriculture Handbook #60,

1954). A high percentage of exchangeable sodium in soils containing clays that swell when wet can cause a soil condition adverse to water movement and plant growth. The exchangeable-sodium percentage (ESP)* is an index of the sodium status of soils. An ESP of 10 to 15 percent is considered excessive if a high percentage of swelling clay minerals is present (Agricultural Handbook #60, 1954).

For sensitive fruits, the tolerance for sodium for irrigation water is for a sodium adsorption ratio (SAR)** of about 4, whereas for general crops and forages a range of 8 to 18 is generally considered usable (NTAC, 1968). It is emphasized that application of these factors must be interpreted in relation to specific soil conditions existing in a given locale and therefore frequently requires field investigation.

Industrial requirements regarding the dissolved solids content of raw waters is quite variable. Table 10 indicates maximum values accepted by various industries for process requirements (NAS, 1974). Since water of almost any dissolved solids concentration can be de-ionized to meet the most stringent requirements, the economics of such treatment are the limiting factor for industry.

---

*$ESP = \frac{100[a + b(SAR)]}{1 \quad [a + b(SAR)]}$

where: a = intercept representing experimental error (ranges from -0.06 to 0.01)
b = slope of regression line (ranges from 0.014 to 0.016)

**SAR = sodium adsorption ratio = $\frac{Na}{[0.5(Ca + Mg)]^{0.5}}$

SAR is expressed as milliequivalents

Table 10

Total Dissolved Solids Concentrations of Surface Waters that have been Used as Sources for Industrial Water Supplies

Industry/Use	Maximum Concentration (mg/l)
Textile	150
Pulp and Paper	1,080
Chemical	2,500
Petroleum	3,500
Primary Metals	1,500
Copper Mining	2,100
Boiler Make-up	35,000

Source: NAS, 1974

REFERENCES CITED:

Agriculture Handbook No. 60, 1954. Diagnosis and improvement of saline and alkali soils. L. A. Richards, editor, U.S. Government Printing Office, Washington, D. C.

Bruvold, W.H., et al., 1969. Consumer assessment of mineral taste in domestic water. Jour. Amer. Water Works Assn., 61:575.

Capurro, L.R.A., 1970. Oceanography for practicing engineers. Barnes and Noble Inc., New York.

Griffith, E.W., 1963. Salt as a possible limiting factor to the Suisan Marsh pheasant population. Annual Report Delta Fish and Wildlife Protection Study, Cooperative Study of California

Lehman, J.A., 1964. Control of corrosion in water systems. Jour. Amer Water Works Assn., 56: 1009.

Lockhart, E.E., et al., 1955. The effect of water impurities on the flavor of brewed coffee. Food Research, 20:598.

Moore, E.W., 1952. Physiological effects of the consumption of saline drinking water. National Res. Council, Div. of Medical Sciences, Bull. San. Engr., and Environment, Appendix E.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

National Research Council, 1954. Sodium restricted diets. Publication 325, Food and Nutrition Board, Washington, D.C.

National Technical Advisory Committee to the Secretary of the Interior, 1968. Water quality criteria. U.S. Government Printing Office, Washington, D.C.

Patterson, W.L. and R.F. Banker, 1968. Effects of highly mineralized water on household plumbing and appliances. Jour. Amer. Water Works Assn., 60:1060.

Public Health Service, 1962. Drinking water standards, 1962. U.S. Govt. Print. Office, Washington, D. C.

Rawson, D.S. and J.E. Moore, 1944. The saline lakes of Saskatchewan. Canadian Jour. of Res., 22: 141.

Ricter, C.O., and A. MacLean, 1939. Salt taste threshold of humans. Am. J. Physiol., 126:1.

Rounsefell, G.A. and W.H. Everhart, 1953. Fishery science, its methods and applications. John Wiley and Sons, Inc., New York.

Sawyer, C.N., 1960. Chemistry for sanitary engineers. McGraw-Hill Book Co., Inc., New York.

Standard methods for the examination of water and wastewater, 13th edition, 1971. APHA, Amer. Water Works Assn., Water Poll. Control Fed., Washington, D. C.

SOLIDS (SUSPENDED, SETTLEABLE) AND TURBIDITY

CRITERIA:

Freshwater fish and other aquatic life:

Settleable and suspended solids should not reduce the depth of the compensation point for photosythetic activity by more then 10 percent from the seasonably established norm for aquatic life.

INTRODUCTION

The term "suspended and settleable solids" is descriptive of the organic and inorganic particulate matter in water. The equivalent terminology used for solids in Standard Methods (APHA, 1971) is total suspended matter for suspended solids, settleable matter for settleable solids, volatile suspended matter for volatile solids and fixed suspended matter for fixed suspended solids. The term "solids" is used in this discussion because of its more common use in the water pollution control literature.

RATIONALE:

Suspended solids and turbidity are important parameters in both municipal and industrial water supply practices. Finished drinking waters have a maximum limit of 1 turbidity unit where the water enters the distribution system. This limit is based on health considerations as it relates to effective chlorine disinfection. Suspended matter provides areas where micro-organisms do not come into contact with the chlorine disinfectant (NAS, 1974). The ability of common water treatment processes (i.e., coagulation, sedimentation, filtration and chlorination) to remove suspended matter to achieve acceptable

final turbidities is a function of the composition of the material as well as its concentration. Because of the variability of such removal efficiency, it is not possible to delineate a general raw water criterion for these uses.

Turbid water interferes with recreational use and aesthetic enjoyment of water. Turbid waters can be dangerous for swimming, especially if diving facilities are provided because of the possibility of unseen submerged hazards and the difficulty in locating swimmers in danger of drowning (NAS, 1974). The less turbid the water the more desirable it becomes for swimming and other water contact sports. Other recreational pursuits such as boating and fishing will be adequately protected by suspended solids criteria developed for protection of fish and other aquatic life.

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

The effects of suspended solids on fish have been reviewed by the European Inland Fisheries Advisory Commission (EIFAC, 1965). This review identified four effects on the fish and fish food populations, namely:

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

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

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

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

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

Plankton and inorganic suspended materials reduce light penetration into the water body reducing the depth of the photic zone. This reduces primary production and decreases fish food. The NAS committee recommended that the depth of light penetration not be reduced by more than 10 percent (NAS, 1974). Additionally, the near surface waters are heated because of the greater heat absorbency of the particulate material which tends to stabilize the water column and prevents vertical mixing (NAS, 1974). Such mixing reductions decrease the dispersion of dissolved oxygen and nutrients to lower portions of the water body.

One partially offsetting benefit of suspended inorganic material in water is the sorption of organic materials such as pesticides. Following this sorption process subsequent sedimentation may remove these materials from the water column into the sediments (NAS, 1974).

Identifiable effects of suspended solids on irrigation use of water include the formation of crusts on top of the soil which inhibits water infiltration, plant emergence and impedes soil aeration; the formation of films on plant leaves which blocks sunlight and impedes 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 (NAS, 1974).

The criteria for freshwater fish and other aquatic life is essentially that proposed by the N.A.S. and the Great Lakes Water Quality Board.

REFERENCES CITED:

Edberg, N. and Hofstan, B.V., 1973. Oxygen uptake of bottom sediment studied in-situ and in the laboratory. Water Research, 7: 1285.

European Inland Fisheries Advisory Commission, 1965. Water quality criteria for European freshwater fish, report on finely divided solids and inland fisheries. Int. Jour. Air Water Poll., 9: 151.

Gammon, J.R., 1970. The effect of inorganic sediment on stream biota. Environmental Protection Agency. Water Poll. Cont. Res. Series, 18050 DWC 12/70, USGPO, Washington, D.C.

Mackenthun, K.M., 1973. Toward a cleaner aquatic environment. U.S. Government Printing Office, Washington, D. C.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Standard Methods for the Examination of Water and Wastewater, 1971. 13th Edition, APHA, Amer. Water Works Assn., Wtr. Poll. Cont. Fed.

Tarzwell, C.M. and A.R. Gaufin, 1953. Some important biological effects of pollution often disregarded in stream surveys, proceedings of the 8th Purdue industrial waste conference. Reprinted in Biology of Water Pollution, 1967. Dept. of Interior, Washington, D. C.

Tebo, L.B., Jr., 1955. Effects of siltation, resulting from improper logging, on the bottom fauna of a small trout stream in the southern Appalachians. The Progressive Fish-Culturist, 17: 64.

SULFIDE - HYDROGEN SULFIDE

CRITERION:

2 ug/l undissociated H_2S for fish and other aquatic life, fresh and marine water

INTRODUCTION:

Hydrogen sulfide is a soluble, highly poisonous, gaseous compound having the characteristic odor of rotten eggs. It is detectable in air by humans at a dilution of 0.002 ppm. It will dissolve in water at 4000 mg/l at 20°C and one atmosphere of pressure. Hydrogen sulfide biologically is an active compound that is found primarily as an anaerobic degradation product of both organic sulfur compounds and inorganic sulfates. Sulfides are constituents of many industrial wastes such as those from tanneries, paper mills, chemical plants and gas works. The anaerobic decomnosition of sewage, sludge beds, algae and other naturally deposited organic material is a major source of hydrogen sulfide.

When soluble sulfides are added to water they react with hydrogen ions to form HS^- or H_2S, the proportion of each

depending on the pH. The toxicity of sulfides derives primarily from H_2S rather than from the hydrosulfide (HS^-) or sulfide ($S^=$) ions. When hydrogen sulfide dissolves in water it dissociates according to the reactions:

$$H_2S \rightleftharpoons HS^- + H^+ \text{ and } HS^- \rightleftharpoons S^= + H^+$$

At pH 9 about 99 percent of the sulfide is in the form of HS^-, at pH 7 the sulfide is equally divided between HS^- and H_2S and at pH 5 about 99 percent of the sulfide is present as H_2S (NAS 1974). The fact that H_2S is oxidized in well-aerated water by natural biological systems to sulfates or is biologically oxidized to elemental sulfur has caused investigators to minimize the toxic effects of H_2S on fish and other aquatic life.

RATIONALE:

The degree of hazard exhibited by sulfide to aquatic animal life is dependent on the temperature, pH and dissolved oxygen. At lower pH values a greater proportion is in the form of the toxic undissociated H_2S. In winter when the pH is neutral or below or when dissolved oxygen levels are low but not lethal to fish, the hazard from sulfides is exacerbated. Fish exhibit a strong avoidance reaction to sulfide. Based on data from experiments with the stickleback, Jones (1964) hypothesized that if fish

encounter a lethal concentration of sulfide there is a reasonable chance they will be repelled by it before they are harmed. This of course assumes that an escape route is open.

Many past data on the toxicity of hydrogen sulfide to fish and other aquatic life have been based on extremely short exposure periods. Consequently these early data have indicated that concentrations between 0.3 and 0.4 mg/l permit fish to survive (Van Horn 1958, Boon and Follis 1967, Theede et al., 1969). Recent long-term data, both in field situations and under controlled laboratory conditions, demonstrate hydrogen sulfide toxicity at lower concentrations.

Colby and Smith (1967) found that concentrations as high as 0.7 mg/l existed within 20 mm of the bottom of sludge beds, and the levels of 0.1 to 0.02 mg/l were common within the first 20 mm of water above this layer. Walleye, Stizostedion vitreum, eggs held in trays in this zone did not hatch. Adelman and Smith (1970) reported that the hatchability of northern pike, Esox lucius, eggs was substantially reduced at 25 ug/l H_2S; at 47 ug/l mortality was almost complete. Northern pike fry had

96-hour LC_{50} values that varied from 17 to 32 ug/l at normal oxygen levels of 6.0 mg/l. The highest concentration of hydrogen sulfide that had no observable effect on eggs and fry was 14 and 4 ug/l, respectively. Smith and Oseid (1972), working on eggs, fry and juveniles of walleyes and white suckers, Catostomus commersoni , and Smith (1971), working on walleyes and fathead minnows, Pimephales promelas , found that safe levels varied from 2.9 ug/l to 12 ug/l with eggs being the least sensitive and juveniles being the most sensitive in short-term tests. In 96-hour bioassays, fathead minnows and goldfish, Carassius auratus, varied greatly in tolerance to hydrogen sulfide with changes in temperature. They were more tolerant at low temperatures (6 to 10°C). Holland, et al. (1960) reported that 1.0 mg/l sulfide caused 100 percent mortality in 72 hours with Pacific salmon.

On the basis of chronic tests evaluating growth and survival, the safe H_2S level for bluegill, Lepomis macrochirus, juveniles and adults was 2 ug/l. Egg deposition in bluegills was reduced after 46 days in 1.4 ug/l H_2S (Smith and Oseid, 1974). White sucker eggs were hatched at 15 ug/l, but juveniles showed growth reductions at 1 ug/l. Safe levels for fathead minnows were between 2 and 3 ug/l. Studies showed that safe levels for Gammarus pseudolimnaeus and Hexagenia limbata were 2 and 15 ug/l, respectively (Oseid and Smith, 1974a, 1974b). Some species typical of normally stressed habitats, Asellus spp., were much more resistant (Oseid and Smith, 1974c).

Sulfide criteria for domestic or livestock use have not been established because the unpleasant odor and taste would preclude such use at hazardous concentrations.

It is recognized that the hazard from hydrogen sulfide to aquatic life is often localized and transient. Available data indicate that water containing concentrations of 2.0 ug/l undissociated H_2S would not be hazardous to most fish and other aquatic wildlife, but concentrations in excess of 2.0 ug/l would constitute a long-term hazard.

REFERENCES CITED:

Adelman, I.R. and L.L. Smith, 1970. Effect of hydrogen sulfide on northern pike eggs and sac fry. Trans. Amer. Fish. Soc., 99: 501.

Boon, C.W. and B.J. Follis, 1967. Effects of hydrogen sulfide on channel catfish. Trans. Amer. Fish. Soc., 96: 31.

Colby, P.V. and L.L. Smith, 1967. Survival of walleye eggs and fry on paper fiber sludge deposits in Rainey River, Minnesota. Trans. Amer. Fish. Soc., 96: 278.

Holland, G.A., et al., 1960. Toxic effects of organic and inorganic pollutants on young salmon and trout. Wash. Dept. Fish. Res., Bull. No. 5, 264 p.

Jones, J.R., 1964. Fish and river pollution. Butterworth and Co., London, England.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D. C.

Oseid, D.M. and L.L. Smith, Jr, 1974a. Chronic toxicity of hydrogen sulfide to Gammarus pseudolimnaeus. Trans. Amer. Fish. Soc., 103 (in press).

Oseid, D.M. and L.L. Smith, 1974b. Long-term effects of hydrogen sulfide on Hexagenia limbata (Ephemeroptera). Environmental Ecology (in press).

Oseid, D.M. and L.L. Smith, 1974c. Factors influencing acute toxicity estimates of hydrogen sulfide to freshwater invertebrates. Water Research 8 (in press).

Smith, L.L., 1971. Influence of hydrogen sulfide on fish and arthropods. Environmental Protection Agency, Project 18050 PCG, Washington, D. C.

Smith, L.L. and D.M. Oseid, 1972. Effects of hydrogen sulfide on fish eggs and fry. Water Research, 6: 711.

Smith, L.L., Jr., and D.M. Oseid, 1974. Effect of hydrogen sulfide on development and survival of eight freshwater fish species, In: The early life history of fish. J.H.S. Blaxter (ed.), p. 415-430. Springer-Verlag, New York, N.Y.

Theede, H., et al., 1969. Studies on the resistance of marine bottom invertebrates to oxygen deficiencies and hydrogen sulfide. Mar. Biol., 2: 325.

Van Horn, W.M., 1958. The effect of pulp and paper mill wastes on aquatic life. Proc. Ontario Indust. Waste Conf., 5: 60.

TAINTING SUBSTANCES

CRITERION:

> Materials should not be present in concentrations that individually or in combination produce undesirable flavors which are detectable by organoleptic tests performed on the edible portions of aquatic organisms.

RATIONALE:

Fish or shellfish with abnormal flavors, colors, tastes or odors are either not marketable or will result in consumer complaints and possible rejection of the food source even though subsequent lots of organisms may be acceptable. Poor product quality can and has seriously affected or eliminated the commercial fishing industry in some areas. Recreational fishing also can be affected adversely by off-flavored fish. For the majority of sport fishermen, the consumption of their catch is part of their recreation and off-flavored catches can result in diversion of the sportsmen to other water bodies. This can have serious economic impact on the established recreation industries such as tackle and bait sales and boat and cottage rental.

Water Quality Criteria, 1972 (NAS, 1974) lists a number of wastewaters and chemical compounds that have been found to lower the palatability of fish flesh. Implicated wastewaters included those from 2,4-D manufacturing plants, kraft and neutral sulfite pulping processes, municipal wastewater treatment plants, oily wastes, refinery wastes, phenolic wastes, and wastes from slaughterhouses. The list of implicated chemical compounds is long; it includes cresol and phenol compounds, kerosene, naphthol, styrene, toluene, and exhaust outboard motor fuel. As little as 0.1 ug/l o-chlorophenol was reported to cause tainting of fish flesh.

Shumway and Palensky (1973) determined estimated threshold concentrations for

twenty-two organic compounds. The values ranged from 0.4 ug/l (2,4-dichlorophenol) to 95,000 ug/l (formaldehyde). An additional twelve compounds were tested, seven of which were not found to impair flavor at or near lethal levels.

Thomas (1973) reviewed the literature on tainting substances and listed serious problems that have occurred; he detailed studies and methodology used in the evaluation of the palatability of fishes in the Ohio River as affected by various waste discharges. The susceptibility of fishes to the accumulation of tainting substances is variable and dependent upon the species, length of exposure, and the pollutant. As little as 5 ug/l of gasoline can impart off-flavors to fish (Boyle, 1967).

REFERENCES CITED:

Boyle, H.W., 1967. Taste/odor contamination of fish from the Ohio River. Fed. Water Poll. Cont. Admin., Cincinnati, Ohio.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. U.S. Government Printing Office, Washington, D.C.

Shumway, D.L. and J.R. Palensky, 1973. Impairment of the flavor of fish by water pollutants. U.S. Environmental Protection Agency, EPA-R3-73-010, U.S. Government Printing Office, Washington, D. C.

Thomas, N.A., 1973. Assessment of fish flesh tainting substances. In: Biological methods for the assessment of water quality, J. Carins and K.L. Dickson (eds.) Amer. Society for Testing and Materials, Tech. Publ. 528, Philadelphia, Pennsylvania.

TEMPERATURE

CRITERIA

Freshwater Aquatic Life

For any time of year, there are two upper limiting temperatures for a location (based on the important sensitive species found there at that time):

1. One limit consists of a maximum temperature for short exposures that is time dependent and is given by the species-specific equation:

$$\text{Temperature}_{(C^o)} = (1/b)(\log_{10}[\text{time}_{(\text{min})}] - a) - 2^oC$$

where: $\log_{10}$ = logarithm to base 10. (common logarithm)

a = intercept on the "y" or logarithmic axis of the line fitted to experimental data and which is available from Appendix II-C, NAS, 1974 for some species.

b = slope of the line fitted to experimental data and available from Appendix II-C, NAS, 1974 for some species.

and

2. The second value is a limit on the weekly average temperature that:

   a. in the cooler months (mid-October to mid-April in the north and December to February in the south) will protect against mortality of important species if the elevated plume temperature is suddenly dropped to the ambient temperature, with the limit being the acclimation temperature minus 2^oC when the lower lethal threshold temperature equals the ambient water temperature (in some regions this limitation may also be applicable in summer).

or

   b. In the warmer months (April through October in the north and March through November in the south) is determined by adding to the physiological optimum temperature (usually for growth) a factor calculated as one-third of the difference between the ultimate upper incipient lethal temperature and the optimum temperature for the most sensitive important species (and appropriate life state) that normally is found at that location and time.

or

c. During reproductive seasons (generally April through June and September through October in the north and March through May and October through November in the south) the limit is that temperature that meets site-specific requirements for successful migration, spawning, egg incubation, fry rearing, and other reproductive functions of important species. These local requirements should supersede all other requirements when they are applicable.

or

d. There is a site-specific limit that is found necessary to preserve normal species diversity or prevent appearance of nuisance organisms.

Marine Aquatic Life

In order to assure protection of the characteristic indigenous marine community of a water body segment from adverse thermal effects:

a) the maximum acceptable increase in the weekly average temperature due to artificial sources is 1° C (1.8° F) during all seasons of the year, providing the summer maxima are not exceeded; and

b) daily temperature cycles characteristic of the water body segment should not be altered in either amplitude or frequency.

Summer thermal maxima, which define the upper thermal limits for the communities of the discharge area, should be established on a site-specific basis. Existing studies suggest the following regional limits:

	Short-term Maximum	Maximum True Daily Mean*
Sub-tropical Regions (south of Cape Canaveral and Tampa Bay, Florida, and Hawaii	32.2° C (90° F)	29.4°C (85°F)
Cape Hatteras, N.C., to Cape Canaveral, Fla.	32.2° C (90° F)	29.4° C (85° F)
Long Island (south shore) to Cape Hatteras, N.C.	30.6° C (87° F)	27.8° C (82° F)

(* True Daily Mean = average of 24 hourly temperature readings.)

Baseline thermal conditions should be measured at a site where there is no unnatural thermal addition from any source, which is in reasonable proximity to the thermal discharge (within 5 miles) and which has similar hydrography to that of the receiving waters at the discharge.

INTRODUCTION

The uses of water by man in and out of its natural situs in the environment are affected by its temperature. Offstream domestic uses and instream recreation are both partially temperature dependent. Likewise, the life associated with the aquatic environment in any location has its species composition and activity regulated by water temperature. Since essentially all of these organisms are so called "cold blooded" or poikilotherms, the temperature of the water regulates their metabolism and ability to survive and reproduce effectively. Industrial uses for process water and for cooling is likewise regulated by the water's temperature. Temperature, therefore, is an important physical parameter which to some extent regulates many of the beneficial uses of water. To quote from the FWPCA (1967), "Temperature, a catalyst, a depressant, an activator, a restrictor, a stimulator, a controller, a killer, is one of the most important and most influential water quality characteristics to life in water."

RATIONALE

The suitability of water for total body immersion is greatly affected by temperature. In temperate climates, dangers from exposure to low temperatures is more prevalent than exposure to elevated water temperatures. Depending on the amount of activity by the swimmer, comfortable temperatures range from 20°C to 30°C. Short durations of lower and higher temperatures can be tolerated by most individuals. For example, for a 30-minute period, temperatures of 10°C or 35°C can be tolerated without harm by most individuals (NAS, 1974).

Temperature also affects the self-purification phenomenon in water bodies and therefore the aesthetic and sanitary qualities that exist. Increased temperatures accelerate the biodegradation of organic material both in the overlying water and in bottom deposits which makes increased demands on the dissolved oxygen resources of a given system. The typical situation is exacerbated by the fact that oxygen becomes less soluble as water temperature increases. Thus, greater demands are exerted on an increasingly scarce resource which may lead to total oxygen depletion and obnoxious septic conditions. These effects have been described by Phelps (1944), Camp (1963), and Velz (1970).

Indicator enteric bacteria, and presumably enteric pathogens, are likewise affected by temperature. It has been shown that both total and fecal coliform bacteria die away more rapidly in the environment with increasing temperatures (Ballentine and Kittrell, 1968).

Temperature effects have been shown for water treatment processes. Lower temperatures reduce the effectiveness of coagulation with alum and subsequent rapid sand filtration. In one study, difficulty was especially pronounced below 5°C (Hannah, et al., 1967). Decreased temperature also decreases the effectiveness of chlorination. Based on studies relating chlorine dosage to temperature, and with a 30-minute contact time, dosages required for equivalent disinfective effect increased by as much as a factor of 3 when temperatures were decreased from 20° C to 10° C (Reid and Carlson, 1974). Increased temperature may increase the odor of water because of the increased volatility of odor-causing compounds (Burnson, 1938). Odor problems associated with plankton may also be aggravated.

The effects of temperature on aquatic organisms have been the subject of several comprehensive literature reviews (Brett, 1956; Fry, 1967; FWPCA, 1967; Kinne, 1970) and annual literature reviews published by the Water Pollution Control Federation (Coutant, 1968, 1969, 1970, 1971; Coutant and Goodyear, 1972; Coutant and Pfuderer, 1973, 1974). Only highlights from the thermal effects on aquatic life are presented here.

Temperature changes in water bodies can alter the existing aquatic community. The dominance of various phytoplankton groups in specific temperature ranges has been shown. For example, from 20°C to 25°C, diatoms predominated; green algae predominated from 30°C to 35°C; and blue-greens predominated above 35°C (Cairns, 1956). Likewise, changes from a cold water fishery to a warm water fishery can occur because temperature may be directly lethal to adults or fry; cause a reduction of activity; or limit reproduction (Brett, 1960).

Upper and lower limits for temperature have been established for many aquatic organisms. Considerably more data exist for upper as opposed to lower limits. Tabulations of lethal temperatures for fish and other organisms are available (Jones, 1964: FWPCA, 1967; NAS, 1974). Factors such as diet, activity, age, general health, osmotic stress, and even weather contribute to the lethality of temperature. The aquatic species, thermal acclimation state and exposure time are considered the critical factors (Parker and Krenkel, 1969).

The effects of sublethal temperatures on metabolism, respiration, behavior, distribution and migration, feeding rate, growth and reproduction have been summarized by De Sylva (1969). Another study has illustrated that inside the tolerance zone there is encompassed a more restrictive temperature range in

which normal activity and growth occur; and yet an even more restrictive zone inside that in which normal reproduction will occur (Brett, 1960).

De Sylva (1969) has summarized available data on the combined effects of increased temperature and toxic materials on fish. The available data indicate that toxicity generally increases with increased temperature and that organisms subjected to stress from toxic materials are less tolerant of temperature extremes.

The tolerance of organisms to extremes of temperature is a function of their genetic ability to adapt to thermal changes within their charactertistic temperature range, the acclimation temperature prior to exposure, and the time of exposure to the elevated temperature (Coutant, 1972). The upper incipient lethal temperature or the highest temperature that 50% of a sample of organisms can survive is determined on the organism at the highest sustainable acclimation temperature. The lowest temperature that 50% of the warm acclimated organisms can survive in is the ultimate lower incipient lethal temperature. True acclimation to changing temperatures requires several days (Brett, 1941). The lower end of the temperature accommodation range for aquatic life is $0^{o}C$ in fresh water and somewhat less for saline waters. However, organisms acclimated to relatively warm water, when subjected to reduced temperatures which under other conditions of acclimation would not be detrimental, may suffer a significant mortality due to thermal shock (Coutant, 1972).

Through the natural changes in climatic conditions, the temperatures of water bodies fluctuate daily, as well as seasonally. These changes do not eliminate indigenous aquatic populations, but affect the existing community structure and the geographical distribution of species. Such temperature changes are necessary to induce the reproductive cycles of aquatic organisms and to regulate other life factors (Mount, 1969).

Artifically induced changes such as the return of cooling water or the release of cool hypolimnetic waters from impoundments may alter indigenous aquatic ecosystems (Coutant, 1972). Entrained organisms may be damaged by temperature increases across cooling water condensers if the increase is sufficiently great or the exposure period sufficiently long. Impingement upon condenser screens, chlorination for slime control or other physical insults damage aquatic life (Raney, 1969; Patrick, 1969 (b)). However, Patrick (1969(a)) has shown that algae passing through condensers are not injured if the temperature of the outflowing water does not exceed 34°C to 34.5°C

In open waters elevated temperatures may affect periphyton, benthic invertebrates, and fish in addition to causing shifts in algal predominance. Trembley (1960) studied the Delaware River downstream from a power plant and concluded that the periphyton population was considerably altered by the discharge.

The number and distribution of bottom organisms decrease as water temperatures increase. The upper tolerance limit for a balanced benthic population structure is approximately 32°C. A large number of these invertebrate species are able to tolerate higher temperatures than those required for reproduction (FWPCA, 1967).

In order to define criteria for fresh waters, Coutant (1972) cited the following as currently defineable requirements:

"1. Maximum sustained temperatures that are consistent with maintaining desirable levels of productivity,

2. Maximum levels of metabolic acclimation to warm temperatures that will permit return to ambient winter temperatures should artificial sources of heat cease,

3. Time dependent temperature limitations for survival of brief exposures to temperature extremes, both upper and lower,

4. Restricted temperature ranges for various states of reproduction including (for fish) gametogenesis, spawning migration, release of gametes, development of the embryo, commencement of independent feeding (and other activities) by juveniles, and temperatures required for metamorphosis, emergence or other activities of lower forms,

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

6. Thermal requirements of downstream aquatic life (in rivers) where upstream diminution of a cold water resource will adversely affect downstream temperature requirements."

The major portion of such information that is available, however, is for freshwater fish species rather than lower forms or marine aquatic life.

The temperature-time duration for short term exposures such that 50 percent of a given population will survive an extreme temperature frequently is expressed mathematically by fitting experimental data with a straight line on a semi-logarithmic plot with time on the logarithmic scale and temperature on the linear scale. (See Fig. 1). In equation form this 50 percent mortality relationship is:

$$\log_{10}(\text{time}_{(\text{minutes})}) = a + b\ (\text{Temperature}_{(^\circ C)})$$

where: $\log_{10}$ = logarithm to base 10 (common logarithm)

a = intercept on the "y" or logarithmic axis of the line fitted to experimental data and which is available from Appendix II-C, NAS, 1974 for some species.

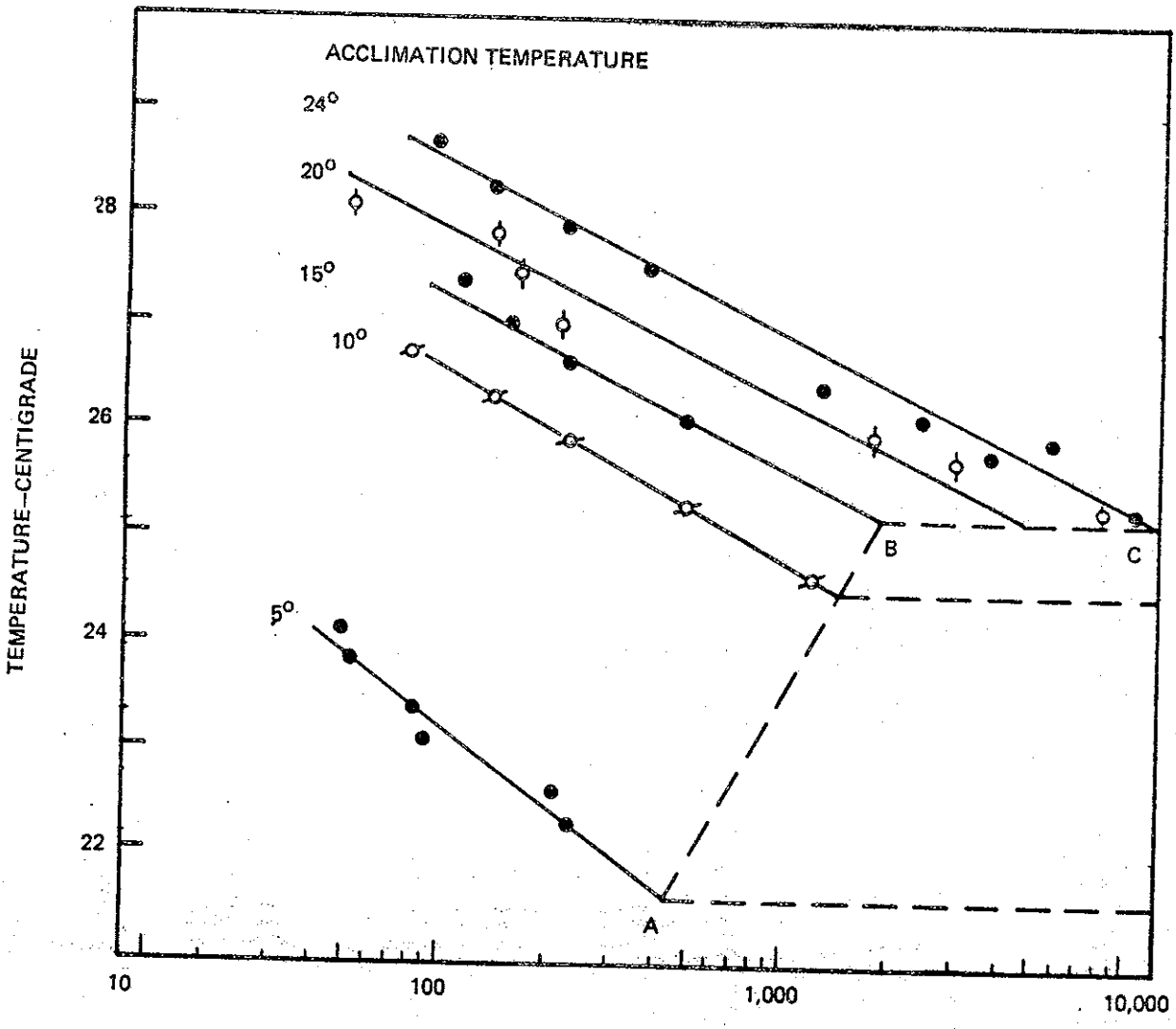
b = slope of the line fitted to experimental data and which is available from Appendix II-C, NAS, 1974 for some species.

To provide a safety factor so that none or only a few organisms will perish, it has been found experimentally that a criterion of 2° C below maximum temperature is usually sufficient (Black, 1953). To provide safety for all the organisms, the temperature causing a median mortality for 50 percent of the population would be calculated and reduced by 2° C in the case of an elevated temperature. Available scientific information includes upper and lower incipient lethal temperatures, coefficients "a" and "b" for the thermal resistance equation, and information on size, life stage, and geographic source of the particular test species (Appendix II-C, NAS, 1974)

Maximum temperatures for an extensive exposure (e.g., more than 1 week) must be divided into those for warmer periods and winter. Other than for reproduction, the most temperature-sensitive life function appears to be growth (Coutant, 1972). Coutant (1972) has suggested that a satisfactory estimate of a limiting maximum weekly mean temperature* is an average of the optimum temperature for growth and the temperature for zero net growth.

---

* maximum weekly mean temperature - true mean temperature for a calendar week which is a higher value than for any other week. Can be determined from continuous measurements, hourly determinations, or some other non-biased statistical method of analyzing temperature data.



AFTER BRETT 1952

Figure 1. MEDIAN RESISTANCE TIMES TO HIGH TEMPERATURES AMONG YOUNG CHINOOK (Oncorhynchus tshawytscha) ACCLIMATED TO TEMPERATURES INDICATED. LINE A-B DENOTES RISING LETHAL THRESHOLD (incipient lethal temperatures) WITH INCREASING ACCLIMATION TEMPERATURE. THIS RISE EVENTUALLY CEASES AT THE ULTIMATE LETHAL THRESHOLD (ultimate upper incipient lethal temperature), LINE B-C. (TAKEN FROM NAS, 1974)

Because of the difficulty in determining the temperature of zero net growth, essentially the same temperature can be derived by adding to the optimum temperature (for growth or other physiological functions) a factor calculated as 1/3 of the difference between the ultimate upper incipient lethal temperature and the optimum temperature (NAS, 1974). In equation form:

$$\begin{array}{l}\text{Maximum weekly} \\ \text{average} \\ \text{temperature}\end{array} = \begin{array}{l}\text{optimum} \\ \text{temperature}\end{array} + 1/3 \begin{pmatrix}\text{ultimate upper} \\ \text{incipient lethal} \\ \text{temperature}\end{pmatrix} - \begin{pmatrix}\text{optimum} \\ \text{temperature}\end{pmatrix}$$

Since temperature tolerance varies with various states of development of a particular species, the criterion for a particular location would be calculated for the most important life form likely to be present during a particular month. One caveat in using the maximum weekly mean temperature is that the limit for short-term exposure must not be exceeded. Example calculations for predicting the summer maximum temperatures for short-term survival and for extensive exposure for various fish species are presented in Table 11. These calculations use the above equations and data from ERL-Duluth, 1976.

The winter maximum temperature must not exceed the ambient water temperature by more than the amount of change a specimen acclimated to the plume temperature can tolerate. Such a change could occur by a cessation of the source of heat or by the specimen being driven from an area by addition of biocides or other factors. However, there are inadequate data to estimate a safety factor for the "no stress" level from cold shocks (NAS, 1974). Figure 2 was developed from available data in the literature (ERL-Duluth, 1976) and can be used for estimating allowable winter temperature increases.

Coutant (1972) has reviewed the effects of temperature on aquatic life reproduction and development. Reproductive events are noted as perhaps the most thermally restricted of all life phases assuming other factors are at or

TABLE 1.1

Example Calculated Values for
Maximum Weekly Average Temperatures for Growth and Short-Term
Maxima for Survival for Juveniles and
Adults During the Summer
(Centigrade and Fahrenheit)

Species	Growth[a]	Maxima[b]
Atlantic Salmon	20 (68)	23 (73)
Bigmouth Buffalo	-	-
Black Crappie	27 (81)	-
Bluegill	32 (90)	35 (95)
Brook Trout	19 (66)	24 (75)
Carp	-	-
Channel Catfish	32 (90)	35 (95)
Coho Salmon	18 (64)	24 (75)
Emerald Shiner	30 (86)	-
Freshwater Drum	-	-
Lake Herring (Cisco)	17 (63)[c]	25 (77)
Largemouth Bass	32 (90)	34 (93)
Northern Pike	28 (82)	30 (86)
Rainbow Trout	19 (66)	24 (75)
Sauger	25 (77)	-
Smallmouth Bass	29 (84)	-
Smallmouth Buffalo	-	-
Sockeye Salmon	18 (64)	22 (72)
Striped Bass	-	-
Threadfin Shad	-	-
White Bass	-	-
White Crappie	28 (82)	-
White Sucker	28 (82)[c]	-
Yellow Perch	29 (84)	-

a - Calculated according to the equation (using optimum temperature for growth)

maximum weekly average temperature for growth = optimum temperature + 1/3 (ultimate incipient lethal temp. - optimum temperature

b - Based on temperature (^oC) = $1/b\ (\log_{10}\ time_{(min.)} - a) - 2^oC$, acclimation at the maximum weekly average temperature for summer growth, and data in Appendix II-C of Water Quality Criteria, 1972 (NAS, 1973).

c - Based on data for larvae (ERL-Duluth, 1976).

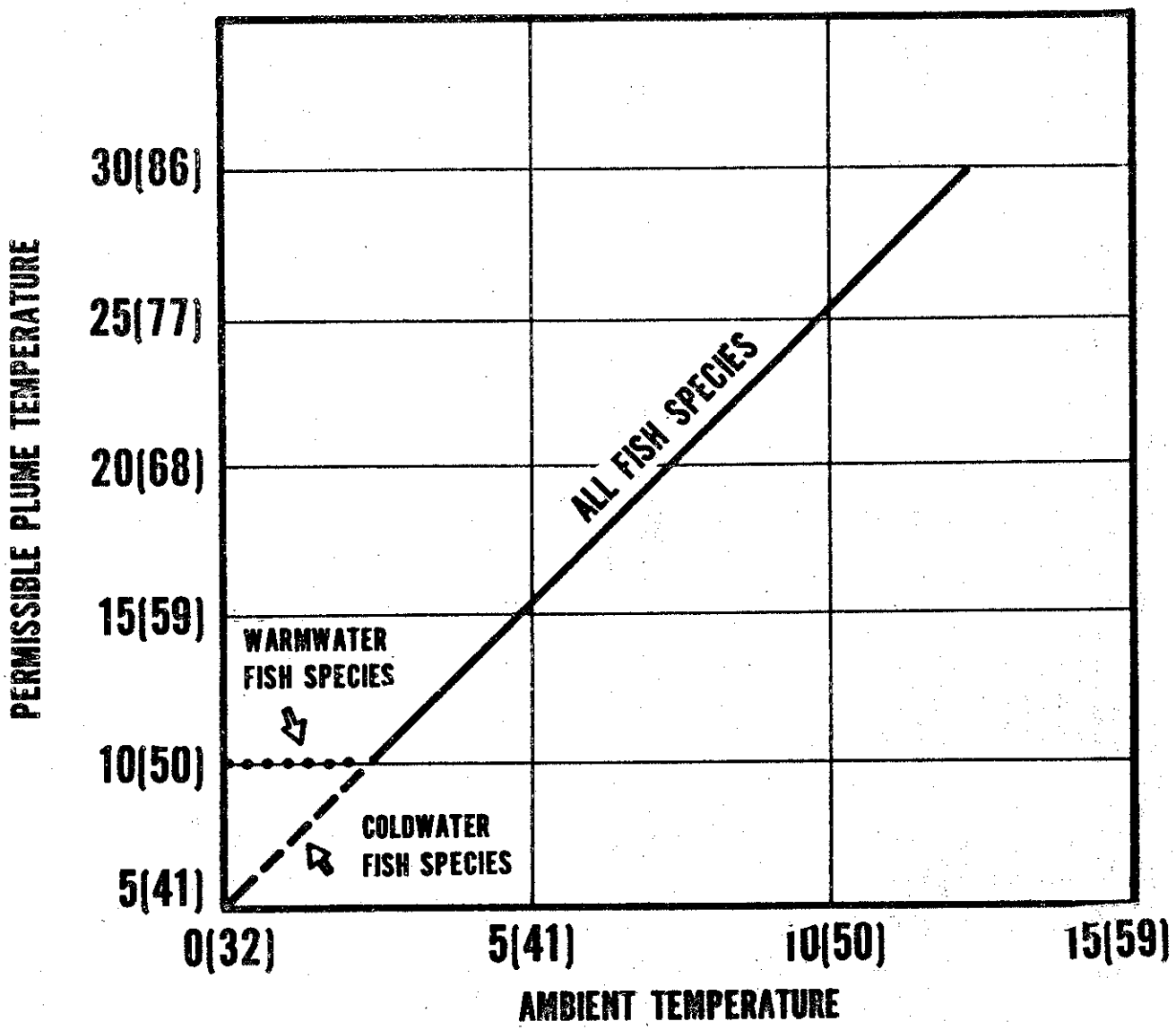


FIGURE 2. GRAPH TO ESTIMATE THE MAXIMUM WEEKLY AVERAGE TEMPERATURE OF PLUMES FOR VARIOUS AMBIENT TEMPERATURES, °C (°F)

near optimum levels. Unnatural short-term temperature fluctuations appear to cause reduced reproduction of fish and invertebrates. There are inadequate data available quantitating the most temperature-sensitive life stages among various aquatic species. Uniform elevation of temperature a few degrees, but still within the spawning range may lead to advanced spawning for spring spawning species and delays for fall spawners. Such changes may not be detrimental unless asynchrony occurs between newly hatched juveniles and their normal food source. Such asynchrony may be most pronounced among anadromous species or other migrants who pass from the warmed area to a normally chilled, unproductive area. Reported temperature data on maximum temperatures for spawning and embryo survival have been summarized in Table 12 (from ERL-Duluth 1976).

Although the limiting effects of thermal addition to estuarine and marine waters are not as conspicuous in the fall, winter and spring as during the summer season of maximum heat stress, nonetheless crucial thermal limitations do exist. Hence, it is important that the thermal additions to the receiving waters be minimized during all seasons of the year. Size of harvestable stocks of commercial fish and shellfish, particularly near geographic limits of the fishery, appear to be markedly influenced by slight changes in the long-term temperature regime (Dow, 1973).

Jefferies and Johnson (1974) studied the relationship between temperature and annual variation in 7-year catch data for winter flounder, Pseudopleuronectes americanus, in Narragansett Bay, Rhode Island. A 78 percent decrease in annual catch correlated closely with a $0.5^{o}C$ increase in the average temperature over the 30-month period between spawning and recruitment into

TABLE 12

Summary of Reported Values for
Maximum Weekly Average Temperature for Spawning and Short-Term
Maxima for Embryo Survival During the Spawning Season
(Centigrade and Fahrenheit)

Species	Spawning[a]		Embryo Survival[b]	
Atlantic Salmon	5	(41)	7	(45)
Bigmouth Buffalo	17	(63)	27	(81)c
Black Crappie	-		-	
Bluegill	25	(77)	34	(93)
Brook Trout	9	(48)	13	(55)
Carp	21	(70)	33	(91)
Channel Catfish	27	(81)	29	(84)
Coho Salmon	10	(50)	13	(55)
Emerald Shiner	24	(75)	28	(82)c
Freshwater Drum	21	(70)	26	(79)
Lake Herring (Cisco)	3	(37)	8	(46)
Largemouth Bass	21	(70)	27	(81)
Northern Pike	11	(52)	19	(66)
Rainbow Trout	9	(48)	13	(55)
Sauger	10	(50)	21	(70)
Smallmouth Bass	17	(63)	-	
Smallmouth Buffalo	17	(63)	21	(70)
Sockeye Salmon	10	(50)	13	(55)
Striped Bass	18	(64)	24	(75)
Threadfin Shad	18	(64)	34	(93)
White Bass	17	(63)	26	(79)
White Crappie	18	(64)	23	(73)
White Sucker	10	(50)	20	(68)
Yellow Perch	12	(54)	20	(68)

a - the optimum or mean of the range of spawning temperatures reported for the species (ERL-Duluth, 1976).

b - the upper temperature for successful incubation and hatching reported for the species (ERL-Duluth, 1976).

c - upper temperature for spawning

the fishery. Sissenwine's 1974 model predicts a 68 percent reduction of recruitment in yellowtail flounder, Limanda ferruginea, with a 1° C long-term elevation in southern New England waters.

Community balance can be influenced strongly by such temperature dependent factors as rates of reproduction, recruitment and growth of each component population. A few degrees elevation in average monthly temperature can appreciably alter a community through changes in inter-species relationships. A 50 percent reduction in the soft-shell clam fishery in Maine by the green crab, Carcinus maenus, illustrates how an increase in winter temperatures can establish new predator-prey relationships. Over a period of four years, there was a natural amelioration of temperature and the monthly mean for the coldest month of each year did not fall below 2° C. This apparently precluded appreciable ice formation and winter cold kill of the green crab and permitted a major expansion of its population, with increased predation of the soft-shell clam resulting (Glude, 1954; Welch, 1968).

Temperature is a primary factor controlling reproduction and can influence many events of the reproductive cycle from gametogenesis to spawning. Among marine invertebrates, initiation of reproduction (gametogenesis) is often triggered during late winter by attainment of a minimum environmental threshold temperature. In some species, availability of adequate food is also a requisite (Pearse, 1970; Sastry, 1975; deVlaming, 1971). Elevated temperature can limit gametogenesis by preventing accumulation of nutrients in the gonads. This problem could be acute during the winter if food availability and feeding activity is reduced. Most marine organisms spawn during the spring and summer; gametogenesis is usually initiated during the previous fall. It should also be noted that there are some species which

spawn only during the fall (herring), while others spawn during the winter and very early spring. At the higher latitudes, winter breeders include such estuarine community dominants as acorn barnacles, Balanus balanus, and B. balanoides, the edible blue mussel Mytilus edulis, sea urchin, Strongylocentrotus drobachiensis, sculpin, and the winter flounder, Pseudopleuronectes americanus. The two boreal barnacles require temperatures below 10°C before egg production will be initiated (Crisp, 1957). It is clear that adaptations for reproduction exist which are dependent on temperature conditions close to the natural cycle.

Juvenile and adult fish usually thermoregulate behaviorally by moving to water having temperatures closest to their thermal preference. This provides a thermal environment which approximates the optimal temperature for many physiological functions, including growth (Neill and Magnuson, 1974). As a consequence, fishes usually are attracted to heated water during the fall, winter, and spring. Avoidance will occur as water temperature exceeds the preferendum by 1 to 3° C (Coutant, 1975). This response precludes problems of heat stress for juvenile and adult fishes during the summer, but several potential problems exist during the other seasons. The possibility of cold shock and death of plume-entrained fish due to winter plant shutdown is well recognized. Also, increased incidence of disease and a deterioration of physiological condition has been observed among plume-entrained fishes, perhaps due to insufficient food (Massengill, 1973). A weight loss of approximately 10% for each 1°C rise in water temperature has been observed in fish when food is absent (Phillips et al., 1960) There may also be indirect adverse effects on the indigenous community due to increased predation pressure if thermal addition leads to a concentration of fish which are dependent on this community for their food.

Fish migration is often linked to natural environmental temperature cycles. In early spring, fish employ temperature as their environmental cue to migrate northward (e.g., menhaden, bluefish) or to move inshore (winter

flounder). Likewise, water temperature strongly influences timing of spawning runs of anadromous fish into rivers (Leggett and Whitney, 1972). In the autumn, a number of juvenile marine fishes and shrimp are dependent on a drop in temperature to trigger their migration from estuarine nursery grounds for oceanic dispersal or southward migration (Lund and Maltezos, 1970; Talbot, 1966).

Thermal discharges should not alter diurnal and tidal temperature variations normally experienced by marine communities. Laboratory studies show thermal tolerance to be enhanced when animals are maintained under a diurnally fluctuating temperature regime rather than at a constant temperature (Costlow and Bookhout, 1971; Furch, 1972; Hoss, et al., 1975). A daily cyclic regime can be protective additionally as it reduces duration of exposure to extreme temperatures (Pearce, 1969; Gonzalez, 1972).

Summer thermal maxima should be established to protect the various marine communities within each biogeographic region. During the summer, naturally elevated temperatures may be of sufficient magnitude to cause death or emigration (Chin, 1961; Glynn, 1968; Vaughn, 1918) This more commonly occurs in tropical and warm temperate zone waters, but has been reported for enclosed bays and shallow waters in other regions as well (Nichols, 1918). Summer heat stress also can contribute to increased incidence of disease or parasitism (Sinderman, 1965); reduce or block sexual maturation (Thorhaug, et al., 1971; deVlaming, 1972); inhibit or block embryonic cleavage of larval development (Calabrese, 1969); reduce feeding and growth of juveniles and adults (Olla and Studholme, 1971);

result in increased predation (Gonzalez, 1972); and reduce productivity of macroalgae and seagrasses (South and Hill, 1970; Zieman, 1970). The general ceilings set forth here are derived from studies delineating limiting temperatures for the more thermally sensitive species or communities of a biogeographic region.

Thermal effects data are presently insufficient to set general temperature limits for all coastal biogeographic regions. The data enumerated in the Appendix, plus any additional data subsequently generated, should be utilized to develop thermal limits which specifically consider communities relevant to given water bodies.

REFERENCES CITED:

Ballentine, R.K. and F.W. Kittrell, 1968. Observations of fecal coliforms in several recent stream pollution studies. Proceedings of the Symposium on Fecal Coliform Bacteria in Water and Wastewater, May 21-22, 1968, Bureau of Sanitary Engineering, California State Department of Public Health.

Black, E.C., 1953. Upper lethal temperatures of some British Columbia freshwater fishes. Jour. Fish. Res. Bd. Can., 10: 196.

Brett, J.R., 1941. Tempering versus acclimation in the planting of speckled trout. Trans. Amer. Fish. Soc., 70: 397.

Brett, J.R., 1956. Some principals in the thermal requirements of fishes. Quarterly Rev. Biol., 31: 75.

Brett, J.R., 1960. Thermal requirements of fish--three decades of study, 1940-1970. In: Biological problems in water pollution. C.M. Tarzwell (ed.) Dept. Health, Education and Welfare, Public Health Service.

Burnson, B., 1938. Seasonal temperature variations in relation to water treatment. Jour. Amer. Water Works Assn., 30: 793.

Cairns, Jr., J., 1956. Effects of increased temperatures on aquatic organisms. Industrial Wastes, 1: 50.

Calabrese, A., 1969. Individual and combined effects of salinity and temperature on embryos and larvae of the coot clam, Mulinia lateralis (say). Biol. Bull. 137, 3: 417.

Camp, T.R., 1963. Water and its impurities. Reinhold Publishing Corp., New York, New York.

Chin, E., 1961. A trawl study of an estuarine nursery area in Galveston Bay with particular reference to penaeid shrimp. Ph.D. Dissertation, University of Washington.

Costlow, Jr., J.D. and C.G. Bookhout, 1971. The effect of cyclic temperatures on larval development in the mud crab, Rhithropanopeus harrisii. In: Fourth European Marine Biology Symposium. D.I. Crisp (Ed.), Cambridge University Press, London.

Coutant, C.C., 1968. Thermal pollution--biological effects: A review of the literature of 1967. Jour. Water Poll. Cont. Fed., 40: 1047.

Coutant, C.C., 1969. Thermal pollution--biological effects: A review of the literature of 1968. Jour. Water Poll. Cont. Fed., 41: 1036.

Coutant, C.C., 1970. Thermal pollution--biological effects: A review of the literature of 1970. Thermal pollution--biological effects: A review of the literature of 1969. Jour. Water Poll. Cont. Fed., 42: 1025.

Coutant, C.C., 1971. Thermal pollution--biological effects: A review of the literature of 1970. Jour. Water Poll. Cont. Fed., 43: 1292.

Coutant, C.C., 1972. Biological aspects of thermal pollution, II. Scientific basis for water temperature standards at power plants. CRC Critical Rev. in Environ. Cont., 3: 1.

Coutant, C.C., 1975. Temperature selection by fish--a factor in power plant impact assessments. In: Symposium on the physical and biological effects on the environment of cooling systems and thermal discharges at nuclear power stations. Int'l Atomic Energy Agency (In Press).

Coutant, C.C., and C.P. Goodyear, 1972. Water pollution--thermal pollution: A review of the literature of 1971. Jour. Water Poll. Cont. Fed., 44: 1250.

Coutant, C.C. and H.A. Pfuderer, 1973. Water pollution--thermal effects: A review of the literature of 1973. Jour. Water Poll. Cont. Fed., 45: 1331.

Coutant, C.C. and H.A. Pfuderer, 1974. Water pollution--thermal effects: A review of the literature of 1974. Jour. Water Poll. Cont. Fed., 46:1476.

Crisp, D.J., 1957. Effect of low temperature on the breeding of marine animals. Nature, 179: 1138.

De Sylva, D.P., 1969. Theoretical considerations of the effects of heated effluents on marine fishes. In: Biological aspects of thermal pollution. P.A. Krenkel and F. C. Parker (eds.), Vanderbilt University Press.

deVlaming, V.L., 1972. The effects of temperature and photoperiod on reproductive cycling of the estuarine gobiid fish, Gillichthys mirabilis. Fishery Bull. 70, 4:1137.

deVlaming, V.L., 1971. The effects of food deprivation and salinity changes on reproductive function in the estuarine gobiid fish, Gillichthys mirabilis. Biol. Bull., 141: 458.

Dow, R.L., 1973. Fluctuations in marine species abundance during climatic cycles. Mar. Tech. Soc. Jour. 7,4:38.

ERL-Duluth, Environmental Research Laboratory, 1976, Procedures for developing temperature criteria for freshwater fish. Ecological Research Series, Report in Preparation.

Federal Water Pollution Control Administration, 1967. Temperature and aquatic life. Laboratory Investigations-No. 6, Technical Advisory and Investigations Branch, Cincinnati, Ohio.

Fry, F.E.J., 1967. Responses of vertebrate poikilotherms to temperature. In: Thermobiology. A.H. Rose (Ed.), Academic Press, New York

Furch, K., 1972. Der Einfluss einer Vorbehandlung mit konstanten und wechselnden temperaturen auf die hitzeresistenz von Gammarus salinus und Idotea balthica. Mar. Biol., 15: 12.

Glude, J.B., 1954. The effects of temperature and predators on the abundance of the soft-shell clam, Mya arenaria, in New England. Trans. Amer. Fish. Soc., 84: 13.

Glynn, P.W., 1968. Mass mortalities of echinoids and other reef flat organisms coincident with midday, low water exposures in Puerto Rico. Mar. Biol. 3, 3: 226.

Gonzalez, J.G., 1972. Seasonal variation in the responses of estuarine populations to heated water in the vicinity of a steam generating plant. Ph.D. Dissertation, Univ. Rhode Island, 142 p.

Hannah, S.A., J.M. Cohen, and G.G. Robeck, 1967. Control techniques for coagulation-filtration. Jour. Amer. Water Works Assn., 59: 1149.

Hoss, D.I., et al., 1975. Effects of temperature, copper and chlorine on fish during simulated entrainment in power plant condenser cooling systems. Symp. Physical and Biological Effects on the Environment of Cooling Systems and Thermal Discharges at Nuclear Power Plants. Int'l. Atomic Energy Agency (In Press).

Jeffries, H.P. and W.C. Johnson, 1974. Seasonal distributions of bottom fishes in the Narragansett Bay area: Seven-year variations in the abundance of winter flounder (Pseudopleuronectes americanus). Jour. Fish. Res. Bd. Can., 31: 1057.

Jones, J.R.E., 1964. Fish and river pollution. Butterworth and Co., Ltd., London.

Kinne, O., 1970. Temperature-animals-invertebrates. In: Marine ecology. O. Kinne (ed.), John Wiley and Sons, New York.

Leggett, W.C. and R.R. Whitney, 1972. Water temperature and the migrations of American shad, Fish. Bull. 70, 3: 659.

Lund, W.A. and G.C. Maltezos, 1970. Movements and migrations of the bluefish, Pomatomus saltatrix, tagged in waters of New York and southern New England. Trans. Amer. Fish. Soc., 99:719

Massengill, R.R., 1973. Change in feeding and body condition of brown bullheads overwintering in the heated effluent of a power plant. Ches. Sci. 14, 2: 138.

Mount, D.I., 1969. Developing thermal requirements for freshwater fishes. In: Biological aspects of thermal pollution. P.A. Krenkel and F.L. Parkers, (eds.), Vanderbilt University Press.

National Academy of Sciences, National Academy of Engineering, 1974. Water quality criteria, 1972. A report of the Committee on Water Quality

Criteria. U.S. Government Printing Office, Washington, D.C.

Neill, W.H. and J.J. Magnuson, 1974. Distributional ecology and behavioral thermoregulation of fishes in relation to heated effluent from a power plant at Lake Monona, Wisconsin. Trans. Amer. Fish. Soc. 103, 4: 663.

Nichols, J.T., 1918. An abnormal winter flounder and others. Copeia No., 55: 37.

Olla, B.L. and A.L. Studholme, 1971. The effect of temperature on the activity of bluefish, Pomatomus saltatrix (1.). Biol. Bull., 141: 337.

Parker, F.L. and P.A. Krenkel, 1969. Thermal pollution: Status of the art. Report No. 3, Vanderbilt University, School of Engineering, Nashville, Tennessee.

Patrick, R., 1969a. Some effects of temperature on freshwater algae. In: Biological aspects of thermal pollution. P.A. Krenkel and F.L. Parker, (eds.), Vanderbilt University Press.

Patrick, R., 1969b. Discussion of engineering aspects, sources, and magnitude of thermal pollution. By P.A. Krenkel and F.L. Parker. In: Biological aspects of thermal pollution. P.A. Krenkel and F.L. Parker, (eds.), Vanderbilt University Press.

Pearce, J.B., 1969. Thermal addition and the benthos, Cape Cod Canal. Ches. Sci., 10: 227.

Pearse, J.S., 1970. Reproductive periodicities of Indo-Pacific invertebrates in the Gulf of Suez. III. The echinoid Diadema setosum (Leske). Bull. Mar. Sci. 20, 3: 697.

Phelps, E.B., 1944. Stream sanitation. John Wiley and Sons, Inc., New York.

Phillips, A.M., Jr., D.L. Livingston and R.F. Dumas, 1960. Effects of starvation on the chemical composition of brook trout. Prog. Fish. Cult., 22: 147.

Reid, L.C. and D.A. Carlson, 1974. Chlorine disinfection of low temperature waters. Jour. Environ. Eng. Div., ASCE, Vol. 100, No. EE2: 339.

Raney, E.C., 1969. Discussion of effects of heated discharges on freshwater fish in Britain. By J.S. Alabaster. In: Biological aspects of thermal pollution. P.A. Krenkel and F.L. Parker (eds.), Vanderbilt University Press.

Sastry, A.N., 1975 Physiology and ecology of reproduction in marine invertebrates. In: F.J. and W.B. Vernberg (eds.), Physiological ecology of estuarine organisms. Belle W. Baruch Library of Marine Science, Univ. So. Carolina Press, Columbia, South Carolina.

Sinderman, C.J. 1965. Effects of environment on several diseases of herring from the western North Atlantic. Spec. Publ. Comm. Northw. Atlantic Fish. No. 6 603-610.

Sissenwine, M.P., 1974. Variability in recruitment and equilibrium catch of the southern New England yellowtail flounder fishery. Jour. du Conseil (In Press).

South, G.E. and R.D. Hill, 1970. Studies on marine algae of Newfoundland. I. Occurrence and distribution of free living Ascophyllum nodosum in Newfoundland. Can. Jour. Bot., 48: 1697.

Talbot, G.B., 1966. Estuarine environmental requirements and limiting factors for striped bass. Trans. Amer. Fish. Soc. Special Pub. 3: 37.

Thorhaug, A., et al., 1971. Laboratory thermal tolerances. In: R.G. Baderond B.A. Roessler (eds.), An ecological study of South Biscayne Bay and Card Sound. Progress Report to USAEC and Fla. Power and Light Co. (Univ. Miami, Coral Gables, Fla.), p. 11-31.

Trembley, F.J., 1960. Research project on effects of condenser discharge water on aquatic life, progress report, 1956-1959. The Institute of Research, Lehigh University.

Vaughan, T.W., 1918. The temperature of the Florida coral-reef tract. Publs. Carnegie Inst. Wash., 213: 319.

Velz, C.J., 1970. Applied stream sanitation. Wiley-Interscience, New York.

Welch, W.R., 1968. Changes in abundance of the green crab, Carcinus maenas (L.), in relation to recent temperature changes. Fish. Bull. 67, 2: 337.

Zieman, J.C., Jr., 1970. The effects of a thermal effluent stress on the sea grasses and macroalgae in the vicinity of Turkey Point, Biscayne Bay, Florida. Ph.D. Dissertation, Univ. Miami, Coral Gables, Fla., 129 p.

Appendix

A Summary of the Thermal Effects Literature for the United States Estuarine and Coastal Biota

Recommended marine thermal criteria are based on scientific evaluation of available data. Representative thermal effects data are summarized here for an array of ecologically diverse marine organisms, grouped by biotic region. Since the summer temperature regime can provide "worst case" thermal conditions, studies dealing with warm-acclimated organisms are cited primarily. Findings of sublethal effects studies are listed also. Twenty-four-hour TLm (median tolerance limit) data have been adjusted by subtracting 2.2°C to estimate the upper thermal protection limit for the life history stage in question (Mihursky, 1969). Recognized biological variables such as recent environmental history, nutritional state, size, sex, and age have been considered for all thermal effects investigations. Likewise, contrasting methods of study were considered. Normally, thermal effects data derived in one biotic region should not be applied to another. Latitudinally separated populations of widely distributed species may exhibit significant genetic variability and usually have experienced different, recent environmental histories. Boundaries for regional ceilings are demarcated by biogeographic provinces. Species composition of the marine system, and most important, responses to elevated

temperature, are generally similar within a region. Boundaries of a biotic province are characterized by significant thermal discontinuities. Boundary areas are maintained during summer or winter due to combined forces of current, wind, and coastal geomorphology. On the east coast, Cape Canaveral, Fla.; Cape Hatteras, North Carolina; and Cape Cod, Massachusetts, represent these boundaries. On the west coast, Point Conception in southern California marks the limit of warm and cold temperate zones.

Boreal Zone, Atlantic Coast: This region extends from Cape Cod, Mass., to the Gulf of Maine. Insufficient data are available for setting regional temperature limits. Upper limits should be determined on a case-by-case basis using best available data for the site and its environs.

In the boreal region, maintenance of a general temperature regime resembling natural conditions is particularly important during winter months. Some boreal species require periods of uninterrupted low water temperatures to fulfill environmental requirements for successful maturation of sexual products, spawning, and subsequent egg and larval survival. Winter flounder, Pseudopleuronectes americanus, have an upper limit for spawning of 5.5°C (Bigelow and Schroeder, 1953). Spawning occurs during the winter.

Ten degrees centigrade is the upper thermal limit for Atlantic salmon, Salmo salar, smolt migration to the sea, which normally occurs in June. Twelve degrees centigrade inhibits maturation of sex products (DeCola, 1970). Development of winter flounder, Pseudopleuronectes americanus, eggs to hatching is reduced 50 percent at 13°C (Rogers, in press). Blood worm, Glycera americana, spawning is induced when temperatures reach 13°C (Creaser, 1973). Fifteen degrees centigrade is the upper limit for spawning Atlantic herring, Clupea harengus, (Hela and Laevastu, 1962) and of an amphipod, Psammonx nobilis, (Scott, 1975). In Atlantic herring, there is above normal incidence of a protozoan disease at 15°C (Sinderman, 1965) and at 16°C, there is a prevalence of erythrocyte degeneration (Sherburne, 1973). Field mortality of yellowtail flounder larvae, Limanda ferruginea, was observed at 17.8°C (Colton, 1959). The protection limit for yearling Atlantic herring, Clupea harengus, (48-hr. TLm - 2.2°C) is 19.0°C (Brawn, 1960). At 21°C, embryonic development ceases in the amphipod, Gammarus duebeni, (Steele and Steele, 1969). Above 21.2°C, spores are killed and growth is reduced in the macroalga, Chondrus crispus, which is commercially harvested as Irish moss (Prince and Kingsbury, 1973).

Cold Temperate Zone, Atlantic Coast: Temperature ceilings are particularly critical in the southern portion of this region (south shore of Long Island to Cape Hatteras, N.C.) where enclosed sounds and large coastal-plain bays and rivers are

prevalent. Maximum temperatures should not exceed 30.6°C. Were temperatures of 30°C to persist for over 4 to 6 hours, appreciable stress or direct mortality would occur among juvenile winter flounder, Pseudopleuronectes americanus; striped mullet larvae, Mugil cephalus; Atlantic silverside eggs and adults, Menidia menidia; adult northern puffer, Sphaeroides maculatus; adult blue mussel, Mytilus edulis; and adult soft shell clam, Mya arenaria. Specific critical temperatures for these species are detailed in Table 13 The adult protection limit (TLm - 2.2°C) is 28.8°C for sand shrimp, Crangon septemspinosa, and 30.8°C for opossum shrimp, Neomysis americanus. Both are important food organisms for fish (Mihursky and Kennedy, 1967). Respiration rate is depressed above 30°C in the mole crab, (Edwards and Irving, 1943). At 31.5°C, there is 67 percent mortality in coot clams when exposed for 6 hours (Kennedy, et al., 1974).

A true daily mean limit of 27.8°C approximates the upper limit for larval growth of the coot clam (27.5°C; Calabrese, 1969). Between 28°C and 30°C juvenile amphipods, Corophium insidiosum, leave their tubes and thereby lose natual protection from predation (Gonzalez, 1972). Such elevated temperatures may also have subtle sublethal effects, such as reducing feeding and growth. In the quahaug, Mercenaria mercenaria, growth is optimum at 20°C (Ansell, 1968). Growth is inhibited above 24°C in a rock weed, Ascophyllum nodosum, (South and Hill, 1970). Prolonged

13

TABLE SELECTED THERMAL REQUIREMENTS & LIMITING TEMPERATURE DATA
COLD TEMPERATE ZONE, ATLANTIC COAST:
South of Long Island, N.Y. to Cape Hatteras, N.C.

Temperature °C	Temperature °F	Effect	Species	Seasonal Occurrence	Reference
30	86.0	Avoidance response breakdown (CTM)	Morone saxatilis (striped bass)	April-November	Gift & Westman, 1971
29.8	85.6	Behavior-reduced feeding and behavior altered	Pomatomus saltatrix (bluefish)	May-October	Olla, et al 1969
29.4	84.9	Survival-eggs (50% optimal survival)	Menidia menidia (Atlantic silverside)	May-June	Everich & Keves, 1973 (unpublished)
29.1	84.3	Survival-larvae (TLm)	Mugil cephalus (striped mullet)	January-April (coastal waters)	Cortenay & Roberts, 1973
29.0	84.2	Survival-adult protection limit (TLm - 2.2°C)	Sphaeroides maculatus (Northern puffer)	January-December	Hoff & Westman, 1966
29.0	84.2	Avoidance response	Brevoortia tyrannus (Atlantic menhaden)	April-October	Meldrim & Gift, 1971
29.2	82.7	Survival-adult protection limit (TLm - 2.2°C)	Menidia menidia (Atlantic silverside)	April-November	Hoff & Westman, 1966
28.0	82.4	Survival-adult limit	Mya arenaria (soft shell clam)	January-December	Pfitzenmeyer (pers comm)
27.5	81.5	Development-upper limit larval development	Mulinia lateralis (coot clam)	March-October	Calabrese, 1969
26.9	80.4	Survival-juvenile protection limit (TLm - 2.2°C)	Pseudopleuronectes americanus (winter flounder)	April-December	Hoff & Westman, 1966
26.5	79.7	Avoidance response	Cynoscinn regalis (sea trout)	May-October	Gift & Westman, 1971
26.0	78.8	Survival-adult	Mytilus edulis (blue mussel)	January-December	Gonzalez, 1972
25.5	77.9	Avoidance response	Leiostomus xanthrus (spot)	January-December	Gift & Westman, 1971
24.8	76.7	Occurrence-maximum temperature for occurrence in Chesapeake Bay	Uronhycis regius (spotted hake)	January-December	Barans, 1972
24.6	76.2	Survival-larvae (TLm)	Menidia menidia (Atlantic silverside)	May-June	Everich & Keves, 1973 (unpublished)

Compiled by ERL-Narragansett, 1976

locomotion is markedly reduced at 22°C in the Jonah crab, Cancer borealis; at 28°C in Cancer irroratus (Jeffries, 1967). An oyster pathogen, Democystidium marinum, readily proliferates above 25°C (Andrews, 1965).

High temperature usually will elicit avoidance response in fishes. Avoidance is triggered at 29°C in Atlantic menhaden, Brevoortia tyrannus, and at 26.5°C in sea trout, Cynoscion regalis, (Meldrin and Gift, 1971). Breakdown of the avoidance response in striped bass, Morone saxatilis, occurs at 30°C (Gift and Westman, 1971). Maximum reported temperature for capture of spotted hake, Urophycis regius, is 24.8°C in the Chesapeake Bay (Barans, 1972).

North of Long Island, a 1.0°C rise above summer ambient provides reasonable protection. For example, maximum short-term temperatures in Narragansett Bay, Rhode Island, usually would not exceed 23.4°C in August, judging from 15-year mean temperature data for Fox Island. Larval Atlantic silverside, juvenile winter flounder, and blue mussel should be protected by that thermal limitation. The thermal protection limit (TLm - 2.2°C) for juvenile winter flounder is 26.9°C (Gift and Westman, 1971

Repeated exposures to 25°C would stress the blue mussel, Mytilus edulis, by causing cessation of feeding (Gonzalez, 1972). Diurnal summer maxima exceeding 22°C can alter normal metabolic rates in embryonic tautog, Tautoga onitis, (Laurence, 1973) and cause feeding problems for adult winter flounder (Olla, 1969) and the sand-collar snail, Polinices duplicata, (Hanks, 1953).

Optimum for summer development of the rock crab, Cancer irroratus, and Jonah crab, C. borealis, larvae is 20°C; at 25°C, mortality precludes completion of larval development (Sastry and Vargo, in press). Between 15 and 20°C, activity of the amphipod, Gammarus oceanicus, is much reduced (Halcrow and Boyd, 1967). Initiation of spawning is often cued by temperature. Blue mussel spawning occurs when spring temperatures reach 12°C (Engle and Loosanoff, 1944). A minimum of 10°C is required for their embryonic development (Brenko and Calabrese, 1969) and spawning occurs at 15°C. Peak spawning runs of American shad, Alosa sapidissima, into rivers occurs at 19.5°C (15 year average, Connecticut River); downstream migration of juveniles occurs as temperature falls below 15.5°C (Leggett and Whitney, 1972). Menhaden migrate at 10°C (Bigelow and Schroeder, 1953); striped bass, Morone saxatilis, migrate into or leave rivers at 6 to 7.5°C (Merriman, 1941). In the fall and winter, fishes congregate in discharge plumes which exceed these temperatures. These fishes exhibit increased incidence of disease and a general loss of physiological condition (Mihursky, et al; 1970).

Warm Temperate Zone, Atlantic and Gulf Coasts: This region extends from Cape Hatteras, N.C., to Cape Canaveral, Florida, and

on the Gulf Coast from Tampa, Florida, to Mexico. The recommended regional ceiling is a short-term maximum of 32.2°C . Prolonged exposures to temperatures near this level would adversely affect portions of the biota. At 33°C, bay anchovy, Anchoa mitchilli, embryonic development is reduced to 50 percent of optimum (Rebel, 1973). The upper limit for growth of juvenile white shrimp, Penaeus setiferus, is 32.5°C (Zein-Eldin and Griffith, 1969). A decline in field abundance of brown shrimp, Penaeus aztecus, at temperatures above 30°C was reported by Chin (1961).

Protection limits (50 percent of optimal survival) of two sardines, Harengula jaguana and Harengula pensacolae, for development of the yolk sac larval stage are 31.4°C and 32.2°C, respectively (Rebel, 1973; Sakensa, et al., 1972). Larval pinfish, Lagodon rhomboides, and spot, Leiostomus xanthurus, exhibit a breakdown in avoidance response mechanisms at 31.0°C and 31.1°C, respectively (Hoss, D.E., et al., 1974). The protection limit (TLm - 2.2°C) for young-of-the-year Atlantic menhaden is 30.8°C (Lewis and Hettler, 1968). Upper limit for adult growth of the quahaug, Mercenaria mercenaria, is 31°C (Ansell, 1968).

Daily mean temperatures continually exceeding 29°C would result in mortality of striped mullet eggs, Mugil cephalus.

Their 96-hour TLm is 26.4°C (Courtenay and Roberts, 1973). Egg and yolk sac larval survival of sea bream, Archosargus rhomboidalis, is reduced to 50 percent of optimal at 29.1°C. For yellowfin menhaden, Brevoortia smithi, exposure to 29.8°C reduced survival of egg and yolk sac larvae to 50 percent of optimal (Rebel, 1973). Sublethal but potentially damaging ecological effects could occur at levels well below 29°C. For example, the upper limit for optimal growth of post-larval brown shrimp, Penaeus aztecus, is 27.5°C (Zein-Eldin and Alrich, 1965). Developing embryos and fry of striped bass cannot tolerate 26.7°C in fresh water (Shannon, 1969). This report may also apply to fry in waters at the head of estuaries. This species spawns in early spring. Elevation of winter temperatures above 20°C in St. Johns River, Florida, could interfere with upstream migration of American shad, Alosa sapidissima, (Leggett and Whitney, 1972).

Sub-Tropical Regions: Ceilings for sub-tropical regions such as south Florida (Cape Canaveral and Tampa southward), and Hawaii are an instantaneous maximum 32.2°C and a true daily mean not exceeding 29.4°C. Ceilings for true tropical sites should be developed from studies of indigenous populations of relevant communities. Physiological variation in thermal adaptations and tolerances have been reported for coral between sub-tropical (Hawaii, 19-22° Lat.) and true tropical sites (Eniwetok Atol, Marshall Islands, 11° Lat.) (Jokiel, et al., in press).

Much of the following thermal effects data represent southern Florida or Hawaiian biota. A review by Zieman and Wood (1975) suggests that the thermal optimum is 26-28°C for tropical marine systems, with chronic exposure to temperatures between 28°C and 30°C causing heat stress. Death of the biota is readily discernible between 30°C and 32°C. Mayer (1914) recognized that nearshore

tropical marine biota normally live at temperatures only a few degrees below their upper lethal limit. A study of elevated temperature effects on the benthic community in Biscayne Bay, Florida, resulted in the following data (Roessler, 1974):

Phylum	Temperature for High Species Diversity (°C)	Temperature for 50 Percent Species Exclusion (°C)
Molluscs	26.7	31.4
Echinoderms	27.2	31.8
Coelenterates	25.9	29.5
Porifera	24.0	31.2

Other thermal data for tropical biota include a 25.4-27.8°C optimum for fouling community larval settlement (Roessler, 1974); 25°C optimum for larval development of Polyonyx gibbesi, a commensal crab (Gore, 1968); 27°C for growth and gonad development in sea urchins, Lytechinus variegatus, and for growth in a snail, Cantharus tinctus, (Albertson, 1973); 27 to 28°C optimum for larval development of pink shrimp, Penaeus duorarum, (Thorhaug, et al., 1971a); and 30°C optium for turtle grass, Thalassia testudinum, productivity (Zieman, 1970). Kuthalingham (1959) studied thermal tolerance of newly hatched larvae of ten tropical marine fishes in the laboratory. When held at a series of constant temperatures for 12 hours immediately following

hatch, optimal survival for all species fell between 28°C to 30°C, but their tolerance limit ranged from 30°C to 32°C.

Thermal stress of the fouling community is seen in a 50 percent reduced settlement rate of larvae at 28°C (Roessler, 1974). Fifty percent reduction in gonadal volume of the sea urchin, Lytechinus variegatus, occurs at 29.9°C (Thorhaug, et al., 1971b). Irreversible plasmyolysis of the macroalga, Valonia ventricosa, at 29.9°C and of Valonia macrophysa at temperatures above 29.7°C has been reported. Survival of developing embryos to the yolk sac larval stage was reduced to 50 percent of optimal at 29.1°C among sea bream, Archosargus rhomboidalis. At 29.8°C, yellowfin menhaden, Brevoortia smithi, and at 31.4°C scaled sardines, Harengula jaguana, suffer similar mortalities during early development (Rebel, 1973). Temperatures in excess of 31°C to 33°C can interfere with embryonic development in six species of mangrove-associated nematodes, even though adults can tolerate an additional 2°C to 7°C (Hopper, et al., 1973). Upper limit for larval (naupliar) metamorphosis in pink shrimp, Penaeus duorarum, is 31.5°C (Thorhaug, et al., 1971b). Upper lethal temperatures include 31.5°C for five species of Valonia (Thorhaug, 1970); death in 3 to 8 hours for five Hawaiian corals at 31-32°C (Edmondson, 1928; Jokiel and Coles, 1974); 32°C Tlm (95-hour) for the sea squirt, Ascidia nigra, and sea urchin, Lytechinus variegatis, (Chesher, 1971). Average daily temperatures near 31°C

for three to ten days result in decreased growth in seagrass, Thallassia testudinum and red macroalgae, Laurencia poitei. Between 32°C and 33°C, health and abundance of these species declines markedly (Thorhaug, 1971, 1973). Replacement of seagrass is slow, especially if rhizomes are damaged due to excessive consumption of stored starch during heat stress (Zieman, 1970). Recovery of Thallassia beds may take decades (Zieman and Wood, 1975).

Pacific Coast: Fewer thermal effects studies have been conducted on West Coast species. However, the concept of seasonal restrictions for temperature elevations above ambient are well supported in several East Coast provinces and is deemed applicable to the West Coast as a general biological principle. Data are not sufficient to develop general regional ceilings. These must be determined on a case-by-case basis until general principles emerge.

The Pacific Coast consists of two distinct biogeographical regions; the cold temperate province ranges north from Point Conception, California and the warm temperate region from Point Conception southward. Published data should provide a general indication of possible adverse effects of excessive thermal discharge on indigenous species.

Pacific Cold Temperate Zone: Some winter and spring spawning temperature ranges include 3°C to 6°C for Pacific herring, Clupea pallasi, (McCauley and Hancock, 1971); 7°C to 8°C for English sole, Parophrys vetulus, (Alderdice and Forrester, 1968); 13°C for May and June spawning of razor clams, Siliqua patula, (McCauley and Hancock, 1971); and 12°C to 14°C for native little neck clams, Protothaca staminea, (Schink and Woelke, 1973). Optimal growth occurs at 10°C in the small filamentous red algae, Antithamnion spp., (West, 1968), and 12°C to 16°C is optimal for growth and reproduction of various red and brown algae, including kelp, Macrocystis pyrifera, (Druehl and Hisiao, 1969). Twelve to 16°C favors sea grasses, Zostera marina and Phyllospadix scouleri (McRoy, 1970). Spawning migration of striped bass, Morone saxatilis, occurs at 15°C to 18°C (Albrecht, 1964); in American shad, Alosa sapidissima, spawning runs occur at 16.0-19.5°C (Leggett & Whitney, 1972). At Vancouver Island, B.C., distribution of a kelp, Laminaria groenlandica , is temperature influenced. The long stipe form is not found above 13°C; the short stipe form does not occur above 17°C. In the laboratory, elevation of temperature to 13°C produces abnormal sporophytes (Druehl, 1967). Dungeness crab, Cancer magister, larval development is optimal at 10 and 13.9°C, survival is reduced at 17.8°C, with no survival to megalops at 21.7°C (Reed, 1969). The Upper thermal limit for razor clam embryonic and larval development is 17°C (McCauley and Hancock, 1971). Upper growth limit

for small filamentous red algae, Antithamnion spp, is 18°C (West, 1968). King salmon migration into the San Joaquin River may be delayed by estuarine temperatures in excess of 17.8°C (Dunham, 1968).

The sea grass, Phyllospadix scouleri, begins to die off (McRoy, 1970), and the pea pod borer, Botula fulcata, ceases to develop at 20°C (Fox and Corcoran, 1957). Twenty degrees centigrade also is the upper limit for embryonic and larval development of the summer-spawning horse clam, Tresus nuttalli, and native little neck clam, Protothaca staminea, (Schink and Woelke, 1973). Upper incipient lethal temperature for the mysid shrimp, Neomysis intermedia, is 21.7°C (Hair, 1971). This value is corroborated by reports of a drop in field populations of this important fish food organism above 22.2°C in the San Joaquin estuary (Heubach, 1969). Twenty-two degrees centigrade is the upper tolerance limit for embryological development of the wooly sculpin, Clinocottus analis, (Hubbs, 1966). A four-hour exposure to 23°C results in significant mortality of the adult razor clam, Siliqua patula, (Woelke, 1971) and the sockeye salmon, Oncorhynchus nerka, (Brett and Alderdice, 1958). Striped bass, Morone saxatilis, are believed to be stressed at temperatures above 23.9°C (Dunham, 1968). Sexual maturation in a gobiid fish, Gillichthys mirabilis, is blocked at high temperatures. Gonadal regression begins at 22°C in females; at 24°C in males.

Gonadal recrudescence will not occur at 24°C or above, regardless of photoperiod (DeVlaming, 1972). The 36-hour TLm for red abalone adults is 23°C when acclimated to 15°C; for the embryos, 26°C, when exposed for 30 hours (Ebert, 1974). Sea urchin, Strongylocentrotus purpuratus, upper tolerance limit is 23.5°C for adults (Gonor, 1968); 25°C is lethal to embryos and renders adults limp and unresponsive after 4 hours (Farmanfarmaian and Giese, 1963).

Pacific Warm Temperate Zone: The thermal threshold for spawning in Pacific sardine, Sardinops caerulea, is 13°C (Marr, 1962). Reports of temperature optima for spawning include 15°C in a ctenophore, Pleurobranchia bachei, (Hirota, 1973); 16°C in the spring spawning wooly sculpin, Clinocottus analis, (Graham, 1970); 17.5°C for northern anchovy, Engraulis mordax; 19°C for opaleye fish, Girella nigricans, (Norris, 1963). Larval survival is best at 16 to 18°C in white abalone, Haliotis sorenseni, (Leighton, 1972).

Limiting effects of temperature include scarcity of the kelp isopod in the beds above 17.8°C (Jones, 1971). Upper limit for growth in Pleurobranchia bachei is 17°C; 20°C is the upper tolerance limit for the adult ctenophore (Hirota, 1973). Twenty degrees centigrade also causes limited survival in recently settled juvenile white abalone (Leighton, 1972). Limiting

effects for wooly sculpin include the upper limit of optimal growth at 21°C; at 22°C, a 50 percent reduction in the successful development of eggs; at 24°C, the upper limit for embryonic development is reached (Hubbs, 1966). Sea Urchins, Strongylocentrotus sp. are weakened or killed at 24°C to 25°C (Leighton, 1971). At 25°C, partial osmoregulatory failure occurs in staghorn sculpin, Leptocottus armatus, at 37.6 o/oo (Morris, 1960). A maximum temperature of occurrence of 25°C is reported for top smelt, Atherinops affinis, by Doudoroff (1945) and northern anchovy, Engraulis mordax, (Baxter, 1967). For topsmelt, the upper limit at which larvae hatch is 26.8°C (Hubbs, 1965).

Natural summer temperatures are stressful to beds of giant kelp, Macrocystis pyrifera, in southern California. This precludes any summer thermal discharge in the vicinity of these beds. Deterioration of surface blades is evident from late June onward, due in part to reduced photosynthesis (Clendenning, 1971). Several weeks' exposure to 18.9°C is harmful to the beds (Jones, 1971), while temperatures over 20°C result in pronounced loss of kelp (North, 1964). Brandt (1923) reported that there was some 60 percent reduction of kelp harvest when the average temperature was 20.65°C and that a bacterial disease, black rot, thrives on kelp at 18-20°C. One-day exposure to 22°C is quite harmful to cultured gametophytes of giant kelp (North, 1972).

REFERENCES CITED APPENDIX:

Albertson, H.D., 1973. A comparison of the upper lethal temperatures of animals of fifty common species from Biscayne Bay. M.S. Thesis, Univ. Miami, Coral Gables, Fla.

Albrecht, A.B., 1964. Some observations on factors associated with survival of striped bass eggs and larvae. Calif. Fish and Game, 50:100.

Alderdice, D.F. and C.R. Forrester, 1968. Some effects of salinity and temperature on early development and survival of the English sole, Parophrys vetulus. Jour. Fish. Res. Board Canada, 27:495.

Andrews, J.D., 1965. Infection experiments in nature with Dermocystidium marinum in Chesapeake Bay. Chesapeake Sci., 6:60.

Ansell, A.D., 1968. The rate of growth of the hard clam, Mercenaria mercenaria (L), throughout the geographical range. Jour. Cons. per. int. Explor. Mer., 31:364.

Barans, C.A., 1972. Spotted hake, Urophycis regius, of the York River and lower Chesapeake Bay. Chesapeake Sci. 13(1):59

Baxter, J.L., 1967. Summary of biological information on the northern anchovy, Engraulis mordax Girard. In: Sumposium on anchovies, genus Engraulis, Lake Arrowhead, Calif. Nov. 23-24, 1964. Calif. Coop. Oceanic Fish. Invest. Rep.,11:110.

Bigelow, H.B. and W.C. Schroeder, 1953. Fishes of the Gulf of Maine. U.S. Fish and Wildlife Ser. Fish. Bull. 74, 53:1

Brandt, R.P., 1923. Potash from kelp: early development and growth of the giant kelp, Macrocystis pyrifera. U.S. Dept. Agr., Dept. Bull. 1191.

Brawn, V.M., 1960. Temperature tolerance of unacclimated herring, Clupea harengus, L.L., Jour. Fish. Res. Board Canada, 17:721.

Brenko, M. and A. Calabrese. 1969. The combined effects of salinity and temperature on larvae of the mussel, Mytilus edulis. Mar. Biol. 4 (3): 224-226.

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

Brett, J.R. and Alderdice, 1958. Unpublished data, p. 526-527, Cited in: Brett, J.R., 1970. Temperature, animals, fishes. In: Kinne (ed.) Marine ecology - a

comprehensive treatise on life in oceans and coastal waters. Vol. 1, environmental factors. Wiley-Interscience, London.

Calabrese, A., 1969. Individual and combined effects of salinity and temperature on embryos and larvae of the coot clam, Mulinia lateralis (say). Biol. Bull., 137(3):417

Chesher, R.H., 1971. Biological impact of a large-scale desalination plant at Key West. EPA Water Poll: Control Res. Ser. 18080 GBX. EPA Office of Research and Monitoring, Washington.

Chin, E., 1961. A trawl study of an estuarine nursery area in Galveston Bay with particular reference to penaeid shrimp. Ph.D. Dissertation, Univ. Washington, 113 p.

Clendenning, K.A., 1971. Photosynthesis and general development in Macrocystis, In: W.J. North (ed.) The biology of giant kelp beds (Macrocystis) in California. Beihefte Zur Nova Hedwigia. J. Crammer, Lehre, Germany.

Colton, J.B., Jr., 1959. A field observation of mortality of marine fish larvae due to warming. Limn. and Oceanog., 4: 219.

Courtenay, W.R., Jr. and, M.H. Roberts, Jr., 1973. Environmental effects of toxaphene toxicity to selected fishes and crustaceans. EPA Ecol. Res. Ser. EPA R3-73-035, EPA Office of Research and Monitoring, Washington, D.C.

Creaser, E.P., Jr., 1973. Reproduction of the blood worm, Glycera dibranchiata, in the Sheepscot estuary, Maine. Jour. Fish. Res. Board Canada, 30 :161.

DeCola, J.M., 1970. Water quality requirements for Atlantic salmon. U.S. Dept. of the Interior, Federal Water Quality Administration, Northeast Region, New England Basins Office, Needham Heights, Massachusetts. Appendices, Mimeo.

deVlaming, V.L., 1972. The effects of temperature and photoperiod on reproductive cycling in the estuarine gobiid fish, Gillichthys mirabilis. Fishery Bull., 70 :1137.

Doudoroff, P., 1945. The resistance and acclimatization of marine fishes to temperature changes. II. Experiments with Fundulus and Atherinops. Scripps Inst. of Oceanography, Series No. 253, p. 194-206

Druehl, L.D. and S.I.C. Hsiao, 1969. Axenic culture of Laminariales in defined media. Phycologia 8 :47

Druehl, L.D., 1967. Distribution of two species of Liminaria as related to some environmental factors. Jour. Phycology 3 :103.

Dunham, L.R., 1968. Recommendations on thermal objectives for water quality control policies on the interstate waters of California. Water Projects Branch Report No. 7. Calif. Dept. Fish and Game.

Ebert, E.E., 1974. Unpublished data. Pacific Gas and Electric Co. Cooperative Research Agreement 6S-1047. Calif. Dept. Fish and Game Marine Culture Laboratory, Monterey, Calif.

Edmonson, C.H., 1928. Ecology of a Hawaiian coral reef. B.P. Bishop Mus. Bull. 45.

Edwards, G.A. and O. Irving, 1943. The influence of temperature and season upon the oxygen consumption of the sand crab, Emerita talpoida Say. Jour. Cell Physiol., 21:169.

Engle, J.B. and V.L. Loosanoff. 1944. On season of attachment of larvae of Mytilus edulis Linn. Ecol. 25:433-440.

Farmanfarmaian, A. and A.C. Giese, 1963. Thermal tolerance and acclimation in the western purple sea urchin, Strongylocentrotus purpuratus. Physiol. Zool., 36: 237.

Fox, D.L. and E.F. Corcoran, 1957. Thermal and osmotic countermeasures against some typical marine fouling organsims. Corrosion, 14:31.

Gift, J.J. and J.R. Westman, 1971. Responses of some estuarine fishes to increasing thermal gradients. Dept. Environ. Sci., Rutgers Univ., New Brunswick, N.J., Mimeo. 154 p.

Gonor, J.J., 1968. Temperature relations of central Oregon marine intertidal invertebrates: A prepublication technical report to the Office of Naval Research Dept. of Oceanography, Oregon State Univ. Reference 68-38.

Gonzalez, J.G., 1972. Seasonal variation in the responses of estuarine populations to heated water in the vicinity of

a steam generating plant. Ph.D. Dissertation, Univ. Rhode Island, 142 p.

Gore, R.H., 1968. The larval development of the commensal crab, Palyonx gibbesi. Haig, 1956 (Crustacea: Decapoda). Biol. Bull., 135:111.

Graham, J.B., 1970. Temperature sensitivity of two species of intertidal fishes. Copeia, 1970:49

Hair, J.R., 1971. Upper lethal temperature and thermal shock tolerances of the opossum shrimp, Neomysis awatschensis, from the Sacramento-San Joaquin Estuary, California. Calif. Fish and Game, 57:17.

Hanks, J. 1953. Comparative studies on the feeding habits of Polinices heros and Polinices duplicata relative to temperature and salinity. Fourth Ann. Conf. on Clam Res., U.S. Fish and Wildlife Serv., Boothbay Harbor, Me., p. 88-95.

Halcrow, K. and C. M. Boyd. 1967. The oxygen consumption and swimming activity of the amphipod, Gammarus oceanicus, at different temperatures. Comp. Biochem. and Physiol. 23: 233-242.

Hela, I. and T. Laevastu., 1962. Fisheries hydrography. Fishing News Ltd., London.

Heubach, W., 1969. Neomysis awatschensis in the Sacramento-San Joaquin River Estuary. Limnol. and Oceanog., 14:533.

Hirota, J., 1973. Quantitative natural history of Plueorbrachia bachei A. Agassiz in La Jolla Bight. Ph.D. Thesis, Univ. Calif., San Diego.

Hoff, J.G. and J.R. Westman. 1966. The temperature tolerance of three species of marine fishes. J. Mar Res. 24(141).

Hopper, B.E., et al., 1973. Effect of temperature on life cycles of nematodes associated with the mangrove, Rizophera mangle, detrital system. Mar. Biol., 23: 293.

Hoss, D.E., et al., 1974 Effects of thermal shock on larval estuarine fish -- Ecological implications with respect to entrainment in power plant cooling systems. In: J.H.S. Blaxter (ed.), Symp. on Early Life History of Fish, Proc. Oban, Scotland, Springer-Verlag, New York, 1973.

Hubbs, C., 1965. Developmental temperature tolerance and rates of four southern California fishes, Fundulus parvipinnis, Atherinops affinis, Leuresthes tenuis, and Hypsoblennius sp. Calif. Fish Game, 51: 113.

Hubbs, C., 1966. Fertilization, initiation of cleavage, and developmental temperature tolerance of the cottid fish, Clinocottus analis. Copeia, 1966:29.

Jeffries, H.P., 1967. Saturation of estuarine zooplankton by congeneric associates. In: G.H. Lauff (ed.) Estuaries. American Association for the Advance of Science, Publ. No. 83, Washington, D. C.

Jokiel, P.L. and S.L. Coles, 1974. Effects of heated effluent on hematypic corals at Kahe Point, Oahu. Pacific Sci., 28,1.

Jokiel, P.L., et al. In press. Effects of thermal loading on Hawaiian reef corals. EPA Ecological Research Series, EPA Office of Research and Development, Washington, D.C.

Jones, L.G., 1971. Studies on selected small herbivorous invertebrates inhabiting Macrocystis canopies and holdfasts in southern California kelp beds. In: W.J. North (ed.) The Biology of Giant Kelp Beds (Macrocystis) in California. Beihefte zur Nova Hedwigia, Heft 32. J. Cramer, Lehre, Germany .

Kennedy, V.S., et al., 1974. Temperature-time relationships for survival of embryos and larvae of Mulinia lateralis (mollusca: Bivalvia). Mar. Biol., 24:137.

Kuthalingam, M.D.K., 1959. Temperature tolerance of the larvae of ten species of marine fishes. Current Sci., 2: 75.

Laurence, G.C. 1973 Influence of temperature on energy utilization of embryonic and postlarval tautog, Tautoga onitis. J. Fish. Res. Bd. Can. 30 (3): 435-442.

Leggett, W.C. and R.R. Whitney., 1972. Water temperature and the migrations of American shad. Fish. Bull., 70:659.

Leighton, D. L., 1971. Grazing activities of benthic invertebrates in southern California kelp beds. In: W.J. North (ed) The biology of giant kelp beds

(Macrocystis) in California. Beihefte zur Nova Hedwigia, Heft 32. J. Cramer, Lehre, Germany.

Leighton, D.L., 1972. Laboratory observations on the early growth of the abalone, Haliotis soreseni, and the effect of temperature on larval development and settling success. Fish. Bull., 70 : 373.

Lewis, R.M. and W.F. Hettler, Jr., 1968. Effect of temperature and salinity on the survival of young Atlantic menhaden, Brevoortia tyrannus. Trans. Amer. Fish. Soc., 97: 344.

Marr, J.C., 1962. The causes of major variations in the catch of the Pacific sardine, Sardinops caerulea (Girard). Proc. World Scientific Meeting on the Biology of Sardines and Related Species, FAO, Rome. 3: 667.

Mayer, A.G., 1914. The effects of temperature upon tropical marine animals. Papers Mar. Biol. Lab. Tortugas, Carnegie Inst. Wash., 6:3.

McCauley, J.E. and D.R. Hancock, 1971. Biology of selected northwest species or species groups, P. 246-303 In: Oregon State

Univ. Oceanography of the Nearshore Coastal Waters of the Pacific Northwest Relating to Possible Pollution. Vol. 1. EPA Water Pollution Control Research Series 16070 EOK. EPA, Water Quality Office, Washington, D.C.

McRoy, C.P., 1970. The biology of eel grass in Alaska. Ph.D. Thesis, Univ. Alaska, p. 156.

Meldrin, J.W. and J.J. Gift, 1971. Temperature preference, avoidance and shock experiments with estuarine fishes. Ichthyological Associates, Ithaca, N.Y., Bull. 7.

Merriman, D. 1941. Studies on the striped bass (Roccus saxatilis) of the Atlantic coast. U.S. Fish & Wildlife Serv., Fish. Bull. 50, Vol. 35, p. 77.

Mihursky, J.A., 1969. Patuxent thermal studies. Natural Res. Inst. Spec. Rept. 1, Univ. Md., p. 20.

Mihursky, J.A. and V.S. Kennedy, 1967. Water temperature criteria to protect aquatic life. In: E.L. Cooper (ed.) Special Publ. 4, Trans. Amer. Fish. Soc., 96 (1) Supplement.

Mihursky, J.A., A.J. McErlean, and V.S. Kennedy, 1970. Thermal pollution, aquaculture and pathobiology in aquatic systems. J. Wildlife Diseases 6: 347-355.

Morris, W., 1960. Temperature, salinity, and southern limits of three species of Pacific cottid fishes. Limn. and Oceanog., 5:175.

Norris, K.S., 1963. The functions of temperature in the ecology of the percoid fish, Girella nigricans (Ayres). Ecol. Monogr., 33:23.

North, W.J., 1964. Experimental transplantation of the giant kelp, Macrocystis pyrifera. In: DeVirville and Feldmann (eds.) IVth Int. Seaweed Symp. (Biarritz, 1961). Perqamon, New York.

North, W.J., 1972. Kelp habitat improvement project. W.M. Keck Lab. of Environ. Health Engr., Cal. Tech., Annual Report, 1 July, 1971 - 30 June, 1972, p. 200.

Olla, B.L., R. Wicklund, and S. Wilk. 1969. Behavior of winter flounder in a natural habitat. Amer. Fish. Soc. Trans. 98: 717-720.

Prince, J.S., and J.M. Kingsbury, 1973. The ecology of Chondrus crispus at Plymouth, Massachusetts. III. Effect of elevated temperature on growth and survival. Biol. Bull., 145: 580.

Rebel, T.P., 1973. Effects of temperature on survival of eggs and yolk sac larvae of four species of marine fishes from south Florida. M.S. Thesis, Univ. Miami, Coral Gables, Florida, p. 53.

Reed, P.H., 1969. Culture methods and effects of temperature and salinity on survival and growth of Dungeness crab

(Cancer magister) larvae in the laboratory. Jour. Fish. Res. Board Can., 26: 389.

Roessler, M.A., 1974. Studies of the effects of thermal pollution in Biscayne Bay, Florida. EPA Ecological Research Series 660/3-74-014. U.S. EPA, Office of Research and Management, Washington, D.C.

Rogers, C.A., 1976 Effects of temperature and salinity on the survival of winter flounder embryos. Fish. Bull., In press.

Saksena, V.P., et al., 1972. Effects of temperature on growth and survival of laboratory-reared larvae of the scaled sardine, Harengula pensacolae. Goode and Bean. Trans. Amer. Fish. Soc., 101: 691.

Sastry, A.N. and S.L. Vargo. In press. Variations in the physiological responses of crustacean larvae to temperature. F.J. Vernberg, A. Calabrese, F.P. Thurberg, W.B. Vernberg (eds.). Physiological Responses of Marine Organisms to Pollutants. Academic Press, N.Y.

Schink, T.D. and C.E. Woelke, 1973. Development of an in situ marine bioassay with clams. EPA Water Pollution Control Research Series 18050 DOJ. EPA, Office Research and Monitoring, Washignton, D.C.

Scott, K.J., 1975. Studies on the ecology of Psammonyx nobilis, (Stimpson) 1853. Ph.D. Dissertation, Univ. of New Hampshire.

Shannon, E.H., 1969. Effect of temperature changes upon developing striped bass eggs and fry. Proc. Twenty-Third Annual Conf. S.E. Assoc. Game and Fish. Comm., p. 265-274.

Sherburne, S.W., 1973. Erythrocyte degeneration in Atlantic herring, Clupea harengus (L.), Fish. Bull., 71: 125.

Sinderman, C.J. 1965. Effects of environment on several diseases of herring from the western North Atlantic. Spec. Publ. Comm. Northw. Atlantic Fish. No. 6, 603-610.

South, G.R. and R.D. Hill, 1970. Studies on marine algae of Newfoundland. I. Occurrence and distribution of free living Ascophyllum nodosum in Newfoundland. Can. Jour. Bot., 48: 1697.

Steele, D.H. and V.J. Steele, 1969. The biology of Gammarus (Crustacea, Amphipoda) in the northwestern Atlantic. I. Gammarus duebeni Lillj. Can Jour. Zool., 47:235.

Thorhaug, A., 1970. Temperature limits of five species of Valonia. Jour. Phycol., 6: 27.

Thorhaug, A., 1971. Grasses and macroalgae, p. X1-63. In: R.G. Bader and M.A. Roessler (eds.) An ecological study of South Biscayne Bay and Card Sound. Progress Rept. to USAEC and Fla. Power and Light Co. (Univ. of Miami, Coral Gables, Fla.).

Thorhaug, A., et al., 1971 a. Refining shrimp culture methods: The effect of temperature on early stages of the commercial pink shrimp. Gulf and Caribbean Fish. Inst., Proc. Twenty-third Annual Session, p. 125.

Thorhaug, A., et al., 1971 b. Laboratory thermal tolerances, p. XI-31 In: R.G. Bader and M.A. Roessler (eds.) An ecological study of South Biscayne Bay and Card Sound. Progress Rept. to USAEC and Fla. Power and Light Co. (Univ. Miami, Coral Gables, Fla.).

Thorahug, A. 1973. Grasses and macroalgae laboratory temperature studies. Annual Report (1972-1973) to the USAEC and Fla. Power and Light (Univ. Miami, Coral Gables, Fla.), 92 p.

West, J.A., 1968. Morphology and reproduction of the red algae, Ocrochaetium pectinatum, in culture . Jour. Phycol., 4:88.

Woelke, C.E., 1971. Some relationships between temperature and Pacific northwest shellfish. Western Assoc. State Game and Fish Comm., Proc. Fifty-first Ann. Conf.

Zieman, J.C. and E.J. Ferguson Wood, 1975. Effects of thermal pollution on tropical-type estuaries with emphasis on Biscayne Bay Florida. In: E.J. Ferguson Wood and R.E. Johannes, Tropical Marine Pollution. Elsevier Oceanography Series No. 12, Elsevier Scientific Publishing Co., N. Y., p 75-98.

Zein-Eldin, J.P. and D.V. Aldrich, 1965. Growth and survival of postlarval Penaeus aztecus under controlled conditions of temperature and salinity. Biol. Bull., 129: 199.

Zein-Eldin, J.P. and G.W. Griffith, 1969. An appraisal of the effects of salinity and temperature on growth and survival of postlarval penaeids. FAO Fish. Rep. 57 Vol. 3: 1015.

Zieman, J.C., Jr., 1970. The effects of a thermal effluent stress on the sea grasses and macroalgae in the vicinity of Turkey point, Biscayne Bay, Florida. Ph.D. Dissertation, Univ. Miami, Coral Gables, Fla.

ZINC

CRITERIA:

5,000 ug/l for domestic water supplies (welfare);

For freshwater aquatic life, 0.01 of the 96-hour LC_{50} as determined through bioassay using a sensitive resident species.

INTRODUCTION:

Zinc usually is found in nature as the sulfide; it is often associated with sulfides of other metals, especially lead, copper, cadmium, and iron. Most other zinc minerals probably have been formed as oxidation products of the sulfide; they represent only minor sources of zinc. Nearly 3,000,000 short tons of recoverable zinc per year are mined in the world; about 500,000 tons of this come from the United States.

Zinc (as metal) is used in galvanizing, i.e., coating (hot dipping) of various iron and steel surfaces with a thin layer of zinc to retard corrosion of the coated metal. In contact with iron, zinc is oxidized preferentially, thus protecting the iron. The second most important use of zinc, reaching major proportions in the last quarter century, is in the preparation of alloys for dye casting. Zinc is used also in brass and bronze alloys, slush castings (in the rolled or extruded state), in the production of zinc oxide and other chemical products, and in photoengraving and printing plates.

Kopp and Kroner (1967) report that in 1,207 positive tests for zinc on samples from U.S. waterways, the maximum observed value was 1,183 ug/l (Cuyahoga River at Cleveland, Ohio) and the mean was 64 ug/l. Dissolved zinc was measured in over 76 percent of all water samples tested. The highest mean zinc value, 205 ug/l, was found in the Lake Erie Basin, whereas the lowest mean zinc value, 16 ug/l, was observed in the California Basin. In seawater, zinc is found at a maximum concentration of about 10 ug/l.

RATIONALE:

Zinc is an essential and beneficial element in human metabolism (Vallee, 1957). The daily requirement of preschool-aged children is 0.3 mg Zn/kg body weight. The daily adult human intake averages 0 to 15 mg/ zinc; deficiency in children leads to growth retardation. Community water supplies have contained 11 to 27 mg/l without harmful effects (Anderson, et al., 1934; Bartow and Weigle, 1932). However, in tests performed by a taste panel, 5 percent of the observers were able to distinguish between water containing 4 mg/l zinc as $ZnSO_4$, which had a bitter or astringent taste, and water containing no zinc salts (Cohen, et al., 1960). Because zinc in water produces undesirable aesthetic effects, the concentration of zinc in domestic water supplies should be below 5 mg/l (5,000 ug/l).

The toxicity of zinc compounds to aquatic animals is modified by several environmental factors, particularly hardness, dissolved oxygen, and temperature. Skidmore (1964), in undertaking a review of the literature on the toxicity of zinc to fish, reported that salts of the

alkaline-earth metals are antagonistic to the action of zinc salts, and salts of certain heavy metals are synergistic in soft water. Both an increase in temperature and a reduction in dissolved oxygen increase the toxicity of zinc. Toxic concentrations of zinc compounds cause adverse changes in the morphology and physiology of fish. Acutely toxic concentrations induce cellular breakdown of the gills, and possibly the clogging of the gills with mucous. Chronically toxic concentrations of zinc compounds, in contrast, cause general enfeeblement and widespread histological changes to many organs, but not to gills. Growth and maturation are retarded.

Using dilution water with calcium of 1.7 mg/l and magnesium of 1.0 mg/l, Affleck (1952) found a 54 percent mortality of rainbow trout fry in 28 days in a zinc concentration of 10 ug/l. Pickering and Henderson (1966) determined the 96-hour LC_{50} of zinc for fathead minnows, Pimephales promelas, and bluegills, Lepomis macrochirus, using static test conditions. For fathead minnows in soft water (20 mg/l $CaCO_3$) the LC_{50} was 870 ug/l, and in hard water (360 mg/l $CaCO_3$) it was 33,000 ug/l. Bluegills were more resistant in both waters. Similarly, the lethal threshold concentration was 3 or 4 times as high for coarse fish as for trout, Salvelinus fontinalis, (Ball, 1967).

The 24-hour LC_{50} of zinc for rainbow trout, Salmo gairdneri, was reduced only 20 percent when the fish were forced to swim at 85 percent of their maximum sustained swimming speed (Herbert and Shurben, 1964). The maximum effect of a reduction in dissolved oxygen from 6 to 7 mg/l to 2 mg/l on the acute toxicity of zinc was a 50-percent increase (Lloyd, 1961; Cairns and Scheier, 1958; Pickering, 1968).

The Atlantic salmon, Salmo salar, was tested in a 168-hour continuous-flow bioassay at 17° C in water with a total hardness of 14 mg/l $CaCO_3$. The incipient lethal level, the level beyond which the organism can no longer survive, was 420 ug/l of zinc (Sprague and Ramsay, 1965).

Brungs (1969) found that in water with a total hardness of 200 mg/l $CaCO_3$, 180 ug/l zinc caused an 83 percent reduction in eggs produced by the fathead minnow, Pimephales promelas, in chronic tests. The tests lasted 10 months and the control test water contained 30 ug/l zinc. The 96-hour continuous-flow TLm was determined to be 9,200 ug/l zinc.

A number of short term fish toxicity data are detailed in Table 14. When referring to this table, the reader should consider the species tested, pH, alkalinity, and hardness (if alkalinity is not given (in most natural waters alkalinity parallels hardness). In general, the salmonids are most sensitive to elemental zinc in soft water; the rainbow trout Salmo gairdneri is the most sensitive in hard waters. The influence of the pH on solubility of zinc complexes, and the resulting toxicity of zinc, is clearly shown in the data presented on the fathead minnow, Pimephales promelas. The influence of pH and other factors on the solubility and form of the zinc preclude the recommendation of a freshwater or marine criteria based on acute toxicities alone.

Wurtz (1962) performed bioassays on young pond snails, Physa heterostropha, in waters with a total hardness of 100 mg/l and 20 mg/l $CaCO_3$. In water with a temperature of 51° F, the soft water tests resulted in a 96-hour LC_{50} of 303 ug/l zinc, whereas the 96-hour LC_{50} in hard water was 434 ug/l zinc. The LC_{50} of a zinc sulfate solution in dilution water with a total hardness of 44 mg/l $CaCO_3$ for a 10-day test to a mayfly, Ephemerella subvaria, was 16,000 ug/l (Warnick and Bell, 1969). In such tests with several heavy metals, the immature insects seem to be less sensitive than many fishes that have been tested.

The 48-hour LC_{50} for Daphnia magna in soft water with a hardness of 45 mg/l $CaCO_3$ and an alkalinity of 42 mg/l has been found to be 100 ug/l; in 70 ug/l zinc, there was a 16 percent reproductive impairment in a 3-week chronic test (Biesinger and Christensen, 1972).

Toxicities of zinc in nutrient solutions have been demonstrated for a number of plants. Hewitt (1948) found that zinc at 16 to 20 mg/l produced iron deficiencies in sugar beets. Hunter and Vergnano (1953) found toxicity to oats at 25 mg/l. Millikan (1947) found that 2.5 mg/l produced iron deficiency in oats. Early (1943) found that the Peking variety of soybeans was killed at 0.4 mg/l, whereas the Manchu variety was killed at 1.6 mg/l zinc.

Although few data are available on the effects of zinc in the marine environment, it is accumulated by some species, and marine animals contain zinc in the range of 6 to 1,500 mg/kg (NTAC, 1968). As a goal, the marine environment should be protected to the same level as the fresh water environment.

TABLE

The acute toxicity (24-, 48-, 96-hr TL50 values) of zinc to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Brook trout (Salvelinus fontinalis)	3.0 g 3.9 g 19.0 g	$ZnSO_4 \cdot 7H_2O$	96	FT*	14.8-15.5	1.38 Zn 2.09 Zn 2.50 Zn	7.3-7.7	42	45	(7)
Brook trout	3.0 g 3.9 g 19.0 g	$ZnSO_4 \cdot 7H_2O$	96	FT	14.8-15.5	5.50 Zn 6.05 Zn 4.92 Zn	7.3-7.7	-	100±10	(7)
Rainbow trout (Salmo gairdneri)	3.9 g 4.9 g 28.4 g	$ZnSO_4 \cdot 7H_2O$	96	FT	14.8-15.5	0.285 Zn 0.506 Zn 0.820 Zn	7.3-7.7	42	45	(7)
Rainbow trout	Juveniles	$ZnSO_4$	96	FT	12.7	0.43 Zn	6.8	25	26	(15)
Rainbow trout	7 g	Zinc	96	FT	11.6-12.4	0.10 Zn	6.8-7.0	17-26	20-25	(4)
Rainbow trout	Fingerlings	Zinc sulfate	48	FT	17.7	0.91 Zn	6.9	41.5	44	(6)
Rainbow trout	1.5 g	Zinc	96	S*	10	0.09 Zn	7	20	20	(5)
Rainbow trout	Juveniles	$ZnSO_4$	96	FT	16.2	7.21 Zn	7.8	238	333	(15)
Rainbow trout	3.9 g 4.9 g 28.4 g	$ZnSO_4 \cdot 7H_2O$	96	FT	14.8-15.5	2.40 Zn 2.66 Zn 1.95 Zn	7.3-7.7	-	100±10	(7)
Rainbow trout	-	Zinc	48	S	15	3.2 Zn	7.6	200	300	(1)

*flow-through bioassay
**static bioassay

The acute toxicity (24-, 48-, 96-hr TL50 values) of zinc to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Cutthroat trout (Salmo clarki)	-	Zinc	24 48 96 24	FT FT FT S	--	0.62 Zn 0.27 Zn 0.09 Zn 0.42 Zn	7	23.9	-	(11)
Chincok salmon (Oncorhynchus tshawytcha)	At hatch	Zinc	96	FT	11.1-12.0	>0.70 Zn	6.8-7.0	17-26	20-25	(4)
	1 month old	Zinc	96	FT	10.8-12.5	0.103 Zn				
Atlantic salmon (Salmo salar)	Juveniles	Zinc	24	FT	15	0.65 Zn	7.1-7.5	-	20	(17)
Fathead minnow (Pimephales promelas)	1-2 g	$ZnSO_4 \cdot 7H_2O$	24 48 96	S	15	0.92 $ZnSO_4 \cdot 7H_2O$ 0.87 $ZnSO_4 \cdot 7H_2O$ 0.87 $ZnSO_4 \cdot 7H_2O$	7.5	18	20	(9)
Fathead minnow	1-2 g	$Zn(C_2H_3O_2)_2$	24 48 96	S	15	1.03 $Zn(C_2H_3O_2)_2$ 0.88 $Zn(C_2H_3O_2)_2$ 0.88 $Zn(C_2H_3O_2)_2$	7.5	18	20	(9)
Fathead minnow	1-2 g	$ZnSO_4 \cdot 7H_2O$	96	FT	25±1	12.5-13.8 Zn^{++} 6.2-13.7 Zn^{++} 4.7-6.1 Zn^{++}	6 7 8	-	50.2 50 50	(8)
Fathead minnow	1-2 g	$ZnSO_4 \cdot 7H_2O$	96	FT	25±1	18.5-25.0 Zn^{++} 12.3-12.5 Zn^{++} 8.1-10.9 Zn^{++}	6 7 8	-	100	(8)

The acute toxicity (24-, 48-, 96-hr TL50 values) of zinc to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Fathead minnow	1-2 g	$ZnSO_4 \cdot 7H_2O$	96	FT	25±1	25.0-39.5 Zn^{++} 13.6-19.3 Zn^{++} 8.2-21.0 Zn^{++}	6 7 8	-	200	(8)
Fathead minnow	1-2 g	$ZnSO_4 \cdot 7H_2O$	24 48 96	S	15	34.5 $ZnSO_4 \cdot 7H_2O$ 33.4 $ZnSO_4 \cdot 7H_2O$ 33.4 $ZnSO_4 \cdot 7H_2O$	7.5 ✓	300	360	(9)
Fathead minnow	Eggs	$ZnSO_4 \cdot 7H_2O$	24 48 96	FT	20	3.95 Zn 2.55 Zn 1.84 Zn	7.5-7.6	44-58	174-198	(10)
Fathead minnow	Fry	$ZnSO_4 \cdot 7H_2O$	24 48 96	FT	20	0.95 Zn 0.95 Zn 0.87 Zn	7.5-7.6	44-58	174-198	(10)
Fathead minnow	45 (mm)	Zinc	24 48 96	S	23.2	8.9 Zn 7.8 Zn 7.6 Zn	6.2	-	166	(12)
Fathead minnow	2-3 g	$ZnSO_4 \cdot 7H_2O$	96	S FT	23	12.5 Zn 9.2 Zn	7.4-7.9	-	203	(2)
Bluegills (Lepomis macrochirus)	1-2 g	$ZnSO_4 \cdot 7H_2O$	24 48 96	S	15	6.48 $ZnSO_4 \cdot 7H_2O$ 5.46 $ZnSO_4 \cdot 7H_2O$ 5.38 $ZnSO_4 \cdot 7H_2O$	7.5	18	20	(9)

The acute toxicity (24-, 48-, 96-hr TL50 values) of zinc to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Bluegills	1-2 g	$ZnCl_2$	24 48 96	S	15	7.24 $ZnCl_2$ 5.76 $ZnCl_2$ 5.37 $ZnCl_2$	7.5	18	20	(9)
Bluegills	37 g	$ZnSO_4$	96	FT	7-9	10.6 Zn^{++}	7.8	35.6	46	(3)
Bluegills	1-2 g	$ZnSO_4 \cdot 7H_2O$	24 48 96	S	15	40.9 $ZnSO_4 \cdot 7H_2O$ 40.9 $ZnSO_4 \cdot 7H_2O$ 40.9 $ZnSO_4 \cdot 7H_2O$	7.5	300	360	(9)
Flagfish (Jordanella floridae)	20 (mm)	$ZnSO_4 \cdot 7H_2O$	96	FT	25±2	1.5 Zn	7.5	42	45	(16)
Banded killifish (Fundulus diaphanus)	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	17	22.6 Zn^{++} 20.7 Zn^{++} 19.1 Zn^{++}	7.8	-	53	(13)
Banded killifish	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	28	23.0 Zn^{++} 20.4 Zn^{++} 19.2 Zn^{++}	8.0	-	55	(14)
Striped bass (Roccus saxatilis)	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	17	11.2 Zn^{++} 10.0 Zn^{++} 6.7 Zn^{++}	7.8	-	53	(13)

The acute toxicity (24-, 48-, 96-hr TL50 values) of zinc to several species of fish in water of various water qualities.
(continued)

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Striped bass	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	28	11.3 Zn^{++} 10.0 Zn^{++} 6.8 Zn^{++}	8.0	-	55	(14)
Pumpkinseed (*Lepomis gibbosus*)	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	17	25.2 Zn^{++} 21.8 Zn^{++} 20.0 Zn^{++}	7.8	-	53	(13)
Pumpkinseed	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	28	25.1 Zn^{++} 21.9 Zn^{++} 20.1 Zn^{++}	8.0	-	55	(14)
White perch (*Roccus americanus*)	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	17	13.6 Zn^{++} 10.2 Zn^{++} 14.3 Zn^{++}	7.8	-	53	(13)
White perch	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	28	13.5 Zn^{++} 10.1 Zn^{++} 14.4 Zn^{++}	8.0	-	55	(14)
Carp (*Cyprinus carpio*)	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	17	14.3 Zn^{++} 9.3 Zn^{++} 7.8 Zn^{++}	7.8	-	53	(13)
Carp	≤20 (cm)	$Zn(NO_3)_2$	24 48 96	S	28	14.4 Zn^{++} 9.2 Zn^{++} 7.8 Zn^{++}	8.0	-	55	(14)

0617

The acute toxicity (24-, 48-, 96-hr TL50 values) of zinc to several species of fish in water of various water qualities.

Species	Size	Compound	Exposure time (hr)	Exposure type	Temperature (°C)	Concentration (mg/l)	pH	Alkalinity	Hardness	Reference
Carp	2.5-3.0 (cm)	Zinc sulfate	48	S	28-30	10-12 $ZnSO_4$	7.0-7.2	46	-	(18)
Goldfish (Carassius auratus)	1-2 g	$ZnSO_4 \cdot 7H_2O$	24 48 96	S	15	9.07 $ZnSO_4 \cdot 7H_2O$ 6.44 $ZnSO_4 \cdot 7H_2O$ 6.44 $ZnSO_4 \cdot 7H_2O$	7.5	18	20	(9)
Guppy (Lebistes reticulatus)	0.1-0.2 g	$ZnSO_4 \cdot 7H_2O$	24 48 96	S	15	2.90 $ZnSO_4 \cdot 7H_2O$ 1.96 $ZnSO_4 \cdot 7H_2O$ 1.27 $ZnSO_4 \cdot 7H_2O$	7.5	18	20	(9)
Southern platyfish (Xiphophorus maculatus)	20.8 (cm)	Zinc sulfate	24 48 96	S	23.2	23.0 Zn 18.0 Zn 12.0 Zn	6.2		166	(12)

*FT = Flow-through.

*S = Static.

REFERENCES CITED in TABLE 15:

(1) Brown, V.B., 1968. The calculation of the acute toxicity of mixtures of poisons to rainbow trout. Water Res. 2:723.

(2) Brungs, W.A., 1969. Chronic toxicity of zinc to the fathead minnow, Pimephales promelas Rafinesque. Trans. Am. Fish. Soc., 98:272.

(3) Cairns, J., Jr., et al., 1971. The effects of pH, solubility, and temperature upon the acute toxicity of zinc to the bluegill sunfish (Lepomis macrochirus Rafinesque). Trans. of the Kansas Acad. of Sci., 74(1).

(4) Chapman, G.A. Unpublished data available at the National Water Quality Laboratory, Duluth, Minnesota.

(5) Garton, R.R., 1972. Biological effects of cooling tower blowdown. 71st National meeting. Amer. Inst. Chem. Engr. Jour., Dallas, Texas.

(6) Herbert, D.W.M., and D.S. Shurben, 1964. The toxicity to fish of mixtures of poisons. I. Salts of ammonia and zinc. Ann. Appl. Biol., 53:33.

(7) Holcombe, G., and D.A. Benoit. Unpublished data available at the National Environmental Research Laboratory, Duluth, Minnesota, 55804.

(8) Mount, D.I., 1966. The effect of total hardness and pH on acute toxicity of zinc to fish. Air/Water Poll., 10:49.

(9) Pickering, Q.H., and C. Henderson, 1966. The acute toxicity of some heavy metals to different species of warm water fishes. Air/Water Poll., 10:453.

(10) Pickering, Q.H., and W.N. Vigor, 1965. The acute toxicity of zinc to eggs and fry of the fathead minnow. Progressive Fish Culturist, 27:153.

(11) Rabe, F.W., and C.W. Sappington, 1970. The acute toxicity of zinc to cutthroat trout Salmo Clarki, pp 1-16. In: Biological Productivity of the coeur d' Alene River as related to water quality. Completion Rept. Water Resources Res. Inst. Univ. Idaho, Moscow. Sport Fish. Abs. 13665 1971.

(12) Rachlin, J.W., and A. Perlmutter, 1968. Response of an inbred strain of platyfish and the fathead minnow to zinc. Progressive Fish Culturist, 30:203.

(13) Rehwoldt, R., et al., 1971. Acute toxicity of copper, nickel, and zinc ions to some Hudson River fish species. Bull. Environ. Contam. toxicol., 6:445.

(14) Rehwoldt, R., et al., 1972. The effect of increased temperature upon the acute toxicity of some heavy metal ions. Bull. Environ. Contam. Toxicol., 8:91.

(15) Sinley, J.R., et al., 1974. The effects of zinc on rainbow trout (Salmo gairdneri) in hard and soft water. Bull. Environ. Contam. Toxicol., 12 No. 2.

(16) Spehar, R.L., 1974. Cadmium and zinc toxicity to Jordanella floridae (Goode and Bean): Effects on growth, reproduction, survival, and behavior. A thesis submitted to the University of Minnesota.

(17) Sprague, J.B., 1964. Lethal concentrations of copper and zinc for young Atlantic salmon. Jour. Fish Res. Bd. Can., 21:17.

(18) Sreenivasan, A., and R.S. Raj, 1963. Toxicity of zinc to fish. Current Sci. (India), 32(8):363; Chem. Abs., 59:143386 (1963).

REFERENCES CITED:

Affleck, R.J., 1952. Zinc poisoning in a trout hatchery. Australian J ur Marine Freshwater Res. 3:142.

Anderson, E.A., et al., 1934. The corrosion of zinc in various waters. Jour. Amer. Water Works Assn., 26:49.

Ball, I.R., 1967. The relative susceptibilities of some species of fresh fish to poisons. I. Ammonia. Water Res. 1:767.

Bartow, E. and O.M. Weigle, 1932. Zinc in water supplies. Indus. Eng. Chem. 24:463.

Biesinger, K.E. and G.M. Christensen, 1972. Effects of various metals on survival, growth, reproduction, and metabolism of Daphnia magna. Jour. Fish. Res. Bd. Canada 29:1691.

Brungs, W.A., 1969. Chronic toxicity of zinc to the fathead minnow, Pimephales promelas Rafinesque. Trans. Amer. Fish. Soc. 98:272.

Cairns, J., Jr. and A. Scheier, 1958. The effect of periodic low oxygen upon the toxicity of various chemicals to aquatic organisms. 12 Ind. Waste Conf. Proc. Purdue Univ. Eng. Extension Series.

Cohen, J.M., et al., 1960. Taste threshold concentrations of metals in drinking water. Jour. Amer. Water Works Assn. 52:660.

Early, E.B., 1943. Minor element studies with soybeans: 1. Varietal reactions to concentrations of zinc in excess of nutritional requirements. Jour. Amer. Soc. Agron. 35:1012.

Herbert, D.W.M. and D.S. Shurben, 1964. The toxicity to fish of mixtures of poisons. I. Salts of Ammonia and zinc. Ann. Appl. Biol. 53:33.

Hewitt, E.J., 1948. Relation of manganese and some other metals to the iron status of plants. Nature, 161:489.

Hunter, J.G. and O. Vergnano, 1953. Trace element toxicities in oat plants. Ann. Appl. Biol. 40:761.

Kopp, J.F. and R.C. Kroner, 1967. Trace metals in waters of the United States. U.S. Dept. Interior, Federal Water Pollution Control Adm., Cincinnati, Ohio.

Lloyd, R., 1961. Effect of dissolved oxygen concentrations on the toxicity of several poisons to rainbow trout (Salmo gairdnerii Richardson). Jour. Exp. Biology 38:447.

Millikan, C.R., 1947. Effect of molybdenum on the severity of toxicity symptoms in flax induced by an excess of either manganese, zinc, copper, nickel, or bobalt in the nutrient solution. Jour. Aust. Inst. Agr. Sci., 13:180.

National Technical Advisory Committee, 1968. Water Quality Criteria. Federal Water Pollution Control Administration, Washington, D.C.

Pickering, Q.H., 1968. Some effects of dissolved oxygen concentrations upon the toxicity of zinc to bluegill Lepomis macrochirus Rafinesque. Water Res. 2:187.

Pickering, Q.H. and C. Henderson, 1966. The acute toxicity of some heavy metals to different species of warm water fishes. International Jour. Air and Water Poll., 10:453.

Skidmore, J.F., 1964. Toxicity of zinc compounds to aquatic animals, with special reference to fish. Quarterly Rev. of Biology. 39:227.

Sprague, J.B. and B.A. Ramsay, 1965. Lethal levels of mixed copper-zinc solutions for juvenile salmon. Jour. Fish. Res. Bd. Canada. 22:425.

Vallee, B.L., 1957. Zinc and its biological significance. Arch. Indust. Health, 16:147.

Warnick, S.L. and H.L. Bell, 1969. The acute toxicity of some heavy metals to different species of aquatic insects. Jour. Water Poll. Control Fed. 41:(Part 1)280.

Wurtz, C.B., 1962. Zinc effects on freshwater mollusks. Nautilus, 76;53.

GLOSSARY

Acutely toxic: Causing death or severe damage to an organism by poisoning during a brief exposure period, normally ninety-six hours or less, although there is no clear line of demarcation between acute and chronic toxicity.

Chronically toxic: Causing death or damage to an organism by poisoning during prolonged exposure, which, depending on the organism tested and the test conditions and purposes, may range from several days, to weeks, months, or years.

Dose equivalent: The product of the absorbed dose from ionizing radiation and such factors to account for differences in biological effectiveness due to the type of radiation and its distribution in the body as specified by the International Commission on Radiological Units and Measurements (ICRU).

EC50: The concentration at which a specified effect is observed under the test conditions in a specified time in fifty percent of the organisms tested. Examples of specified effects are hemorrhaging, decreased feeding, dilation of pupils, and altered swimming patterns.

Gross alpha particle activity: The total radioactivity due to alpha particle emission as inferred from measurements on a dry sample exclusive of the contribution, if any, due to radon and uranium.

Gross beta particle activity: The total radioactivity due to beta particle emission as inferred from measurements on a dry sample exclusive of the contribution, if any, due to potassium-40 and other naturally occurring radionuclides.

LC25: The concentration of a toxicant that is lethal (fatal) to twenty-five percent of the organisms tested under the test conditions in a specified time.

LC50: The concentration of a toxicant which is lethal (fatal) to fifty percent of the organisms tested under the test conditions in a specified time.

LD50: The dose of a toxicant that is lethal (fatal) to fifty percent of the organisms tested under the test conditions in a specified time. A dose is the quantity actually administered to the organism and is not identical with a concentration, which is the amount of toxicant in a unit of test medium rather than the amount ingested by or administered to the organism.

Liter (l): The volume occupied by one kilogram of water at a pressure of 760 mm of mercury and a temperature of 4 C. A liter is 0.9463 quart.

Man-made beta particle and photon emitters: All radionuclides emitting beta particles and/or photons listed in Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air or Water for Occupational Exposure, NBS Handbook 69, except the daughter products of thorium-232, uranium-235, and uranium-238.

Microgram per liter (ug/l): The concentration at which one millionth of a gram (10^{-6}g) is contained in a volume of one liter. There are 453.59 grams in a pound.

Microgram per kilogram (ug/kg): The concentration at which one millionth of a gram (one microgram) is contained in a mass of one kilogram. A kilogram is 2.2046 pounds.

Milligram per kilogram (mg/kg): The concentration at which one thousandth of a gram (one milligram) is contained in a mass of one kilogram. A gram contains 1000 milligrams.

Milligram per liter (mg/l): The concentration at which one milligram (10^{-3}g) is contained in a volume of one liter.

Milliliter (ml): A volume equal to one thousandth of a liter.

Most Probable Number (MPN): The statistically determined number which represents the number of individuals most likely present in a given sample or aliquot, based on test data.

Nanogram per liter (ng/l): The concentration at which one billionth of a gram (10^{-9}g) is contained in a volume of one liter.

Part per million (ppm): A concentration at which one unit is contained in a total of a million units. Any units may be used (e.g., weight, volume) but in any given application identical units should be used (e.g., grams per million grams or liters per million liters).

Parts per thousand (o /oo): A concentration at which one unit is contained in a total of a thousand units. The rules for using this term are the same as those for parts per million. Normally, this term is used to specify the salinity of estuarine or sea waters.

Picocurie (pCi): That quantity of radioactive material producing 2.22 nuclear transformations per minute.

Rem: The unit of dose equivalent from ionizing radiation to the total body or any internal organ or organ system. A millirem (mrem) is 1/1000 of a rem (0.001 rem).

TL50: - Median Tolerance Limit: The concentration of a test material at which just fifty percent of the test animals are able to survive under test conditions for a specified period of exposure.

TLm : Synonymous with TL_{50}.