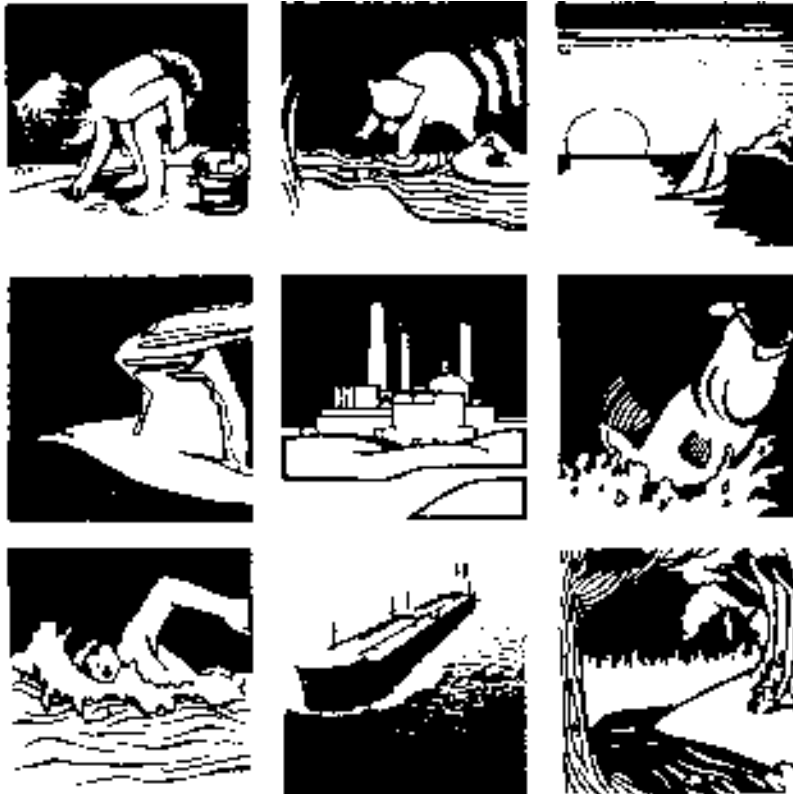




QUALITY CRITERIA for WATER 1986



TO INTERESTED PARTIES

Section 304(a)(1) of the Clean Water Act (33 U.S.C. 1314(a)(1)) requires the Environmental Protection Agency (EPA) to publish and periodically update ambient water quality criteria. These criteria are to accurately reflect 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, aesthetics, 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 organic and inorganic sedimentation for varying types of receiving waters. These criteria are not rules and they do not have regulatory impact. Rather, these criteria present scientific data and guidance of the environmental effects of pollutants which can be useful to derive regulatory requirements based on considerations of water quality impacts. When additional data has become available, these summaries have been updated to reflect the latest Agency recommendations on acceptable limits for aquatic life and human health protection.

Periodically EPA and its predecessor agencies has issued ambient water quality criteria, beginning in 1968 with the "Green Book" followed by the 1973 publication of the "Blue Book" (Water Quality Criteria 1972). In 1976, the "Red Book" (Quality

Criteria for Water) was published. On November 28, 1980 (45 FR 79318), and February 15, 1984 (49 FR 5831), EPA announced through Federal Register notices, the publication of 65 individual ambient water quality criteria documents for pollutants listed as toxic under section 307(a)(1) of the Clean Water Act. On July 29, 1985 (50 FR 30784), EPA published additional water quality criteria documents.

The development and publication of ambient water quality criteria has been pursued over the past 10 years and is an ongoing process. EPA expects to publish about 10 final criteria documents each year. Some of these will update and revise existing criteria recommendations and others will be issued for the first time.

In a continuing effort to provide those who use EPA's water quality and human health criteria with up-to-date criteria values and associated information, this document Quality Criteria for Water 1986 was assembled. This document includes summaries of all the contaminants for which EPA has developed criteria recommendations (Appendix A-C). The appropriate appendix is identified at the end of each summary. A more detailed description of these procedures can be found in the appropriate Appendix. Copies of this document can be obtained by contacting the U.S. Government Printing Office at:

U.S. Government Printing Office
Superintendent of Documents
N. Capitol and H Street N.W.
Washington, D.C. 20401

A fee is charged for this document.

Copies of the complete background ambient water quality

criteria documents containing all the data used to develop the criteria recommendations summarized herein and the "Red Book", including complete bibliographies are available only from:

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161

Telephone: (703) 487-4650

The NTIS order numbers for the criteria documents can be found in the Index. A fee is charged for copies of these documents.

As new criteria are developed and existing criteria revised, updated criteria summaries will be made available once a year to those who purchase this document through the U.S. Government Printing office. You will automatically be placed on the mailing list to receive annual updates. The cost for receiving annual updates is included in the purchase price of the document.

Quality Criteria for Water, 1986 is designed to be easily updated to reflect EPA's continuing work to present the latest scientific information and practices. Our planned schedule for future criteria development in the next few years is attached for your information.

The Agency is currently developing Acceptable Daily Intake (ADI) or Verified Reference Dose (RfD) values on a number of chemicals for Agency-wide use. Based upon this new analysis the values have changed significantly for 5 chemicals from those used in the original human health criteria calculation done in 1980. The chemicals affected are as follows:

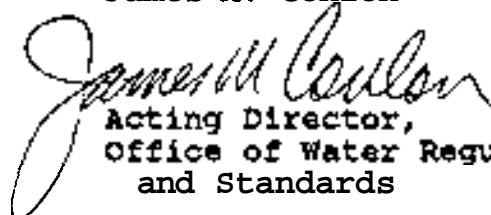
<u>chemical</u>	<u>1980 WQC</u>	<u>Draft RfD</u>
1. cyanide	200 ug/L	.02 mg/kg/day
2. Ethylbenzene	1.4 mg/L	.01 mg/kg/day
3. Nitrobenzene	19.8 mg/L	.0005 mg/kg/day
4. Phenol	3.5 mg/L	0.1 mg/kg/day
5. Toluene	14.3 mg/L	0.3 mg/kg/day

FOR **FURTHER** INFORMATION CONTACT:

Dr. Frank Gostomski at the above address or by phoning (202) 245-3030.

It is EPA's goal to continue to develop and make available ambient water quality criteria reflecting the latest scientific practices and information. In this way we can continue to improve and protect the quality of the Nation's waters.

James M. Conlon


 Acting Director,
 Office of Water Regulations
 and Standards

DRAFT CRITERIA DOCUMENTS TO BE PROPOSED

LATE FY 86/EARLY FY 87

Diethyhexylphthalate
1,2,4, Trichlorobenzene
Silver
Phenanthrene
2,4,5, Trichlorophenol
Organotins
Tributyltin
Selenium (no saltwater criteria)
Hexachlorobenzene
Antimony III
Acrolein (no saltwater criteria)

~~LATE FY 87~~/EARLY 88

Thallium (no saltwater criteria)
Tetrachloroethylene (no saltwater criteria)
Phenol
Toluene
Chloroform (no saltwater criteria)
Aniline
Acrylonitrile
Hexachlorocyclopentadiene (no saltwater criteria)
Dimethylphenol
Hexachlorobutadiene (no saltwater criteria)

- Both lists will incorporate aquatic and human health values.
- All above are toxic pollutants except for organotins and aniline which are non-conventionals.

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ACENAPHTHENE

CRITERIA:

Aquatic Life

The available data for acenaphthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 1,700 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of acenaphthene to sensitive freshwater aquatic animals but toxicity to freshwater algae occur at concentrations as low as 520 ug/L.

The available data for acenaphthene indicate that acute and chronic toxicity to saltwater aquatic life occurs at concentrations as low as 970 and 710 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to algae occurs at concentrations as low as 500 ug/L.

Human Health

Sufficient data are not available for acenaphthene to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor quality of ambient water the estimated level is 0.02 mg/L. It should be recognized that organoleptic data, have limitations as a basis for establishing water quality criteria, and have no demonstrated relationship to potential adverse human health effects.

ACROLEIN

CRITERIA:

Aquatic Life

The available data for acrolein indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 68 and 21 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for acrolein indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 55 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of acrolein to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of acrolein ingested through contaminated aquatic organisms, the ambient water criterion is determined to be 320 ug/L.

For the protection of human health from the toxic properties of acrolein ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 780 ug/L.

ACRYLONITRILE

CRITERIA:

Aquatic Life

The available data for acrylonitrile indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as **7,550** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of acrylonitrile to sensitive freshwater aquatic life but mortality occurs at concentrations as low as 2,600 ug/L with a fish species exposed for 30 days.

Only one saltwater species has been tested with acrylonitrile and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects resulting from exposure to acrylonitrile through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 0.58 ug/L, 0.058 ug/L, and 0.006 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption

of water, the levels are 6.5 ug/L, 0.65 ug/L, and 0.065 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

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.

**(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY**

ALKALINITY

CRITERION:

20 mg/L or more as CaCO_3 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 calcination, 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 biocarbonate will complex some toxic heavy metals and reduce their toxicity markedly. For these reasons, the National Technical Advisory Committee (NATC, 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 (NATC, 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

TABLE I*

Maximum Alkalinity In Waters Used As A Source
Of Supply Prior To Treatment

Industry	Alkalinity <u>mg/L as CaCO₃</u>
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

water and make the iron unavailable to .plants. Such deficiencies induce chlorosis and further plant damage. Usually alkalinity must exceed 6 mg/L before such effects are noticed, however.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

*ALDRIN-DIELDRIN

CRITERIA:

Aquatic Life

Dieldrin

For dieldrin the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0019 ug/L as a 24-hour average, and the concentration should not exceed 1.0 ug/L at any time.

For dieldrin the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0019 ug/L as a 24-hour average, and the concentration should not exceed 0.71 ug/L at any time.

Aldrin

For freshwater aquatic life the concentration of aldrin should not exceed 4.0 ug/L at any time. No data are available concerning the chronic toxicity of aldrin to sensitive freshwater aquatic life.

For saltwater aquatic life the concentration of aldrin should not exceed 1.3 ug/L at any time. No data are available concerning the chronic toxicity of aldrin to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to aldrin through ingestion of contaminated water and contaminated aquatic organisms, the

*Indicates suspended, canceled or restricted by U.S. EPA Office of Pesticides and Toxic Substances

ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} and 10^{-7} . The corresponding recommended criteria are 0.74 ng/L, 0.074 ng/L, and 0.0074 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 0.79 ng/L, 0.079 ng/L, and 0.0079 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to dieldrin through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} and 10^{-7} . The corresponding recommended criteria are 0.71 ng/L, 0.071 ng/L, and 0.0071 ng/L, respectively. If these above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 0.76 ng/L, 0.076 ng/L, and 0.0076 ng/L, respectively.

AMMONIA

SUMMARY -

All concentrations used herein are expressed as un-ionized ammonia (NH_3), because NH_3 , not the ammonium ion (NH_4^+) has been demonstrated to be the principal toxic form of ammonia. The data used in deriving criteria are predominantly from flow through tests in which ammonia concentrations were measured. Ammonia was reported to be acutely toxic to freshwater organisms at concentrations (uncorrected for pH) ranging from 0.53 to 22.8 mg/L NH_3 for 19 invertebrate species representing 14 families and 16 genera and from 0.083 to 4.60 mg/L NH_3 for 29 fish species from 9 families and 18 genera. Among fish species, reported 96-hour LC50 ranged from 0.083 to 1.09 mg/L for salmonids and from 0.14 to 4.60 mg/L NH_3 for nonsalmonids. Reported data from chronic tests on ammonia with two freshwater invertebrate species, both daphnids, showed effects at concentrations (uncorrected for pH) ranging from 0.304 to 1.2 mg/L NH_3 , and with nine freshwater fish species, from five families and seven genera, ranging from 0.0017 to 0.612 mg/L NH_3 .

Concentrations of ammonia acutely toxic to fishes may cause loss of equilibrium, hyperexcitability, increased breathing, cardiac output and oxygen uptake, and, in extreme cases, convulsions, coma, and death. At lower concentrations ammonia has many effects on fishes, including a reduction in hatching success, reduction in growth rate and morphological development, and pathologic changes in tissues of gills, livers, and kidneys.

Several factors have been shown to modify acute NH_3 toxicity in fresh water. Some factors alter the concentration of un-ionized ammonia in the water by affecting the aqueous ammonia equilibrium, and some factors affect the toxicity of un-ionized ammonia itself, either ameliorating or exacerbating the effects of ammonia. Factors that have been shown to affect ammonia toxicity include dissolved oxygen concentration, temperature, pH, previous acclimation to ammonia, fluctuating or intermittent exposures, carbon dioxide concentration, salinity, and the presence of other toxicants.

The most well-studied of these is pH; the acute toxicity of NH_3 has been shown to increase as pH decreases. Sufficient data exist from toxicity tests conducted at different pH values to formulate a mathematical expression to describe pH-dependent acute NH_3 toxicity. The very limited amount of data regarding effects of pH on chronic NH_3 toxicity also indicates increasing NH_3 toxicity with decreasing pH, but the data are insufficient to derive a broadly applicable toxicity/pH relationship. Data on temperature effects on acute NH_3 toxicity are limited and somewhat variable, but indications are that NH_3 toxicity to fish is greater as temperature decreases. There is no information available regarding temperature effects on chronic NH_3 toxicity.

Examination of pH and temperature-corrected acute NH_3 toxicity values among species and genera of freshwater organisms showed that invertebrates are generally more tolerant than fishes, a notable exception being the fingernail clam. There is no clear trend among groups of fish; the several most sensitive

tested species and genera include representatives from diverse families (Salmonidae, Cyprinidae, Percidae, and Centrarchidae). Available chronic toxicity data for freshwater organisms also indicate invertebrates (cladocerans, one insect species) to be more tolerant than fishes, again with the exception of the fingernail clam. When corrected for the presumed effects of temperature and pH, there is also no clear trend among groups of fish for chronic toxicity values, the most sensitive species including representatives from five families (Salmonidae, Cyprinidae, Ictaluridae, Centrarchidae, and Catostomidae) and having chronic values ranging by not much more than a factor or two. The range of acute-chronic ratios for 10 species from 6 families was 3 to 43, and acute-chronic ratios were higher for the species having chronic tolerance below the median. Available data indicate that differences in sensitivities between warm and coldwater families of aquatic organisms are inadequate to warrant discrimination in the national ammonia criterion between bodies of water with "warm" and "coldwater" fishes; rather, effects of organism sensitivities on the criterion are most appropriately handled by site-specific criteria derivation procedures.

Data for concentrations of NH_3 toxic to freshwater phytoplankton and vascular plants, although limited, indicate that freshwater plant species are appreciably more tolerant to NH_3 than are invertebrates or fishes. The ammonia criterion appropriate for the protection of aquatic animals will therefore in all likelihood be sufficiently protective of plant life.

Available acute and chronic data for ammonia with saltwater organisms are very limited, and insufficient to derive a saltwater criterion. A few saltwater invertebrate species have been tested, and the prawn *Macrobrachium rosenbergii* was the most sensitive. The few saltwater fishes tested suggest greater sensitivity than freshwater fishes. Acute toxicity of NH_3 appears to be greater at low pH values, similar to findings in freshwater. Data for saltwater plant species are limited to diatoms, which appear to be more sensitive than the saltwater invertebrates for which data are available.

More quantitative information needs to be published on the toxicity of ammonia to aquatic life. Several key research needs must be addressed to provide a more complete assessment of ammonia toxicity. These are: (1) acute tests with additional saltwater fish species and saltwater invertebrate species; (2) life-cycle and early life-stage tests with representative freshwater and saltwater organisms from different families, with particular attention to trends of acute-chronic ratios; (3) fluctuating and intermittent exposure tests with a variety of species and exposure patterns; (4) more complete tests of the individual and combined effects of pH and temperature, especially for chronic toxicity; (5) more histopathological and histochemical research with fishes, which would provide a rapid means of identifying and quantifying sublethal ammonia effects; and (6) studies on effects of dissolved and suspended solids on acute and chronic toxicity.

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if:

(1) the 1-hour* average concentration of un-ionized ammonia (in mg/L NH_3) does not exceed, more often than once every 3 years on the average, the numerical value given by $0.52/\text{FT}/\text{FPH}/2$,

where:

$$\text{FT} = 10 - 0.03(20 - \text{TCAP}); \text{TCAP} \leq T \leq 30$$

$$10 - 0.03(20 - T); 0 \leq T \leq \text{TCAP}$$

$$\text{FPH} = 1 \quad ; 8 < \text{pH} < 9$$

$$\frac{1 + 10^{-7.4 - \text{pH}}}{1.25} \quad ; 6.5 < \text{pH} < 8$$

$\text{TCAP} = 20$ C: Salmonids or other sensitive coldwater species present

$= 25$ C; Salmonids and other sensitive coldwater species absent

(*An averaging period of 1 hour may not be appropriate if excursions of concentrations to greater than 1.5 times the average occur during the hour; in such cases, a shorter averaging period may be needed.)

(2) the 4-day average concentration of un-ionized ammonia (in mg/L NH_3) does not exceed, more often than once every 3 years on the average, the average* numerical value given by $0.80/\text{FT}/\text{FPH}/\text{RATIO}$, where FT and FPH are as above and:

$$\text{RATIO} = 16 \quad ; 7.7 \leq \text{pH} \leq 9$$

$$= 24 \quad \frac{10^{-7.7-\text{pH}}}{1+10^{-7.4-\text{pH}}} \quad ; 6.5 \leq \text{pH} \leq 7.7$$

TCAP = 15 C; Salmonids or other sensitive
coldwater species present

= 20 C: Salmonids and other sensitive
coldwater species absent

(*Because these formulas are nonlinear in pH and temperature, the criterion should be the average of separate evaluations of the formulas reflective of the fluctuations of flow, pH, and temperature within the averaging period; it is not appropriate in general to simply apply the formula to average pH, temperature, and flow.)

The extremes for temperature (0, 30) and pH (6.5, 9) given in the above formulas are absolute. It is not permissible with current data to conduct any extrapolations beyond these limits. In particular, there is reason to believe that appropriate criteria at pH > 9 will be lower than the plateau between pH 8 and 9 given above.

Criteria concentrations for the pH range 6.5 to 9.0 and the temperature range 0 C to 30 C are provided in the following tables. Total ammonia concentrations equivalent to each unionized ammonia concentration are also provided in these tables. There are limited data on the effect of temperature on chronic toxicity. EPA will be conducting additional research on the effects of temperature on ammonia toxicity in order to fill perceived data gaps. Because of this uncertainty, additional site-specific information should be developed before these

criteria are used in wasteload allocation modeling. For example, the chronic criteria tabulated for sites lacking salmonids are less certain at temperatures much below 20 C than those tabulated at temperatures near 20 C. Where the treatment levels needed to meet these criteria below 20 C may be substantial, use of site-specific criteria is strongly suggested. Development of such criteria should be based upon site-specific toxicity tests.

Data available for saltwater species are insufficient to derive a criterion for saltwater.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to ammonia exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems respectively.

(1) One-hour average concentrations for ammonia.*

pH	0 C	5 C	10 C	15 C	20 C	25 C	30 C
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A. Salmonids or Other Sensitive Coldwater Species Present

Un-ionized Ammonia (mg/liter NH₃)

6.54	0.0091	0.0129	0.0182	0.026	0.036	0.036	0.036
6.75	0.0149	0.021	0.030	0.042	0.059	0.059	0.059
7.00	0.023	0.033	0.046	0.066	0.093	0.093	0.093
7.35	0.034	0.048	0.068	0.095	0.135	0.135	0.135
7.50	0.045	0.064	0.091	0.128	0.181	0.181	0.181
7.75	0.056	0.080	0.113	0.159	0.22	0.22	0.22
8.00	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.35	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.54	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.15	0.065	0.092	0.130	0.184	0.26	0.26	0.26
9.00	0.065	0.092	0.130	0.184	0.26	0.36	0.26

Total Ammonia (mg/liter NH₃)

6.50	35	33	31	30	29	20	14.3
6.75	32	30	28	27	27	18.6	13.3
7.00	28	26	25	24	23	16.4	11.6
7.25	23	22	20	19.7	19.2	13.4	9.5
7.50	17.4	16.3	15.5	14.9	14.6	10.1	7.3
7.75	12.2	11.4	10.9	10.5	10.3	7.25 N	5.2
8.00	8.0	7.5	7.1	6.9	6.8	4.8	3.5
8.25	4.5	4.2	4.1	4.0	3.9	2.8	2.1
8.50	2.6	2.4	2.3	2.3	2.3	1.71	1.28
8.75	1.47	1.40	1.37	1.38	1.42	1.07	0.83
9.00	0.86	0.83	0.83	0.86	0.91	0.72	0.58

B. Salmonids and Other Sensitive Coldwater Species Absent

Un-ionized Ammonia (mg/liter NH₃)

6.50	0.0091	0.0129	0.0182	0.026	0.036	0.051	0.051
6.75	0.0149	0.021	0.030	0.042	0.059	0.084	0.084
7.00	0.023	0.033	0.046	0.066	0.093	0.131	0.131
7.25	0.034	0.048	0.068	0.095	0.135	0.190	0.190
7.50	0.045	0.064	0.091	0.128	0.181	0.26	0.26
7.75	0.056	0.080	0.113	0.159	0.22	0.32	0.32
8.00	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.25	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.50	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.75	0.065	0.092	0.130	0.184	0.26	0.37	0.37
9.00	0.065	0.092	0.130	0.184	0.36	0.37	0.37

Total Ammonia (mg/liter NH₃)

6.50	35	33	31	30	29	29	20
6.75	32	30	28	27	27	26	18.6
7.00	28	26	25	24	23	23	16.4
7.25	23	22	20	19.7	19.2	19.0	13.5
7.50	17.4	16.3	15.5	14.9	14.6	14.5	10.3
7.75	12.2	11.4	10.9	10.5	10.5	10.2	7.3
8.00	8.0	7.5	7.1	6.9	6.8	6.8	4.9
8.25	4.5	4.2	4.1	4.0	3.9	4.0	2.9
8.50	2.6	2.4	2.3	2.3	2.3	2.4	1.81
8.75	1.47	1.40	1.31	1.38	1.42	1.52	1.18
9.00	0.86	0.83	0.83	0.86	0.91	1.01	0.82

* To convert these values to mg/liter N, multiply by 0.822.

(2) 4-day average concentrations for ammonia.*

pH	0 C	5 C	10 C	15 C	20 C	is C	30 C
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A. Salmonids or Other Sensitive Coldwater Species PresentUn-ionized Ammonia (mg/liter NH_3)

6.50	0.0007	0.0009	0.0013	0.0019	0.0019	0.0019	0.0019
6.75	0.0012	0.0017	0.0023	0.0033	0.0033	0.0033	0.0033
7.00	0.0021	0.0029	0.0042	0.0059	0.0059	0.0059	0.0059
7.25	0.0037	0.0052	0.0074	0.0105	0.0105	0.0105	0.0105
7.50	0.0066	0.0093	0.0132	0.0186	0.0186	0.0186	0.0186
7.75	0.0109	0.0153	0.022	0.031	0.031	0.031	0.031
8.00	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.25	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.50	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.75	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
9.00	0.0126	0.0171	0.025	0.035	0.035	0.035	0.035

Total Ammonia (mg/liter NH_3)

6.50	2.5	2.4	2.2	2.2	1.49	1.04	0.73
6.75	25	24	2.2	2.2	1.49	1.04	0.73
7.00	2.5	24	2.3	2.2	1.49	1.04	0.74
7.25	25	24	2.2	2.2	1.50	1.04	0.74
7.50	25	24	2.2	2.2	1.50	1.05	0.74
7.75	23	22	2.1	2.0	1.40	0.99	0.71
8.00	1.53	1.44	1.37	1.33	0.93	0.66	0.47
8.25	0.87	0.82	0.78	0.76	0.54	0.39	0.28
8.50	0.49	0.47	0.45	0.44	0.32	0.23	0.17
8.75	0.28	0.27	0.26	0.27	0.19	0.15	0.11
9.00	0.16	0.16	0.16	0.16	0.13	0.10	0.08

B. Salmonids and Other Sensitive Coldwater Species Absent†Un-ionized Ammonia (mg/liter NH_3)

6.50	0.0007	0.0009	0.0013	0.0019	0.0026	0.0026	0.0026
6.75	0.0012	0.0017	0.0023	0.0033	0.0047	0.0047	0.0047
7.00	0.0021	0.0029	0.0042	0.0059	0.0083	0.0083	0.0083
7.25	0.0037	0.0052	0.0074	0.0105	0.0148	0.0148	0.0148
7.50	0.0066	0.0093	0.0132	0.0186	0.026	0.026	0.026
7.75	0.0109	0.0153	0.022	0.031	0.043	0.043	0.043
8.00	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.25	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.50	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.75	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
9.00	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050

Total Ammonia (mg/liter NH_3)

6.50	25	2.4	2.2	2.2	2.1	1.46	1.20	1.03	0.85
6.75	25	2.4	2.2	2.3	2.1	1.47	1.21	1.04	0.85
7.00	2.5	2.4	2.2	2.2	2.1	1.47	1.21	1.04	0.85
7.25	25	2.4	2.2	2.2	2.1	1.48	1.22	1.05	0.86
7.50	25	2.4	2.2	2.2	2.1	1.49	1.22	1.06	0.87
7.75	23	2.2	2.1	2.0	1.98	1.39	1.14	1.00	0.82
8.00	1.53	1.44	1.37	1.33	1.31	0.93	0.76	0.67	0.55
8.25	0.87	0.82	0.78	0.76	0.76	0.54		0.40	
8.50	0.49	0.47	0.45	0.44	0.45	0.33		0.35	
8.75	0.28	0.27	0.26	0.27	0.27	0.21		0.16	
9.00	0.16	0.16	0.16	0.16	0.17	0.14		0.11	

* To convert these values to mg/liter N, multiply by 0.822.

† Site-specific criteria development is strongly suggested at temperatures above 20 C because of the limited data available to generate the criteria recommendation, and at temperatures below 20 C because of the limited data and because small changes in the criteria may have significant impact on the level of treatment required in meeting the recommended criteria.

The Agency acknowledges that the Criterion Continuous Concentration stream flow averaging period used for steady-state wasteload allocation modeling may be as long as 30 days in situations involving POTWs designed to remove ammonia where limited variability of effluent pollutant concentration and resultant concentrations in receiving waters can be demonstrated. In cases where low variability can be demonstrated, longer averaging periods for the ammonia Criterion Continuous Concentration (e.g., 30-day averaging periods) would be acceptable because the magnitude and duration of exceedences above the Criterion Continuous Concentration would be sufficiently limited. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985a).

(50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

ANTIMONY

CRITERIA:

Aquatic Life

The available data for antimony indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 9,000 and 1,600 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to algae occurs at concentrations as low as 610 ug/L.

No saltwater organisms have been adequately tested with antimony, and no statement can be made concerning acute or chronic toxicity.

Human Health

For the protection of human health from the toxic properties of antimony ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 146 ug/L.

For the protection of human health from the toxic properties of antimony ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 45 mg/L.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

ARSENIC

AQUATIC LIFE SUMMARY:

The chemistry of arsenic in water is complex and the form present in solution is dependent on such environmental conditions as Eh, pH, organic content, suspended solids, and sediment. The relative toxicities of the various forms of arsenic apparently vary from species to species. For inorganic arsenic(III) acute values for 16 freshwater animal species ranged from 812 ug/L for a cladoceran to 97,000 ug/L for a midge, but the three acute-chronic ratios only ranged from 4.660 to 4.862. The five acute values for inorganic arsenic(V) covered about the same range, but the single acute-chronic ratio was 28.71. The six acute values for MSMA ranged from 3,243 to 1,403,000 ug/L. The freshwater residue data indicated that arsenic is not bioconcentrated to a high degree but that lower **forms** of aquatic life may accumulate higher arsenic residues than fish. The low bioconcentration factor and short half-life of arsenic in fish tissue suggest that residues should not be a problem to predators of aquatic life.

The available data indicate that freshwater plants differ a great deal as to their sensitivity to arsenic(III) and arsenic(V). In comparable tests, the alga, Selenastrum capricornutum, was 45 times more sensitive to arsenic(V) than to arsenic(III), although other data present conflicting information on the sensitivity of this alga to arsenic(V). Many plant values for inorganic arsenic(III) were in the same range as the available chronic values for freshwater animals; several

plant values for arsenic(V) were lower than the one available chronic value.

The other toxicological data revealed a wide range of toxicity based on tests with a variety of freshwater species and endpoints. Tests with early life stages appeared to be the most sensitive indicator of arsenic toxicity. Values obtained from this type of test with inorganic arsenic(III) were lower than chronic values contained in Table 2. For example, an effect concentration of 40 ug/L was obtained in a test on inorganic arsenic(III) with embryos and larvae of a toad.

Twelve species of saltwater animals have acute values for inorganic arsenic(III) from 232 to 16,030 ug/L and the single acute-chronic ratio is 1.945. The only values available for inorganic arsenic(V) are for two invertebrate and are between 2,000 and 3,000 ug/L. Arsenic(III) and arsenic(V) are equally toxic to various species of saltwater algae, but the sensitivities of the species range from 19 ug/L to more than 1,000 ug/L. In a test with an oyster, a BCF of 350 was obtained for inorganic arsenic(III).

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of arsenic(III) does not exceed 190 ug/L more than once every 3 years on the

average and if the 1-hour average concentration does not exceed 360 ug/L more than once every 3 years on the average.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of arsenic(III) does not exceed 36 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 69 ug/L more than once every 3 years on the average. This criterion might be too high wherever *Skeletonema costatum* or *Thalassiosira aestivalis* are ecologically important.

Not enough data are available to allow derivation of numerical national water quality criteria for freshwater aquatic life for inorganic arsenic(V) or any organic arsenic compound. Inorganic arsenic(V) is acutely toxic to freshwater aquatic animals at concentrations as low as 850 ug/L and an acute-chronic ratio of 28 was obtained with the fathead minnow. Arsenic(V) affected freshwater aquatic plants at concentrations as low as 48 ug/L. Monosodium methanearsenate (MSMA) is acutely toxic to aquatic animals at concentrations as low as 1,900 ug/L, but no data are available concerning chronic toxicity to animals or toxicity to plants.

Very few data are available concerning the toxicity of any form of arsenic other than inorganic arsenic(III) to saltwater aquatic life. The available data do show that inorganic arsenic(V) is acutely toxic to saltwater animals at concentrations as low as 2,319 ug/L and affected some saltwater

plants at 13 to 56 ug/L. No data are available concerning the chronic toxicity of any form of arsenic other than inorganic arsenic(III) to saltwater aquatic life.

EPA believes that a measurement such as "acid-soluble" would provide a more scientifically correct basis upon which to establish criteria for metals. The criteria were developed on this basis. However, at this time, no EPA approved methods for such a measurement are available to implement the criteria through the regulatory programs of the Agency and the States. The Agency is considering development and approval of methods for a measurement such as acid-soluble. Until available, however, EPA recommends applying the criteria using the total recoverable method. This has two impacts: (1) certain species of some metals cannot be analyzed directly because the total recoverable method does not distinguish between individual oxidation states, and (2) these criteria may be overly protective when based on the total recoverable method.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to arsenic(III) exceeds the criterion. a stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation

model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

HUMAN HEALTH CRITERIA:

For the maximum protection of human health from the potential carcinogenic effects due to exposure of arsenic through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-6} , and 10^{-7} . The corresponding criteria are 22 ng/L, 2.2 ng/L, and .22 ng/L, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 175 ng/L, 17.5 ng/L, and 1.75 ng/L, respectively. Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on an "acceptable" risk level.

(45 F.R. 79318 Nov. 28, 1980) (50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

ASBESTOS

CRITERIA:

Aquatic Life

No freshwater organisms have been tested with any asbestiform mineral and no statement can be made concerning acute or chronic toxicity

No saltwater organisms have been tested with any asbestiform mineral and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to asbestos through ingestion of water and contaminated aquatic organisms, the ambient water concentration should be zero. The estimated levels which would result in increased lifetime cancer risks of 10^{-5} , 10^{-6} , and 10^{-7} are 300,000 fibers/L, 30,000 fibers/L, and 3,000 fibers/L, respectively. Estimates for consumption of aquatic organisms only, excluding the consumption of water cannot be made.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR **METHODOLOGY**

BACTERIA

CRITERIA

Freshwater Bathing

Based on a statistically sufficient number of samples (generally not less than 5 samples equally spaced over a 30-day period), the geometric mean of the indicated bacterial densities should not exceed one or the other of the following:⁽¹⁾

E. coli	126 per 100 ml; or
enterococci	33 per 100 ml;

no sample should exceed a one sided confidence limit (C.L.) calculated using the following as guidance:

designated bathing beach	75% C.L.
moderate use for bathing	82% C.L.
light use for bathing	90% C.L.
infrequent use for bathing	95% C.L.

based on a site-specific log standard deviation, or if site data are insufficient to establish a log standard deviation, then using **0.4** as the log standard deviation for both indicators.

Marine Water Bathing

Based on a statistically sufficient number of samples (generally not less than **5** samples equally spaced over a 30-day period), the geometric mean of the enterococci densities should not exceed 35 per 100 ml; no sample should exceed a one sided confidence limit using the following as guidance:

designated bathing beach	75% C.L.
moderate use for bathing	82% C.L.
light use for bathing	90% C.L.
infrequent use for bathing	95% C.L.

based on a site-specific log standard deviation, or if site data are insufficient to establish a log standard deviation, then using 0.7 as the log standard deviation.

Note (1) - Only one indicator should be used. The Regulatory agency should select the appropriate indicator for its conditions.

Shellfish Harvesting Waters

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

RATIONALE

Bathing Waters

A recreational water quality criterion can be defined as a "quantifiable relationship between the density of an indicator in the water and the potential human health risks involved in the water's recreational use." From such a definition, a criterion can be adopted which establishes upper limits for densities of indicator bacteria in waters that are associated with acceptable health risks for swimmers.

The Environmental Protection Agency, in 1972, initiated a series of studies at marine and fresh water bathing beaches which were designed to determine if swimming in sewage-contaminated marine and fresh water carries a health risk for bathers; and, if so, to what type of illness. Additionally, the Agency wanted to determine which bacterial indicator is best correlated to swimming-associated health effects and if the relationship is strong enough to provide a criterion. (1)

The quantitative relationships between the rates of swimming-associated health effects and bacterial indicator densities were determined using regression analysis. Linear relationships were estimated from data grouped on the basis of summers or trials with similar indicator densities. The data for each summer were analyzed by pairing the geometric mean indicator density for a summer bathing season at each beach with the corresponding swimming-associated gastrointestinal illness rate for the same summer. The swimming-associated illness rate was determined by subtracting the gastrointestinal illness rate in nonswimmers from that for swimmers. These two variables from multiple beach sites were used to calculate a regression coefficient, y-intercept and 95% confidence intervals for the paired data. In the marine studies the total number of points for use in regression analysis was increased by collecting trial days with similar indicator densities from each study location and placing them into groups. The swimming-associated illness rate was determined as before, by subtracting the nonswimmer illness rate of all the individuals included in the grouped trial days from the swimmer illness rate during these same grouped trial days. The grouping by trial days with similar indicator densities approach was not possible with the freshwater data because the variation of bacterial indicator densities in freshwater samples was not large enough to allow such an adjustment to be made. For the saltwater studies the results of the regression analyses of illness rates against indicator density data was very similar using the "by summer" or "by grouped trial days" approaches.

The methods used to enumerate the bacterial indicator densities which showed the best relationship to swimming-associated gastroenteritis rates were specifically developed for the recreational water quality studies.'

These membrane filter methods have successfully undergone precision and bias testing by the EPA Environmental Monitoring and Support Laboratory. (2)

Several monitoring situations to assess bacterial quality are encountered by regulatory agencies. The situation needing the most rigorous monitoring is the designated swimming beach. Such areas are frequently lifeguard protected, provide parking and other public access and are heavily used by the public. Public beaches of this type were used by EPA in developing the relationship described in this document.

Other recreational activities may involve bodies of water which are regulated by individual State water quality standards. These recreational resources may be natural wading ponds used by children or waters where incidental full body contact occurs because of water skiing or other similar activities.

EPA's evaluation of the bacteriological data indicated that using the fecal coliform indicator group at the maximum geometric mean of 200 per 100 ml, recommended in Quality Criteria for Water would cause an estimated 8 illness per 1,000 swimmers at fresh water beaches and 19 illness per 1,000 swimmers at marine beaches. These relationships are only approximate and are based on applying ratios of the geometric means of the various indicators from the EPA studies to the 200 per 100 ml fecal

coliform criterion. However, these are EPA's best estimates of the accepted illness rates for areas which apply the EPA fecal coliform criterion.

The E. coli and enterococci criteria presented in Table 1 were developed using these currently accepted illness rates. The equations developed by Dufour⁽³⁾ and Cabelli⁽⁴⁾ were used to calculate the geometric mean indicator densities corresponding to the accepted gastrointestinal illness rates. These densities are for steady state dry weather conditions. The beach is in noncompliance with the criteria if the geometric mean of several bacterial density samples exceeds the value listed in Table 1.

Noncompliance is also signaled by an unacceptably high value for any single bacterial sample. The maximum acceptable bacterial density for a single sample is set higher than that for the geometric mean, in order to avoid necessary beach closings based on single samples. In deciding whether a beach should be left open, it is the long term geometric mean bacterial density that is of interest. Because of day-to-day fluctuations around this mean, a decision based on a single sample (or even several samples) may be erroneous, i.e., the sample may exceed the recommended mean criteria even though the long-term geometric mean is protective, or may fall below the maximum even if this mean is in the nonprotective range.

To set the single sample maximum, it is necessary to specify the desired chance that the beach will be left open when the protection is adequate. This chance, or confidence level, was based on Agency judgment. For the simple decision rule considered here, a smaller confidence level corresponds to a more

stringent (i.e. lower) single sample maximum. Conversely, a greater confidence level corresponds to less stringent (i.e. higher) maximum values. This technique reduces the chances of single samples inappropriately indicating violations of the recommended criteria.

By using a control chart analogy (5) and the actual log standard deviations from the EPA studies, single sample maximum densities for various confidence levels were calculated. EPA then assigned qualitative use intensities to those confidence levels. A low confidence level (75%) was assigned to designated beach areas because a high degree of caution should be used to evaluate water quality for heavily used areas. Less intensively used areas would allow less restrictive single sample limits. Thus, 95% confidence might be appropriate for swimmable water in remote areas. Table 1 summarizes the results of these calculations. These single sample maximum levels should be recalculated for individual areas if significant differences in log standard deviations occur.

The levels displayed in Table 1 depend not only on the assumed standard deviation of log densities, but also on the chosen level of acceptable risk. While this level was based on the historically accepted risk, it is still arbitrary insofar as the historical risk was itself arbitrary.

Currently EPA is not recommending a change in the stringency of its bacterial criteria for recreational waters. Such a change does not appear warranted until more information based on greater experience with the new indicators can be accrued. EPA and the

State Agencies can then evaluate the impacts of change in terms of beach closures and other restricted uses.

Shellfish Harvesting Waters

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 disease 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. Thus, attention has been directed toward the adoption of the fecal coliform test to measure more accurately 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. 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 15 and the 90th percentile should not exceed 43 for a 5-tube, 3-dilution method.

EPA is currently (1986) co-sponsoring, with the National

Oceanic and Atmospheric Administration, research into the application of the enterococci and E. coli indicators for assessing the quality of shellfish harvesting waters. The Food and Drug Administration is also reviewing the results of these studies. A change to the new indicators may be forthcoming if the studies show a correlation between gastrointestinal disease and the consumption of raw shellfish from waters with defined densities of the new indicators. However, these studies have not sufficiently progressed to justify any change at this time. Thus, evaluation of the microbiological suitability of waters for recreational taking of shellfish should be based upon the fecal coliform bacterial levels. (6)

CRITERIA FOR INDICATOR FOR BACTERIOLOGICAL DENSITIES

		Single sample Maximum Allowable Density (4), (5)				
Acceptable Swimming Associated Gastro- enteritis Rate per 1000 swimmers		Steady State Geometric Mean Indicator Density	Designated Beach Area (upper 75% C.L.)	Moderate Full Body contact Recreation (upper 82% C.L.)	Lightly Used Full m y Contact Recreation (upper 90% C. L.)	Infrequently Used Full m y Contact Recreation (upper 95% C. L.)
Freshwater						
enterococci	8	33(1)	61	89	108	151
E. coli	8	126(2)	235	298	406	576
Marine Water						
enterococci	19	35(3)	104	124	276	500

Notes:

- (1) Calculated to nearest whole number using equation:

$$(\text{mean enterococci density}) = \text{antilog}_{10} \frac{\text{illness rate}/1000 \text{ people} + 6.28}{9.40}$$
- (2) Calculated to nearest whole number using equation:

$$(\text{mean E. coli density}) = \text{antilog}_{10} \frac{\text{illness rate}/1000 \text{ people} + 11.74}{9.40}$$
- (3) Calculated to nearest whole number using equation:

$$(\text{mean enterococci density}) = \text{antilog}_{10} \frac{\text{illness rate}/1000 \text{ people} - 0.20}{12.17}$$
- (4) Single sample limit = $\text{antilog}_{10} (\text{Log}_{10} \text{ indicator geometric mean density}/100 \text{ ml}) + \text{Factor determined from } x (\text{log}_{10} \text{ stand. areas under the Normal probability curve for the assumed level of probability})$

The appropriate factors for the indicated one sided confidence levels are:

75% C.L. = .675
 82% CL = .935
 90% C.L. = 1.28
 95% C.L. = 1.65

- (5) Based on the observed log standard deviations during the EPA studies: 0.4 for freshwater E coli and enterococci; and 0.7 for marine water enterococci. Each jurisdiction should establish its own standard deviation for its conditions which would then vary the single sample limit.

References

1. Ambient Water Quality Criteria for Bacteria - 1986, EPA 440/5-84-002, U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, DC. (NTIS access #: PB 86-158-045)
2. Test Methods for *Escherichia coli* and *Enterococci* in Water By The Membrane Filter procedure, EPA 600/4-85-076, U.S. Environmental Protection Agency, Cincinnati, OH. (NTIS access #: PB 86-158-052)
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4. Cabelli, V. J. 1981. Health Effects Criteria for Marine Recreational Waters. EPA-600/1-80-031, U.S. Environmental Protection Agency, Cincinnati, OH.
5. ASTM. 1951. Manual on Quality Control of Materials. Special Technical Publication 15-C, American Society for Testing and Materials, Philadelphia, PA.
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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, 1965; NAS, 1974). However, barium ions generally are thought to be rapidly precipitated or removed from solution by absorption 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 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; 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 (Sollman, 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 2 liters of water. From other 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 fresh water conditions, a restrictive criterion for aquatic life appears unwarranted.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

BENZENE

CRITERIA:

Aquatic Life

The available data for benzene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 5,300 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of benzene to sensitive freshwater aquatic life.

The available data for benzene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 5,100 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of benzene to sensitive saltwater aquatic life, but adverse effects occur at concentrations as low as 700 ug/L with a fish species exposed for 168 days.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to benzene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may **result** in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 6.6 ug/L, 0.66 ug/L, and

0.066 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 400 ug/L, 40.0 ug/L, and 4.0 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

BENZIDINE

CRITERIA:

Aquatic Life

The available data for benzidine indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as **2,500** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of benzidine to sensitive freshwater aquatic life.

No saltwater organisms have been tested with benzidine and no statement can be made concerning acute and chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to benzidine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 1.2 ng/L, 0.12 ng/L, and 0.01 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are **5.3** ng/L, 0.53 ng/L, and **0.05** ng/L, respectively.

BERYLLIUM

CRITERIA:

Aquatic Life

The available data for beryllium indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 130 and 5.3 $\mu\text{g/L}$, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Hardness has a substantial effect on acute toxicity.

The limited saltwater data base available for beryllium does not permit any statement concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to beryllium through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 37 ng/L , 3.7 ng/L , and 0.37 ng/L , respectively. If these estimates are made for

consumption of aquatic organisms only, excluding consumption of water, the levels are 641 ng/L, 64.1 ng/L, and 6.41 ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

BORON

CRITERION:

750 mg/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 seawater is reported as 45 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 ug/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 ug/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.

CADMIUM

AQUATIC LIFE SUMMARY:

Freshwater acute values for cadmium are available for species in 44 genera and range from 1.0 ug/L for rainbow trout to 28,000 ug/L for a mayfly. The antagonistic effect of hardness on acute toxicity has been demonstrated with five species. Chronic tests have been conducted on cadmium with 12 freshwater fish species and 4 invertebrate species with chronic values ranging from 0.15 ug/L for Daphnia magna to 156 ug/L for the Atlantic salmon. Acute-chronic ratios are available for eight species and range from 0.9021 for the chinook salmon to 433.8 for the flagfish.

Freshwater aquatic plants are affected by cadmium at concentrations ranging from 2 to 7,400 ug/L. These values are in the same range as the acute toxicity values for fish and invertebrate species, and are considerably above the chronic values. Bioconcentration factors (BCFs) for cadmium in fresh water range from 164 to 4,190 for invertebrates and from 3 to 2,213 for fishes.

Saltwater acute values for cadmium and five species of fishes range from 577 ug/L for larval Atlantic silverside to 114,000 ug/L for juvenile mummichog. Acute values for 30 species of invertebrates range from 15.5 ug/L for a mysid to 135,000 ug/L for an oligochaete worm. The acute toxicity of cadmium generally increases as salinity decreases. The effect of temperature seems to be species-specific. Two life-cycle tests with Mysidopsis bahia under different test conditions resulted in similar chronic values of 8.2 and 7.1 ug/L, but the acute-chronic ratios were 1.9 and 15, respectively. The acute values appear to

reflect effects of salinity and temperature, whereas the few available chronic values apparently do not. A life-cycle test with Mysidopsis bigelowi also resulted in a chronic value of 7.1 ug/L and an acute-chronic ratio of 15. Studies with microalgae and macroalgae revealed effects at 22.8 to 860 ug/L.

BCFs determined with a variety of saltwater invertebrates ranged from 5 to 3,160. BCFs for bivalve molluscs were above 1,000 in long exposures, with no indication that steady-state had been reached. Cadmium mortality is cumulative for exposure periods beyond 4 days. Chronic cadmium exposure resulted in significant effects on the growth of bay scallops at 78 ug/L and on reproduction of a copepod at 44 ug/L.

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration (in ug/L) of cadmium does not exceed the numerical value given by $e^{(0.7852[\ln(\text{hardness})]-3.490)}$ more than once every 3 years on the average and if the one-hour average concentration (in ug/L) does not exceed the numerical value given by $e^{(1.128[\ln(\text{hardness})]-3.828)}$ more than once every 3 years on the average. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 the 4-day average concentrations of cadmium are 0.66, 1.1, and 2.0 ug/L, respectively, and the 1-hour average concentrations are

1.8, 3.9 and 8.6 ug/L. If brook trout, brown trout, and striped bass are as sensitive as some data indicate, they might not be protected by this criterion.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of cadmium does not exceed 9.3 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 43 ug/L more than once every 3 years on the average. The little information that is available concerning the sensitivity of the American lobster to cadmium indicates that this important species might not be protected by this criterion. In addition, data suggest that the acute toxicity of cadmium is salinity dependent: therefore, the 1-hour average concentration might be underprotective at low salinities and overprotective at high salinities.

EPA believes that a measurement such as "acid-soluble" would provide a more scientifically correct basis upon which to establish criteria for metals. The criteria were developed on this basis. However, at this time, no EPA-approved methods for such a measurement are available to implement the criteria through the regulatory programs of the Agency and the States. The Agency is considering development and approval of methods for a measurement such as acid-soluble. Until available, however, EPA recommends applying the criteria using the total recoverable method. This has two impacts: (1) certain species of some metals

cannot be analyzed directly because the total recoverable method does not distinguish between individual oxidation states, and (2) these criteria may be overly protective when based on the total recoverable method.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to cadmium exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems, respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

HUMAN HEALTH CRITERIA:

The ambient water quality criterion for cadmium is recommended to be identical to the existing drinking water

standard which is 10 ug/L. Analysis of the toxic effects data resulted in a calculated level which is protective *of* human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value *is* comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams *of* aquatic organisms was not derived.

(45 F.R. 79318 Nov. 28, 1980) (50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

CARBON TETRACHLORIDE

CRITERIA:

Aquatic Life

The available data for carbon tetrachloride indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 35,200 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of carbon tetrachloride to sensitive freshwater aquatic life.

The available data for carbon tetrachloride indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 50,000 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of carbontetrachloride to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to carbon tetrachloride through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 4.0 ug/L, 0.40ug/L, and 0.04 ug/L, respectively. If these estimates are made for

consumption of aquatic organisms only, excluding consumption of water, the levels are 69.4 ug/L, 6.94 ug/L, and 0.69 ug/L respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

CHLORDANE

CRITERIA:

Aquatic Life

For chlordane the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0043 ug/L as a 24-hour average, and the concentration should not exceed 2.4 ug/L at any time.

For chlordane the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0040 ug/L as a 24-hour average, and the concentration should not exceed 0.09 ug/L at any time.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to chlordane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 4.6 ng/L, 0.46 ng/L, and 0.046 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 4.8 ng/L, 0.48 ng/L, and 0.048 ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

CHLORINATED BENZENES

CRITERIA:

Aquatic Life

The available data for chlorinated benzenes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 250 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of the more toxic of the chlorinated benzenes to sensitive freshwater aquatic life, but toxicity occurs at concentrations as low as 50 ug/L for a fish species exposed for 7.5 days.

The available data for chlorinated benzenes indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 160 and 129 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

Monochlorobenzene

For comparison purposes, two approaches were used to derive criterion levels for monochlorobenzene. Based on available toxicity data, for the protection of public health the derived level is 488 ug/L. Using available organoleptic data, to control undesirable taste and odor quality of ambient water the estimated level is 20 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing water quality criteria, and have no demonstrated relationship to potential adverse human health effects.

CHLORINATED ETHANES

CRITERIA:

Aquatic Life

The available freshwater data for chlorinated ethanes indicate that toxicity increases greatly with increasing chlorination, and that acute toxicity occurs at concentrations as low as 118,000 ug/L for 1,2-dichloroethane, 18,000 ug/L for two trichloroethanes, 9,320 ug/L for two tetrachloroethanes, 7,240 ug/L for pentachloroethane, and 980 ug/L for hexachloroethane. Chronic toxicity occurs at concentrations as low as 20,000 ug/L for 1,2-dichloroethane, 9,400 ug/L for 1,1,2-trichloroethane, 2,400 ug/L for 1,1,2,2-tetrachloroethane, 1,100 ug/L for pentachloroethane, and 540 ug/L for hexachloroethane. Acute and chronic toxicity would occur at lower concentrations among species that are more sensitive than those tested.

The available saltwater data for chlorinated ethanes indicate that toxicity increases greatly with increasing chlorination and that acute toxicity to fish and invertebrate species occurs at concentrations as low as 113,000 ug/L for 1,2-dichloroethane, 31,200 ug/L for 1,1,1-trichloroethane, 9,020 ug/L for 1,1,2,2-tetrachloroethane, 390 ug/L for pentachloroethane, and 940 ug/L for hexachloroethane. Chronic toxicity occurs at concentrations as low as 281 ug/L for pentachloroethane. Acute and chronic toxicity would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to 1,2-dichloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 9.4 ug/L, 0.94 ug/L, and **0.094** ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 2,430 ug/L, 243 ug/L, and 24.3 ug/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to 1,1,2-trichloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 6.0 ug/L, 0.6 ug/L, and 0.06 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 418 ug/L, 41.8 ug/L, and 4.18 ug/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to 1,1,2,2-tetrachloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 1.7 ug/L, 0.17 ug/L, and 0.017 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 107 ug/L, 10.7 ug/L, and 1.07 ug/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to hexachloroethane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 19 ug/L, 1.9 ug/L, and 0.19 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 87.4 ug/L, 8.74 ug/L, and 0.87 ug/L, respectively.

For the protection of human health from the toxic properties of 1,1,1-trichloroethane ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 18.4 mg/L.

For the protection of human health from the toxic properties of 1,1,1-trichloroethane ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 1.03 ug/l.

Because of insufficient available data for monochloroethane, 1,1-dichloroethane, 1,1,1,2-tetrachloroethane, and pentachloroethane, satisfactory criteria cannot be derived at this time, using the present guidelines.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

CHLORINATED NAPHTHALENES

CRITERIA:

Aquatic Life

The available data for chlorinated naphthalenes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as **low** as 1,600 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of chlorinated naphthalenes to sensitive freshwater aquatic life.

The available data for chlorinated naphthalenes, indicate that acute toxicity to saltwater aquatic life occurs at concentrations **as** low as **7.5** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of chlorinated naphthalenes to sensitive saltwater aquatic life.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time because of insufficient available data for chlorinated naphthalenes.

CHLORINE

SUMMARY:

Thirty-three freshwater species in 28 genera have been exposed to TRC and the acute values range from 28 ug/L for Daphnia magna to 710 ug/L for the threespine stickleback. Fish and invertebrate species had similar ranges of sensitivity. Freshwater chronic tests have been conducted with two invertebrate and one fish species and the chronic values for these three species ranged from less than 3.4 to 26 ug/L, with acute-chronic ratios from 3.7 to greater than 78.

The acute sensitivities of 24 species of saltwater animals in 21 genera have been determined for CPO, and the LC50 range from 26 ug/L for the eastern oyster to 1,418 ug/L for a mixture of two shore crab species. This range is very similar to that observed with freshwater species, and fish and invertebrate species had similar sensitivities. Only one chronic test has been conducted with a saltwater species, Menidia peninsulae, and in this test the acute chronic ratio was 1.162.

The available data indicate that aquatic plants are more resistant to chlorine than fish and invertebrate species.

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected

unacceptably if the 4-day average concentration of total residual chlorine does not exceed 11 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 19 ug/L more than once every 3 years on the average.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of chlorine-produced oxidants does not exceed 7.5 ug/L more than once every 3 years on the average and if the one-hour average concentration does not exceed 13 ug/L more than once every 3 years on the average.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to chlorine exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for

the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems, respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

(50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

CHLORINATED PHENOLS

CRITERIA:

Aquatic Life

The available freshwater data for chlorinated phenols indicate that toxicity generally increases with increasing chlorination, and that acute toxicity occurs at concentrations as low **as** 30 ug/L for 4-chloro-3-methylphenol to greater than **500,000** ug/L for other compounds. Chronic toxicity occurs at concentrations as low as 970 ug/L for **2,4,6-trichlorophenol**. Acute and chronic toxicity would occur at lower concentrations among species that are more sensitive than those tested.

The available saltwater data for chlorinated phenols indicate that toxicity generally increases with increasing chlorination and that acute toxicity occurs at concentrations as low as 440 ug/L for 2,3,5,6-tetrachlorophenol and 29,700 ug/L for 4-chlorophenol. Acute toxicity would occur at lower concentrations **among** species that are more sensitive than those tested. No data are available concerning the chronic toxicity of chlorinated phenols to sensitive saltwater aquatic life.

Human Health

Sufficient data are not available for 3-chlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water, the estimated level is 0.1 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated

relationship to potential adverse human health effects.

Sufficient data are not available for 4-chlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 0.1 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 2,3-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 0.04 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 2,5-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 0.5 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 2,6-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control

undesirable taste and odor qualities of ambient water the estimated level is 0.2 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 3,4-dichlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 0.3 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

For comparison purposes, two approaches were used to derive criterion levels for 2,4,5-trichlorophenol. Based on available toxicity data, to protect public health the derived level is 2.6 mg/L. Using available organoleptic data, to control undesirable taste and odor quality of ambient water the estimated level is 1.0 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

For the maximum protection of human health from the potential carcinogenic effects of exposure to 2,4,6-trichlorophenol through the ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this

chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 12 ug/L, 1.2 ug/L, and 0.12 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 36 ug/L, 3.6 ug/L, and 0.36 ug/L, respectively. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 2 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 2,3,4,6-tetrachlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 1.0 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 2-methyl-4-chlorophenol to derive a criterion level which would protect against any potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 1,800 ug/L. It

should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion and have no demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 3-methyl-4-chlorophenol to derive a criterion level which would protect against any potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 3,000 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

Sufficient data are not available for 3-methyl-6-chlorophenol to derive a criterion level which would protect against any potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 20 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

CHLOROALKYL ETHERS

CRITERIA :

Aquatic Life

The available data for chloroalkyl ethers indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 238,000 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. NO definitive data are available concerning the chronic toxicity of chloroalkyl ethers to sensitive freshwater aquatic life.

No saltwater organism has been tested with any chloroalkyl ether and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

For the protection of human health from the toxic properties of bis(2-chloroisopropyl) ether ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 34.7 ug/L.

For the protection of human health from the toxic properties of bis(2-chloroisopropyl) ether ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 4.36 mg/L.

For the maximum protection of human health from the potential carcinogenic effects of exposure to bis(chloromethyl) ether through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero

level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 37.6×10^{-6} ug/L, 3.76×10^{-6} ug/L, and 0.376×10^{-6} ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 18.4×10^{-3} ug/L, 1.84×10^{-3} ug/L, and 0.184×10^{-3} ug/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to bis(2-chloroethyl) ether through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 0.30 ug/L, 0.030 ug/L, and 0.003 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 13.6 ug/L, 1.36 ug/L, and 0.136 ug/L, respectively.

CHLOROFORM

CRITERIA:

Aquatic Life

The available data for chloroform indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as **28,900** ug/L, and would occur at lower concentrations among species that are more sensitive than the three tested species. Twenty-seven-day **LC50** values indicate that chronic toxicity occurs at concentrations as low as 1,240 ug/L, and could occur at lower concentrations among species or other life stages that are more sensitive than the earliest life cycle stages of the rainbow trout. The data base for saltwater species is limited to one test and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to chloroform through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are **1.90** ug/L, **0.19** ug/L, and **0.019** ug/L, respectively. If these

estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 157 ug/L, 15.7 ug/L, and 1.57 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

CHLOROPHENOXY

2,4-D; 2

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 19th and final dose of 3600 mg (67 mg/kg) produced mild symptoms.

The long-term no-effects levels (mg/kg/day) are listed for the rat and the dog. Those 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.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

CHROMIUM (VI)

AQUATIC LIFE SUMMARY:

Acute toxicity values for chromium(VI) are available for freshwater animal species in 27 genera and range from 23.07 ug/L for a cladoceran to 1,870,000 ug/L for a stonefly. These species include a wide variety of animals that perform a wide spectrum of ecological functions. All five tested species of daphnids are especially sensitive. The few data that are available indicate that the acute toxicity of chromium(VI) decreases as hardness and pH increase.

The chronic value for both rainbow trout and brook trout is 264.6 ug/L, which is much lower than the chronic value of 1,987 ug/L for the fathead minnow. The acute-chronic ratios for these three fishes range from 18.55 to 260.8. In all three chronic tests a temporary reduction in growth occurred at low concentrations. Six chronic tests with five species of daphnids gave chronic values that range from <2.5 to 40 ug/L and the acute-chronic ratios range from 1.130 to >9.680. Except for the fathead minnow, all the chronic tests were conducted in soft water. Green algae are quite sensitive to chromium(VI). The bioconcentration factor obtained with rainbow trout is less than 3. Growth of chinook salmon was reduced at a measured concentration of 16 ug/L.

The acute toxicity of chromium (VI) to 23 saltwater vertebrate and invertebrate species ranges from 2,000 ug/L for a polychaete worm and a mysid to 105,000 ug/L for the mud snail. The chronic values for a polychaete range from <13 to 36.74 ug/L, whereas

that for a mysid is 132 ug/L. The acute-chronic ratios range from 15.38 to >238.5. Toxicity to macroalgae was reported at 1,000 and 5,000 ug/L. Bioconcentration factors for chromium(VI) range from 125 to 236 for bivalve molluscs and polychaetes.

CHROMIUM (III)

Acute values for chromium(III) are available for 20 freshwater animal species in 1a genera ranging from 2,221 ug/L for a mayfly to 71,060 ug/L for caddisfly. Hardness has a significant influence on toxicity, with chromium(III) being more toxic in soft water.

A life-cycle test with Daphnia magna in soft water gave a chronic value of 66 ug/L. In a comparable test in hard water the lowest test concentration of 44 ug/L inhibited reproduction of Daphnia magna, but this effect may have resulted from ingested precipitated chromium. In a life-cycle test with the fathead minnow in hard water the chronic value was 1,025 ug/L. Toxicity data are available for only two freshwater plant species. A concentration of 9,900 ug/L inhibited growth of roots of Eurasian watermilfoil. A freshwater green alga was affected by a concentration of 397 ug/L in soft water. No bioconcentration factor has been measured for chromium(III) with freshwater organisms.

Only two acute values are available for chromium (III) in saltwater 10,300 ug/L for the eastern oyster and 31,500 ug/L for the mummichog. In a chronic test effects were not observed on a polychaete worm at 50,400 ug/L at pH = 7.9, but acute lethality occurred when pH = 4.5. Bioconcentration factors for saltwater

organisms and chromium(III) range from 86 to 153, similar to the bioconcentration factors for chromium(VI) and saltwater species.

NATIONAL CRITERIA:

CHROMIUM(VI)

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of chromium(VI) does not exceed 11 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 16 ug/L more than once every 3 years on the average.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms, and their uses should not be affected unacceptably if the 4-day average concentration of chromium(VI) does not exceed 50 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 1,100 ug/L more than once every 3 years on the average. Data suggest that the acute toxicity of chromium (VI) is salinity dependent; therefore, the 1-hour average concentration might be underprotective at low salinities.

CHROMIUM(III)

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not

be affected unacceptably if the 4-day average concentration (in ug/L) of chromium(III) does not exceed the numerical value given by $e^{(0.8190[\ln(\text{hardness})]+1.561)}$ more than once every 3 years on the average and if the 1-hour average concentration (in ug/L) does not exceed the numerical value given by $e^{(0.8190[\ln(\text{hardness})]+3.688)}$ more than once every 3 years on the average. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 the **4-day** average concentrations of chromium(III) are 120, 210, and 370 ug/L, respectively, and the 1-hour average concentrations are 980, 1,700, and 3,100 ug/L.

No saltwater criterion can be derived for chromium(III), but 10,300 ug/L is the EC50 for eastern oyster embryos, whereas **50,400** ug/L did not affect a polychaete worm in a life-cycle test.

EPA believes that a measurement such as "**acid-soluble**" would provide a more scientifically correct basis upon which to establish criteria for minerals. The criteria were developed on this basis. However, at this time, no EPA-approved methods for such a measurement are available to implement the criteria through the regulatory programs of the Agency and the States. The Agency is considering development and approval of methods for a measurement such as acid-soluble. Until available, however, EPA recommends applying the criteria using the total recoverable method. This has two impacts: (1) certain species of some metals cannot be analyzed directly because the total recoverable method does not distinguish between individual

oxidation states, and (2) these criteria may be overly protective when based on the total recoverable method.

The recommended exceedence frequency of 3 years is the 'Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to chromium exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected or require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 or the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems, respectively. These matters are discussed in more detail in the Technical Support Document for water Quality-Based Toxics Control (U.S. EPA, 1985).

HUMAN HEALTH CRITERIA:

For the protection of human health from the toxic properties of Chromium III ingested through water and contaminated aquatic

organisms, the ambient water criterion is determined to be 170 mg/L.

For the protection of human health from the toxic properties of Chromium III ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 3433 mg/L.

The ambient water quality criterion for total Chromium VI is recommended to be identical to the existing drinking water standard which is 50 ug/L. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

(45 F.R. 79318 Nov. 28, 1980) (50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

2-CHLOROPHENOL

CRITERIA:

Aquatic Life

The available data for 2-chlorophenol indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 4,380 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of 2-chlorophenol to sensitive freshwater aquatic life, but flavor impairment occurs in one species of fish at concentrations as low as 2,000 ug/L.

No saltwater organisms have been tested with 2-chlorophenol and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

Sufficient data are not available for 2-chlorophenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 0.1 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

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 human 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 human 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, polyhydroxyl 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 mu 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 standard or blue end of the spectrum (Birge and Juday, 1930). In

TABLE 2.

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

<u>Industry or Industrial Use</u>	<u>Color units</u>
Boiler make up	1,200
cooling water	1,200
Pulp and paper	360
Chemical and allied products	500
Petroleum	25

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 standardized (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.

*COPPER

AQUATIC LIFE SUMMARY:

Acute toxicity data are available for species in 41 genera of freshwater animals. At a hardness of 50 mg/L the genera range in sensitivity from 16.74 ug/L for Ptychocheilus to 10,240 ug/L for Acroneuria. Data for eight species indicate that acute toxicity decreases as hardness increases. Additional data for several species indicate that toxicity also decreases with increases in alkalinity and total organic carbon.

Chronic values are available for 15 freshwater species and range from 3.873 ug/L for brook trout to 60.36 ug/L for northern pike. Fish and invertebrate species seem to be about equally sensitive to the chronic toxicity of copper.

Toxicity tests have been conducted on copper with a wide range of freshwater plants and the sensitivities are similar to those of animals. Complexing effects of the test media and a lack of good analytical data make interpretation and application of these results difficult. Protection of animal species, however, appears to offer adequate protection of plants. Copper does not appear to bioconcentrate very much in the edible portion of freshwater aquatic species.

The acute sensitivities of saltwater animals to copper range from 5.8 ug/L for the blue mussel to 600 ug/L for the green crab. A chronic life-cycle test has been conducted with a mysid, and adverse effects were observed at 77 ug/L but not at 38 ug/L, which resulted in an acute-chronic ratio of 3.346. Several

*Indicates suspended, canceled or restricted by U.S. EPA Office of Pesticides and Toxic Substances

saltwater algal species have been tested, and effects were observed between 5 and 100 ug/L. Oysters can bioaccumulate copper up to 28,200 times, and become bluish-green, apparently without significant mortality. In long-term exposures, the bay scallop was killed at 5 ug/L.

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration (in ug/L) of copper does not exceed the numerical value given by $e^{(0.8545[\ln(\text{hardness})]-1.465)}$ more than once every 3 years on the average and if the 1-hour average concentration (in ug/L) does not exceed the numerical value given by $e^{(0.9422[\ln(\text{hardness})]-1.464)}$ more than once every 3 years on the average. For example, at hardnesses of 50, 100, and **200** mg/L as CaCO_3 the 4-day average concentrations of copper are 6.5, 12, and 21 ug/L, respectively, and the 1-hour average concentrations are 9.2, 18, and 34 ug/L.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the 1-hour average concentration of copper does not exceed 2.9 ug/L more than once every 3 years on the average.

EPA believes that a measurement such as "**acid-soluble**" would

provide a more scientifically correct basis upon which to establish criteria for metals. The criteria were developed on this basis. However, at this time, no EPA approved methods for such a measurement are available to implement the criteria through the regulatory programs of the Agency and the States. The Agency is considering development and approval of methods for a measurement such as acid-soluble. Until available, however, EPA recommends applying the criteria using the total recoverable method. This has two impacts: (1) certain species of some metals cannot be analyzed directly because the total recoverable method does not distinguish between individual oxidation states, and (2) these criteria may be overly protective when based on the total recoverable method.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to copper exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in developing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for

Criterion Maximum Concentration design flow and 745 or 7Q10 for the Criterion Continuous Concentration (CCC) design flow in steady-state models for unstressed and stressed systems respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

HUMAN HEALTH CRITERIA:

Sufficient data is not available for copper to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 1 mg/L. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

(45 F.R. 79318 Nov. 28, 1980) (50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

CYANIDE

AQUATIC LIFE SUMMARY:

Data on the acute toxicity of free cyanide (the sum of cyanide present as HCN and CN⁻, expressed as CN) are available for a wide variety of freshwater species that are involved in diverse community functions. The acute sensitivities ranged from 44.73 ug/L to 2,490 ug/L, but all of the species with acute sensitivities above 400 ug/L were invertebrates. A long-term survival, and a partial and life-cycle test with fish gave chronic values of 13.57, 7.849, and 16.39 ug/L, respectively. Chronic values for two freshwater invertebrate species were 18.33 and 34.06 ug/L. Freshwater plants were affected at cyanide concentrations ranging from 30 ug/L to 26,000 ug/L.

The acute toxicity of free cyanide to saltwater species ranged from 4.893 ug/L to >10,000 ug/L and invertebrates were both the most and least sensitive species. Long-term survival in an early life-stage test with the sheepshead minnow gave a chronic value of 36.12 ug/L. Long-term survival in a mysid life-cycle test resulted in a chronic value of 69.71 ug/L. Tests with the red macroalga, Champia parvula, showed cyanide toxicity at 11 to 25 ug/L, but other species were affected at concentrations up to 3,000 ug/L.

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their **Uses** indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected

unacceptably if the 4-day average concentration of cyanide does not exceed 5.2 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 22 ug/L more than once every 3 years on the average.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the 1-hour average concentration of cyanide does not exceed 1.0 ug/L more than once every 3 years on the average.

EPA believes that a measurement such as "acid soluble" would provide a more scientifically correct basis upon which to establish criteria for cyanide. The criteria were developed on this basis. However, at this time, no EPA-approved methods for such a measurement are available to implement the criteria through the regulatory programs of the Agency and the States.

The Agency is considering development and approval of methods for a measurement such as acid soluble. Until available, however, EPA recommends applying the criteria using the total recoverable method. These criteria may be overly protective when based on the total recoverable method.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to cyanide exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to

recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

HUMAN HEALTH CRITERIA

The ambient water quality criterion for cyanide is recommended to be identical to the existing drinking water standard which is 200 ug/L. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

NOTE: The U.S. EPA is currently developing Acceptable Daily Intake (ADI) or Verified Reference Dose (RfD) values for Agency-wide use for this chemical. The new value should be substituted when it becomes available. The January, 1986, draft Verified Reference Dose document cites an RfD of .02 mg/kg/day for free cyanide.

~~DDT AND~~ METABOLITES

CRITERIA:

Aquatic Life

DDT

For DDT and its metabolites the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0010 ug/L as a 24-hour average and the concentration should not exceed 1.1 ug/L at any time.

For DDT and its metabolites the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0010 ug/L as a 24-hour average and the concentration should not exceed 0.13 ug/L at any time.

TDE

The available data for TDE indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 0.6 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of TDE to sensitive freshwater aquatic life.

The available data for TDE indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 3.6 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of TDE to sensitive saltwater aquatic life.

DDE

The available data for DDE indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as

1,050 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of DDE to sensitive freshwater aquatic life.

The available data for DDE indicate that acute toxicity to saltwater aquatic life occurs in concentrations as low as 14 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of DDE to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to DDT through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} and 10^{-7} . The corresponding recommended criteria are 0.24 ng/L, 0.024 ng/L, and 0.0024 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 0.24 ng/L, 0.024 ng/L, and 0.0024 ng/L, respectively.

DEMETON

CRITERION:

0.1 ug/L for freshwater and marine aquatic life

RATIONALE:

Static LC50 bioassays yielded toxicity values for the organo-phosphorus 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, 1982; 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 LC50 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, 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 LC50 data for invertebrates apparently are limited to a single, nominal concentration static exposure of Gammarus fasciatus (Sanders, 1972). These 24- and 96-hour LC50 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 subacute concentrations of organophosphates.

There are few data on the toxicity of demeton to marine organisms. Butler (1964) reported a 48-hour EC50 of 63 ug/L for

the pink shrimp, Peneaus duorarum, and a 24-hour LC50 of 550 ug/L for the spot, Leiostomus xanthurus.

Chronic demeton toxicity data for freshwater organism are not currently available. Since no data are 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 10 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.

DICHLOROBENZENES

CRITERIA:

Aquatic Life

The available data for dichlorobenzenes indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 1,120 and 763 ug/L, respectively, and **would** occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichlorobenzenes indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 1,970 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the ~~chronic~~ toxicity of dichlorobenzenes to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of dichlorobenzene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be **400 ug/L**.

For the protection of human health from the toxic properties of dichlorobenzenes ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be **2.6 mg/L**.

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SEE APPENDIX B FOR METHODOLOGY

DICHLOROBENZIDINE

CRITERIA:

Aquatic Life

The data base available for dichlorobenzidines and freshwater organisms is limited to one test on bioconcentration of 3,3-dichlorobenzidine, and therefore, no statement can be made concerning acute or chronic toxicity.

No saltwater organisms have been tested with any dichlorobenzidine, and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to dichlorobenzidine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 0.103 ug/L, 0.010 ug/L, and 0.001 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 0.204 ug/L, 0.020 ug/L, and 0.002 ug/L, respectively.

DICHLOROETHYLENES

CRITERIA:

Aquatic Life

The available data for dichloroethylenes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,600 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of dichloroethylenes to sensitive freshwater aquatic life.

The available data for dichloroethylenes indicate that acute and chronic toxicity to saltwater aquatic life occurs at concentrations as low as **224,000** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dichloroethylenes to sensitive saltwater aquatic life.

Human Health

1,1-Dichloroethylene

For the maximum protection of human health from the potential carcinogenic effects of exposure to 1,1 dichloroethylene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 0.33 ug/L, 0.033 ug/L, and 0.003 ug/L, respectively. If

these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 18.5 ug/L, 1.85 ug/L, and 0.185 ug/L, respectively.

1,2-Dichloroethylene

Using the present guidelines, a satisfactory criterion cannot be derived at this time because of insufficient available data for 1,2-dichloroethylene.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

2,4-DICHLOROPHENOL

CRITERIA:

Aquatic Life

The available data for 2,4-dichlorophenol indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 2,020 and 365 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Mortality to early life stages of one species of fish occurs at concentrations as low as 70 ug/L.

Only one test has been conducted with saltwater organisms and 2,4-dichlorophenol and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for 2,4-dichlorophenol. Based on available toxicity data, to protect public health the derived level is 3.09 mg/L. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 0.3 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

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SEE APPENDIX B FOR METHODOLOGY

DICHLOROPROPANES/DICHLOROPROPENES

CRITERIA:

Aquatic Life

The available data for dichloropropanes indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 23,000 and 5,700 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropene indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 6,060 and **244** ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropane indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 10,300 and 3,040 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 790 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dichloropropene to sensitive saltwater aquatic life.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time because of insufficient available data for dichloropropanes.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

2,4-DIMETHYLPHENOL

CRITERIA

Aquatic Life

The available data for 2,4-dimethylphenol indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 2,120 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dimethylphenol to sensitive freshwater aquatic life.

No saltwater organisms have been tested with 2,4-dimethyl-phenol and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

Sufficient data are not available for 2,4-dimethylphenol to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor quality of ambient water the estimated level is 400 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

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SEE APPENDIX B FOR METHODOLOGY

DINITROTOLUENE

CRITERIA:

Aquatic Life

The available data for dinitrotoluenes indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 330 and 230 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dinitrotoluenes indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 590 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dinitrotoluenes to sensitive saltwater aquatic life but a decrease in algal cell numbers occurs at concentrations as low as 370 ug/L.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to 2,4-dinitrotoluene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} and 10^{-7} . The corresponding recommended criteria are 1.1 ug/L, 0.11 ug/L, and 0.011 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption

of water, the levels are 91 ug/L, 9.1 ug/L, and 0.91 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

DIPHENYLHYDRAZINE

CRITERIA:

Aquatic Life

The available data for 1,2-diphenylhydrazine indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 270 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of 1,2-diphenylhydrazine to sensitive freshwater aquatic life.

No saltwater organisms have been tested with 1,2-diphenylhydrazine and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to diphenylhydrazine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 422 ng/L, 42 ng/L, and 4 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 5.6 ug/L, 0.56 ug/L, and 0.056 ug/L, respectively.

ENDOSULFAN

CRITERIA:

Aquatic Life

For endosulfan the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.056 ug/L as a 24-hour average and the concentration should not exceed 0.22 ug/L at any time.

For endosulfan the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0087 ug/L as a 24-hour average and the concentration should not exceed 0.034 ug/L at any time.

Human Health

For the protection of human health from the toxic properties of endosulfan ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 74 ug/L.

For the protection of human health from the toxic properties of endosulfan ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 159 ug/L.

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SEE APPENDIX B FOR METHODOLOGY

*ENDRIN

CRITERIA:

Aquatic Life

For endrin the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0023 ug/L as a 24-hour average, and the concentration should not exceed 0.18 ug/L at any time.

For endrin the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0023 ug/L as a 24-hour average, and the concentration should not exceed 0.037 ug/L at any time.

Human Health

The ambient water quality criterion for endrin is recommended to be identical to the existing water standard which is 1.0 ug/L. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from assumption of **6.5** g of aquatic organisms was not derived.

*Indicates suspended, canceled or restricted by U.S. EPA Office of Pesticides and Toxic Substances

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

ETHYLBENZENE

CRITERIA:

Aquatic Life

The available data for ethylbenzene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 32,000 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of ethylbenzene to sensitive freshwater aquatic life.

The available data for ethylbenzene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 430 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of ethylbenzene to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of ethylbenzene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 1.4 mg/L.

For the protection of human health from the toxic properties of ethylbenzene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 3.28 mg/L,

(45 F.R. 79318, November 28, 1980)
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NOTE: The U.S. EPA is currently developing Acceptable Daily Intake (ADI) or Verified Reference Dose (RfD) values for Agency-wide use for this chemical. The new value should be substituted when it becomes available. The January, 1986, draft Verified Reference Dose document cites an RfD of 0.1 mg/kg/day for ethylbenzene.

FLUORANTHENE

CRITERIA:

Aquatic Life

The available data for fluoranthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 3,980 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of fluoranthene to sensitive-freshwater aquatic life.

The available data for fluoranthene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 40 and 16 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the protection of human health from the toxic properties of fluoranthene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 42 ug/L.

For the protection of human health from the toxic properties of fluoranthene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 54 ug/L.

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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 100 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 pressures is very low. Dissolved nitrogen (N_2), which comprises roughly 80 percent of the earth's atmosphere, is nearly inert biologically and is the most significant cause of gas bubble disease in aquatic animals. Dissolved oxygen, which is extremely bioactive, is consumed by the metabolic processes of the organism and is less important in causing serious gas bubble disease though it may be involved in initiating emboli formation in the blood (Nebeker et al. 1976a).

Percent saturation of water containing a given amount of gas varies with the absolute temperature and with the pressure. Because 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 because of 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 because of 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 because of 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; 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 (1m), 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 nonsalmonids.

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 collected 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. 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 of 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),

GUTHION

CRITERION:

0.01 ug/L for freshwater and marine aquatic life.

RATIONALE:

Ninety-six-hour LC50 values for fish exposed to the organophosphorus pesticide guthion range from 4 to 4270 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 one week (Meyer, 1965). Analysis of plankton and pond water 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 30C in pond water and in a phosphate buffer protected from light in the laboratory. The half-life in pond water was 1.2 days whereas that in the laboratory solution was 10 days. The more rapid degradation in pond water 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 Gakskatter (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, 1960; Carter, 1971). Inhibition of brain AChE of fishes by 46 percent or more has been associated with harmful effects in exposures to there 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 twentyfold accumulation. LC50 toxicity values for birds are relatively high and range from 70 to 2,000 mg/kg (Tucker and Crabtree, 1970).

Ninety-six-hour LC50 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, 1972). Sanders (1972) exposed the grass shrimp, Palaemonetes kadiakensis, to guthion in a continuous flow bioassay for up to 20 days and found that the 5- and 20-day LC50 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 LC50 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 LC50 for the white mullet, Mugil

curema, was found to be 5.5 ug/L guthion (Butler, 1963). The 96-hour LC50 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 LC50 for the fish, Pleuronectes limanda, to be 10 to 30 ug/L. The 48-hour LC50 for the European shrimp, Crangon crangon, was found to be 0.33 ug/L guthion (Portman, 1972). Butler (1963) found that the 24-hour EC50 for blue crab, Callinectes sapidus, was 550 ug/L and the 48-hour EC50 for pink shrimp, Penaeus duorarum, was 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 .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.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

HALOETHERS

CRITERIA:

Aquatic Life

The available data for haloethers indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 360 and 122 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

No saltwater organisms have been tested with any haloether and therefore, no statement can be made concerning acute or chronic toxicity.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time because of insufficient available data for haloethers.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

HALOMETHANES

CRITERIA:

Aquatic Life

The available data for halomethanes indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 11,000 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of halomethanes to sensitive freshwater aquatic life.

The available data for halomethanes indicate that acute and chronic toxicity to saltwater aquatic life occurs at concentrations as low as 12,000 and 6,400 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. A decrease in algal cell numbers occurs at concentrations as low as 11,500 ug/L.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to chloromethane, bromomethane, dichloromethane, bromodichloromethane, tribromomethane, dichlorodifluoromethane, trichlorofluoromethane, or combinations of these chemicals through ingestion of contaminated water and aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime

are estimated at 10^{-5} , 10^{-6} and 10^{-7} . The corresponding recommended criteria are 1.9 ug/L, 0.19 ug/L, and 0.019 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 157 ug/L, 15.7 ug/L, and 1.57 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

HARDNESS

INTRODUCTION:

Water hardness is caused by the polyvalent metallic ions dissolved in water. In fresh water these are primarily 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).

TABLE 3.

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 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 (1968) and 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 whereas 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 LC50 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

TABLE 4.

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

<u>Industry</u>	<u>Maximum Concentration</u> <u>mg/L as CaCO₃</u>
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.

than hardness.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

HEPTACHLOR

CRITERIA:

Aquatic Life

For heptachlor the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0038 ug/L as a 24-hour average, and the concentration should not exceed 0.52 ug/L at any time.

For heptachlor the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0036 ug/L as a 24-hour average, and the concentration should not exceed 0.053 ug/L at any time.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to heptachlor through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 2.00 ng/L, 0.20 ng/L, and 0.020 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 2.04 ng/L, 0.20 ng/L, and 0.020. ng/L, respectively.

HEXACHLOROBUTADIENE

CRITERIA:

Aquatic Life

The available data for hexachlorobutadiene indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 90 and 9.3 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for hexachlorobutadiene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 32 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of hexachlorobutadiene to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to hexachlorobutadiene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 4.47 ug/L, 0.45 ug/L, and 0.045 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding

consumption *of* water, the levels are 500 ug/L, 50 ug/L, and 5.0 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

HEXACHLOROCYCLOHEXANE

CRITERIA:

Aquatic Life

Lindane

For lindane the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.080 ug/L as a 24-hour average and the concentration should not exceed 2.0 ug/L at any time.

For saltwater aquatic life the concentration of lindane should not exceed 0.16 ug/L at any time. No data are available concerning the chronic toxicity of lindane to sensitive saltwater aquatic life.

BHC

The available data for a mixture of isomers of **BHC** indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 100 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of a mixture of isomers of **BHC** to sensitive freshwater aquatic life.

The available data for a mixture of isomers of **BHC** indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 0.34 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of a mixture of isomers of **BHC** to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 22 ng/L, 2.2 ng/L, and .22 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 74 ng/L, 7.4 ng/L, and .74 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 134 ng/L, 13.4 ng/L, and 1.34 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 450 ng/L, 45.0 ng/L, and 4.50 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of γ -hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 186 ng/L, 18.6 ng/L, and 1.86 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 625 ng/L, 62.5 ng/L, and 6.25 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to technical-hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 52 ng/L, 5.2 ng/L, and .52 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 174 ng/L, 17.4 ng/L, and 1.74 ng/L, respectively.

Using the present guidelines, satisfactory criteria cannot be derived at this time for d- and e- hexachlorocyclohexane because of insufficient available data.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

HEXACHLOROCYCLOPENTADIENE

CRITERIA:

Aquatic Life

The available data for hexachlorocyclopentadiene indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 7.0 and 5.2 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for hexachlorocyclopentadiene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 7.0 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of hexachlorocyclopentadiene to sensitive saltwater aquatic life.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for hexachlorocyclopentadiene. Based on available toxicity data, to protect public health the derived level is **206** ug/L. Using available organoleptic data, to control undersirable taste and odor quality of ambient water the estimated level is 1 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing water quality criteria, and have no demonstrated relationship to potential adverse human health effects.

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; this is true especially 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 little effect on aquatic

life.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

ISOPHORONE

CRITERIA:

Aquatic Life

The available data for isophorone indicate that acute toxicity to freshwater aquatic life occurs at concentrations **as** low as **117,000** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of isophorone to sensitive freshwater aquatic life.

The available data for isophorone indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as **12,900** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of isophorone to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of isophorone ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be **5.2 mg/L**.

For the protection of human health from the toxic properties of isophorone ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be **520 mg/L**.

LEAD

AQUATIC LIFE SUMMARY:

The acute toxicity of lead to several species of freshwater animals has been shown to decrease as the hardness of water increases. At a hardness of 50 mg/L the acute sensitivities of 10 species range from 142.5 ug/L for an amphipod to 235,900 ug/L for a midge. Data on the chronic effects of lead on freshwater animals are available for two fish and two invertebrate species. The chronic toxicity of lead also decreases as hardness increases and the lowest and highest available chronic values (12.26 and 128.1 ug/L) are both for a cladoceran, but in soft and hard water, respectively. Acute-chronic ratios are available for three species and range from 18 to 62. Freshwater algae are affected by concentrations of lead above 500 ug/L, based on data for four species. Bioconcentration factors are available for four invertebrate and two fish species and range from 42 to 1,700.

Acute values are available for 13 saltwater animal species and range from 315 ug/L for the mummichog to 27,000 ug/L for the soft shell clam. A chronic toxicity test was conducted with a mysid; unacceptable effects were observed at 37 ug/L but not at 17 ug/L and the acute-chronic ratio for this species is 124.8. A species of macroalgae was affected at 20 ug/L. Available bioconcentration factors range from 17.5 to 2,570.

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of

Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration (in $\mu\text{g/L}$) of lead does not exceed the numerical value given by $e^{(1.273[\ln(\text{hardness})]-4.705)}$ more than once every 3 years on the average and if the 1-hour average concentration (in $\mu\text{g/L}$) does not exceed the numerical value given by $e^{(1.273[\ln(\text{hardness})]-1.460)}$ more than once every 3 years on the average. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 the 4-day average concentrations of lead are 1.3, 3.2, and 7.7 $\mu\text{g/L}$, respectively, and the 1-hour average concentrations are 34, 82, and 200 $\mu\text{g/L}$.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of lead does not exceed 5.6 $\mu\text{g/L}$ more than once every 3 years on the average and if the 1-hour average concentration does not exceed 140 $\mu\text{g/L}$ more than once every three years on the average.

EPA believes that a measurement such as **"acid-soluble"** would provide a more scientifically correct basis upon which to establish criteria for metals. The criteria were developed on this basis. However, at this time, no EPA-approved methods for such a measurement are available to implement the criteria through the regulatory programs of the Agency and the States. The Agency is considering development and approval of methods for a measurement such as acid-soluble. Until available, however, EPA recommends applying the criteria using the total recoverable

method. This has two impacts: (1) Certain species of some metals cannot be analyzed directly because the total recoverable method does not distinguish between individual oxidation states, and (2) these criteria may be overly protective when based on the total recoverable method.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to lead exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however,¹ and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems, respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

HUMAN HEALTH CRITERIA:

The ambient water quality criterion for lead is recommended to be identical to the existing drinking water standard which is 50 ug/L. Analysis of the toxic effects data resulted in a calculated level which is protective to human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 grams of aquatic organisms was not derived.

(45 F.R. 79318 Nov. 28, 1980) (50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

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 LC50 between 120 and 265 ug/L for 4 species of salmonids. Macek and McAllister (1970) found a 96-hour LC50 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 LC50 for bluegill, Lepomis macrochirus, in a flow-through system at 110 ug/L. Macek and McAllister (1970) reported a similar 96-hour LC50 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 LC50 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 LC50 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 1 to 2 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 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 LC50 for several marine fishes at 20 °C in static, aerated seawater. The 96-hour LC50 values (in ug/L) were: Menidia menidia, 125; Mugil cephalus, 550; Fundulus majalis, 250; Fundulus heteroclitus, 240; Sphaeroides maculatus, 3250; Anquilla rostrata, 82; and Thalassoma bifasciatum, 27. Katz (1961) reported the static 24-hour LC50 for Gasterosteus aculeatus in 25 o/oo saltwater as 76.9 ug/L active ingredient. The 96-hour LC50 for striped bass,

Marone 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 LC50 (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 LC50 for fertilized eggs of oysters was estimated by Davis and Hidu (1969) to be 9.07 mg/L and the 14-day LC50 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 AChE and because such inhibition may be additive with repeated exposures and may be caused by any of the organophosphorus 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.

MANGANESE

CRITERIA:

50 ug/L for domestic water supplies (welfare):

100 ug/L for protection of consumers of marine molluscs.

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 molluscs, 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 is rarely 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.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

*MERCURY

AQUATIC LIFE SUMMARY:-

Data are available on the acute toxicity of mercury(II) ,to 28 genera of freshwater animals. Acute values for invertebrate species range from 2.2 ug/L for *Daphnia pulex* to 2,000 ug/L for three insects. Acute values for fishes range from 30 ug/L for the guppy to 1,000 ug/L for the Mozambique tilapia. Few data are available for various organomercury compounds and mercurous nitrate, and they all appear to be 4 to 31 times more acutely toxic than mercury(II).

Available chronic data indicate that methylmercury is the most chronically toxic of the tested mercury compounds. Tests on methylmercury with Daphnia magna and brook trout produced chronic values less than 0.07 ug/L. For mercury(II) the chronic value obtained with ~~Daphnia magna~~ was about 1.1 ug/L and the acute-chronic ratio was 4.5. In both a life-cycle test and an early life-stage test on mercuric chloride with the fathead minnow, the chronic value was less than 0.26 ug/L and the acute-chronic ratio was over 600.

Freshwater plants show a wide range of sensitivities to mercury, but the most sensitive plants appear to be less sensitive than the most sensitive freshwater animals to both mercury(II) and methylmercury. A bioconcentration factor of 4,994 is available for mercury(II), but the bioconcentration factors for methylmercury range from 4,000 to 85,000.

*Indicates suspended, canceled or restricted by U.S. EPA
Office of Pesticides and Toxic Substances

Data on the acute toxicity of mercuric chloride are available for **29** genera of saltwater animals, including annelids, molluscs, crustaceans, echinoderms, and fishes. Acute values range from 3.5 ug/L for a mysid to 1,678 ug/L for winter flounder. Fishes tend to be more resistant and molluscs and crustaceans tend to be more sensitive to the acute toxic effects of mercury(II). Results of a life-cycle test with the mysid show that mercury(II) at a concentration of 1.6 ug/L significantly affected time of first spawn and productivity; the resulting acute-chronic ratio was 3.1.

Concentrations of mercury that affected growth and photosynthetic activity of one saltwater diatom and six species of brown algae range from 10 to 160 ug/L. Bioconcentration factors of 10,000 and 40,000 have been obtained for mercuric chloride and methylmercury with an oyster.

NATIONAL CRITERIA:

Derivation of a water quality criterion for mercury is more complex than for most metals because of methylation of mercury in sediment, in fish, and in the food chain of fish. Apparently almost all mercury currently being discharged is mercury(II). Thus mercury(II) should be the only important possible cause of acute toxicity and the Criterion Maximum Concentrations can be based on the acute values for mercury(II).

The best available data concerning long-term exposure of fish to mercury(II) indicates that concentrations above 0.23 ug/L caused statistically significant effects on the fathead minnow and caused the concentration of total mercury in the whole body

to exceed 1.0 mg/kg. Although it is not known what percent of the mercury in the fish was methylmercury, it is also not known whether uptake from food would increase the concentration in the fish in natural situations. Species such as rainbow trout, coho salmon, and especially the bluegill, might suffer chronic effects and accumulate high residues of mercury about the same as the fathead minnow.

With regard to long-term exposure to methylmercury, McKim et al. (1976) found that brook trout can exceed the FDA action level without suffering statistically significant adverse effects on survival, growth, or reproduction. Thus for methylmercury the Final Residue Value would be substantially lower than the Final Chronic Value.

Basing a freshwater criterion on the Final Residue Value of 0.012 ug/L derived from the bioconcentration factor of 81,700 for methylmercury with the fathead minnow (Olson et al. 1975) essentially assumes that all discharged mercury is methylmercury. On the other hand, there is the possibility that in field situations uptake from food might add to the uptake from water. Similar considerations apply to the derivation of the saltwater criterion of 0.025 ug/L using the BCF of 40,000 obtained for methylmercury with the Eastern oyster (Kopfler, 1974). Because the Final Residue Values for methylmercury are substantially below the Final Chronic Values for mercury(II), it is probably not too important that many fishes, including the rainbow trout, coho salmon, bluegill, and haddock might not be adequately protected by the freshwater and saltwater Final Chronic Values for mercury(II).

In contrast to all the complexities of deriving numerical criteria for mercury, monitoring for unacceptable environmental effects should be relatively straightforward. The most sensitive adverse effect will probably be exceedence of the FDA action level. Therefore, existing discharges should be acceptable if the concentration of methylmercury in the edible portion of exposed consumed species does not exceed the FDA action level.

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of mercury does not exceed 0.012 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 2.4 ug/L more than once every 3 years on the average. If the 4-day average concentration exceeds 0.012 ug/L more than once in a 3-year period, the edible portion of consumed species should be analyzed to determine whether the concentration of methylmercury exceeds the FDA action level.

The procedures described in the Guidelines indicate that, except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of mercury does not exceed 0.025 ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 2.1 ug/L more than once every 3 years on the average. If the 4-day average concentration exceeds 0.025 ug/L more than once

in a 3-year period, the edible portion of consumed species should be analyzed to determine whether the concentration of methylmercury exceeds the FDA action level.

EPA believes that a measurement such as "acid-soluble" would provide a more scientifically correct basis upon which to establish criteria for metals. The criteria were developed on this basis. However, at this time, no EPA approved-methods for such a measurement are available to implement the criteria through the regulatory programs of the Agency and the States. The Agency is considering development and approval of methods for a measurement such as acid-soluble. Until available, however, EPA recommends applying the criteria using the total recoverable method. This has two impacts: (1) certain species of some metals cannot be analyzed directly because the total recoverable method does not distinguish between individual oxidation states, and (2) these criteria may be overly protective when based on the total recoverable method.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to mercury exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems respectively. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985).

HUMAN HEALTH CRITERIA

For the protection of human health from the toxic properties of mercury ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 144 ng/L.

For the protection of human health from the toxic properties of mercury ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 146 ng/L.

NOTE: These values include the consumption of freshwater, estuarine, and marine species.

(45 F.R. 79318 Nov. 28, 1980) (50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

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, and that the average person weighs 70 kg and consumes 2 liters of water per day, the formula for calculating a criterion is $2.0 \text{ mg/kg} \times 0.2 \times 70 \text{ kg} \times 1/100 \times 1/2 = 0.14 \text{ mg/L}$. 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 8 months exhibited reduced growth. The 36-hour LC50 concentration was 7.5 and 22 ug/L for the fathead minnow and yellow perch, respectively. Korn and Earnest (1974) obtained a 96-hour LC50

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 LC50 value of 0.5 ug/L for the crayfish, *Orconectes nais*. Merna and Eisele (1973) obtained a 96-hour LC50 value of 0.61 ug/L for the scud, *Gammarus pseudolimnaeus* and 96-hour LC50's 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 1 year. The near extinction of one species of scud, *Hyalella 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 bioaccumulative 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 10 times lower than the level causing effects on some invertebrate populations in a 1-year dosing of a natural stream.

Bahner and Nimmo (1974) found the 96-hour LC50 of methoxychlor for the pink shrimp, Penaeus duorarum to be 3.5 ug/L and the 30-day LC50 to be 1.3 ug/L. Using an application factor of 0.01 with the pink shrimp's 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, Marcanaria marcanaria 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.

MIREX

CRITERION:

0.001 ug/L for freshwater and marine aquatic life.

RATIONAL??:

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, 14.1 percent soybean oil and 85 percent corncob grits. The mirex bait often is applied at a rate of 1.4 kg/ha, equivalent to 4.2 grams of toxicant per hectare.

Relatively few studies have been made of the effects of mirex on freshwater invertebrates ~~og~~ yhrdr, only Dudke 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 14 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

bayl, 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 2 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/ha) in 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 and 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. (1988) 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 58 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 supplies 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. Navgi 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/ha). 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 0.78 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 the 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 (Stoke et al. 1978) 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 seawater was lethal to juvenile pink shrimp, Penaeus duorarum, in a 3-week exposure (Lowe et al. 1971). In static tests with larval stages (megalopal) of the mud crab, Rhithropanopeus harrisi, reduced survival was observed in 0.1 ug/L mirex (Bookhout et al. 1972). In three of four 28-day seasonal flow-through experiments, Tagatz et al. (1975) found reduced survival of Callinectes sapidus, Penaeus duorarum, and grass shrimp, Palaemonetes ~US&, 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), stated 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.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C **FOR** METHODOLOGY

NAPHTHALENE

CRITERIA:

Aquatic Life

The available data for naphthalene indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 2,300 and 620 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for naphthalene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 2,350 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of naphthalene to sensitive saltwater aquatic life.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time because of insufficient available data for naphthalene.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

NICKEL

CRITERIA:

Aquatic Life

For total recoverable nickel the criterion (in ug/L) to protect freshwater aquatic life as derived using the Guidelines is the numerical value given by $e^{(0.76[\ln(\text{hardness})]+1.06)}$ as a 24-hour average, and the concentration (in ug/L) should not exceed the numerical value given by $e^{(0.76[\ln(\text{hardness})]+4.02)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 the criteria are 56, 96, and 160 ug/L, respectively, as 24-hour averages, and the concentrations should not exceed 1,100, 1,800, and 3,100 ug/L, respectively, at any time.

For total recoverable nickel the criterion to protect saltwater aquatic life as derived using the Guidelines is 7.1 ug/L as a 24-hour average, and the concentration should not exceed 140 ug/L at any time.

Human Health

For the protection of human health from the toxic properties of nickel ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 632 ug/L.

For the protection of human health from the toxic properties of nickel ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 4.77 mg/L.

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 either is 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 feed lot discharges. Diffuse sources of 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" concentration nitrates are rapidly excreted in the urine. High intake of nitrates constitutes a hazard primarily to warmblooded animals under conditions that are favorable to 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, consequently impairing transport.

The reaction of nitrite with hemoglobin can be hazardous in infants under 3 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 2,000 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 exceed this amount, but only one U.S. case of infant methemoglobinemia associated with a public water supply has ever been reported (Virgil 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 systems 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 LC50 values for chinook salmon, Oncorhynchus tshawytscha, were 1,310 and 1,080 mg/L nitrate nitrogen in fresh water and 990 and 900 mg/L nitrate nitrogen in 15 ‰ saline water. For fingerling rainbow trout, Salmo gairdneri, the respective 96-hour and 7-day LC50 values were 1,360 and 1,060 mg/L nitrate nitrogen in fresh water, and 1,050 and 900 mg/L nitrate nitrogen in 15 ‰ saline water. Trama (1954) reported that the 96-hour LC50 for bluegills, Lepomis macrochirus, at 20°C was **2,000** 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 LC50 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 phoxinus, that suffered a 50 percent mortality within 1.5 hours of exposure to 2,030 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 LC50 of 1.6 mg/L nitrite nitrogen, and 48- and 96-hour LC50 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, Carpionodes 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 CaCO₃; alkalinity = 176 mg/L CaCO₃; pH = 7.9) on rainbow trout, Salmo gairdneri, of four different sizes, and obtained 96-hour LC50 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 LC50 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 warmwater fish (Knepp and Arkin, 1973); (2) nitrite nitrogen at or below 5 mg/L should be protective of most warmwater 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 coldwater fish could rarely occur in nature, restrictive criteria are not recommended.

NITROBENZENE

CRITERIA:

Aquatic Life

The available data for nitrobenzene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 27,000 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of nitrobenzene to sensitive freshwater aquatic life.

The available data for nitrobenzene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 6,680 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of nitrobenzene to sensitive saltwater aquatic life.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for nitrobenzene. Based on available toxicity data, to protect public health the derived level is 19.8 mg/L. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 30 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

NOTE: The U.S. EPA is currently developing Acceptable Daily Intake (ADI) or Verified Reference Dose (RfD) values for Agency-wide use for this chemical. The new value should

be substituted when it becomes available. The January, 1986, draft Verified Reference Dose document cites an RfD of .0005 mg/kg/day for nitrobenzene.

NITROPHENOLS

CRITERIA:

Aquatic Life

The available data for nitrophenols indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 230 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrophenols to sensitive freshwater aquatic life but toxicity to one species of algae occurs at concentrations as low as 150 ug/L.

The available data for nitrophenols indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 4,850 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrophenols to sensitive saltwater aquatic life.

Human Health

Because of insufficient available data for mono- and trinitrophenols, satisfactory criteria cannot be derived at this time, using the present guidelines.

For the protection of human health from the toxic properties of dinitrophenols and 2,4-dinitro-o-cresol ingested through water and contaminated aquatic organisms, the ambient water criteria are determined to be 70 ug/L and 13.4 ug/L, respectively.

For the protection of human health from the toxic properties of dinitrophenols and 2,4-dinitro-o-cresol ingested through contaminated aquatic organisms alone, the ambient water criteria

are determined to be 14.3 mg/L and 765 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

NITROSAMINES

CRITERIA:

Aquatic Life

The available data for nitrosamines indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as **5,850** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrosamines to sensitive freshwater aquatic life.

The available data for nitrosamines indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 3,300,000 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of nitrosamines to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to N-nitrosodiethylamine and all other nitrosamines except those listed below, through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are **8.0** ng/L, **0.8** ng/L, and **0.08** ng/L, respectively. If these estimates are made for

consumption of aquatic organisms only, excluding consumption of water, the levels are 12,400 ng/L, 1,240 ng/L, and 124 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to N-nitrosodimethylamine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 14 ng/L, 1.4 ng/L, and 0.14 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 160,000 ng/L, 16,000 ng/L, and 1,600 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to N-nitrosodibutylamine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 64 ng/L, 6.4 ng/L, and 0.64 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 5,868 ng/L, 587

ng/L, and **58.7** ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to N-nitrosopyrrolidine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 160 ng/L, 16 ng/L, and **1.6** ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are **919,000** ng/L, **91,900** ng/L, and **9,190** ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to N-nitrosodiphenylamine through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are **49,000** ng/L, **4,900** ng/L, and **490** ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 161,000 ng/L, **16,100** ng/L, and **1,610** ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

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 oil 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 nonvolatile, 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 2,500 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 LC50 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 and 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 $\mu\text{g/L}$ (see Table 7).

Table 7 summarizes some of the sublethal toxicities for various petroleum pollutants and aquatic species. In addition to sublethal effects reported at the 10 to 100 $\mu\text{g/L}$ level, it has been shown that petroleum products can harm aquatic life at concentrations as low as 1 $\mu\text{g/L}$ (Jacobson and Boylan, 1973).

Bioaccumulation of petroleum products presents two especially important public health problems: (1) the tainting of edible,

aquatic 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 also has 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. Jacobson 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 Univ., 1956; Mckee and Wolf, 1963). Oils of animal or vegetable origin generally are nontoxic 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 because of lack of mobility; (b) lethal effects on fish by coating epithelial surfaces of gills, thus preventing respiration; (c) potential fishkills resulting from 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 shorelines 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 nontoxic 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 nonpetroleum oils of vegetable or animal origin. This same recommendation applies to floating oils of petroleum origin since they too may produce similar effects.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

DISSOLVED OXYGEN

NATIONAL CRITERIA:

The national criteria for ambient dissolved oxygen concentrations for the protection of freshwater aquatic life are presented in Table 1. The criteria are derived from the production impairment estimates which are based primarily upon growth data and information on temperature, disease, and pollutant stresses. The average dissolved oxygen concentrations selected are values **0.5** mg/L above the slight production impairment values and represent values between no production impairment and slight production impairment. Each criterion may thus be viewed as an estimate of the threshold concentration below which detrimental effects are expected.

Criteria for coldwater fish are intended to apply to waters containing a population of one or more species in the family Salmonidae (Bailey et al., 1970) or to waters containing other coldwater or coolwater fish deemed by the user to be closer to salmonids in sensitivity than to most warmwater species. Although the acute lethal limit for salmonids is at or below 3 mg/L, the coldwater minimum has been established at **4** mg/L because a significant proportion of the insect species common to salmonid habitats are less tolerant of acute exposures to low dissolved oxygen than are salmonids. Some coolwater species may require more protection than that afforded by the other life stage criteria for warmwater fish and it may be desirable to protect sensitive coolwater species with the coldwater criteria. Many states have more stringent dissolved oxygen standards for cooler waters, waters that contain either

salmonids, nonsalmonid coolwater fish, or the sensitive centrarchid, the smallmouth bass. The warmwater criteria are necessary to protect early life stages of warmwater fish as sensitive as channel catfish and to protect other life stages of fish as sensitive as largemouth bass. Criteria for early life stages are intended to apply only where and when these stages occur. These criteria represent dissolved oxygen concentrations which EPA believes provide a reasonable and adequate degree of protection for freshwater aquatic life.

The criteria do not represent assured no-effect levels. However, because the criteria represent worst case conditions (i.e. for wasteload allocation and waste treatment plant design), conditions will be better than the criteria nearly all of the time at most sites. In situations where criteria conditions are just maintained for considerable periods the proposed criteria represent some risk of production impairment. This impairment would depend on innumerable other factors. If slight production impairment or a small but undefinable risk of moderate impairment is unacceptable, then one should use the "no production impairment" values given in the document as means and the "slight production impairment" values as minima. The table which presents these concentrations is reproduced here as table 2.

The criteria do represent dissolved oxygen concentrations believed to protect the more sensitive populations of organisms against potentially damaging production impairment. The dissolved oxygen concentrations in the criteria are intended to be protective at typically high seasonal environmental temperatures for the appropriate taxonomic and life stage classi-

Table 1. Water quality criteria for ambient dissolved oxygen concentration.

	<u>Coldwater Criteria</u>		<u>Warmwater Criteria</u>	
	Early Life Stages^{1,2}	Other Life Stages	Early Life Stages ²	Other Life Stages
30 Day Mean	NA ³	6.5	NA	5.5
7 Day Mean	9.5 (6.5)	NA	6.0	NA
7 Day Mean Minimum	NA	5.0	NA	4.0
1 Day Minimum ^{4,5}	8.0 (5.0)	4.0	5.0	3.0

These are water column concentrations recommended to achieve the required intergravel dissolved oxygen concentrations shown in parentheses. The 3 mg/L differential is discussed in the criteria document. For species that have early life stages exposed directly to the water column, the figures in parentheses apply.

Includes all embryonic and larval stages and all juvenile forms to 30-days following hatching.

3 NA (not applicable).

For highly manipulatable discharges, further restrictions apply (see page 37)

All minima should be considered as instantaneous concentrations to be achieved at all times.

fications, temperatures which are often higher than those used in the research from which the criteria were generated, especially for other than early life stages.

Where natural conditions alone create dissolved oxygen concentrations less than 110 percent of the applicable criteria means or minima or both, the minimum acceptable concentration is

90 percent of the natural concentration. These values are similar to those presented graphically by Doudoroff and Shumway (1970) and those calculated from Water Quality Criteria 1972 (NAS/NAE, 1973). Absolutely no anthropogenic dissolved oxygen depression in the potentially lethal area below the 1-day minima should be allowed unless special care is taken to ascertain the tolerance of resident species to low dissolved oxygen.

If daily cycles of dissolved oxygen are essentially sinusoidal, a reasonable daily average is calculated from the day's high and low dissolved oxygen values. A time-weighted average may be required if the dissolved oxygen cycles are decidedly non-sinusoidal. Determining the magnitude of daily dissolved oxygen cycles requires at least two appropriately timed measurements daily, and characterizing the shape of the cycle requires several more appropriately spaced measurements.

Once a series of daily mean dissolved oxygen concentrations are calculated, an average of these daily means can be calculated (Table 3). For embryonic, larval, and early life stages, the averaging period should not exceed 7 days. This short time is needed to adequately protect these often short duration, most sensitive life stages. Other life stages can probably be adequately protected by 30-day averages. Regardless of the averaging period, the average should be considered a moving average rather than a calendar-week or calendar-month average.

Table 2. Dissolved Oxygen Concentrations (mg/L) Versus Quantitative Level of Effect.

1. Salmonid Waters

a. Embryo and Larval Stages

No Production Impairment	=	11* (8)
Slight Production Impairment	=	9* (6)
Moderate Production Impairment	=	8* (5)
Severe Production Impairment	=	7* (4)
Limit to Avoid Acute Mortality	=	6* (3)

(* Note: These are water column concentrations recommended to achieve the required intergravel dissolved oxygen concentrations shown in parentheses. The 3 mg/L difference is discussed in the criteria document.)

b. Other Life Stages

No Production Impairment	=	8
light Production Impairment	=	6
Moderate Production Impairment	=	5
Severe Production Impairment	=	4
Limit to Avoid Acute Mortality	=	3

2. Nonsalmonid Waters

a. Early Life Stages

No Production Impairment	=	6.5
Slight Production Impairment	=	5.5
Moderate Production Impairment	=	5
Severe Production Impairment	=	4.5
Limit to Avoid Acute Mortality	=	4

b. Other Life Stages

No Production Impairment	=	6
Slight Production Impairment	=	5
Moderate Production Impairment	=	4
Severe Production Impairment	=	3.5
Limit to Avoid Acute Mortality	=	3

3. Invertebrates

No Production Impairment	=	8
Some Production Impairment	=	5
Acute Mortality Limit	=	4

Table 3. Sample calculations for determining daily means and 7-day mean dissolved oxygen concentrations (30-day averages are calculated in a similar fashion using 30 days data).

Dissolved Oxygen (mg/L)

Day	Daily Max.	Daily Min.	Daily Mean
1	9.0	7.0	8.0
2	10.0	7.0	8.5
3	11.0	8.0	9.5 ^b
4	12.0 ^a	8.0	9.5
5	10.0	8.0	9.0
6	11.0	9.0	10.0
7	12.0 ^a	10.0	10.5 ^c
		57.0	65.0
1-day Minimum		7.0	
7-day Mean Minimum		8.1	
7-day Mean			9.3

a Above air saturation concentration (assumed to be 11.0 mg/L for this example).

b $(11.0 + 8.0)2.$

c $(11.0 + 10.0)2.$

The criteria have been established on the basis that the maximum dissolved oxygen value actually used in calculating any daily mean should not exceed the air saturation value. This consideration is based primarily on analysis of studies of **cycling dissolved oxygen and the growth of largemouth bass** (Stewart et al., 1967), which indicated that high dissolved oxygen levels (> 6 mg/L) had no beneficial effect on growth.

During periodic cycles of dissolved oxygen concentrations, minima lower than acceptable constant exposure levels are tolerable so long as:

1. the average concentration attained meets or exceeds the criterion;
2. the average dissolved oxygen concentration is calculated as recommended in Table 3; and
3. the minima are not unduly stressful and clearly are not lethal.

A daily minimum has been included to make certain that no acute mortality of sensitive species occurs as a result of lack of oxygen. Because repeated exposure to dissolved oxygen concentrations at or near the acute lethal threshold will be stressful and because stress can indirectly produce mortality or other adverse effects (e.g., through disease), the criteria are designed to prevent significant episodes of continuous or regularly recurring exposures to dissolved oxygen concentrations at or near the lethal threshold. This protection has been achieved by setting the daily minimum for early life stages at the subacute lethality threshold, by the use of a 7-day averaging period for early life stages, by stipulating a 7-day mean minimum value for other life stages, and by recommending additional limits for manipulatable discharges.

The previous **EPA** criterion for dissolved oxygen published in Quality Criteria for Water (USEPA, 1976) was a minimum of 5 mg/L (usually applied as a 7Q10) which is similar to the current criterion minimum except for other life stages of warmwater fish which now allows a 7-day mean minimum of 4 mg/L. The new criteria are similar to those contained in the 1968 "Green Book" of the Federal Water Pollution Control Federation (FWPCA, 1968).

A. The Criteria and Monitoring and Design Conditions

The acceptable mean concentrations should be attained most of the time, but some deviation below these values would probably not cause significant harm. Deviations below the mean will probably be serially correlated and hence apt to occur on consecutive days. The significance of deviations below the mean will depend on whether they occur continuously or in daily cycles, the former being more adverse than the latter. Current knowledge regarding such deviations is limited primarily to laboratory growth experiments and by extrapolation to other activity-related phenomena.

Under conditions where large daily cycles of dissolved oxygen occur, it is possible to meet the criteria mean values and consistently violate the mean minimum criteria. Under these conditions the mean minimum criteria will clearly be the limiting regulation unless alternatives such as nutrient control can dampen the daily cycles.

The significance of conditions which fail to meet the recommended dissolved oxygen criteria depend largely upon five factors: (1) the duration of the event; (2) the magnitude of the dissolved oxygen depression; (3) the frequency of recurrence; (4) the proportional area of the site failing to meet the criteria, and (5) the biological significance of the site where the event occurs. Evaluation of an event's significance must be largely case- and site-specific. Common sense would dictate that the magnitude of the depression would be the single most important factor in general, especially if the acute value is violated. A

logical extension of these considerations is that the event must be considered in the context of the level of resolution of the monitoring or modeling effort. Evaluating the extent, duration, and magnitude of an event must be a function of the spatial and temporal frequency of the data. Thus, a single deviation below the criterion takes on considerably less significance where continuous monitoring occurs than where sampling is comprised of once-a-week grab samples. This is so because based on continuous monitoring the event is provably small, but with the much less frequent sampling the event is not provably small and can be considerably worse than indicated by the sample. The frequency of recurrence is of considerable interest to those modeling dissolved oxygen concentrations because the return period, or period between recurrences, is a primary modeling consideration contingent upon probabilities of receiving water volumes, waste loads, temperatures, etc. It should be apparent that return period cannot be isolated from the other four factors discussed above. Ultimately, the question of return period may be decided on a site-specific basis taking into account the other factors (duration, magnitude, areal extent, and biological significance) mentioned above. Future studies of temporal patterns of dissolved oxygen concentrations, both within and between years, must be conducted to provide a better basis for selection of the appropriate return period.

In conducting wasteload allocation and treatment plant design computations, the choice of temperature in the models will be important. Probably the best option would be to use temperatures consistent with those expected in the receiving water over the

critical dissolved oxygen period for the biota.

B. The Criteria and Manipulatable Discharges

If daily minimum DOs are perfectly serially correlated, i.e., if the annual lowest daily minimum dissolved oxygen concentration is adjacent in time to the next lower daily minimum dissolved oxygen concentration and one of these two minima is adjacent to the third lowest daily minimum dissolved oxygen concentration, etc., then in order to meet the 7-day mean minimum criterion it is unlikely that there will be more than three or four consecutive daily minimum values below the acceptable 7-day mean minimum. Unless the dissolved oxygen pattern is extremely erratic, it is also unlikely that the lowest dissolved oxygen concentration will be appreciably below the acceptable 7-day mean minimum or that daily minimum values below the 7-day mean minimum will occur in more than one or two weeks each year. For some discharges, the distribution of dissolved oxygen concentrations can be manipulated to varying degrees. Applying the daily minimum to manipulatable discharges would allow repeated weekly cycles of minimum acutely acceptable dissolved oxygen values, a condition of unacceptable stress and possible adverse biological effect. For this reason, the application of the one day minimum criterion to manipulatable discharges must limit either the frequency of occurrence of values below the acceptable 7-day mean minimum or must impose further limits on the extent of excursions below the 7-day mean minimum. For such controlled discharges, it is recommended that the occurrence of daily minima below the acceptable 7-day mean

minimum be limited to 3 weeks per year or that the acceptable one-day minimum be increased to 4.5 mg/L for coldwater fish and 3.5 mg/L for warmwater fish, Such decisions could be site-specific based upon the extent of control and serial correlation.

PARATHION

CRITERION:

0.04 ug/L for freshwater and marine aquatic life.

RATIONALE:

Acute static LC50 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. Environ. Prot. Agency, 1975). In flowing water exposures, Spacie (1975) obtained 96-hour LC50 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 LC50 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, Ambystoma 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 effects 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 phosphorus 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-hydrolyzing capability in mosquitofish, Gambusia

affinis (Ludke, 1970).

Parathion has been found acutely toxic to aquatic invertebrates at under 1 ug/L e.g., a 50-hour LC50 of 0.8 ug/L for Daphnia magna; 48-hour LC50 of 0.6 ug/L for Daphnia pulex; 48-hour LC50 of 0.37 for Simocephalus serrulatus (a daphnid) (Sanders and Cope, 1966); a 5-day LC50 of 0.93 ug/L for the larval stonefly, Acroneuria pacifica (Jensen and Gaufin, 1964); and a 96-hour LC50 of 0.43 ug/L for the larval caddisfly Hydropsyche californica (Gaufin et al. 1965). Mulla and Khasawinah (1969) obtained a 24-hour LC50 of 0.5 ug/L for 4th instar larvae of the midge Tanypus grodhausi. Spacie (1975) obtained 96-hour LC50'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 Chironomus 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. Environ. Prot. Agency, 1975).

Few longer exposures have been conducted. Jensen and Gaufin (1964) obtained 30-day LC50's for Pteronarcys californica and Acroneuria pacifica of 2.2 and 0.44 ug/L, respectively. Spacie (1975) found the 3-week LC50 for Daphnia magna to be 0.14 ug/L. Statistically significant reproductive impairment occurred at concentrations above 0.08 ug/L. A 43-day LC50 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 1 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 seawater 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 5,000, 2,000, 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 EC50 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 LC50 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 0.04 ug/L is recommended for marine and freshwater aquatic life.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

PENTACHLOROPHENOL

CRITERIA:

Aquatic Life

The available data for pentachlorophenol indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 55 and 3.2 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for pentachlorophenol indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 53 and 34 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for pentachlorophenol. Based on available toxicity data, to protect public health the derived level is 1.01 mg/L. Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 30 ug/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

pH

CRITERIA:

Range

5 - 9	Domestic water supplies (welfare)
6.5 - 9.0	Freshwater aquatic life
6.5 - 8.5	Marine aquatic life (but not more than 0.2 units outside of normally occurring range.)

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: $\text{pH} = -\log_{10} (\text{H}^+)$, 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 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:



Butterfield (1984) 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 fresh water fish has been published by the European Inland Fisheries Advisory Commission (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 CO₂ 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 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) stoneflies (four species), dragonflies (two species), and mayflies (one species). All are important fish food organisms. The 30-day TL50 values ranged from 2.45 to 5.38 with the caddisflies being the most tolerant and the mayflies being 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 invertebrates 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 that are also toxic. Ammonia has been shown to be 10 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 seawater, 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 seawater 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 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 freshwater criteria, rapid pH fluctuations that are caused by 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.

PHENOL

CRITERIA:

Aquatic Life

The available data for phenol indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 10,200 and 2,560 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for phenol indicate that toxicity to saltwater aquatic life occurs at concentrations as low as 5,800 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of phenol to sensitive saltwater aquatic life.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for phenol. Based on available toxicity data, to protect public health the derived level is 3.5 mg/L.

Using available organoleptic data, to control undesirable taste and odor qualities of ambient water the estimated level is 0.3 mg/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criterion, and have no demonstrated relationship to potential adverse human health effects.

NOTE: The U.S. EPA is currently developing Acceptable Daily Intake (ADI) or Verified Reference Dose (RfD) values for Agency-wide use for this chemical. The new value should **be substituted** when **it** becomes available. The January, 1986, draft Verified Reference Dose document cites an RfD of 0.1 mg/kg/day for phenol.

PHOSPHORUS

CRITERION::

0.10 ug/L yellow (elemental) phosphorus for marine or estuarine water.

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 sand. The phosphate rock beds lie within a few feet of the surface and mining is accomplished by using hydraulic water jets and a washing operation that separates the phosphates 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 3.5 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 a 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 or recycling within the biotic community, while the remainder may be deposited with sediments. Much of the material that combines 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 were 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 p₄ 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 p₄ 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 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, such waters would need more stringent nutrient limits. 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	Oligotrophic or Permissible Loading	Eutrophic or Critical Loading
(meters/year)	(grams/meter ² /year)	(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 populations 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: 1. Naturally occurring phenomena may limit the development of plant nuisances. 2. Technological or cost-effective limitations may help control introduced pollutants. 3. Waters may be highly laden with natural silts or colors which reduce the penetration of sunlight needed for plant photosynthesis. 4. Some waters morphometric features of steep banks, great depth, and substantial flows contribute to a history of no plant problems. 5. Waters may be managed primarily for waterfowl or other wildlife. 7. In some waters nutrient other than phosphorus is limiting to plant growth: the level and nature of such limiting nutrient would not be expected to increase to an extent that would influence eutrophication. 6. In some waters 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.

PHTHALATE ESTERS

CRITERIA:

Aquatic Life

The available data for phthalate esters indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as **940** and **3** ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for phthalate esters indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as **2,944** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of phthalate esters to sensitive saltwater aquatic life but toxicity to one species of algae occurs at concentrations as low as **3.4** ug/L.

Human Health

For the protection of human health from the toxic properties of dimethyl phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be **313** mg/L.

For the protection of human health from the toxic properties of dimethyl phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be **2.9** g/l.

For the protection of human health from the toxic properties of diethyl phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 350 mg/L.

For the protection of human health from the toxic properties of diethyl phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 1.8 g/l.

For the protection of human health from the toxic properties of dibutyl phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 34 mg/L.

For the protection of human health from the toxic properties of dibutyl phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 154 mg/L.

For the protection of human health from the toxic properties of di-2-ethylhexyl phthalate ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 15 mg/L.

For the protection of human health from the toxic properties of di-2-ethylhexyl phthalate ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 50 mg/L.

POLYCHLORINATED BIPHENYLS

CRITERIA:

Aquatic Life

For polychlorinated biphenyls the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.014 ug/L as a 24-hour average. The concentration of 0.014 ug/L is probably too high because it is based on bioconcentration factors measured in laboratory studies, but field studies apparently produce factors at least 10 times higher for fishes. The available data indicate that acute toxicity to freshwater aquatic life probably will occur only at concentrations above 2.0 ug/L and that the 24-hour average should provide adequate protection against acute toxicity.

For polychlorinated biphenyls the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.030 ug/L as a 24-hour average. The concentration of 0.030 ug/L is probably too high because it is based on bioconcentration factors measured in laboratory studies, but field studies apparently produce factors at least 10 times higher for fishes. The available data indicate that acute toxicity to saltwater aquatic life probably will only occur at concentrations above 10 ug/L and that the 24-hour average criterion should provide adequate protection against acute toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to polychlorinated biphenyls through ingestion of contaminated water and contaminated aquatic

organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 0.79 ng/L, 0.079 ng/L, and 0.0079 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 0.79 ng/L, 0.079 ng/L, and 0.0079 ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

POLYNUCLEAR AROMATIC HYDROCARBONS

CRITERIA :-

Aquatic Life

The limited freshwater data base available for polynuclear aromatic hydrocarbons, mostly from short-term bioconcentration studies with two compounds, does not permit a statement concerning acute or chronic toxicity.

The available data for polynuclear aromatic hydrocarbons indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 300 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of polynuclear aromatic hydrocarbons to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to polynuclear aromatic hydrocarbons through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 28.0 ng/L, 2.8 ng/L, and **0.28** ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 311.0 ng/L,

31.1 ng/L, and 3.11 ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

SELENIUM

CRITERIA:

Aquatic Life

For total recoverable inorganic selenite the criterion to protect freshwater aquatic life as derived using the Guidelines is 35 ug/L as a 24-hour average, and the concentration should not exceed 260 ug/L at any time.

For total recoverable inorganic selenite the criterion to protect saltwater aquatic life as derived using the Guidelines is 54 ug/L as a 24-hour average, and the concentration should not exceed 410 ug/L at any time.

The available data for inorganic selenate indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 760 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of inorganic selenate to sensitive freshwater aquatic life.

No data are available concerning the toxicity of inorganic selenate to saltwater aquatic life.

Human Health

The ambient water quality criterion for selenium is recommended to be identical to the existing water standard which is 10 ug/L. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present

standard. For this reason a selective criterion based on **exposure solely** from consumption of 6.5 grams of aquatic organisms was not derived.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR **METHODOLOGY**

SILVER

CRITERIA:

Aquatic Life

For freshwater aquatic life the concentration (in ug/L) of total recoverable silver should not exceed the numerical value given by $e^{(1.72[\ln(\text{hardness})] - 6.52)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/L **as** CaCO_3 , the concentration of total recoverable silver should not exceed 1.2, **4.1**, and 13 ug/L, respectively, at any time. The available data indicate that chronic toxicity to freshwater aquatic life may occur at concentrations as **low** as 0.12 ug/L.

For saltwater aquatic life the concentration of total recoverable silver should not exceed **2.3** ug/L at any time. No data are available concerning the chronic toxicity of silver to sensitive saltwater aquatic life.

Human Health

The ambient water quality criterion for silver is recommended to be identical to the existing water standard, which **is** 50 ug/L. Analysis of the toxic effects data resulted in **a** calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value **is** comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of **6.5** grams of aquatic organisms was not derived.

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 1,283 to 1,333 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. The 1962 PHS Drinking Water Standards 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 tastes at lower concentrations than other constituents. Chloride ions have frequently been cited as having a low taste threshold in water. Data from Richter 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 1,215 mg/L. Lockhart, et al. (1955) evaluated the effect of chlorides on water used for brewing coffee indicated threshold concentrations for chloride ranging 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 nonmetallic materials, using pH control and corrosion inhibitors, or some form of galvanic or impressed electrical current systems (Lehmann, 1964). In household systems water piping, wastewater piping, water heaters, faucets, toilet flushing mechanisms, garbage grinders and both clothes and dishwashing machines incur damage.

By using water with 1,750 mg/L dissolved solids as compared with 250 mg/L, service life was reduced 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.

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 1968 NTAC Report 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 ‰. Such concentrated seawater is caused by evaporation and lack of exchange with the Gulf of Mexico (Rounsafell and Everhart, 1953).

Estuarine species of fish are tolerant of salinity changes ranging from fresh to brackish to seawater. 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 ‰. Therefore, estuarine segments containing salinities below about 12.5 ‰ produce most of the seed oysters for planting (Rounsefell and Everhart, 1953). Based on similar examples, the 1968 NTAC Report 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 ‰ with means as high as 1.26 ‰ (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 1968 NTAC Report 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 depends not only 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 detri-
mental effects will usually
be noticed-----

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

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
Boiler Make-up	35,000

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.

$$*ESP = \frac{100 [a + b(SAR)]}{1 + [a + b(SAR)]}$$

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)

$$**SAR = \text{sodium adsorption ratio} = \frac{Na}{[0.5(Ca + Mg)]^{0.5}}$$

SAR is expressed as milliequivalents

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

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 photosynthetic activity by more than 10 percent from the seasonally 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 microorganisms 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 in 1965 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 versus 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 flourish 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 in 1974 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 and 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 criterion for freshwater fish and other aquatic life are essentially that proposed by the National Academy of Sciences and the Great Lakes Water Quality Board.

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SEE APPENDIX C FOR METHODOLOGY

SULFIDE = HYDROGEN SULFIDE

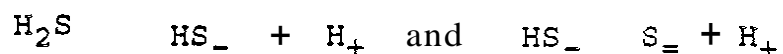
CRITERION:

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 4,000 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 decomposition 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:



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 have been found 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 H₂S; at 41 ug/L mortality was almost complete. Northern pike fry had 96-hour LC50 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), Safe levels in working on walleyes and fathead minnows, Pimephales promelas, were found to vary 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 H₂S 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 H₂S (Smith and Oseid, 1974). White sucker eggs were hatched at 15 ug/L, but juveniles showed growth reductions at 1 ug/L. Safe level 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 (Osleid 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.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

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 22 organic compounds. The values ranged from 0.4 ug/L (2,4-dichlorophenol) to 95,000 ug/L (formaldehyde). An additional 12 compounds were tested, 7 of which were not found to impair flavor at or near lethal levels.

Thomas (1973) reviewed the literature review on tainting substances revealed serious problems that have occurred. Detailed studies and methodology used to evaluate the palatability of fishes in the Ohio River as affected by various waste discharges showed that 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).

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

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:

$$\text{Temperature } (C_0) = (1/b) (\log_{10} [\text{time (min)}] - a) + 20 C$$

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 for some species from Appendix 11-C, National Academy of Sciences 1974 document.

b = slope of the line fitted to experimental data and available for some species from Appendix 11-C, of the National Academy of Sciences document.

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 importtr 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 Pto c 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 resulting from 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 are 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. The Federal Water Pollution Control Administration in 1967 called temperature a

catalyst, a depressant, an activator, a restrictor, a stimulator, a controller, a killer, 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), Carp (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 (Bumson, 1938). Odor problems associated with plankton may also be aggravated.

The effects of temperature on aquatic organisms have been the subject of comprehensive literature reviews (Brett, 1956; Fry, 1967; FWPCA, 1967; Kine, 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 coldwater 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 accumulation state and exposure time are considered the critical factors (Parker and Xrenkel, 1969).

The effects of sublethal temperatures on metabolism, respiration, behavior, distribution and migration, feeding rate, growth, and reproduction have been summarized by Be 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 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 characteristic 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 percent of a sample of organisms can survive is determined on the organism at the highest sustainable acclimation temperature. The lowest temperature that 50 percent 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° C in fresh water and somewhat less for saline waters. However, organisms acclimated to relatively warm water, when subjected to reduced temperatures that under other conditions of acclimation would not be detrimental, may suffer a significant mortality caused by 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 geographic distribution of species. Such temperature changes are necessary to induce the reproductive cycles of aquatic organisms and to regulate other life factors (Mount, 1969).

Artificially 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 34.5° C.

In open waters elevated temperatures may affect periphyton, benthic invertebrates, and fish, in addition to causing shifts in algal dominance. Trembley (1960) studies of the Delaware River downstream from a power plant 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 definable 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 coldwater 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 of 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:

$$\log^{10} (\text{time (minutes)}) = a + b (\text{Temperature (}^{\circ} \text{C)})$$

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 for some species from Appendix II-C, of the National Academy of Sciences document.

b = slope of the line fitted to experimental data and which is available for some species from Appendix II-C, of the National Academy of Sciences document.

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 of 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 may be an average of the optimum temperature for growth and the temperature for zero net growth.

Because of the difficulty in determining the temperature of zero net growth, essentially the same temperature can be derived by adding to the optimum essentially to temperature (for growth or other physiological functions) a factor calculated as one-third of the difference between the ultimate upper incipient lethal temperature and the optimum temperature (NAS, 1974). In equation form:

$$\begin{array}{lcl} \text{Maximum weekly} & & \text{(ultimate upper optimum)} \\ \text{average =} & \text{optimum} + 1/3 & \text{(incipient lethal - temperature)} \\ \text{temperature} & \text{temperature} & \text{(temperature)} \end{array}$$

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 EPA's Environmental Research Laboxatory in Duluth.

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 near optimum levels. Natural short-term temperature fluctuations appear to cause reduced reproduction of fish and invertebrates.

TABLE 11.-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)	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)		
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 temperature - optimum temperature).

b - Based on temperature ($^{\circ}\text{C}$) = $1/b (\log^{10} \text{time}_{(\text{min.})} - a)$
20 $^{\circ}$ C, acclimation at the maximum weekly average temperature for summer growth, and data in Appendix II-C of Water Quality Criteria, published by National Academy of Sciences.

c - Based on data for larvae (ERL-Duluth, 1976).

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, revealed that a 78 percent decrease in annual catch correlated closely with a 0.5°C increase in the average

temperature over the 30-month period between spawning and recruitment into 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.

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,		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.

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 interspecies relationships. A 50 percent reduction in the softshell 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 4 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 softshell 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 some species spawn only during the fall (herring), while others 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 warmer 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 resulting from 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 because of insufficient food (Massengill, 1973). A weight loss of approximately 10 percent 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 because of 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.). 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 (Glynn, 1968; Vaughn, 1961). 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 used to develop thermal limits which specifically consider communities relevant to given water bodies.

2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN

CRITERIA:

Aquatic Life

Not enough data are available concerning the effects of 2,3,7,8-TCDD on aquatic life and its uses to allow derivation of national criteria. The available information indicates that acute values for some freshwater animal species are >1.0 ug/L; some chronic values are <0.01 ug/L; and the chronic value for rainbow trout is <0.001 ug/L. Because exposures of some species of fishes to 0.01 ug/L for <6 days resulted in substantial mortality several weeks later, derivation of aquatic life criteria for 2,3,7,8-TCDD may require special consideration. Predicted bioconcentration factors (BCFs) for 2,3,7,8-TCDD range from 3,000 to 900,000, but the available measured BCFs range from 390 to 13,000. If the BCF is 5,000, concentrations >0.00001 ug/L should result in concentrations in edible freshwater and saltwater fish and shellfish that exceed levels identified in a U.S. FDA health advisory. If the BCF is $>5,000$ or if uptake in a field situation is greater than that in laboratory tests, the value of 0.00001 ug/L will be too high.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of 2,3,7,8-TCDD exposure through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero. This criterion is

based on the nonthreshold assumption for 2,3,7,8-TCDD. However, zero may not be an attainable level at this time.

(49 F.R. 5831, February 15, 1984)
SEE APPENDIX B FOR METHODOLOGY

TETRACHLOROETHYLENE

CRITERIA:

Aquatic Life

The available data for tetrachloroethylene indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 5,280 and 840 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for tetrachloroethylene indicate that acute and chronic toxicity to saltwater aquatic life occurs at concentrations as low as 10,200 and 450 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to tetrachloroethylene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 8.0 ug/L, 0.80 ug/L, and 0.08 ug/L, respectively. If these estimates are made for

consumption of aquatic organisms only, excluding consumption of water, the levels are 88.5 ug/L, 8.85 ug/L, and 0.88 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

THALLIUM

CRITERIA:

Aquatic Life

The available data for thallium indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 1,400 and 40 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to one species of fish occurs at concentrations as low as 20 ug/L after 2,600 hours of exposure.

The available data for thallium indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 2,130 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of thallium to sensitive saltwater aquatic life.

Human Health

For the protection of human health from the toxic properties of thallium ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 13 ug/L.

For the protection of human health from the toxic properties of thallium ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 48 ug/L.

TOLUENE

CRITERIA:

Aquatic Life

The **available** data for toluene indicate ~~that~~ acute toxicity to freshwater aquatic life occurs at concentrations as low as **17,500** ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of toluene to sensitive freshwater aquatic life.

The available data for toluene indicate that acute and chronic toxicity to saltwater aquatic life occurs at concentrations as low as **6,300** and 5,000 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For the protection of human health from the toxic properties of toluene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be **14.3** mg/L.

For the protection of human health from the toxic properties of toluene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be **424** mg/L.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

NOTE: The U.S. EPA is currently developing Acceptable Daily Intake (ADI) or Verified Reference Dose (RfD) values for Agency-wide use for this chemical. The new value should be substituted when it becomes available. The January, **1986**, draft Verified Reference Dose document cites an RfD of 0.3 mg/kg/day for toluene.

TOXAPHENE

CRITERIA:

Aquatic Life

For toxaphene the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.013 ug/L as a 24-hour average, and the concentration should not exceed 1.6 ug/L at any time.

For saltwater aquatic life the concentration of toxaphene should not exceed 0.070 ug/L at any time. No data are available concerning the chronic toxicity of toxaphene to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to toxaphene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 7.1 ng/L, 0.71 ng/L, and 0.07 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 7.3 ng/L, 0.73 ng/L, and 0.01 ng/L, respectively.

TRICHLOROETHYLENE

CRITERIA:

Aquatic Life

The available data for trichloroethylene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 45,000 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trichloroethylene to sensitive freshwater aquatic life but the behavior of one species is adversely affected at concentrations as low as **21,900 ug/L**.

The available data for trichloroethylene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as **2,000 ug/L** and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of trichloroethylene to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to trichloroethylene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are **27 ug/L**, **2.7 ug/L**, and **0.27 ug/L**, respectively. If these estimates are made for

consumption of aquatic organisms only, excluding consumption of water, the levels are 807 ug/L, 80.7 ug/L, and 8.07 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

VINYL CHLORIDE

CRITERIA:

Aquatic Life

No freshwater organisms have been tested with vinyl chloride and no statement can be made concerning acute or chronic toxicity.

No saltwater organisms have been tested with vinyl chloride and no statement can be made concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to vinyl chloride through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 20 ug/L, 2.0 ug/L, and 0.2 ug/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 5,246 ug/L, 525 ug/L, and 52.5 ug/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

ZINC

CRITERIA:

Aquatic Life

For total recoverable zinc the criterion to protect freshwater aquatic life as derived using the Guidelines is 47 ug/L as a 24-hour average and the concentration (in ug/L) should not exceed the numerical value given by $e^{(0.83 [\ln(\text{hardness})] + 1.95)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 the concentration of total recoverable zinc should not exceed 180, 320, and 570 ug/L at any time.

For total recoverable zinc the criterion to protect saltwater aquatic life as derived using the Guidelines is 58 ug/L as a 24-hour average and the concentration should not exceed 170 ug/L at any time.

Human Health

Sufficient data are not available for zinc to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, to control undesirable taste and odor quality of ambient water the estimated level is 5 mg/L. It should be recognized that organoleptic data have limitations as a basis for establishing a water quality criteria, and have no demonstrated relationship to potential adverse human health effects.

APPENDIX A

DERIVATION OF THE 1985 CRITERION

Derivation of numerical national water quality criteria for the protection of aquatic organisms and their uses is a complex process that uses information from many areas of aquatic toxicology. After a decision is made that a national criterion is needed for a particular material, all available information concerning toxicity to, and bioaccumulation by, aquatic organisms is collected, reviewed for acceptability, and sorted. If enough acceptable data on acute toxicity to aquatic animals are available, they are used to estimate the highest 1-hour average concentration that should not result in unacceptable effects on aquatic organisms and their uses. If justified, this concentration is made a function of a water quality characteristic such as pH, salinity, or hardness. Similarly, data on the chronic toxicity of the material to aquatic animals are used to estimate the highest 4-day average concentration that should not cause unacceptable toxicity during a long-term exposure. If appropriate, this concentration is also related to a water quality characteristic.

Data on toxicity to aquatic plants are examined to determine whether plants are likely to be unacceptably affected by concentrations that should not cause unacceptable effects on animals. Data on bioaccumulation by aquatic organisms are used to determine if residues might subject edible species to restrictions by the U.S. Food and Drug Administration or if such residues might harm some wildlife consumers of aquatic life. All other available data are examined for adverse effects that might

be biologically important.

If a thorough review of the pertinent information indicates that enough acceptable data are available, numerical national water quality criteria are derived for fresh water or saltwater or both to protect aquatic organisms and their uses from unacceptable effects due to exposures to high concentrations for short periods of time, lower concentrations for longer periods of time, and combinations of the two.

I. Collection of Data

- A. Collect all available data on the material concerning (a) toxicity to, and bioaccumulation by, aquatic animals and plants, (b) FDA action levels [12], and (c) chronic feeding studies and long-term field studies with wildlife species that regularly consume aquatic organisms.
- B. All data that are used should be available in typed, dated, and signed hard copy (publication, manuscript, letter, memorandum, etc.) with enough supporting information to indicate that acceptable test procedures were used and that the results are probably reliable. In some cases it may be appropriate to obtain additional written information from the investigator, if possible. Information that is confidential or privileged or otherwise not available for distribution should not be used.
- C. Questionable data, whether published or

unpublished, should not be used. For example, data should usually be rejected if they are from tests that did not contain a control treatment, tests in which too many organisms in the control treatment died or showed signs of stress or disease, and tests in which distilled or deionized water was used as the dilution water without addition of appropriate salts.

- D. Data on technical grade materials may be used if appropriate, but data on formulated mixtures and emulsifiable concentrates of the material of concern should not be used.
- E. For some highly volatile, hydrolyzable, or degradable materials it is probably appropriate to use only results of flow-through tests in which the concentrations of test material in the test solutions were measured often enough using acceptable analytical methods.
- F. Data should be rejected if they were obtained using:
 - 1. Brine shrimp, because they usually occur naturally only in water with salinity greater than 35 g/kg.
 - 2. Species that do not have reproducing wild populations in North America (See Appendix 1).
 - 3. Organisms that were previously exposed to substantial concentrations of the test material or other contaminants.

- G. Questionable data, data on formulated mixtures and emulsifiable concentrates, and data obtained with nonresident species or previously exposed organisms may be used to provide auxiliary information but should not be used in the derivation of criteria.

11. Required Data

- A. Certain data should be available to help ensure that each of the four major kinds of possible adverse effects receives adequate consideration. Results of acute and chronic toxicity tests with representative species of aquatic animals are necessary so that data available for tested species can be considered a useful indication of the sensitivities of appropriate untested species. Fewer data concerning toxicity to aquatic plants are required because procedures for conducting tests with plants and interpreting the results of such tests are not as well developed. Data concerning bioaccumulation by aquatic organisms are required only if relevant data are available concerning the significance of residues in aquatic organisms.
- B. To derive a criterion for freshwater aquatic organisms and their uses, the following should be available:
1. Results of acceptable acute tests (see Section

IV) with at least one species of freshwater animal in at least eight different families such that all of the following are included:

- a. the family Salmonidae in the class Osteichthyes
- b. a second family in the class Osteichthyes, preferably a commercially or recreationally important warmwater species (e.g., bluegill, channel catfish, etc.)
- c. a third family in the phylum Chordata (may be in the class Osteichthyes or may be an amphibian, etc.)
- d. a planktonic crustacean (e.g., cladoceran, copepod, etc.)
- e. a benthic crustacean (e.g., ostracod, isopod, amphipod, crayfish, etc.)
- f. an insect (e.g., mayfly, dragonfly, damselfly, stonefly, caddisfly, mosquito, midge, etc.)
- g. a family in a phylum other than Arthropoda or Chordata (e.g., Rotifera, Annelida, Mollusca, etc.)
- h. a family in any order of insect or any phylum not already represented.

- 2. Acute-chronic ratios (see Section VI) with species of aquatic animals in at least three different families provided that of the three species:

- a. at least one is a fish
 - b. at least one is an invertebrate
 - c. at least one is an acutely sensitive freshwater species (the other two may be saltwater species).
 3. Results of at least one acceptable test with a freshwater alga or vascular plant (see Section VIII). If plants are among the aquatic organisms that are most sensitive to the material, results of a test with a plant in another phylum (division) should also be available.
 4. At least one acceptable bioconcentration factor determined with an appropriate freshwater species, if a maximum permissible tissue concentration is available (see Section IX) .
- C. To derive a criterion for saltwater aquatic organisms and their uses, the following should be available:
1. Results of acceptable acute tests (see Section IV) with at least one species of saltwater animal in at least eight different families such that all of the following are included:
 - a. two families in the phylum Chordata
 - b. a family in a phylum other than Arthropoda or Chordata

- c. either the Mysidae or Penaeidae family
 - d. three other families not in the phylum Chordata (may include Mysidae or Penaeidae, whichever was not used above)
 - e. any other family.
2. Acute-chronic ratios (see section VI) with species of aquatic animals in at least three different families provided that of the three species:
- a. at least one is a fish
 - b. at least one is an invertebrate
 - c. at least one is an acutely sensitive saltwater species (the other one may be a freshwater species).
3. Results of at least one acceptable test with a saltwater alga or vascular plant (see Section VIII. If plants are among the aquatic organisms most sensitive to the material, results of a test with a plant in another phylum (division) should also be available.
4. At least one acceptable bioconcentration factor determined with an appropriate saltwater species, if a maximum permissible tissue concentration is available (see Section IX).
- D. If all the required data are available, a numerical criterion can usually be derived, except in special cases. For example, derivation of a criterion
-

might not be possible if the available acute-chronic ratios vary by more than a factor of 10 with no apparent pattern. Also, if a criterion is to be related to a water quality characteristic T (see Sections V and VII), more data will be necessary.

Similarly, if all required data are not available, a numerical criterion should not be derived except in special cases. For example, even if not enough acute and chronic data are available, it might be possible to derive a criterion if the available data clearly indicate that the Final Residue Value should be much lower than either the Final Chronic Value or the Final Plant Value.

- E. Confidence in a criterion usually increases as the amount of available pertinent data increases. Thus, additional data are usually desirable.

III. Final Acute Value

- A. Appropriate measures of the acute (short-term) toxicity of the material to a variety of species of aquatic animals are used to calculate the Final Acute Value. The Final Acute Value is an estimate of the concentration of the material corresponding to a cumulative probability of 0.05 in the acute toxicity values for the genera with which acceptable acute tests have been conducted on the material. However, in some cases, if the Species

Mean Acute Value of a commercially or recreationally important species is lower than the calculated Final Acute Value, then that Species Mean Acute Value replaces the calculated Final Acute Value in order to provide protection for that important species.

- B. Acute toxicity tests should have been conducted using acceptable procedures [13].
- C. Except for tests with saltwater annelids and mysids, results of acute tests during which the test organisms were fed should not be used, unless data indicate that the food did not affect the toxicity of the test material.
- D. Results of acute tests conducted in unusual dilution water, e.g., dilution water in which total organic carbon or particulate matter exceeded 5 mg/L, should not be used, unless a relationship is developed between acute toxicity and organic carbon or particulate matter or unless data show that organic carbon, particulate matter, etc., do not affect toxicity.
- E. Acute values should be based on endpoints which reflect the total severe acute adverse impact of the test material on the organisms used in the test. Therefore, only the following kinds of data on acute toxicity to aquatic animals should be used:

1. Tests with daphnids and other cladocerans should be started with organisms less than 24 hours old and tests with midges should be stressed with second- or third-instar larvae. The result should be the 48-hr **EC50** based on percentage of organisms immobilized plus percentage of organisms killed. If such an **EC50** is not available from a test, the 48-hr **LC50** should be used in place of the desired 48-hr **EC50**. An **EC50** or **LC50** of longer than 48 hours can be used as long as the animals were not fed and the control animals were acceptable at the end of the test.
2. The result of a test with embryos and larvae of barnacles, bivalve molluscs (clams, mussels, oysters, and scallops), sea urchins, lobsters, crabs, shrimp, and abalones should be the 96-hr **EC50** based on the percentage of organisms with incompletely developed shells plus the percentage of organisms killed. If such an **EC50** is not available from a test, the lower of the 96-hr **EC50** based on the percentage of organisms with incompletely developed shells and the 96-hr **LC50** should be used in place of the desired 96-hr **EC50**. If the duration of the test was between 48 and 96 hours, the **EC50** or **LC50** at the end of the test should be used.

3. The acute values from tests with all other freshwater and saltwater animal species and older life stages of barnacles, bivalve molluscs, sea urchins, lobsters, crabs, shrimps, and abalones should be the 96-hr EC50 based on the percentage of organisms exhibiting loss of equilibrium plus the percentage of organisms immobilized plus the percentage of organisms killed. If such an EC50 is not available from a test, the 96-hr LC50 should be used in place of the desired 96-hr EC50.
4. Tests with single-celled organisms are not considered acute tests, even if the duration was 96 hours or less.
5. If the tests were conducted properly, acute values reported as "greater than" values and those which are above the solubility of the test material should be used, because rejection of such acute values would unnecessarily lower the Final Acute Value by eliminating acute values for resistant species.

F. If the acute toxicity of the material to aquatic animals apparently has been shown to be related to a water quality characteristic such as hardness or particulate matter for freshwater animals or

salinity **or** particulate matter for saltwater animals, a Final Acute Equation should be derived based on that water quality characteristic. Go to Section V.

- G. **If** the available data indicate that one **or** more life stages are at least a factor of 2 more resistant than one **or** more other life stages of the same species, the data **for** the more resistant life stages should not be used in the calculation of the Species Mean Acute Value (SMAV) because a species can only be considered protected from acute toxicity if all life stages are protected.
- H. The agreement of the data within and between species should be considered. Acute values that appear to be questionable in comparison with other acute and chronic data for the same species and for other species in the same genus probably should not be used in calculation of a Species Mean Acute Value. For example, if the acute values available **For a** species or genus differ by more than **a** factor of 10, some **or** all of the values probably should not be used in calculations.
- I. **For** each species for which at least one acute value is available, the Species Mean Acute Value should be calculated as the geometric mean of the results of all flow-through tests in which the concentrations of test material were measured. **For a** species **for** which no such result is available,

the Species Mean Acute Value should be calculated as the geometric mean of all available acute values, i.e., results of flow-through tests in which the concentrations were not measured and results of static and renewal tests based on initial concentrations of test material (nominal concentrations are acceptable for most test materials if measured concentrations are not available).

NOTE: Data reported by original investigators should not be rounded off. Results of all intermediate calculations should be rounded [14] to four significant digits.

NOTE: The geometric mean of N numbers is the N^{th} root of the product of the N numbers. Alternatively, the geometric mean can be calculated by adding the logarithms of the N numbers, dividing the sum by N , and taking the antilog of the quotient. The geometric mean of two numbers is the square root of the product of the two numbers, and the geometric mean of one number is that number. Either natural (base 0) or common (base 10) logarithms can be used to calculate geometric means as long as they are used consistently within each set of data, i.e., the antilog used must match the logarithm Used.

NOTE: Geometric means, rather than arithmetic means, are used here because the distributions of sensitivities of individual organisms in toxicity tests on most materials and the distributions of sensitivities of species within a genus are more likely to be lognormal than normal. Similarly, geometric means are used for acute-chronic ratios and bioconcentration factors because quotients are likely to be closer to lognormal than normal distributions. In addition, division of the geometric mean of a set of numerators by the geometric mean of the set of corresponding denominators will result in the geometric mean of the set of corresponding quotients.

- J. For each genus for which one or more Species Mean Acute Values are available, the Genus Mean Acute Value should be calculated as the geometric mean of the Species Mean Acute Values available for the genus.
- K. Order the Genus Mean Acute Value from high to low.
- L. Assign ranks, R , to the Genus Mean Acute Value from "1" for the lowest to "N" for the highest. If two or more Genus Mean Acute Values are identical, arbitrarily assign them successive ranks.
- M. Calculate the cumulative probability, P , for each Genus Mean Acute Value as $R/(N+1)$.

N. Select the four Genus Mean Acute Value which have cumulative probabilities closest to 0.05 (if there are less than 59 Genus Mean Acute Value, these will always be the four lowest Genus Mean Acute Values).

O. Using the selected Genus Mean Acute Values and **Es**, calculate:

$$S_2 = \frac{E(\ln \text{GMAV})_2 - ((E \ln \text{GMAV}))_2/4}{(P) - ((E / ^p))_2/4}$$

$$L = (E(\ln \text{GMAV}) - S(E(/ ^p)))/4$$

$$A = S(/ ^{0.05}) + L$$

$$\text{FAV} = e^A$$

(See [11] for development of the calculation procedure and Appendix 2 for example calculation and computer program.)

NOTE: Natural logarithms (logarithms to base **e**, denoted as **ln**) are used herein merely because they are easier to use on some hand calculators and computers than common (base 10) logarithms. Consistent use of either will produce the same result.

P. If for a commercially or recreationally important species the geometric mean of the acute values from flow-through tests in which the concentrations of test material were measured is lower than the calculated Final Acute Value, then that geometric mean should be used as the Final Acute Value instead of the calculated Final Acute

Value.

Q. Go to section VI.

IV. Final Acute Equation

- A. When enough data are available to show that acute toxicity to two or more species is similarly related to a water quality characteristic, the relationship should be taken into account as described in Sections B-G below or using analysis of covariance {15,16}. The two methods are equivalent and produce identical results. The manual method described below provides an understanding of this application of covariance analysis, but computerized versions of covariance analysis are much more convenient for analyzing large data tests. If two or more factors affect toxicity, multiple regression analysis should be used.
- B. For each species for which comparable acute toxicity values are available at two or more different values of the water quality characteristic, perform a least squares regression of the acute toxicity values on the corresponding values of the water quality characteristic to obtain the slope and its 95 percent confidence limits for each species.

NOTE: Because the best documented relationship fitting these data is that between hardness and acute toxicity of metals in fresh water and a log-log

relationship, geometric means and natural logarithms of both toxicity and water quality are used in the rest of this section. For relationships based on other water quality characteristics such as pH, temperature, or salinity, no transformation or a different transformation might fit the data better, and appropriate changes will be necessary throughout this section.

- c. Decide whether the data for each species are useful, taking into account the range and number of the tested values of the water quality characteristic and the degree of agreement within and between species. For example, a slope based on six data points might be of limited value if it is based only on data for a very narrow range of values of the water quality characteristic. A slope based on only two data points, however, might be useful if it is consistent with other information and if the two points cover a broad enough range of the water quality characteristic. In addition, acute values that appear to be questionable in comparison with other acute and chronic data available for the same species and for other species in the same genus probably should not be used. For example, if after adjustment for the water quality characteristic, the acute values available for a species or genus

differ by more than a factor of 10, probably some or all of the values should be rejected. If useful slopes are not available for at least one fish and one invertebrate or if the available slopes are too dissimilar or if too few data are available to adequately define the relationship between acute toxicity and the water quality characteristic, return to Section IV.G, using the results of tests conducted under conditions and in waters similar to those commonly used for toxicity tests with the species.

- D. Individually for each species calculate the geometric mean of the available acute values and then divide each of the acute values for species by the mean for the species. This normalizes the values so that the geometric mean of the normalized values for each species individually and for any combination of species is 1.0.
- E. Similarly normalize the values of the water quality characteristic for each species individually.
- F. Individually for each species perform a least squares regression of the normalized acute toxicity values on the corresponding normalized values of the water quality characteristic. The resulting slopes and 95 percent confidence limits will be identical to those obtained in Section B.

Now, however, if the data are actually plotted, the line of best fit for each individual species will go through the point 1,1 in the center of the graph.

- G. Treat all the normalized data as if they were all for the same species and perform a least squares regression of all the normalized acute values on the corresponding normalized values of the water quality characteristic to obtain the pooled acute slope, V , and its 95 percent confidence limits. If all the normalized data are actually plotted, the line of best fit will go through the point 1,1 in the center of the graph.

- H. For each species calculate the geometric mean, W , of the acute toxicity values and the geometric mean, X , of the values of the water quality characteristic. (These were calculated in steps D and E.)

- I. For each species calculate the logarithm, Y , of the Species Mean Acute Value at a selected value, Z , of the water quality characteristic using the equation:

$$Y = \ln W - V(\ln X - \ln Z).$$

- J. For each species calculate the SMAV at Z using the equation: $SMAV = e^Y$.

NOTE: Alternatively, the Species Mean Acute Values at Z can be obtained by skipping step H using the

equations in steps I and J to adjust each acute value individually to Z, and then calculating the geometric mean of the adjusted values for each species individually. This alternative procedure allows an examination of the range of the adjusted acute values for each species.

- K. Obtain the Final Acute Value at Z by using the procedure described in Section IV.J-0.
- L. If the Species Mean Acute Value at Z of a commercially or recreationally important species is lower than the calculated Final Acute Value at Z, then that Species Mean Acute Value should be used as the Final Acute Value at Z instead of the calculated Final Acute Value.
- M. The Final Acute Equation is written as: Final Acute Value = $e^{(V[\ln(\text{water quality characteristic})] + \ln A - V[\ln Z])}$, where V = pooled acute slope and A = Final Acute Value at Z. Because V, A, and Z are known, the Final Acute Value can be calculated for any selected value of the water quality characteristic.

V. Final Chronic Value

- A. Depending on the data that are available concerning chronic toxicity to aquatic animals, the Final Chronic Value might be calculated in the same manner as the Final Acute Value or by dividing the Final Acute Value by the Final Acute-

Chronic Ratio. In some cases it may not be possible to calculate a Final Chronic Value.

NOTE : As the name implies, the acute-chronic ratio is a way of relating acute and chronic toxicities. The acute-chronic ratio is basically the inverse of the application factor, but this new name is better because it is more descriptive and should help prevent confusion between 'application factors' and 'safety factors.' Acute-chronic ratios and application factors are ways of relating the acute and chronic toxicities of a material to aquatic organisms. Safety factors are used to provide an extra margin of safety beyond the known or estimated sensitivities of aquatic organisms. Another advantage of the acute-chronic ratio is that it will usually be greater than 1; this should avoid the confusion as to whether a large application factor is one that is close to unity or one that has a denominator that is much greater than the numerator.

- B. Chronic values should be based on results of flow-through (except renewal is acceptable for daphnids) chronic tests in which the concentrations of test material in the test solutions were properly measured at appropriate times during the test.
- C. Results of chronic tests in which survival,

growth, or reproduction in the control treatment was unacceptably low should not be used. The limits of acceptability will depend on the species.

- D. Results of chronic tests conducted in unusual dilution water, e.g., dilution water in which total organic carbon or particulate matter exceeded 5 mg/L, should not be used, unless a relationship is developed between chronic toxicity and organic carbon or particulate matter or unless data show that organic carbon, particulate matter, etc., do not affect toxicity.
- E. Chronic values should be based on endpoints and lengths of exposure appropriate to the species. Therefore, only results of the following kinds of chronic toxicity tests should be used:
 - 1. Life-cycle toxicity **tests** consisting of exposures of each of **two or** more groups of individuals of a species **to** a different concentration of the test material throughout a life cycle. To ensure that all life stages and life processes are exposed, tests with fish should begin with embryos or newly hatched young less than 48 hours old, continue through maturation and reproduction, and should end not less than **24** days (90 days for **salmonids**) after the hatching of the next generation. Tests with **daphnids** should begin

with young less than 24 hours old and last for not less than 21 days. Tests with mysids should begin with young less than 24 hours old and continue until 7 days past the median time of first brood release in the controls. For fish, data should be obtained and analyzed on survival and growth of adults and young, maturation of males and females, eggs spawned per female, embryo viability (salmonids only), and hatchability. For daphnids, data should be obtained and analyzed on survival and young per female. **For** mysids, data should be obtained and analyzed on survival, growth, and young per female.

2. Partial life-cycle toxicity tests consisting of exposures of each of two or more groups of individuals of a species of fish to a concentration of the test material through most portions of a life cycle. Partial life-cycle tests are allowed with fish species that require more than a year to reach sexual maturity, so that all major life stages can be exposed to the test material in less than 15 months. Exposure to the test material should begin with immature juveniles at least 2 months prior to active gonad development, continue through maturation and reproduction,

and end not less than **24** days (90 days for salmonids) after the hatching of the next generation. Data should be obtained and analyzed on survival and growth of adults and young, maturation of males and females, eggs spawned per female, embryo viability (salmonids only), and hatchability.

3. Early life-stage toxicity tests consisting of 28- to 32-day (**60** days post hatch for salmonids) exposures of the early life stages of a species of fish from shortly after fertilization through embryonic, larval, and early juvenile development. Data should be obtained and analyzed on survival and growth.

NOTE: Results of an early life-stage test are used as predictions of results of life-cycle and partial life-cycle tests with the same species. Therefore, when results of a life-cycle or partial life-cycle test are available, results of an early life-stage test with the same species should not be used. Also, results of early life-stage tests in which the incidence of mortalities or abnormalities increased substantially near the end of the test should not be used because results of such tests are possibly not good predictions of the results of comparable life-cycle or partial life-cycle tests.

F. A chronic value may be obtained by calculating the geometric mean of the lower and upper chronic limits from a chronic test or by analyzing chronic data using regression analysis. A lower chronic limit is the highest tested concentration (a) in an acceptable chronic test, (b) which did not cause an unacceptable amount of adverse effect on any of the specified biological measurements, and (c) below which no tested concentration caused an unacceptable effect. An upper chronic limit is the lowest tested concentration (a) in an acceptable chronic test, (b) which did cause an unacceptable amount of adverse effect on one or more of the specified biological measurements, and (c) above which all tested concentrations also caused such an effect.

NOTE: Because various authors have used a variety of terms and definitions to interpret and report results of chronic tests, reported results should be reviewed carefully. The amount of effect that is considered unacceptable is often based on a statistical hypothesis test, but might also be defined in terms of a specified percent reduction from the controls. A small percent reduction (e.g., 3 percent) might be considered acceptable even if it is statistically significantly different from the control, whereas a large

percent reduction (e.g., 30 percent) might be considered unacceptable even if it is not statistically significant.

- G. If the chronic toxicity of the material to aquatic animals apparently has been shown to be related to a water quality characteristic such as hardness or particulate matter for freshwater animals or salinity or particulate matter for saltwater animals, a Final Chronic Equation should be derived based on that water quality characteristic. Go to Section VII.
- H. If chronic values are available for species in eight families as described in Sections III.B.1 or III.C.1, a Species Mean Chronic Value (SMCV) should be calculated for each species for which at least one chronic value is available by calculating the geometric mean of all chronic values available for the species, and appropriate Genus Mean Chronic Values should be calculated. The Final Chronic Value should then be obtained using the procedure described in Section IV.J-0. Then go to Section VI.M.
- I. For each chronic value for which at least one corresponding appropriate acute value is available, calculate an acute-chronic ratio, using **for** the numerator the geometric mean of the results **of** all acceptable flow-through (except static is acceptable for daphnids) acute tests in

the same dilution water and in which the concentrations were measured. For fish, the acute test(s) should have been conducted with juveniles. The acute test(s) should have been part of the same study as the chronic test. If acute tests were not conducted as part of the same study, acute tests conducted in the same laboratory and dilution water, but in a different study, may be used. If no such acute tests are available, results of acute tests conducted in the same dilution water in a different laboratory may be used. If no such acute tests are available, an acute-chronic ratio should not be calculated.

- J. For each species, calculate the species mean acute-chronic ratio as the geometric mean of all acute-chronic ratios available for that species.
- K. For some materials the acute-chronic ratio seems to be the same for all species, but for other materials the ratio seems to increase or decrease as the Species Mean Acute Value (SMAV) increases. Thus the Final Acute-Chronic Ratio can be obtained in four ways, depending on the data available:
 - 1. If the Species Mean Acute-Chronic ratio Seems to increase or decrease as the Species Mean Acute Value increases, the Final Acute-Chronic Ratio should be calculated as the geometric mean of the acute-chronic ratios for species

whose Species Mean Acute Values are close to the Final Acute Value.

2. If no major trend is apparent and the acute-chronic ratios for a number of species are within a factor of 10, the Final Acute-Chronic Ratio should be calculated as the geometric mean of all the Species Mean Acute-Chronic Ratios available for both freshwater and saltwater species.
3. For acute tests conducted on metals and possibly other substances with embryos and larvae of barnacles, bivalve molluscs, sea urchins, lobsters, crabs, shrimp, and abalones (see Section IV.E.2), it is probably appropriate to assume that the acute-chronic ratio is 2. Chronic tests are very difficult to conduct with most such species, but it is likely that the sensitivities of embryos and larvae would determine the results of life-cycle tests. Thus, if the lowest available Species Mean Acute Values were determined with embryos and larvae of such species, the Final Acute-Chronic Ratio should probably be assumed to be 2, so that the Final Chronic Value is equal to the Criterion Maximum Concentration (see Section XI.E)

4. If the most appropriate Species Mean Acute-Chronic Ratios are less than **2.0**, and especially if they are less than 1.0, acclimation has probably occurred during the chronic test. Because continuous exposure and acclimation cannot be assured to provide adequate protection in field situations, the Final Acute-Chronic Ratio should be assumed to be **2**, so that the Final Chronic Value is equal to the Criterion Maximum Concentration (see Section XI.B).

If the available Species Mean Acute-Chronic Ratios do not fit one of these cases, a Final Acute-Chronic Ratio probably cannot be obtained, and a Final Chronic Value probably cannot be calculated.

- L. Calculate the Final Chronic Value by dividing the Final Acute Value by the Final Acute-Chronic Ratio. If there was a Final Acute Equation rather than a Final Acute Value, see also Section VII.A.
- M. If the Species Mean Chronic Value of a commercially or recreationally important species is lower than the calculated Final Chronic Value, then that species Mean Chronic Value should be used as the Final Chronic Value instead of the calculated Final Chronic Value.
- N. Go to Section VIII.

VI. Final Chronic Equation

- A. A Final Chronic Equation can be derived in two ways. The procedure described here in Section A will result in the chronic slope being the same as the acute slope. The procedure described in Sections B-N usually will result in the chronic slope being different from the acute slope.
 1. If acute-chronic ratios are available for enough species at enough values of the water quality characteristic to indicate that the acute-chronic ratio is probably the same for all species and is probably independent of the water quality characteristic, calculate the Final Acute-Chronic Ratio **as** the geometric mean of the available Species Mean Acute-Chronic Ratios.
 2. Calculate the Final Chronic Value at the selected value x of the water quality characteristic by dividing the Final Acute Value at x (see Section V.M) by the Final Acute-Chronic Ratio.
 3. Use V = pooled acute slope (see section V.M) as L = pooled chronic slope.
 4. Go to Section VII.M.
- B. When enough data are available to show that chronic toxicity to at least one species is related to a water quality characteristic, the

relationship should be taken into account as described in Sections B-G or using analysis of covariance [15,16]. The two methods are equivalent and produce identical results. The manual method described below provides an understanding of this application of covariance analysis, but computerized versions of covariance analysis are much more convenient for analyzing large data sets. If two or more factors affect toxicity, multiple regression analysis should be used.

For each species for which comparable chronic toxicity values are available at two or more different values of the water quality characteristic, perform a least squares regression of the chronic toxicity values on the corresponding values of the water quality characteristic to obtain the slope and its 95 percent confidence limits for each species.

NOTE: Because the best documented relationship fitting these data is that between hardness and acute toxicity of metals in freshwater and a log-log relationship, geometric means and natural logarithms of both toxicity and water quality are used in the rest of this section. For relationships based on other water quality characteristics such as pH, temperature, or

salinity, no transformation ~~or~~ a different transformation might fit the data better, and appropriate changes will be necessary throughout this section. It ~~is~~ probably preferable, but not necessary, to use the same transformation that was used with the acute values in Section V.

- D. Decide whether the data for each species are useful, taking into account the range and number of the tested values of the water quality characteristic and the degree of agreement within and between species. For example, a slope based on six data points might be of limited value if it is based ~~only~~ on data ~~for~~ a very narrow range of values ~~of~~ the water quality characteristic. A slope based on only two data points, however, might be useful if it ~~is~~ consistent with other information and if the two points cover a broad enough range of the water quality characteristic. In addition, chronic values that appear to be questionable in comparison with other acute and chronic data available ~~for~~ the same species and for other species in the same genus probably should not be used. ~~For~~ example, if after adjustment for the water quality characteristic, the chronic values available for a species ~~or~~ genus differ by more than a factor of 10, probably some ~~or~~ all of the values should be rejected. If

a useful chronic slope is not available for at least one species or if the available slopes are too dissimilar or if too few data are available to adequately define the relationship between chronic toxicity and the water quality characteristic, it might be appropriate to assume that the chronic slope is the same as the acute slope, which is equivalent to assuming that the acute-chronic ratio is independent of the water quality characteristic. Alternatively, return to Section VII, using the results of tests conducted under conditions and in waters similar to those commonly used for toxicity tests with the species.

- E. Individually for each species calculate the geometric mean of the available chronic values and then divide each chronic value for a species by the mean for the species. This normalizes the chronic values so that the geometric mean of the normalized values for each species individually and for any combination of species is 1.0.
- F. Similarly normalize the values of the water quality characteristic for each species individually.
- G. Individually for each species perform a least squares regression of the normalized chronic toxicity values on the corresponding normalized values of the water quality characteristic. The resulting slopes and the 95 percent confidence

limits will be identical to those obtained in Section B. Now, however, if the data are actually plotted, the line of best fit for each individual species will go through the point 1,1 in the center of the graph.

H. Treat all the normalized data as if they were all for the same species and perform a least squares regression of all the normalized chronic values on the corresponding normalized values of the water quality characteristic to obtain the pooled chronic slope, L , and its **95** percent confidence limits. If all the normalized data are actually plotted, the line of best fit will go through the point 1,1 in the center of the graph.

I. For each species calculate the geometric mean, M , of the toxicity values and the geometric mean, P , of the values of the water quality characteristic. (These were calculated in steps E and F.)

J. For each species calculate the logarithm, Q , of the Species Mean Chronic Value at a selected value, Z , of the water quality characteristic using the equation: $Q = \ln M - L(\ln P - \ln Z)$.

NOTE: Although it is not necessary, it will usually be best to use the same value of the water quality characteristic here as was used in section V.I.

K. For each species calculate a Species Mean Chronic Value at Z using the equation: $SMCV = e^Q$.

NOTE: Alternatively, the Species Mean Chronic Values at Z can be obtained by skipping step J, using the equations in steps J and K to adjust each acute value individually to Z , and then calculating the geometric means of the adjusted values for each species individually. This alternative procedure allows an examination of the range of the adjusted chronic values for each species.

L. Obtain the Final Chronic Value at Z by using the procedure described in Section IV.J-0.

M. If the Species Mean Chronic Value at Z of a commercially or recreationally important species is lower than the calculated Final Chronic Value at Z , then that Species Mean Chronic Value should be used as the Final Chronic Value at Z instead of the calculated Final Chronic Value.

- N. The Final Chronic Equation is written as: Final Chronic Value = $e(L[\ln(\text{water quality characteristic})] + \ln S - L[\ln Z])$, where L = pooled chronic slope and S = Final Chronic Value at Z. Because L, S and Z are known, the Final Chronic Value can be calculated for any selected value of the water quality characteristic.

VII. Final Plant Value

- A. Appropriate measures of the toxicity of the material to aquatic plants are used to compare the relative sensitivities of aquatic plants and animals. Although procedures for conducting and interpreting the results of toxicity tests with plants are not well developed, results of tests with plants usually indicate that criteria which adequately protect aquatic animals and their uses will probably also protect aquatic plants and their uses.
- B. A plant value is the result of a 96-hr test conducted with an alga or a chronic test conducted with an aquatic vascular plant.
- NOTE: A test of the toxicity of a metal to a plant usually should not be used if the medium contained an excessive amount of a complexing agent, such as EDTA, that might affect the toxicity of the metal. Concentrations of EDTA above about 200 ug/L should probably be considered excessive.

- C. The Final Plant Value should be obtained by selecting the lowest result from a test with an important aquatic plant species in which the concentrations of test material were measured and the endpoint was biologically important.

VIII. Final Residue Value

- A. The Final Residue Value is intended to (a) prevent concentrations in commercially or recreationally important aquatic species from affecting marketability because of exceedence of applicable FDA action levels and (b) protect wildlife, including fishes and birds, that consume aquatic organisms from demonstrated unacceptable effects. The Final Residue Value is the lowest of the residue values that are obtained by dividing maximum permissible tissue concentrations by appropriate bioconcentration or bioaccumulation factors. A maximum permissible tissue concentration is either (a) an FDA action level [12] for fish oil or for the edible portion of fish or shellfish, or (b) a maximum acceptable dietary intake based on observations on survival, growth, or reproduction in a chronic wildlife feeding study or a long-term wildlife field study. If no maximum permissible tissue concentration is available, go to Section X because no Final Residue Value can be derived.

B. Bioconcentration Factors (BCFs) and bioaccumulation factors (BAFs) are quotients of the concentration of a material in one or more tissues of an aquatic organism divided by the average concentration in the solution in which the organism had been living. A BCF is intended to account **only** for net uptake directly from water, and thus almost has to be measured in a laboratory test. Some uptake during the bioconcentration test might not be directly from water **if** the food **sorbs** some of the test material before **it** is eaten by the test organisms. A BAF **is** intended to account for net uptake from both food and water in a **real-world** situation. A BAF almost has **to** be measured in a field situation in which predators accumulate the material directly **from** water and by consuming prey **that** itself could have accumulated the material from both food and water. The BCF and BAF are probably similar for a material with a low BCF, but the BAF is probably higher than the BCF for materials with high BCFs. Although BCFs are not **too** difficult **to** determine, very few BAFs have been measured acceptably because **it** is necessary **to** make enough measurements of the concentration of the material in water **to** show that **it** was reasonably constant for a long enough period of time over the range of territory inhabited **by** the

organisms. Because so few acceptable **BAFs** are available, only **BCFs** will be discussed further. However, if an acceptable **BAF** is available for a material, it should be used instead of any available **BCFs**.

- C. If a maximum permissible tissue concentration is available for a substance (e.g., parent material, parent material plus metabolites, etc.), the tissue concentration used in the calculation of the **BCF** should be for the same substance. otherwise the tissue concentration used in the calculation of the **BCF** should be that of the material and its metabolites which are structurally similar and are not much more soluble in water than the parent material.
- D. 1. A **BCF** should be used only if the test was flow-through, the **BCF** was calculated based on measured concentrations of the test material in tissue and in the test solution, and the exposure continued at least until either apparent steady-state or 28 days was reached. Steady-state is reached when the **BCF** does not change significantly over a period of time, such a 2 days or 16 percent of the length of the exposure, whichever is longer. The **BCF** used from a test should be the highest of (a) the apparent steady-state **BCF**, if apparent steady-state was reached, (b) the highest **BCF**

obtained, if apparent steady-state was not reached, and (c) the projected steady-state BCF, if calculated.

2. Whenever a BCF is determined for a lipophilic material, the percent lipids should also be determined in the tissue(s) for which the BCF was calculated.
3. A BCF obtained from an exposure that adversely affected the test organisms may be used only if it is similar to a BCF obtained with unaffected organisms of the same species at lower concentrations that did not cause adverse effects.
4. Because maximum permissible tissue concentrations are almost never based on dry weights, a BCF calculated using dry tissue weights must be converted to a wet tissue weight basis. If no conversion factor is reported with the BCF, multiply the dry weight BCF by 0.1 for plankton and by 0.2 for individual species of fishes and invertebrates [17].
5. If more than one acceptable BCF is available for a species, the geometric mean of the available values should be used, except that if the BCFs are from different lengths of exposure and the BCF increases with length of

exposure, the BCF for the longest exposure should be used.

- E. If enough pertinent data exist, several residue values can be calculated by dividing maximum permissible tissue concentrations by appropriate BCFs :
1. For each available maximum acceptable dietary intake derived from a chronic feeding study or a long-term field study with wildlife, including birds and aquatic organisms, the appropriate BCF is based on the whole body of aquatic species which constitute' or represent a major portion of the diet of the tested wildlife species.
 2. For an FDA action level for fish or shellfish, the appropriate BCF is the highest geometric mean species BCF for the edible portion (muscle for decapods, muscle with or without skin for fishes, adductor muscle for scallops, and total soft tissue for other bivalve molluscs) of a consumed species. The highest species BCF is used because FDA action levels are applied on a species-by-species basis.
- F. For lipophilic materials, it might be possible to calculate additional residue values. Because the steady-state BCF for a lipophilic material Seems to be proportional to percent lipids from one tissue to another and from one species to another

[18-20], extrapolations can be made from tested tissues or species to untested tissues or species on the basis of percent lipids.

1. For each BCF for which the percent lipids is known for the same tissue for which the BCF was measured, normalize the BCF to a 1 percent lipid basis by dividing the BCF by the percent lipids. This adjustment to a 1 percent lipid basis is intended to make all the measured BCFs for a material comparable regardless of the species or tissue with which the BCF was measured.
2. calculate the geometric mean normalized BCF. Data for both saltwater and freshwater species should be used to determine the mean normalized BCF, unless the data show that the normalized BCFs are probably not similar.
3. Calculate all possible residue values by dividing the available maximum permissible tissue concentrations by the mean normalized BCF and by the percent lipids values appropriate to the maximum permissible tissue concentrations, i.e.,

$$\text{Residue value} = \frac{(\text{maximum permissible tissue concentration})}{(\text{mean normalized BCF})(\text{appropriate percent lipids})}$$
$$\text{Residue value} = (\text{mean normalized BCF}) (\text{appropriate percent lipids})$$

- a. For an FDA action level for fish oil, the

appropriate percent lipids value is 100,

b. For an FDA action level for fish, the appropriate percent lipids value is 11 for freshwater criteria and 10 for saltwater criteria because FDA action levels are applied on a species-by-species basis to commonly consumed species. The highest lipid contents in the edible portions of important consumed species are about 11 percent for both the freshwater chinook salmon and lake trout and about 10 percent for the saltwater Atlantic herring [21],

c. For a maximum acceptable dietary intake derived from a chronic feeding study or a long-term field study with wildlife, the appropriate percent lipids is that of an aquatic species or group of aquatic species which constitute a major portion of the diet of the wildlife species.

G. The Final Residue Value is obtained by selecting the lowest of the available residue values.

NOTE: In some cases the Final Residue Value will not be low enough. For example, a residue value calculated from an FDA action level will probably result in an average concentration in the edible portion of a fatty species that is at the action level. Some individual organisms, and possibly some species, will have residue concentrations higher than the mean value but no mechanism has been devised to provide appropriate additional protection. Also, some chronic feeding studies and long-term field studies with wildlife identify concentrations that cause adverse effects but do not identify concentrations which do not cause adverse effects; again, no mechanism has been devised to provide appropriate additional protection. These are some of the species and uses that are not protected at all times in all places.

x- other Data

Pertinent information that could not be used in earlier sections might be available concerning adverse effects on aquatic organisms and their uses. The most important of these are data on cumulative and delayed toxicity, flavor impairment, reduction in survival, growth, or reproduction, or any other adverse effect that has been shown to be biologically important. Especially important are data for species for which no

other data are available. Data from behavioral, biochemical, physiological, microcosm, and field studies might also be available. Data might be available from tests conducted in unusual dilution water (see IV.D and VI.D), from chronic tests in which the concentrations were not measured (see VI.B), from tests with previously exposed organisms (see II.F), and from tests on formulated mixtures or emulsifiable concentrates (see II.D). Such data might affect a criterion if the data were obtained with an important species, the test concentrations were measured, and the endpoint was biologically important.

XI. Criterion

- A. A criterion consists of two concentrations: the Criterion Maximum Concentration and the Criterion Continuous Concentration.
- B. The Criterion Maximum Concentration (CMC) is equal to one-half the Final Acute Value.
- C. The Criterion Continuous Concentration (CCC) is equal to the lowest of the Final Chronic Value, the Final Plant Value, and the Final Residue Value, unless other data (see Section X) show that a lower value should be used. If toxicity is related to a water quality characteristic, the Criterion continuous concentration is obtained from the Final Chronic Equation, the Final Plant Value, and the Final Residue Value by selecting

the one, or the combination, that results in the lowest concentrations in the usual range of the water quality characteristic, unless other data (see Section X) show that a lower value should be used.

D. Round [14] both the Criterion Maximum Concentration and the Criterion Continuous Concentration to two significant digits.

E. The criterion is stated as:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, (1) aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentration of (2) does not exceed (3) ug/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed (4) ug/L more than once every 3 years on the average.

where (1) = insert "freshwater" or "saltwater"

(2) = insert name of material

(3) = insert the Criterion Continuous
Concentration

(4) = insert the Criterion Maximum
Concentration.

XII. Final Review

A. The derivation of the criterion should be carefully reviewed by rechecking each step of the Guidelines. Items that should be especially checked are:

1. If unpublished data are used, are they well documented?
2. Are all required data available?
3. Is the range of acute values for any species greater than a factor of 10?
4. Is the range of Species Mean Acute Values for any genus greater than a factor of 10?
5. Is there more than a factor of 10 difference between the four lowest Genus Mean Acute Values?
6. Are any of the four lowest Genus Mean Acute Values questionable?
7. Is the Final Acute Value reasonable in comparison with the Species Mean Acute Values and Genus Mean Acute Values?
8. For any commercially or recreationally important species, is the geometric mean of the acute values from flow-through tests in which the concentrations of test material were measured lower than the Final Acute Value?
9. Are any of the chronic values questionable?

10. Are chronic values available for acutely sensitive species?
11. Is the range of acute-chronic ratios greater than a factor of 10?
12. Is the Final Chronic Value reasonable in comparison with the available acute and chronic data?
13. Is the measured or predicted chronic value for any commercially or recreationally important species below the Final Chronic Value?
14. Are any of the other data important?
15. Do any data look like they might be outliers?
16. Are there any deviations from the Guidelines?
Are they acceptable?

B. On the basis of **all** available pertinent laboratory and field information, determine if the criterion is consistent with sound scientific evidence. If it is not, another criterion, either higher or lower, should be derived using appropriate modifications of these Guidelines.

APPENDIX B

SUMMARY OF THE 1980 AQUATIC LIFE GUIDELINES

The Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and its Uses were developed to describe an objective, internally consistent, and appropriate way of ensuring that water quality criteria for aquatic life would provide, on the average, a reasonable amount of protection. The resulting criteria are not intended to provide 100 percent protection of all species and all uses of aquatic life all of the time, but they are intended to protect most species in a balanced, healthy aquatic community.

Minimum data requirements are identified in four areas; acute toxicity to animals (eight data points), chronic toxicity to animals (three data points), toxicity to plants, and residues. Data on acute toxicity are needed for a variety of fish and invertebrate species and are used to derive a Final Acute Value. By taking into account the number and relative sensitivities of the tested species, the Final Acute Value is designed to protect most, but not necessarily all, of the tested and untested species.

Data on chronic toxicity to animals can be used to derive a Final Chronic Value by two different means. If chronic values are available for a specified number and array of species, a Final Chronic Value can be calculated directly. If not, an acute-chronic ratio is derived and then used with the Final Acute Value to obtain the Final Chronic Value.

The Final Plant Value is obtained by selecting the lowest plant toxicity value based on measured concentrations.

The Final Residue Value is intended to protect wildlife which consume aquatic organisms and the marketability of aquatic organisms. Protection of the marketability of aquatic organisms is, in actuality, protection of a use of that water body (commercial fishery). Two kinds of data are necessary to calculate the Final Residue Value: a bioconcentration factor (BCF) and a maximum permissible tissue concentration, which can be an FDA action level or can be the result of a chronic wildlife feeding study. For lipid-soluble pollutants, the BCF is normalized for percent lipids and then the Final Residue Value is calculated by dividing the maximum permissible tissue concentration by the normalized BCF and by an appropriate percent lipid value. BCFs are normalized for percent lipids since the BCF measured for any individual aquatic species is generally proportional to the percent lipids in that species.

If sufficient data are available to demonstrate that one or more of the final values should be related to a water quality characteristic, such as salinity, hardness, or suspended solids, the final value(s) are expressed as a function of that characteristic.

After the four final values (Final Acute Value, Final Chronic Value, Final Plant Value, and Final Residue Value) have been obtained, the criterion is established with the Final Acute value becoming the maximum value and the lowest of the other three values becoming the 24-hour average value. All of the data used to calculate the four final values and any additional pertinent information are then reviewed to determine if the criterion is reasonable. If sound scientific evidence indicates that the

criterion should be raised or lowered, appropriate changes are made as necessary.

The November 28, 1980, Guidelines have been revised from the earlier published versions (43 FR 21506, May 18, 1978; 43 FR 29028, July 5, 1978; 44 FR 15926, March 15, 1979). Details have been added in many places and the concept of a minimum data base has been incorporated. In addition, three adjustment factors and the species sensitivity factor have been deleted. These modifications were the result of the Agency's analysis of public comments and comments received from the Science Advisory Board on earlier versions of the Guidelines. These comments and the Resultant modifications are addressed fully in Appendix D to this notice.

Criteria for the Protection of Human Health

Interpretation of the Human Health Criteria

The human health criteria issued today are summarized in Appendix A of this Federal Register notice. Criteria for the protection of human health are based on their carcinogenic, toxic, or organoleptic (taste and odor) properties. The meanings and practical uses of the criteria values are distinctly different depending on the properties on which they are based.

The objective of the health assessment portions of the criteria documents is to estimate ambient water concentrations which, in the case of noncarcinogens, prevent adverse health effects in humans, and in the case of suspect or proven carcinogens, represent various levels of incremental cancer risk.

Health assessments typically contain discussions of four elements: exposure, pharmacokinetics, toxic effects, and criterion formulation.

The exposure section summarizes information on exposure routes: ingestion directly from water, indirectly from consumption of aquatic organisms found in ambient water, other dietary sources, inhalation, and dermal contact. Exposure assumptions are used to derive human health criteria. **Most** criteria are based solely on exposure from consumption of water containing a specified concentration of a toxic pollutant and through consumption of aquatic organisms which are assumed to have bioconcentrated pollutants from the water in which they live. Other multimedia routes of exposure such as air, nonaquatic diet, or dermal are not factored into the criterion formulation for the vast majority of pollutants because of lack of data. The criteria are calculated using the combined aquatic exposure pathway and also using the aquatic organism ingestion exposure route alone. In criteria reflecting both the water consumption and aquatic organism ingestion routes of exposure, the relative exposure contribution varies with the propensity of a pollutant to bioconcentrate, with the consumption of aquatic organisms becoming more important as the bioconcentration factor (BCF) increases. As additional information on total exposure is assembled for pollutants for which criteria reflect only the two specified aquatic exposure routes, adjustments in water concentration values may be made. The demonstration of significantly different exposure patterns will become an element

of a process to adapt/modify human health-based criteria to local conditions, somewhat analogous to the aquatic life criteria modification process discussed previously. It is anticipated that States at their discretion will be able to set appropriate human health criteria based on this process.

Specific health-based criteria are developed only if a weight of evidence supports the occurrence of the toxic effect and if dose/response data exist from which criteria can be estimated. The pharmacokinetics section reviews data on absorption, distribution, metabolism, and excretion to assess the biochemical fate of the compounds in the human and animal system. The toxic effects section reviews data on acute, subacute, and chronic toxicity, synergistic and antagonistic effects, and specific information on mutagenicity, teratogenicity, and carcinogenicity. From this review, the toxic effect to be protected against is identified taking into account the quality, quantity, and weight of evidence characteristic of the data. The criterion formulation section reviews the highlights of the text and specifies a rationale for criterion development and the mathematical derivation of the criterion number.

Within the limitations of time and resources, current published information of significance was incorporated into the human health assessments. Review articles and reports were used for data evaluation and synthesis. Scientific judgment was exercised in reviewing and evaluating the data in each criteria document and in identifying the adverse effects for which protective criteria were published.

Criteria for suspect or proven carcinogens are presented as concentrations in water associated with a range of incremental cancer risks to man. Criteria for noncarcinogens represent levels at which exposure to a single chemical is not anticipated to produce adverse effects in man. In a few cases, organoleptic (taste and odor) data form the basis for the criterion. While this type of criterion does not represent a value which directly affects human health, it is presented as an estimate of the level of a pollutant that will not produce unpleasant taste or odor either directly from water consumption or indirectly by consumption of aquatic organisms found in ambient waters. A criterion developed in this manner is judged to be as useful as other types of criteria in protecting designated water uses. In addition, where data are available, toxicity-based criteria are also presented for pollutants with derived organoleptic criteria. The choice of criteria used in water quality standards for these pollutants will depend upon the designated use to be protected. In the case of a multiple use water body, the criterion protecting the most sensitive use will be applied. Finally, for several pollutants no criteria are recommended because insufficient information is available for quantitative criterion formulation.

Risk Extrapolation

Because methods do not exist to establish the presence of a threshold for carcinogenic effects, EPA's policy is that there is no scientific basis for estimating "safe" levels for carcinogens. The criteria for carcinogens, therefore, state that the

recommended concentration for maximum protection of human health is zero. In addition, the Agency has presented a range of concentrations corresponding to incremental cancer risks of to 10^{-6} (one additional case of cancer in populations ranging from 10 million to 100,000, respectively). Other concentrations representing different risk levels may be calculated by use of the Guidelines. The risk estimate range is presented for information purposes and does not represent an Agency judgment on a "acceptable" risk level.

Summary of the Human Health Guidelines

The health assessments and corresponding criteria were derived based on Guidelines and Methodology Used in the Preparation of Health Effect Assessment Chapters of the Consent Decree Water Criteria Documents (the Guidelines) developed by EPA'S Office of Research and Development. The estimation of health risk associated with human exposure to environmental pollutants requires predicting the effect of low doses for up to a lifetime in duration. A combination of epidemiological and animal dose/response data is considered the preferred basis for quantitative criterion derivation.

No-effect (noncarcinogen) or specified risk (carcinogen) concentrations were estimated by extrapolation from animal toxicity or human epidemiology studies using the following basic exposure assumptions: a 70-kilogram male person (Report of the Task Group on Reference Man, International Commission for Radiation Protection, November 23, 1957) as the exposed individual; the average daily consumption of freshwater and

estuarine fish and shellfish products equal to 6.5 grams/day; and the average ingestion of 2 liters/day of water (Drinking Water and Health, National Academy of Sciences, National Research Council, 1977). Criteria based on these assumptions are estimated to be protective of an adult male who experiences average exposure conditions.

Two basic methods were used to formulate health criteria, depending on whether the prominent adverse effect was cancer or other toxic manifestations. The following sections detail these methods.

Carcinogens

Extrapolation of cancer responses from high to low doses and subsequent risk estimation from animal data are performed using a linearized multi-stage model. This procedure **is** flexible enough to fit all monotonically-increasing dose response data, since it incorporates several adjustable parameters. The multi-stage model is a linear nonthreshold model as was the "one-hit" model originally used in the proposed criteria documents. The linear nonthreshold concept has been endorsed by the four agencies in the Interagency Regulatory Liaison Group and **is** less likely to underestimate risk at the low doses typical of environmental exposure than other models that could be used. Because of the uncertainties associated with dose response, animal-to-human extrapolation, and other unknown factors; because of the use of average consumptions; and because of the serious public health consequences that could result if risks were underestimated, EPA believes that it is prudent to use conservative methods to

estimate risk in the water quality criteria program. The linearized multistage model is more systematic and invokes fewer arbitrary assumptions than the "one-hit" procedure previously used.

It should be noted that extrapolation models provide estimates of risk since a variety of assumptions are built into any model. Models using widely different assumptions may produce estimates ranging over several orders of magnitude. Since there is at present no way to demonstrate the scientific validity of any model, the use of risk extrapolation models is a subject of debate in the scientific community. However, risk extrapolation is generally recognized as the only tool available at this time for estimating the magnitude of health hazards associated with nonthreshold toxicants and has been endorsed by numerous Federal agencies and scientific organizations, including EPA's Carcinogen Assessment Group, the National Academy of Sciences, and the Interagency Regulatory Liaison Group, as a useful means of assessing the risks of exposure to various carcinogenic pollutants.

Noncarcinogens

Health criteria based on toxic effects of pollutants other than carcinogenicity are estimates of concentrations which are not expected to produce adverse effects in humans. They are based upon Acceptable Daily Intake (ADI) levels and are generally derived using no-observed-adverse-effect-level data from animal studies although human data are used wherever available. The ADI is calculated using safety factors to account for uncertainties

inherent in extrapolation from animal to man. In accordance with the National Research Council recommendations (Drinking Water and Health, National Academy of Sciences, National Research Council, 1977), safety factors of 10, 100, or 1,000 are used, depending on the quality and quantity of data. In some instances extrapolations are made from inhalation studies or limits to approximate a human response from ingestion using the Stokinger-Woodward model (Journal of American Water Works Association, 1958). Calculations of criteria from ADIs are made using the standard exposure assumptions (2 liters of water, 6.5 grams of edible aquatic products, and an average body weight of 70 kg).

APPENDIX C

THE PHILOSOPHY OF THE 1976 WATER QUALITY CRITERIA

Water quality criteria specify concentrations of water constituents which, if not exceeded, are expected to support an organic ecosystem suitable for the higher uses of water. Such criteria are derived from scientific facts obtained from experimental or in situ observations that depict organic 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 strategy 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 and 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 1/2 or 1/10th of some effect levels constitutes 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, their availability to the researcher, and 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 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 manmade 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 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 use water quality criteria involve 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 to virtually 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 affect 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 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 either have 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 waterskiing, and permitting an appropriate factor of safety to ensure protection for essential warm-or coldwater 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 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.

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QUALITY CRITERIA FOR WATER 1986

UPDATE #1

*ALDRIN-DIELDRIN

CRITERIA:

Aquatic Life

Dieldrin

For dieldrin the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0019 ug/L as **a** 24-hour average, and the concentration should not exceed **2.5** ug/L at any time.

For dieldrin the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0019 ug/L as a 24-hour average, and the concentration should not exceed 0.71 ug/L at any time.

Aldrin

For freshwater aquatic life the concentration of aldrin should not exceed 3.0 ug/L at any time. No data are available concerning the chronic toxicity of aldrin to sensitive freshwater aquatic life.

For saltwater aquatic life the concentration of aldrin should not exceed 1.3 ug/L at any time. No data are available concerning the chronic toxicity of aldrin to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to aldrin through ingestion of contaminated water and contaminated aquatic organisms, the
*Indicates suspended, canceled or restricted by **U.S. EPA** office
of Pesticides and Toxic Substances

ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} and 10^{-7} . The corresponding recommended criteria are 0.74 ng/L, 0.074 ng/L, and 0.0074 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 0.79 ng/L, 0.079 ng/L, and 0.0079 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to dieldrin through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} and 10^{-7} . The corresponding recommended criteria are 0.71 ng/L, 0.071 ng/L, and 0.0071 ng/L, respectively. If these above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 0.76 ng/L, 0.076 ng/L, and 0.0076 ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

AMMONIA

SUMMARY:

All concentrations used herein are expressed as un-ionized ammonia (NH_3), because NH_3 , not the ammonium ion (NH_4^+) has been demonstrated to be the principal toxic form of ammonia. The data used in deriving criteria are predominantly from flow through tests in which ammonia concentrations were measured. Ammonia was reported to be acutely toxic to freshwater organisms at concentrations (uncorrected for pH) ranging from 0.53 to 22.8 mg/L NH_3 for 19 invertebrate species representing 14 families and 16 genera and from 0.083 to 4.60 mg/L NH_3 for 29 fish species from 9 families and 18 genera. Among fish species, reported 96-hour LC50 ranged from 0.083 to 1.09 mg/L for salmonids and from 0.14 to 4.60 mg/L NH_3 for nonsalmonids. Reported data from chronic tests on ammonia with two freshwater invertebrate species, both daphnids, showed effects at concentrations (uncorrected for pH) ranging from 0.304 to 1.2 mg/L NH_3 , and with nine freshwater fish species, from five families and seven genera, ranging from 0.0017 to 0.612 mg/L NH_3 .

Concentrations of ammonia acutely toxic to fishes may cause loss of equilibrium, hyperexcitability, increased breathing, cardiac output and oxygen uptake, and, in extreme cases, convulsions, coma, and death. At lower concentrations ammonia has many effects on fishes, including a reduction in hatching success, reduction in growth rate and morphological development, and pathologic changes in tissues of gills, livers, and kidneys.

Several factors have been shown to modify acute NH_3 toxicity in fresh water. Some factors alter the concentration of un-ionized ammonia in the water by affecting the aqueous ammonia equilibrium, and some factors affect the toxicity of un-ionized ammonia itself, either ameliorating or exacerbating the effects of ammonia. Factors that have been shown to affect ammonia toxicity include dissolved oxygen concentration, temperature, pH, previous acclimation to ammonia, fluctuating or intermittent exposures, carbon dioxide concentration, salinity, and the presence of other toxicants.

The most well-studied of these is pH; the acute toxicity of NH_3 has been shown to increase as pH decreases. Sufficient data exist from toxicity tests conducted at different pH values to formulate a mathematical expression to describe pH-dependent acute NH_3 toxicity. The very limited amount of data regarding effects of pH on chronic NH_3 toxicity also indicates increasing NH_3 toxicity with decreasing pH, but the data are insufficient to derive a broadly applicable toxicity/pH relationship. Data on temperature effects on acute NH_3 toxicity are limited and somewhat variable, but indications are that NH_3 toxicity to fish is greater as temperature decreases. There is no information available regarding temperature effects on chronic NH_3 toxicity.

Examination of pH and temperature-corrected acute NH_3 toxicity values among species and genera of freshwater organisms showed that invertebrates are generally more tolerant than fishes, a notable exception being the fingernail clam. There is no clear trend among groups of fish; the several most sensitive

tested species and genera include representatives from diverse families (Salmonidae, Cyprinidae, Percidae, and Centrarchidae). Available chronic toxicity data for freshwater organisms also indicate invertebrates (cladocerans, one insect species) to be more tolerant than fishes, again with the exception of the fingernail clam. When corrected for the presumed effects of temperature and pH, there is also no clear trend among groups of fish for chronic toxicity values, the most sensitive species including representatives from five families (Salmonidae, Cyprinidae, Ictaluridae, Centrarchidae, and Catostomidae) and having chronic values ranging by not much more than a factor of two. The range of acute-chronic ratios for 10 species from 6 families was 3 to 43, and acute-chronic ratios were higher for the species having chronic tolerance below the median. Available data indicate that differences in sensitivities between warm and coldwater families of aquatic organisms are inadequate to warrant discrimination in the national ammonia criterion between bodies of water with "warm" and "coldwater" fishes: rather, effects of organism sensitivities on the criterion are most appropriately handled by site-specific criteria derivation procedures.

Data for concentrations of NH_3 toxic to freshwater phytoplankton and vascular plants, although limited, indicate that freshwater plant species are appreciably more tolerant to NH_3 than are invertebrates or fishes. The ammonia criterion appropriate for the protection of aquatic animals will therefore in all likelihood be sufficiently protective of plant life.

Available acute and chronic data for ammonia with saltwater organisms are very limited, and insufficient to derive a saltwater criterion. A few saltwater invertebrate species have been tested, and the prawn Macrobrachium rosenbergii was the most sensitive. The few saltwater fishes tested suggest greater sensitivity than freshwater fishes. Acute toxicity of NH_3 appears to be greater at low pH values, similar to findings in freshwater. Data for saltwater plant species are limited to diatoms, which appear to be more sensitive than the saltwater invertebrates for which data are available.

More quantitative information needs to be published on the toxicity of ammonia to aquatic life. Several key research needs must be addressed to provide a more complete assessment of ammonia toxicity. These are: (1) acute tests with additional saltwater fish species and saltwater invertebrate species; (2) life-cycle and early life-stage tests with representative freshwater and saltwater organisms from different families, with particular attention to trends of acute-chronic ratios; (3) fluctuating and intermittent exposure tests with a variety of species and exposure patterns; (4) more complete tests of the individual and combined effects of pH and temperature, especially for chronic toxicity; (5) more histopathological and histochemical research with fishes, which would provide a rapid means of identifying and quantifying sublethal ammonia effects; and (6) studies on effects of dissolved and suspended solids on acute and chronic toxicity.

NATIONAL CRITERIA:

The procedures described in the Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if:

(1) the 1-hour* average concentration of un-ionized ammonia (in mg/L NH_3) does not exceed, more often than once every 3 years on the average, the numerical value given by $0.52/\text{FT}/\text{FPH}/2$,

where:

$$\text{FT} = 10^{0.03 (20 - \text{TCAP})}; \text{TCAP} \leq T \leq 30$$

$$10^{0.03 (20 - T)}; 0 \leq T \leq \text{TCAP}$$

$$\text{FPH} = 1 \quad ; \quad 8 < \text{pH} < 9$$

$$\frac{1 + 10^{7.4 - \text{pH}}}{1.25} \quad ; \quad 6.5 \leq \text{pH} \leq 7.7$$

$\text{TCAP} = 20 \text{ }^\circ\text{C}$; Salmonids or other sensitive coldwater species present

$= 25 \text{ }^\circ\text{C}$; Salmonids and other sensitive coldwater species absent

(*An averaging period of 1 hour may not be appropriate if excursions of concentrations to greater than 1.5 times the average occur during the hour; in such cases, a shorter averaging period may be needed.)

(2) the 4-day average concentration of un-ionized ammonia (in mg/L NH_3) does not exceed, more often than once every 3 years on the average, the average* numerical value given by $0.80/\text{FT}/\text{FPH}/\text{RATIO}$, where FT and FPH are as above and:

$$\text{RATIO} = 16 \quad ; 7.7 \leq \text{pH} \leq 9$$

$$= 24 \frac{10^{7.7-\text{pH}}}{1+10^{7.4-\text{pH}}} \quad ; 6.5 \leq \text{pH} \leq 7.7$$

TCAP = 15 C; Salmonids or other sensitive coldwater species present

= 20 C; Salmonids and other sensitive coldwater species absent

(*Because these formulas are nonlinear in pH and temperature, the criterion should be the average of separate evaluations of the formulas reflective of the fluctuations of flow, pH, and temperature within the averaging period; it is not appropriate in general to simply apply the formula to average pH, temperature, and flow.)

The extremes for temperature (0, 30) and pH (6.5, 9) given in the above formulas are absolute. It is not permissible with current data to conduct any extrapolations beyond these limits. In particular, there is reason to believe that appropriate criteria at pH > 9 will be lower than the plateau between pH 8 and 9 given above.

Criteria concentrations for the pH range 6.5 to 9.0 and the temperature range 0 C to 30 C are provided in the following tables. Total ammonia concentrations equivalent to each unionized ammonia concentration are **also** provided in these tables. There are limited data on the effect of temperature on chronic toxicity. EPA will be conducting additional research on the effects of temperature on ammonia toxicity in order to fill perceived data gaps. Because of this uncertainty, additional site-specific information should be developed before these

criteria are used in wasteload allocation modeling. For example, the chronic criteria tabulated for sites lacking salmonids are less certain at temperatures much below 20 C than those tabulated at temperatures near 20 C. Where the treatment levels needed to meet these criteria below 20 C may be substantial, use of site-specific criteria is strongly suggested. Development of such criteria should be based upon site-specific toxicity tests.

Data available for saltwater species are insufficient to derive a criterion for saltwater.

The recommended exceedence frequency of 3 years is the Agency's best scientific judgment of the average amount of time it will take an unstressed system to recover from a pollution event in which exposure to ammonia exceeds the criterion. A stressed system, for example, one in which several outfalls occur in a limited area, would be expected to require more time for recovery. The resilience of ecosystems and their ability to recover differ greatly, however, and site-specific criteria may be established if adequate justification is provided.

The use of criteria in designing waste treatment facilities requires the selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other factors may make their use impractical, in which case one should rely on a steady-state model. The Agency recommends the interim use of 1Q5 or 1Q10 for Criterion Maximum Concentration design flow and 7Q5 or 7Q10 for the Criterion Continuous Concentration design flow in steady-state models for unstressed and stressed systems respectively.

12) 4-day average concentrations for ammonia.*

pH	0 C	5 C	10 C	15 C	20 C	25 C	30 C
A. Salmonids or Other Sensitive Coldwater Species Present							
Un-ionized Ammonia (mg/liter NH ₃)							
6.50	0.0007	0.0009	0.0013	0.0019	0.0019	0.0019	0.0019
6.75	0.0012	0.0017	0.0023	0.0033	0.0033	0.0033	0.0033
7.00	0.0021	0.0029	0.0042	0.0059	0.0059	0.0059	0.0059
7.25	0.0037	0.0052	0.0074	0.0105	0.0105	0.0105	0.0105
7.50	0.0066	0.0093	0.0132	0.0186	0.0186	0.0186	0.0186
7.75	0.0105	0.0153	0.022	0.031	0.031	0.031	0.031
8.00	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.25	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.50	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
8.75	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
9.00	0.0126	0.0177	0.025	0.035	0.035	0.035	0.035
Total Ammonia (mg/liter NH ₃)							
6.50	2.5	2.4	2.2	2.2	1.49	1.04	0.73
6.75	2.5	2.4	2.2	2.2	1.49	1.04	0.73
7.00	2.5	2.4	2.2	2.2	1.49	1.04	0.74
7.25	2.5	2.4	2.2	2.2	1.50	1.04	0.74
7.50	2.5	2.4	2.2	2.2	1.50	1.05	0.74
7.75	2.3	2.2	2.1	2.0	1.40	0.99	0.71
8.00	1.53	1.44	1.37	1.33	0.93	0.66	0.47
8.25	0.87	0.82	0.70	0.76	0.54	0.39	0.28
8.50	0.49	0.47	0.45	0.44	0.32	0.23	0.17
8.75	0.28	0.27	0.26	0.27	0.19	0.15	0.11
9.00	0.16	0.16	0.16	0.16	0.13	0.10	0.08
B. Salmonids and Other Sensitive Coldwater Species Absent†							
Un-ionized Ammonia (mg/liter NH ₃)							
6.50	0.0007	0.0009	0.0013	0.0019	0.0026	0.0026	0.0026
6.75	0.0012	0.0017	0.0023	0.0033	0.0047	0.0047	0.0047
7.00	0.0021	0.0029	0.0042	0.0059	0.0083	0.0083	0.0083
7.25	0.0037	0.0052	0.0074	0.0105	0.0148	0.0148	0.0148
7.50	0.0066	0.0093	0.0132	0.0186	0.026	0.026	0.026
7.75	0.0105	0.0153	0.022	0.031	0.043	0.043	0.043
8.00	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.25	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.50	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
8.75	0.0120	0.0177	0.025	0.035	0.050	0.050	0.050
9.00	0.0126	0.0177	0.025	0.035	0.050	0.050	0.050
Total Ammonia (mg/liter NH ₃)							
6.50	2.5	2.4	2.2	2.2	2	1.46	1.03
6.75	2.5	2.4	2.2	2.2	2.1	1.47	1.04
7.00	2.5	2.4	2.2	2.2	2.1	1.47	1.04
7.25	2.5	2.4	2.2	2.2	2.1	1.48	1.05
7.50	2.5	2.4	2.2	2.2	2	1.49	1.06
7.75	2.3	2.2	2.1	2.0	1.98	1.39	1.00
8.00	1.53	1.44	1.37	1.33	1.31	0.93	0.67
8.25	0.87	0.82	0.78	0.76	0.76	0.54	0.40
8.50	0.49	0.47	0.45	0.44	0.45	0.33	0.25
8.75	0.28	0.27	0.26	0.27	0.27	0.21	0.16
9.00	0.16	0.16	0.16	0.16	0.17	0.14	0.11

* To convert these values to mg/liter N, multiply by 0.822.

† Site-specific criteria development is strongly suggested at temperatures above 20 C because of the limited data available to generate the criteria recommendation, and at temperatures below 20 C because of the limited data and because small changes in the criteria may have significant impact on the level of treatment required in meeting the recommended criteria.

11) One-hour average concentrations for ammonia.*

pH	0 C	5 C	10 C	15 C	20 C	25 C	30 C
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A. Salmonids or Other Sensitive Coldwater Species Present

Un-ionized Ammonia (mg/liter NH_3)

6.50	0.0091	0.0129	0.0182	0.026	0.036	0.036	0.036
6.75	0.0149	0.021	0.030	0.042	0.059	0.059	0.059
7.00	0.023	0.033	0.046	0.066	0.093	0.093	0.093
7.25	0.034	0.048	0.068	0.095	0.135	0.135	0.135
7.50	0.043	0.064	0.091	0.128	0.181	0.181	0.181
7.75	0.056	0.080	0.113	0.159	0.22	0.22	0.22
8.00	0.065	0.092	0.130	0.184	0.26	0.26	0.36
8.25	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.50	0.065	0.092	0.130	0.184	0.26	0.26	0.26
8.75	0.065	0.092	0.130	0.184	0.26	0.26	0.26
9.00	0.065	0.092	0.130	0.184	0.36	0.26	0.26

Total Ammonia (mg/liter NH_3)

6.50	35	33	31	30	29	20	14.3
6.75	32	30	28	27	27	18.6	13.2
7.00	28	26	25	24	23	16.4	11.6
7.25	23	22	20	19.7	19.2	13.4	9.5
7.50	17.4	16.3	15.5	14.9	14.6	10.2	7.3
7.75	12.2	11.4	10.9	10.5	10.3	7.2	5.2
8.00	8.0	7.5	7.1	6.9	6.8	4.8	3.5
8.25	4.5	4.2	4.1	4.0	3.9	2.8	2.1
8.50	2.6	2.4	2.3	2.3	2.3	1.71	1.28
8.75	1.47	1.40	1.37	1.38	1.42	1.07	0.83
9.00	0.86	0.83	0.83	0.86	0.91	0.72	0.58

B. Salmonids and Other Sensitive Coldwater Species Absent

Un-ionized Ammonia (mg/liter NH_3)

6.50	0.0091	0.0129	0.0182	0.026	0.036	0.051	0.051
6.75	0.0149	0.021	0.030	0.042	0.059	0.084	0.084
7.00	0.023	0.033	0.046	0.066	0.093	0.131	0.131
7.25	0.034	0.048	0.068	0.095	0.135	0.190	0.190
7.50	0.043	0.064	0.091	0.128	0.181	0.26	0.26
7.75	0.056	0.080	0.113	0.159	0.22	0.32	0.32
8.00	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.25	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.50	0.065	0.092	0.130	0.184	0.26	0.37	0.37
8.75	0.065	0.092	0.130	0.184	0.26	0.37	0.37
9.00	0.065	0.092	0.130	0.184	0.26	0.37	0.37

Total Ammonia (mg/liter NH_3)

6.50	35	33	31	30	29	29	20
6.75	32	30	28	27	27	26	18.6
7.00	28	26	25	24	23	23	16.4
7.25	23	22	20	19.7	19.2	19.0	13.5
7.50	17.4	16.3	15.5	14.9	14.6	14.5	10.3
7.75	12.2	11.4	10.9	10.5	10.3	10.2	7.3
8.00	8.0	7.5	7.1	6.9	6.8	6.8	4.9
8.25	4.5	4.2	4.1	4.0	3.9	4.0	2.9
8.50	2.6	2.4	2.3	2.3	2.3	2.4	1.81
8.75	1.47	1.40	1.37	1.38	1.42	1.52	1.18
9.00	0.86	0.83	0.83	0.86	0.91	1.01	0.82

* To convert these values to mg/liter N , multiply by 0.822.

The Agency acknowledges that the Criterion Continuous Concentration stream flow averaging period used for steady-state wasteload allocation modeling may be as long as 30 days in situations involving POTWs designed to remove ammonia where limited variability of effluent pollutant concentration and resultant concentrations in receiving waters can be demonstrated. In cases where low variability can be demonstrated, longer averaging periods for the ammonia Criterion Continuous Concentration (e.g., 30-day averaging periods) would be acceptable because the magnitude and duration of exceedences above the Criterion Continuous Concentration would be sufficiently limited. These matters are discussed in more detail in the Technical Support Document for Water Quality-Based Toxics Control (U.S. EPA, 1985a).

(50 F.R. 30784, July 29, 1985)
SEE APPENDIX A FOR METHODOLOGY

BERYLLIUM

CRITERIA:

Aquatic Life

The available data for beryllium indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 130 and 5.3 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Hardness has a substantial effect on acute toxicity.

The limited saltwater data base available for beryllium does not permit any statement concerning acute or chronic toxicity.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to beryllium through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 68 ng/L, 6.8 ng/L, and 0.68 ng/L, respectively. If these estimates are made for

consumption of aquatic organisms only, excluding consumption of water, the levels are 1170 ng/L, 117.0 ng/L, and 11.71 ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

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 seawater 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 ug/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 ug/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.

(QUALITY CRITERIA ~~FOR~~ WATER, JULY 1976) PB-263943
SEE APPENDIX C ~~FOR~~ METHODOLOGY

CHLORINATED BENZENES

CRITERIA

Aquatic Life

The available data for chlorinated benzenes indicate that acute toxicity to fresh water aquatic life at concentrations as low as 250 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of the more toxic of the chlorinated benzenes to sensitive freshwater aquatic life but toxicity occurs at concentrations as low as 50 ug/L for a fish species exposed for 7.5 days.

The available data for chlorinated benzenes indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 160 and 129 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

Human Health

For comparison purposes, two approaches were used to derive criterion levels for monochlorobenzene. Based on available toxicity data, for the protection of public health, the derived level is 488 ug/L. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 20 ug/L. It should be recognized that organoleptic data as a basis for establishing a water quality criteria have limitations and have no demonstrated relationship to potential adverse human health effects.

Trichlorobenzenes

Due to the insufficiency in the available information for the trichlorobenzenes, a criterion cannot be derived at this time using the present guidelines.

1,2,4,5-Tetrachlorobenzene

For the protection of human health from the toxic properties of 1,2,4,5-tetrachlorobenzene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 38 ug/L.

For the protection of human health from the toxic properties of 1,2,4,5-tetrachlorobenzene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 48 ug/L.

Pentachlorobenzene

For the protection of human health from the toxic properties of pentachlorobenzene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 74 ug/L.

For the protection of human health from the toxic properties of pentachlorobenzene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 85 ug/L.

Hexachlorobenzene

For the maximum protection of human health from the potential carcinogenic effects due to exposure of hexachlorobenzene through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero based on the non-threshold assumption for this chemical. However, zero

level may not be attainable at the present time. Therefor, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 7.2 ng/L, 0.72 ng/L, and 0.072 ng/L, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 7.4 ng/L, 0.74 ng/L and 0.074. ng/L respectively.

(45 F.R. 79316, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

DICHLOROPROPANES/DICHLOROPROPENES

CRITERIA:

Aquatic Life

The available data for dichloropropanes indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 23,000 and 5,700 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropene indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 6,060 and 244 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropane indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 10,300 and 3,040 ug/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested.

The available data for dichloropropene indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 790 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of dichloropropene to sensitive saltwater aquatic life.

Human Health

Using the present guidelines, a satisfactory criterion cannot be derived at this time because of insufficient available data for dichloropropanes.

For the protection of human health from the toxic properties of dichloropropenes ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 87 ug/L.

For the protection of human health from the toxic properties of dichloropropenes ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 14.1 mg/L.

(45 F.R. 79318, November **28**, 1980)
SEE APPENDIX B FOR METHODOLOGY

*ENDRIN

CRITERIA:

Aquatic Life

For endrin the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0023 $\mu\text{g/L}$ as a 24-hour average, and the concentration should not exceed 0.18 $\mu\text{g/L}$ at any time.

For endrin the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0023 $\mu\text{g/L}$ as a 24-hour average, and the concentration should not exceed 0.037 $\mu\text{g/L}$ at any time.

Human Health

The ambient water quality criterion for endrin is recommended to be identical to the existing water standard which is 1.0 $\mu\text{g/L}$. Analysis of the toxic effects data resulted in a calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 g of aquatic organisms was not derived.

*Indicates suspended, canceled or restricted by U.S. EPA Office of Pesticides and Toxic Substances

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

HEPTACHLOR

CRITERIA:

Aquatic Life

For heptachlor the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0038 ug/L **as** a 24-hour average, and the concentration should not exceed 0.52 ug/L at any time.

For heptachlor the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.0036 ug/L as a 24-hour average, and the concentration should not exceed 0.053 ug/L at any time.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to heptachlor through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero, based on the non threshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 2.78 ng/L, 0.28 ng/L, and 0.026 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 2.85 ng/L, 0.29 ng/L, and 0.029 ng/L, respectively.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

HEXACHLOROCYCLOHEXANE

CRITERIA :

Aquatic Life

Lindane

For lindane the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.080 ug/L **as a** 24-hour average and the concentration should not exceed **2.0** ug/L at any time.

For saltwater aquatic life the concentration of lindane should not exceed 0.16 ug/L at any time. No data are available concerning the chronic toxicity of lindane to sensitive saltwater aquatic life.

BHC

The available data for a mixture of isomers of **BHC** indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 100 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of a mixture of isomers of **BHC** to sensitive freshwater aquatic life.

The available data for a mixture of isomers of **BHC** indicate that acute toxicity to saltwater aquatic life occurs at concentrations as low as 0.34 ug/L and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of a mixture of isomers of **BHC** to sensitive saltwater aquatic life.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of exposure to alpha-hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-6} and 10^{-7} . The corresponding recommended criteria are 92 ng/L, 9.2 ng/L, and .92 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 310 ng/L, 31.0 ng/L, and 3.10 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to beta-hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 163 ng/L, 16.3 ng/L, and 1.63 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 547 ng/L, 54.7 ng/L, and 5.47 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects due to exposure of gamma-hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 186 ng/L, 18.6 ng/L, and 1.86 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 625 ng/L, 62.5 ng/L, and 6.25 ng/L, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to technical-hexachlorocyclohexane through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentrations should be zero, based on the nonthreshold assumption for this chemical. However, zero level may not be attainable at the present time. Therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 123 ng/L, 12.3 ng/L, and 1.23 ng/L, respectively. If these estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 414 ng/L, 41.4 ng/L, and 4.14 ng/L, respectively.

Using the present guidelines, satisfactory criteria cannot be derived at this time for delta and epsilon hexachlorocyclohexane because of insufficient available data.

(45 F.R. 79318, November 28, 1980)
SEE APPENDIX B FOR METHODOLOGY

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 kg/ha, 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 2 bait particles each. From this report it is obvious that mirex is extremely toxic to these species of crayfish. Mortality and accumulation increase 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/ha) in 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 and 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. Nagvi 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/ha). 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 post-anal fin of the 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 seawater was lethal to juvenile pink shrimp, Penaeus duorarum, in a 3-week exposure (Lowe et al. 1971). In static tests with larval stages (megalopal) of the mud crab, Rhithropanopeus harrisi, reduced survival was observed in 0.1 ug/L mirex (Bookhout et al. 1972). In three of four 28-day seasonal flow-through experiments, Tagatz et al. (1975) found reduced survival of Callinectes sapidus, Penaeus duorarum, 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), stated 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.

(QUALITY CRITERIA FOR WATER, JULY 1976) PB-263943
SEE APPENDIX C FOR METHODOLOGY

NICKEL

CRITERIA:

Aquatic Life

For total recoverable nickel the criterion (in ug/L) to protect freshwater aquatic life as derived using the Guidelines is the numerical value given by $e^{(0.76[\ln(\text{hardness})]+1.06)}$ as a 24-hour average, and the concentration (in ug/L) should not exceed the numerical value given by $e^{(0.76[\ln(\text{hardness})]+4.02)}$ at any time. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 the criteria are 56, 96, and 160 ug/L, respectively, as 24-hour averages, and the concentrations should not exceed 1,100, 1,800, and 3,100 ug/L, respectively, at any time.

For total recoverable nickel the criterion to protect saltwater aquatic life as derived using the Guidelines is 7.1 ug/L as a 24-hour average, and the concentration should not exceed 140 ug/L at any time.

Human Health

For the protection of human health from the toxic properties of nickel ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 13.4 ug/L.

For the protection of human health from the toxic properties of nickel ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 100. ug/L.

2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN

CRITERIA:

Aquatic Life

Not enough data are available concerning the effects of 2,3,7,8-TCDD on aquatic life and its uses to allow derivation of national criteria. The available information indicates that acute values for some freshwater animal species are $>1.0 \text{ ug/L}$; some chronic values are $<0.01 \text{ ug/L}$; and the chronic value for rainbow trout is $<0.001 \text{ ug/L}$. Because exposures of some species of fishes to 0.01 ug/L for <6 days resulted in substantial mortality several weeks later, derivation of aquatic life criteria for 2,3,7,8-TCDD may require special consideration. Predicted bioconcentration factors (BCFs) for 2,3,7,8-TCDD range from 3,000 to 900,000, but the available measured BCFs range from 390 to 13,000. If the BCF is 5,000, concentrations $>0.00001 \text{ ug/L}$ should result in concentrations in edible freshwater and saltwater fish and shellfish that exceed levels identified in a U.S. FDA health advisory. If the BCF is $>5,000$ or if uptake in a field situation is greater than that in laboratory tests, the value of 0.00001 ug/L will be too high.

Human Health

For the maximum protection of human health from the potential carcinogenic effects of 2,3,7,8-TCDD exposure through ingestion of contaminated water and contaminated aquatic organisms, the ambient water concentration should be zero. This criterion is

based on the nonthreshold assumption for 2,3,7,8-TCDD. However, zero may not be an attainable level at this time. Therefore, the levels that may result in an increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6} , and 10^{-7} . The corresponding recommended criteria are 1.3×10^{-7} , 1.3×10^{-8} and 1.3×10^{-9} ug/L, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 1.4×10^{-7} , 1.4×10^{-8} and 1.4×10^{-9} ug/L, respectively. If these estimates are made for consumption of water only, the levels are 2.2×10^{-6} , 2.2×10^{-7} and 2.2×10^{-8} ug/L, respectively.

(49 F.R. 5831, February 15, 1984)
SEE APPENDIX B FOR METHODOLOGY

ALKALINITY

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

OFFICE OF WATER REGULATIONS AND STANDARDS

CRITERIA AND STANDARDS DIVISION

UPDATE #2

to

"QUALITY CRITERIA FOR WATER 1986"

May 1, 1987

This is the second update to the EPA document "Quality Criteria for Water 1986". Included in this package are criteria summaries for contaminants that were recently revised as well as a criteria summary for a new contaminant. Several hand corrections are also included.

<u>Revised</u>	<u>New</u>	<u>Hand corrections</u>
INDEX	CHLORPYRIFOS	AMMONIA
SUMMARY CHART		CYANIDE
NICKEL		CHLORINATED ETHANES
PARATHION		
PENTACHLOROPHENOL		
TOXAPHENE		
ZINC		

Directions:

- Replace sections that have been revised with new sections.
- Insert new section alphabetically.
- Make the identified hand corrections.

For additional information contact:

EPA's Criteria and Standards Division
at (202) 475-7315.

HAND CORRECTIONS

Ammonia - page 5, center page, third line of equation should be changed from

"FPH = 1 ; 8 < pH < 9"

to

"FPH = 1 ; 8 ≤ pH ≤ 9"

Cyanide - last page, include the following after the last line

"(45 F.R. 79318, Nov. 28, 1980) (50 F.R. 30784, July 29, 1985)"
"SEE APPENDIX A FOR METHODOLOGY"

Chlorinated ethanes - last page, change

"1.03 ug/l"

to

"1.03 g/L"

Water Quality Criteria Summary

Adapted. This chart is for general information; please use current documents or detailed information in "Quality Criteria for Water 1995" for regulatory purposes.

Chemical Name	CONCENTRATIONS IN $\mu\text{g/L}$						UNITS PER LITER			Date Received	Date Analyzed	
	Identify Pollutant	Carcinogen	Fresh Acute Effects	Fresh Chronic Effects	Marine Acute Effects	Marine Chronic Effects	Water and Fish Ingestion	Fish Con- sumption Only	Drinking Water M.C.L.			
ACETAPHENONE	Y	N	1,700	520	800	710	100 μg 0.0001 mg/L	700 μg 0.0007 mg/L		1980 FR 1980 FR 1980 FR	1	
ACETYLCHOLINE	Y	Y	1,700	7,400						1980 FR 1980 FR 1980 FR	1	
ALOPH	Y	Y	30		1.3		0.07 mg/L	0.07 mg/L		1980 FR 1978 FR 1980 FR	16	
ALUMINUM	N	N		30,000						1980 FR 1978 FR 1980 FR	24	
AMMONIA	N	N	CONCENTRATIONS ARE AND TEMPERATURE DEPENDENT—SEE DOCUMENT									
ANTHRAQUENONE	Y	N	1,000	1,000			10 μg 3.20 mg/L	40,000 μg 17 mg/L	0.06 mg/L	1980 FR 1980 FR 1980 FR	1	
ANTHRACENE (PENT)	Y	Y	100	100	12,500	113				1980 FR 1980 FR 1980 FR	21	
ANTHRACENE (TRIO)	Y	Y	100	100	80	80				1980 FR 1980 FR 1980 FR	21	
ASBESTOS	Y	Y					20 mg/L			1980 FR 1980 FR 1980 FR	21	
BACTERIA	N	N	FOR PRIMARY RECREATION AND SHELLFISH USES—SEE DOCUMENT						<1,000		1980 FR	20
BARIUM	N	N			5,100	100	1 mg 0.04 mg/L	10 mg/L 0.53 mg/L	3 mg/L	1978 FR 1980 FR 1980 FR	6	
BENZENE	Y	Y	1,300				0.12 mg/L	0.53 mg/L		1980 FR 1980 FR 1980 FR	9	
BENZYLALCOHOL	Y	Y	1,300	5.3			0.04 mg/L	117 mg/L		1980 FR 1980 FR 1980 FR	8	
BENZYLALCOHOL	Y	N	1,000		0.34					1980 FR 1980 FR 1980 FR	21	
CADMIUM	Y	N	2.0	1.7	43	0.3	10 μg		0.010 mg/L			
CARBON TETRACHLORIDE	Y	N	125,000		10,000		0.4 mg/L	0.84 mg/L		1980 FR 1980 FR 1980 FR	1	
CHLORALHYDRATE	Y	Y	2.4	0.0043	0.00	0.004	0.44 mg/L	3.44 mg/L		1980 FR 1980 FR 1980 FR	12	
CHLORINATED BENZENE	Y	Y	250	50	100	100				1980 FR 1980 FR 1980 FR	1	
CHLORINATED NAPHTHALENES	Y	N	1,000		7.5					1980 FR 1980 FR 1980 FR	1	
CHLORINE	N	N	10	11	13	7.5				1980 FR 1980 FR 1980 FR	20	
CHLOROALCOHOL, ETHYLENE	Y	N	1,000,000							1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,2-)	Y	Y					0.03 mg/L 0.14 mg/L	1.00 mg/L 0.74 mg/L		1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,4-)	Y	Y	100,000	11,200			0.03 mg/L 0.14 mg/L	1.00 mg/L 0.74 mg/L		1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,3-)	Y	Y	100,000	11,200			0.03 mg/L 0.14 mg/L	1.00 mg/L 0.74 mg/L		1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,2-)	Y	Y	100,000	11,200			0.03 mg/L 0.14 mg/L	1.00 mg/L 0.74 mg/L		1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,3-)	Y	Y	100,000	11,200			0.03 mg/L 0.14 mg/L	1.00 mg/L 0.74 mg/L		1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,4-)	Y	Y	100,000	11,200			0.03 mg/L 0.14 mg/L	1.00 mg/L 0.74 mg/L		1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,2-)	Y	Y	100							1980 FR 1980 FR 1980 FR	1	
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CHLOROBENZENE, ETHYLENE (1,3-)	Y	Y	100							1980 FR 1980 FR 1980 FR	1	
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CHLOROBENZENE, ETHYLENE (1,3-)	Y	Y	100							1980 FR 1980 FR 1980 FR	1	
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CHLOROBENZENE, ETHYLENE (1,3-)	Y	Y	100							1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,4-)	Y	Y	100							1980 FR 1980 FR 1980 FR	1	
CHLOROBENZENE, ETHYLENE (1,2-)	Y	Y										

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CHLORPYRIFOS

Summary

The acute values for eighteen freshwater species in fifteen genera range from 0.11 ug/L for an amphipod to greater than 806 ug/L for two fishes and a snail. The bluegill is the most acutely sensitive fish species with an acute value of 10 ug/L, but seven invertebrate genera are more sensitive. Smaller organisms seem to be more acutely sensitive than larger ones.

Chronic toxicity data are available for one freshwater species, the fathead minnow. Unacceptable effects occurred in second generation larvae at 0.12 ug/L, which was the lowest concentration tested. The resulting acute-chronic ratio was greater than 1,417.

Little information is available on the toxicity of chlorpyrifos to freshwater plants, although algal blooms frequently follow field applications of chlorpyrifos. The only available bioconcentration test on chlorpyrifos with a freshwater species was with the fathead minnow and resulted in a bioconcentration factor of 1,673.

The acute toxicity of chlorpyrifos has been determined for 15 species of saltwater animals in 12 genera with the acute values ranging from 0.01 ug/L for the Korean shrimp, Palaemon macrodactylus, to 1.911 ug/L for larvae of the eastern oyster, Crassostrea virginica. Arthropods are particularly sensitive to chlorpyrifos. Among the 10 species of fish tested, the 96-hr LC50s range from **0.58** ug/L for striped **bass** to **520** ug/L for **gulf**

toadfish. Fish larvae are more sensitive than other life stages. Growth of the mysid, Mysidopsis bahia, was reduced at 0.004 ug/L in a life-cycle test. In early life-stage tests, the California grunion, Leuresthes tenuis, was the most sensitive of the six fishes, with growth being reduced at 0.30 ug/L. Of the seven acute-chronic ratios that have been determined with saltwater species, the five lowest range from 1.374 to 12.50, whereas the highest is 228.5.

Concentrations of chlorpyrifos affecting six species of saltwater phytoplankton range from 138 to 10,000 ug/L. BCFs ranged from 100 to 5,100 when the gulf toadfish was exposed to concentrations increasing from 1.4 to 150 ug/L. Steady-state BCFs averaged from 100 to 757 for five fishes exposed in early life-stage tests.

National Criteria

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of chlorpyrifos does not exceed 0.041 ug/L more than once every **three years on the average and if the one-hour average** concentration does not exceed 0.083 ug/L more than once every three years on the average.

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of

Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of chlorpyrifos does not exceed 0.0056 ug/L more than once every three years on the average and if the one-hour average concentration does not exceed 0.011 ug/L more than once every three years on the average.

Three years is the Agency's best scientific judgment of the average amount of time aquatic ecosystems should be provided between excursions. The resiliences of ecosystems and their abilities to recover differ greatly, however, and site-specific allowed excursion frequencies may be established if adequate justification is provided.

Use of criteria for developing water quality-based permit limits and for designing waste treatment facilities requires selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other considerations might make their use impractical, in which case one must rely on a steady-state model.

(51 F.R. 43665, December 3, 1986)
SEE APPENDIX A FOR METHODOLOGY

NICKEL

Summary

Acute values with twenty-one freshwater species in **18** genera range from 1,101 ug/L for a cladoceran to 43,240 ug/L for a fish. Fishes and invertebrates are both spread throughout the range of sensitivity. Acute values with four species are significantly correlated with hardness. Data are available concerning the chronic toxicity of nickel to two invertebrates and two fishes in freshwater. Data available for two species indicate that chronic toxicity decreases as hardness increases. The measured chronic values ranged from 14.77 ug/L with Daphnia magna in soft water to 526.7 ug/L with the fathead minnow in hard water. Five acute-chronic ratios are available for two species in soft and hard water and range from 14 to 122.

Nickel appears to be quite toxic to freshwater algae, with concentrations as low as 50 ug/L producing significant inhibition. Bioconcentration factors for nickel range from 0.8 for fish muscle to 193 for a cladoceran. ,

Acute values for twenty-three saltwater species in twenty genera range from **151.7 ug/L** with juveniles of a mysid of to 1,100,000 ug/L with juveniles and adults of a clam. The acute values for the four species of fish range from **7,598** to 350.000 ug/L. The acute toxicity of nickel appears to be related to salinity, but the form of the relationship appears to be species-dependent.

Mysidopsis bahia is the only saltwater species with which an acceptable chronic test has been conducted on nickel. Chronic

exposure to 141 ug/L and greater resulted in reduced survival and reproduction. The measured acute-chronic ratio was 5.478.

Bioconcentration factors in saltwater range from 261.8 with a oyster to 675 with a brown alga.

National Criteria

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of nickel (in ug/L) does not exceed the numerical value given by $e^{(0.8460[\ln(\text{hardness})]+1.1645)}$ more than once every three years on the average and if the one-hour average concentration (in ug/L) does not exceed the numerical value given by $e^{(0.8460[\ln(\text{hardness})]+3.3612)}$ more than once every three years on the average. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 the four-day average concentrations of nickel are 88, 160, and 280 ug/L, respectively, and the one-hour average concentrations are 790, 1400, and 2500 ug/L.

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of nickel does not exceed 8.3 ug/L more than once every three years on the average and if the one-hour average concentration does not exceed

75 ug/L more than once every three years on the average.

"Acid-soluble" is probably the best measurement at present for expressing criteria for metals and the criteria for nickel were developed on this basis. However, at this time, no EPA approved method for such a measurement is available to implement criteria for metals through the regulatory programs of the Agency and the States. The Agency is considering development and approval of a method for a measurement such as "acid-soluble." Until one is approved, however, EPA recommends applying criteria for metals using the total recoverable method. This has two impacts: (1) certain species of some metals cannot be measured because the total recoverable method cannot distinguish between individual oxidation States, and (2) in some cases these criteria might be overly protective when based on the total recoverable method.

Three years is the Agency's best scientific judgment of the average amount of time aquatic ecosystems should be provided between excursions. The resiliences of ecosystems and their abilities to recover differ greatly, however, and site-specific allowed excursion frequencies may be established if adequate justification is provided.

Use of criteria for developing water quality-based permit limits and for designing waste treatment facilities requires selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other considerations might make their use impractical, in which case one must rely on a steady-state model.

(51 F.R. 43665, December 3, 1986)
SEE APPENDIX A FOR METHODOLOGY

PARATHION

Summary

The acute values for thirty-seven freshwater species in thirty-one genera range from 0.04 ug/L for an early instar of a crayfish, Orconectes nais, to 5,230 ug/L for two species of tubificid worms. For Daphnia magna, the chronic value and acute-chronic ratio are 0.0990 ug/L and 10.10 respectively. Chronic toxicity values are available for two freshwater fish species, the bluegill and the fathead minnow, with chronic values of 0.24 ug/L and 6.3 ug/L, and acute-chronic ratios of 2.121 and 79.45 respectively. Two freshwater algae were affected by toxaphene concentrations of 30 and 390 ug/L, respectively. Bioconcentration factors determined with three fish species ranged from 27 to 573.

The acute values that are available for saltwater species are 11.5 and 17.8 ug/L for the Korean shrimp, Palaeomon macrodactylus, and 17.8 ug/L for the striped bass, Morone saxatilis. No data are available concerning the chronic toxicity of parathion to saltwater species, toxicity to saltwater plants, or bioaccumulation by saltwater species. Some data indicate that parathion is acutely lethal to commercially important saltwater shrimp at concentrations as low as 0.24 ug/L. Measurement of acetylcholinesterase (AChE) in fish tissue might be useful for diagnosing fish kills caused by parathion.

National Criteria

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of

Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of parathion does not exceed 0.013 ug/L more than once every three years on the average and if the one-hour average concentration does not exceed 0.065 ug/L more than once every three years on the average.

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" require the availability of specified data for the derivation of a criterion. A saltwater criterion for parathion cannot be derived because very few of the required data are available.

Three years is the Agency's best scientific judgment of the average amount of time aquatic ecosystems should be provided between excursions. The resiliences of ecosystems and their abilities to recover differ greatly, however, and site-specific allowed excursion frequencies may be established if adequate justification is provided.

Use of criteria for developing water quality-based permit limits and for designing waste treatment facilities requires selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other considerations might make their use impractical, in which case one must rely on a steady-state model.

(51 F.R. 43665, December 3, 1986)
SEE APPENDIX A FOR METHODOLOGY

PENTACHLOROPHENOL (PCP)

Summary

The acute and chronic toxicity of PCP to freshwater animals increased as pH and dissolved oxygen concentration of the water decreased. Generally, toxicity also increased with increased temperature. The estimated acute sensitivities of 36 species at pH = 6.5 ranged from 4.355 ug/L for larval common carp to >43,920 ug/L for a crayfish. At pH = 6.5, the lowest and highest estimated chronic values of <1.835 and 79.66 ug/L, respectively, were obtained with different cladoceran species. Chronic toxicity to fish was affected by the presence of impurities, with industrial-grade PCP being more toxic than purified samples. Mean acute-chronic ratios for six freshwater species ranged from 0.8945 to >15.79, but the mean ratios for the four most acutely sensitive species only range 0.8945 to 5.035. Freshwater algae were affected by concentrations as low as 7.5 ug/L, whereas vascular plants were affected at 189 ug/L and above. Bioconcentration factors ranged from 7.3 to 1,066 for three species of fish.

Acute toxicity values from tests with 18 species of saltwater animals, representing 17 genera, range from 22.63 ug/L for late yolk-sac larvae of the Pacific herring, Clupea narenqus pallasi, to 18,000 ug/L for adult blue mussels, Mytilus edulis. The embryo and larval stages of invertebrates and the late larval premetamorphosis stage of fish appear to be the most sensitive life stages to PCP. With few exceptions, fish are more sensitive

than invertebrates to PCP. Salinity, temperature, and pH have a slight effect on the toxicity of PCP to some saltwater animals.

Life-cycle toxicity tests have been conducted with the sheepshead minnow, Cyprinodon variegatus, and the polychaete worm, Ophrvotrocha diadema. The chronic value for the minnow is 64.31 ug/L and the acute-chronic ratio is 6.873. Unfortunately, no statistical analysis of the data from the test with the worm is available.

The EC50s for saltwater plants range from 17.40 ug/L for the diatom, Skeletonema costatum, to 3.600 ug/L for the green alga, Dunaliella tertiolecta. Apparent steady-state BCFs are available for the eastern oyster, Crassostrea virginica, and two saltwater fishes and range from 10 to 82.

National Criteria

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration (in ug/L) of pentachlorophenol does not exceed the numerical value given by $e^{[1.005(\text{pH})-5.290]}$ more than once every three years on the average and if the one-hour average concentration (in ug/L) does not exceed the numerical value given by $e^{[1.005(\text{pH})-4.830]}$ more than once every three years on the average. For example, at pH = 6.5, 7.8, and 9.0 the four-day average concentrations of pentachlorophenol are 3.5, 13, and 43 ug/L, respectively, and the

one-hour average concentrations are 5.5, 20, and 68 ug/L. At pH = 6.8, a pentachlorophenol concentration of 1.74 ug/L caused a 50% reduction in the growth of yearling sockeye salmon in a 56-day test.

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of pentachlorophenol does not exceed 7.9 ug/L more than once every three years on the average and if the one-hour average concentration does not exceed 13 ug/L more than once every three years on the average.

Three years is the Agency's best scientific judgment of the average amount of time aquatic ecosystems should be provided between excursions. The resiliences of ecosystems and their abilities to recover differ greatly, however, and site-specific allowed excursion frequencies may be established if adequate justification is provided.

Use of criteria for developing water quality-based permit limits and for designing waste treatment facilities requires selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other considerations might make their use impractical, in which case one must rely on a steady-state model.

(51 F.R. 43665, December 3, 1986)
SEE APPENDIX A FOR METHODOLOGY

TOXAPHENE

Summary

The acute sensitivities of 36 freshwater species in 28 genera ranged from 0.8 ug/L to 500 ug/L. Such important fish species as the channel catfish, largemouth bass, chinook and coho salmon, brook, brown and rainbow trout, striped bass, and bluegill had acute sensitivities ranging from 0.8 ug/L to 10.8 ug/L. Chronic values for four freshwater species range from less than 0.039 ug/L for the brook trout to **0.1964** ug/L for the channel catfish. The growth of algae was affected at 100 to 1,000 ug/L, and bioconcentration factors from laboratory tests ranged from 3.100 to **90,000**. Concentrations in lake trout in the Great Lakes have frequently exceeded the U.S. FDA action level of 5 mg/kg, even though the concentrations in the water seem to be only 1 to **4** ng/L. These concentrations in lake water are thought to have resulted from toxaphene being transported to the Great Lakes from remote sites, the locations of which are not well known.

The acute toxicity of toxaphene to 15 species of saltwater animals ranges from 0.53 for pinfish, Lagodon rhomoides, to **460.000** ug/L for the adults of the clam, Rangia cuneata. Except for resistant species tested at concentrations greater than toxaphene's water solubility, acute values for most species were within a factor of 10. The toxicity of toxaphene was found to decrease slightly with increasing salinity for adult blue crabs, Callinectes sapidus, whereas no relationship between toxicity and salinity was observed with the three spine stickleback,

Gasterosteus aculeatus. Temperature significantly affected the toxicity of toxaphene to blue crabs.

Early life-stage toxicity tests have been conducted with the sheepshead minnow, Cyprinodon variegatus, and the longnose killifish, Fundulus similis, whereas life-cycle tests have been conducted with the sheepshead minnow and a mysid. For the sheepshead minnow, chronic values of 1.658 ug/L from the early life-stage test and 0.7141 ug/L from the life-cycle toxicity test are similar to the 96-hr LC50 of 1.1 ug/L. Killifish are more chronically sensitive with effects noted at 0.3 ug/L. In the life-cycle test with the mysid, no adverse effects were observed at the highest concentration tested, which was only slightly below the 96-hr LC50, resulting in an acute-chronic ratio of 1.132.

Toxaphene is bioconcentrated by an oyster, Crassostrea virginica, and two fishes, C. variegatus and F. similis, to concentrations that range from 9,380 to 70.140 times that in the test solution.

National Criteria

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of toxaphene does not exceed 0.0002 ug/L more than once every three years on the average and if the one-hour average concentration does not

exceed 0.73 ug/L more than once every three years on the average. If the concentration of toxaphene does exceed 0.0002 ug/L, the edible portions of consumed species should be analyzed to determine whether the concentration of toxaphene exceeds the **FDA** action level of 5 mg/kg. If the channel catfish is as acutely sensitive as some data indicate it might be, it will not be protected by this criterion.

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of toxaphene does not exceed 0.0002 ug/L more than once every three years on the average and if the one-hour average concentration does not exceed 0.21 ug/L more than once every three years on the average. If the concentration of toxaphene does exceed 0.0002 ug/L, the edible portions of consumed species should be analyzed to determine whether the concentration of toxaphene exceeds the **FDA** action level of 5 mg/kg.

Three years is the Agency's best scientific judgment of the average amount of time aquatic ecosystems should be provided between excursions. The resiliences of ecosystems and their abilities to recover differ greatly, however, and site-specific allowed excursion frequencies may be established if adequate justification is provided.

Use of criteria for developing water quality-based permit limits and for designing waste treatment facilities requires

selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other considerations might make their use impractical, in which case one must rely on a steady-state model.

(51 F.R. 43665, December 3, 1986)
SEE APPENDIX A FOR METHODOLOGY

ZINC

Summary

Acute toxicity values are available for 43 species of freshwater animals and data for eight species indicate that acute toxicity decreases as hardness increases. When adjusted to a hardness of 50 mg/L, sensitivities range from 50.70 ug/L for Ceriodaphnia reticulata to 88,960 ug/L for a damselfly. Additional data indicate that toxicity increases as temperature increases. Chronic toxicity data are available for nine freshwater species. Chronic values for two invertebrates ranged from 46.73 ug/L for Daphnia magna to >5,243 ug/L for the caddisfly, Clistoronla magnificia. Chronic values for seven fish species ranged from 36.41 ug/L for the flagfish, Jordanelia floridae, to 854.7 ug/L for the brook trout, Salvelinus fontinalis. Acute-chronic ratios ranged from 0.2614 to 41.20. but the ratios for the sensitive species were all less than 7.3.

The sensitivity range of freshwater plants to zinc is greater than that for animals. Growth of the alga, Selenastrum capricornutum, was inhibited by 30 ug/L. On the other hand, with several other species of green algae, 4-day EC50s exceeded 200,000 ug/L. Zinc **was** found to bioaccumulate in freshwater animal tissues from 51 to 1,130 times the concentration present in the water.

Acceptable acute toxicity values for zinc are available for 33 species of saltwater animals including 26 invertebrates and 7 fish. LC50s range from 191.5 ug/L for cabezon, Scorpanichthys

marmoratus to 320.400 ug/L for adults of another clam, Macoma balthica. Early life stages of saltwater invertebrates and fishes are generally more sensitive to zinc than juveniles and adults. Temperature has variable and inconsistent effects on the sensitivity of saltwater invertebrates to zinc. The sensitivity of saltwater vertebrate animals to zinc decreases with increasing salinity, but the magnitude of the effect is species-specific.

A life-cycle test with the mysid, Mysidopsis bahia, found unacceptable effects at 120 ug/L, but not at 231 ug/L, and the acute-chronic ratio was 2.997. Seven species of saltwater plants were affected at concentrations ranging from 19 to 10,100 ug/L. Bioaccumulation data for zinc are available for seven species of saltwater algae and five species of saltwater animals. Steady-state zinc bioconcentration factors for the twelve species range from 3.692 to 23.820.

National Criteria

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of zinc (in ug/L) does not exceed the numerical value given by $e^{(0.8473[\ln(\text{hardness})]+0.7614)}$ more than once every three years on the average and if the one-hour average concentration (in ug/L) does not exceed the numerical value given by $e^{(0.8473[\ln(\text{hardness})]+0.8604)}$ more than once every three years on the

average. For example, at hardnesses of 50, 100, and 200 mg/L as CaCO_3 , the four-day average concentrations of zinc are 59, 110 and 190 ug/L, respectively, and the one-hour average concentrations are 65, 120, and 210 ug/L. If the striped bass is as sensitive as some data indicate, it will not be protected by this criterion.

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate, that except possibly where a locally important species is very sensitive, saltwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of zinc does not exceed 86 ug/L more than once every three years on the average and if the one-hour average concentration does not exceed 95 ug/L more than once every three years on the average.

"Acid-soluble" is probably the best measurement at present for expressing criteria for metals and the criteria for zinc were developed on this basis. However, at this time no EPA approved method for such a measurement is available to implement criteria for metals through the regulatory programs of the Agency and the States. The Agency is considering development and approval of a method for a measurement such as "acid-soluble." Until one is approved, however, EPA recommends applying criteria for metals using the total recoverable method. This has two impacts: (1) certain species of some metals cannot be measured because the total recoverable method cannot distinguish between individual oxidation States, and (2) in some cases these criteria might be overly protective when based on the total recoverable method.

Three years is the Agency's best scientific judgment of the average amount of time aquatic ecosystems should be provided between excursions. The resiliences of ecosystems and their abilities to recover differ greatly, however, and site-specific allowed excursion frequencies may be established if adequate justification is provided.

Use of criteria for developing water quality-based permit limits and for designing waste treatment facilities requires selection of an appropriate wasteload allocation model. Dynamic models are preferred for the application of these criteria. Limited data or other considerations might make their use impractical, in which case one must rely on a steady-state model.

(52 F.R. 6213, March 2, 1987)
SEE APPENDIX A FOR METHODOLOGY



United States
Environmental Protection
Agency

Office of Water
Office of Science and Technology
(4304T) 2006

National Recommended Water Quality Criteria

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

	Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/Source
			CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
1	Antimony	7440360					5.6 B	640 B	65FR66443
2	Arsenic	7440382	340 A,D,K	150 A,D,K	69 A,D,bb	36 A,D,bb	0.018 C,M,S	0.14 C,M,S	65FR31682 57FR60848
3	Beryllium	7440417					Z		65FR31682
4	Cadmium	7440439	2.0 D,E,K,bb	0.25 D,E,K,bb	40 D,bb	8.8 D,bb	Z		EPA-822-R-01-001 65FR31682
5a	Chromium (III)	16065831	570 D,E,K	74 D,E,K			Z Total		EPA820/B-96-001 65FR31682
5b	Chromium (VI)	18540299	16 D,K	11 D,K	1,100 D,bb	50 D,bb	Z Total		65FR31682
6	Copper	7440508	13 D,E,K,cc	9.0 D,E,K,cc	4.8 D,cc,ff	3.1 D,cc,ff	1,300 U		65FR31682
7	Lead	7439921	65 D,E,bb,gg	2.5 D,E,bb,gg	210 D,bb	8.1 D,bb			65FR31682
8a	Mercury	7439976	1.4 D,K,hh	0.77 D,K,hh	1.8 D,ee,hh	0.94 D,ee,hh		0.3 mg/kg J	62FR42160
8b	Methylmercury	22967926							EPA823-R-01-001
9	Nickel	7440020	470 D,E,K	52 D,E,K	74 D,bb	8.2 D,bb	610 B	4,600 B	65FR31682
10	Selenium	7782492	L,R,T	5.0 T	290 D,bb,dd	71 D,bb,dd	170 Z	4200	62FR42160 65FR31682 65FR66443
11	Silver	7440224	3.2 D,E,G		1.9 D,G				65FR31682

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

	Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
			CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
12	Thallium	7440280					0.24	0.47	68FR75510
13	Zinc	7440666	120 D,E,K	120 D,E,K	90 D,bb	81 D,bb	7,400 U	26,000 U	65FR31682 65FR66443
14	Cyanide	57125	22 K,Q	5.2 K,Q	1 Q,bb	1 Q,bb	140 jj	140 jj	EPA820/B-96-001 57FR60848 68FR75510
15	Asbestos	1332214					7 million fibers/L I		57FR60848
16	2,3,7,8-TCDD (Dioxin)	1746016					5.0E-9 C	5.1E-9 C	65FR66443
17	Acrolein	107028					190	290	65FR66443
18	Acrylonitrile	107131					0.051 B,C	0.25 B,C	65FR66443
19	Benzene	71432					2.2 B,C	51 B,C	IRIS 01/19/00 &65FR66443
20	Bromoform	75252					4.3 B,C	140 B,C	65FR66443
21	Carbon Tetrachloride	56235					0.23 B,C	1.6 B,C	65FR66443
22	Chlorobenzene	108907					130 Z,U,	1,600 U	68FR75510
23	Chlorodibromomethane	124481					0.40 B,C	13 B,C	65FR66443
24	Chloroethane	75003							

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

	Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
			CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
25	2-Chloroethylvinyl Ether	110758							
26	Chloroform	67663					5.7 C,P	470 C,P	62FR42160
27	Dichlorobromomethane	75274					0.55 B,C	17 B,C	65FR66443
28	1,1-Dichloroethane	75343							
29	1,2-Dichloroethane	107062					0.38 B,C	37 B,C	65FR66443
30	1,1-Dichloroethylene	75354					330	7,100	68FR75510
31	1,2-Dichloropropane	78875					0.50 B,C	15 B,C	65FR66443
32	1,3-Dichloropropene	542756					0.34 c	21 c	68FR75510
33	Ethylbenzene	100414					530	2,100	68FR75510
34	Methyl Bromide	74839					47 B	1,500 B	65FR66443
35	Methyl Chloride	74873							65FR31682
36	Methylene Chloride	75092					4.6 B,C	590 B,C	65FR66443
37	1,1,2,2-Tetrachloroethane	79345					0.17 B,C	4.0 B,C	65FR66443
38	Tetrachloroethylene	127184					0.69 c	3.3 c	65FR66443
39	Toluene	108883					1,300 z	15,000	68FR75510
40	1,2-Trans-Dichloroethylene	156605					140 z	10,000	68FR75510

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	Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
			CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
41	1,1,1-Trichloroethane	71556					Z		65FR31682
42	1,1,2-Trichloroethane	79005					0.59 B,C	16 B,C	65FR66443
43	Trichloroethylene	79016					2.5 C	30 C	65FR66443
44	Vinyl Chloride	75014					0.025 C,kk	2.4 C,kk	68FR75510
45	2-Chlorophenol	95578					81 B,U	150 B,U	65FR66443
46	2,4-Dichlorophenol	120832					77 B,U	290 B,U	65FR66443
47	2,4-Dimethylphenol	105679					380 B	850 B,U	65FR66443
48	2-Methyl-4,6-Dinitrophenol	534521					13	280	65FR66443
49	2,4-Dinitrophenol	51285					69 B	5,300 B	65FR66443
50	2-Nitrophenol	88755							
51	4-Nitrophenol	100027							
52	3-Methyl-4-Chlorophenol	59507					U	U	
53	Pentachlorophenol	87865	19 F,K	15 F,K	13 bb	7.9 bb	0.27 B,C	3.0 B,C,H	65FR31682 65FR66443
54	Phenol	108952					21,000 B,U	1,700,000 B,U	65FR66443
55	2,4,6-Trichlorophenol	88062					1.4 B,C	2.4 B,C,U	65FR66443
56	Acenaphthene	83329					670 B,U	990 B,U	65FR66443

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	Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
			CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
57	Acenaphthylene	208968							
58	Anthracene	120127					8,300 B	40,000 B	65FR66443
59	Benzidine	92875					0.000086 B,C	0.00020 B,C	65FR66443
60	Benzo(a)Anthracene	56553					0.0038 B,C	0.018 B,C	65FR66443
61	Benzo(a)Pyrene	50328					0.0038 B,C	0.018 B,C	65FR66443
62	Benzo(b)Fluoranthene	205992					0.0038 B,C	0.018 B,C	65FR66443
63	Benzo(ghi)Perylene	191242							
64	Benzo(k)Fluoranthene	207089					0.0038 B,C	0.018 B,C	65FR66443
65	Bis(2-Chloroethoxy)Methane	111911							
66	Bis(2-Chloroethyl)Ether	111444					0.030 B,C	0.53 B,C	65FR66443
67	Bis(2-Chloroisopropyl)Ether	108601					1,400 B	65,000 B	65FR66443
68	Bis(2-Ethylhexyl)Phthalate ^x	117817					1.2 B,C	2.2 B,C	65FR66443
69	4-Bromophenyl Phenyl Ether	101553							
70	Butylbenzyl Phthalate ^w	85687					1,500 B	1,900 B	65FR66443
71	2-Chloronaphthalene	91587					1,000 B	1,600 B	65FR66443
72	4-Chlorophenyl Phenyl Ether	7005723							

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

	Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
			CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
73	Chrysene	218019					0.0038 _{B,C}	0.018 _{B,C}	65FR66443
74	Dibenzo(a,h)Anthracene	53703					0.0038 _{B,C}	0.018 _{B,C}	65FR66443
75	1,2-Dichlorobenzene	95501					420	1,300	68FR75510
76	1,3-Dichlorobenzene	541731					320	960	65FR66443
77	1,4-Dichlorobenzene	106467					63	190	68FR75510
78	3,3'-Dichlorobenzidine	91941					0.021 _{B,C}	0.028 _{B,C}	65FR66443
79	Diethyl Phthalate ^W	84662					17,000 _B	44,000 _B	65FR66443
80	Dimethyl Phthalate ^W	131113					270,000	1,100,000	65FR66443
81	Di-n-Butyl Phthalate ^W	84742					2,000 _B	4,500 _B	65FR66443
82	2,4-Dinitrotoluene	121142					0.11 _C	3.4 _C	65FR66443
83	2,6-Dinitrotoluene	606202							
84	Di-n-Octyl Phthalate	117840							
85	1,2-Diphenylhydrazine	122667					0.036 _{B,C}	0.20 _{B,C}	65FR66443
86	Fluoranthene	206440					130 _B	140 _B	65FR66443
87	Fluorene	86737					1,100 _B	5,300 _B	65FR66443
88	Hexachlorobenzene	118741					0.00028 _{B,C}	0.00029 _{B,C}	65FR66443

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
		CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
89	Hexachlorobutadiene	87683				0.44 B,C	18 B,C	65FR66443
90	Hexachlorocyclopentadiene	77474				40 U	1,100 U	68FR75510
91	Hexachloroethane	67721				1.4 B,C	3.3 B,C	65FR66443
92	Ideno(1,2,3-cd)Pyrene	193395				0.0038 B,C	0.018 B,C	65FR66443
93	Isophorone	78591				35 B,C	960 B,C	65FR66443
94	Naphthalene	91203						
95	Nitrobenzene	98953				17 B	690 B,H,U	65FR66443
96	N-Nitrosodimethylamine	62759				0.00069 B,C	3.0 B,C	65FR66443
97	N-Nitrosodi-n-Propylamine	621647				0.0050 B,C	0.51 B,C	65FR66443
98	N-Nitrosodiphenylamine	86306				3.3 B,C	6.0 B,C	65FR66443
99	Phenanthrene	85018						
100	Pyrene	129000				830 B	4,000 B	65FR66443
101	1,2,4-Trichlorobenzene	120821				35	70	68FR75510
102	Aldrin	309002	3.0 G	1.3 G		0.000049 B,C	0.000050 B,C	65FR31682 65FR66443
103	alpha-BHC	319846				0.0026 B,C	0.0049 B,C	65FR66443
104	beta-BHC	319857				0.0091 B,C	0.017 B,C	65FR66443

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
		CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
105 gamma-BHC (Lindane)	58899	0.95 K		0.16 G		0.98	1.8	65FR31682 68FR75510
106 delta-BHC	319868							
107 Chlordane	57749	2.4 G	0.0043 G,aa	0.09 G	0.004 G,aa	0.00080 B,C	0.00081 B,C	65FR31682 65FR66443
108 4,4'-DDT	50293	1.1 G,ii	0.001 G,aa,ii	0.13 G,ii	0.001 G,aa,ii	0.00022 B,C	0.00022 B,C	65FR31682 65FR66443
109 4,4'-DDE	72559					0.00022 B,C	0.00022 B,C	65FR66443
110 4,4'-DDD	72548					0.00031 B,C	0.00031 B,C	65FR66443
111 Dieldrin	60571	0.24 K	0.056 K,O	0.71 G	0.0019 G,aa	0.000052 B,C	0.000054 B,C	65FR31682 65FR66443
112 alpha-Endosulfan	959988	0.22 G,Y	0.056 G,Y	0.034 G,Y	0.0087 G,Y	62 B	89 B	65FR31682 65FR66443
113 beta-Endosulfan	33213659	0.22 G,Y	0.056 G,Y	0.034 G,Y	0.0087 G,Y	62 B	89 B	65FR31682 65FR66443
114 Endosulfan Sulfate	1031078					62 B	89 B	65FR66443
115 Endrin	72208	0.086 K	0.036 K,O	0.037 G	0.0023 G,aa	0.059	0.060	65FR31682 68FR75510
116 Endrin Aldehyde	7421934					0.29 B	0.30 B,H	65FR66443

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/ Source
		CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
117 Heptachlor	76448	0.52 G	0.0038 G,aa	0.053 G	0.0036 G,aa	0.000079 B,C	0.000079 B,C	65FR31682 65FR66443
118 Heptachlor Epoxide	1024573	0.52 G,V	0.0038 G,V,aa	0.053 G,V	0.0036 G,V,aa	0.000039 B,C	0.000039 B,C	65FR31682 65FR66443
119 Polychlorinated Biphenyls PCBs:			0.014 N,aa		0.03 N,aa	0.000064 B,C,N	0.000064 B,C,N	65FR31682 65FR66443
120 Toxaphene	8001352	0.73	0.0002 aa	0.21	0.0002 aa	0.00028B,C	0.00028 B,C	65FR31682 65FR66443

Footnotes:

- A This recommended water quality criterion was derived from data for arsenic (III), but is applied here to total arsenic, which might imply that arsenic (III) and arsenic (V) are equally toxic to aquatic life and that their toxicities are additive. In the arsenic criteria document (EPA 440/5-84-033, January 1985), Species Mean Acute Values are given for both arsenic (III) and arsenic (V) for five species and the ratios of the SMAVs for each species range from 0.6 to 1.7. Chronic values are available for both arsenic (III) and arsenic (V) for one species; for the fathead minnow, the chronic value for arsenic (V) is 0.29 times the chronic value for arsenic (III). No data are known to be available concerning whether the toxicities of the forms of arsenic to aquatic organisms are additive.
- B This criterion has been revised to reflect The Environmental Protection Agency's q1* or RfD, as contained in the Integrated Risk Information System (IRIS) as of May 17, 2002. The fish tissue bioconcentration factor (BCF) from the 1980 Ambient Water Quality Criteria document was retained in each case.
- C This criterion is based on carcinogenicity of 10⁻⁶ risk. Alternate risk levels may be obtained by moving the decimal point (e.g., for a risk level of 10⁻⁵, move the decimal point in the recommended criterion one place to the right).
- D Freshwater and saltwater criteria for metals are expressed in terms of the dissolved metal in the water column. The recommended water quality criteria value was calculated by using the previous 304(a) aquatic life criteria expressed in terms of total recoverable metal, and multiplying it by a conversion factor (CF). The term "Conversion Factor" (CF) represents the recommended conversion factor for converting a metal criterion expressed as the total recoverable fraction in the water column to a criterion expressed as the dissolved fraction in the water column. (Conversion Factors for saltwater CCCs are not currently available. Conversion factors derived for saltwater CMCs have been used for both saltwater CMCs and CCCs). See "Office of Water Policy and Technical Guidance on Interpretation and Implementation of Aquatic Life Metals Criteria," October 1, 1993, by Martha G. Prothro, Acting Assistant Administrator for

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

Water, available from the Water Resource center, USEPA, 401 M St., SW, mail code RC4100, Washington, DC 20460; and 40CFR§131.36(b)(1).

Conversion Factors applied in the table can be found in Appendix A to the Preamble- Conversion Factors for Dissolved Metals.

- E The freshwater criterion for this metal is expressed as a function of hardness (mg/L) in the water column. The value given here corresponds to a hardness of 100 mg/L. Criteria values for other hardness may be calculated from the following: $CMC (dissolved) = \exp\{m_A [\ln(hardness)] + b_A\}$ (CF), or $CCC (dissolved) = \exp\{m_C [\ln(hardness)] + b_C\}$ (CF) and the parameters specified in Appendix B- Parameters for Calculating Freshwater Dissolved Metals Criteria That Are Hardness-Dependent.
- F Freshwater aquatic life values for pentachlorophenol are expressed as a function of pH, and are calculated as follows: $CMC = \exp(1.005(pH) - 4.869)$; $CCC = \exp(1.005(pH) - 5.134)$. Values displayed in table correspond to a pH of 7.8.
- G This Criterion is based on 304(a) aquatic life criterion issued in 1980, and was issued in one of the following documents: Aldrin/Dieldrin (EPA 440/5-80-019), Chlordane (EPA 440/5-80-027), DDT (EPA 440/5-80-038), Endosulfan (EPA 440/5-80-046), Endrin (EPA 440/5-80-047), Heptachlor (EPA 440/5-80-052), Hexachlorocyclohexane (EPA 440/5-80-054), Silver (EPA 440/5-80-071). The Minimum Data Requirements and derivation procedures were different in the 1980 Guidelines than in the 1985 Guidelines. For example, a “CMC” derived using the 1980 Guidelines was derived to be used as an instantaneous maximum. If assessment is to be done using an averaging period, the values given should be divided by 2 to obtain a value that is more comparable to a CMC derived using the 1985 Guidelines.
- H No criterion for protection of human health from consumption of aquatic organisms excluding water was presented in the 1980 criteria document or in the *1986 Quality Criteria for Water*. Nevertheless, sufficient information was presented in the 1980 document to allow the calculation of a criterion, even though the results of such a calculation were not shown in the document.
- I This criterion for asbestos is the Maximum Contaminant Level (MCL) developed under the Safe Drinking Water Act (SDWA).
- J This fish tissue residue criterion for methylmercury is based on a total fish consumption rate of 0.0175 kg/day.
- K This recommended criterion is based on a 304(a) aquatic life criterion that was issued in the *1995 Updates: Water Quality Criteria Documents for the Protection of Aquatic Life in Ambient Water*, (EPA-820-B-96-001, September 1996). This value was derived using the GLI Guidelines (60FR15393-15399, March 23, 1995; 40CFR132 Appendix A); the difference between the 1985 Guidelines and the GLI Guidelines are explained on page iv of the 1995 Updates. None of the decisions concerning the derivation of this criterion were affected by any considerations that are specific to the Great Lakes.
- L The $CMC = 1/[(f1/CMC1) + (f2/CMC2)]$ where f1 and f2 are the fractions of total selenium that are treated as selenite and selenate, respectively, and CMC1 and CMC2 are 185.9 Fg/l and 12.82 Fg/l, respectively.
- M EPA is currently reassessing the criteria for arsenic.
- N This criterion applies to total pcbs, (e.g., the sum of all congener or all isomer or homolog or Aroclor analyses.)
- O The derivation of the CCC for this pollutant (Endrin) did not consider exposure through the diet, which is probably important for aquatic life occupying upper trophic levels.
- P Although a new RfD is available in IRIS, the surface water criteria will not be revised until the National Primary Drinking Water Regulations: Stage 2 Disinfectants and Disinfection Byproducts Rule (Stage 2 DBPR) is completed, since public comment on the relative source contribution (RSC) for chloroform is anticipated.
- Q This recommended water quality criterion is expressed as Fg free cyanide (as CN)/L.
- R This value for selenium was announced (61FR58444-58449, November 14, 1996) as a proposed GLI 303(c) aquatic life criterion. EPA is currently working on this criterion and so this value might change substantially in the near future.
- S This recommended water quality criterion for arsenic refers to the inorganic form only.

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- T This recommended water quality criterion for selenium is expressed in terms of total recoverable metal in the water column. It is scientifically acceptable to use the conversion factor (0.996- CMC or 0.922- CCC) that was used in the GLI to convert this to a value that is expressed in terms of dissolved metal.
- U The organoleptic effect criterion is more stringent than the value for priority toxic pollutants.
- V This value was derived from data for heptachlor and the criteria document provides insufficient data to estimate the relative toxicities of heptachlor and heptachlor epoxide.
- W Although EPA has not published a completed criteria document for butylbenzyl phthalate it is EPA's understanding that sufficient data exist to allow calculation of aquatic criteria. It is anticipated that industry intends to publish in the peer reviewed literature draft aquatic life criteria generated in accordance with EPA Guidelines. EPA will review such criteria for possible issuance as national WQC.
- X There is a full set of aquatic life toxicity data that show that DEHP is not toxic to aquatic organisms at or below its solubility limit.
- Y This value was derived from data for endosulfan and is most appropriately applied to the sum of alpha-endosulfan and beta-endosulfan.
- Z A more stringent MCL has been issued by EPA. Refer to drinking water regulations (40 CFR 141) or Safe Drinking Water Hotline (1-800-426-4791) for values.
- aa This criterion is based on a 304(a) aquatic life criterion issued in 1980 or 1986, and was issued in one of the following documents: Aldrin/Dieldrin (EPA 440/5-80-019), Chlordane (EPA 440/5-80-027), DDT (EPA 440/5-80-038), Endrin (EPA 440/5-80-047), Heptachlor (EPA 440/5-80-052), Polychlorinated biphenyls (EPA 440/5-80-068), Toxaphene (EPA 440/5-86-006). This CCC is currently based on the Final Residue Value (FRV) procedure. Since the publication of the Great Lakes Aquatic Life Criteria Guidelines in 1995 (60FR15393-15399, March 23, 1995), the Agency no longer uses the Final Residue Value procedure for deriving CCCs for new or revised 304(a) aquatic life criteria. Therefore, the Agency anticipates that future revisions of this CCC will not be based on the FRV procedure.
- bb This water quality criterion is based on a 304(a) aquatic life criterion that was derived using the 1985 Guidelines (*Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses*, PB85-227049, January 1985) and was issued in one of the following criteria documents: Arsenic (EPA 440/5-84-033), Cadmium (EPA-822-R-01-001), Chromium (EPA 440/5-84-029), Copper (EPA 440/5-84-031), Cyanide (EPA 440/5-84-028), Lead (EPA 440/5-84-027), Nickel (EPA 440/5-86-004), Pentachlorophenol (EPA 440/5-86-009), Toxaphene, (EPA 440/5-86-006), Zinc (EPA 440/5-87-003).
- cc When the concentration of dissolved organic carbon is elevated, copper is substantially less toxic and use of Water-Effect Ratios might be appropriate.
- dd The selenium criteria document (EPA 440/5-87-006, September 1987) provides that if selenium is as toxic to saltwater fishes in the field as it is to freshwater fishes in the field, the status of the fish community should be monitored whenever the concentration of selenium exceeds 5.0 Fg/L in salt water because the saltwater CCC does not take into account uptake via the food chain.
- ee This recommended water quality criterion was derived on page 43 of the mercury criteria document (EPA 440/5-84-026, January 1985). The saltwater CCC of 0.025 ug/L given on page 23 of the criteria document is based on the Final Residue Value procedure in the 1985 Guidelines. Since the publication of the Great Lakes Aquatic Life Criteria Guidelines in 1995 (60FR15393-15399, March 23, 1995), the Agency no longer uses the Final Residue Value procedure for deriving CCCs for new or revised 304(a) aquatic life criteria.
- ff This recommended water quality criterion was derived in *Ambient Water Quality Criteria Saltwater Copper Addendum* (Draft, April 14, 1995) and was promulgated in the Interim final National Toxics Rule (60FR22228-22237, May 4, 1995).
- gg EPA is actively working on this criterion and so this recommended water quality criterion may change substantially in the near future.
- hh This recommended water quality criterion was derived from data for inorganic mercury (II), but is applied here to total mercury. If a substantial portion of the mercury in the water column is methylmercury, this criterion will probably be under protective. In addition, even though inorganic mercury is converted to

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR PRIORITY TOXIC POLLUTANTS

methylmercury and methylmercury bioaccumulates to a great extent, this criterion does not account for uptake via the food chain because sufficient data were not available when the criterion was derived.

- ii This criterion applies to DDT and its metabolites (i.e., the total concentration of DDT and its metabolites should not exceed this value).
- jj This recommended water quality criterion is expressed as total cyanide, even though the IRIS RFD we used to derive the criterion is based on free cyanide. The multiple forms of cyanide that are present in ambient water have significant differences in toxicity due to their differing abilities to liberate the CN-moiety. Some complex cyanides require even more extreme conditions than refluxing with sulfuric acid to liberate the CN-moiety. Thus, these complex cyanides are expected to have little or no 'bioavailability' to humans. If a substantial fraction of the cyanide present in a water body is present in a complexed form (e.g., $\text{Fe}_4[\text{Fe}(\text{CN})_6]_3$), this criterion may be over conservative.
- kk This recommended water quality criterion was derived using the cancer slope factor of 1.4 (LMS exposure from birth).

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR NON PRIORITY POLLUTANTS

Non Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/Source
		CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
1 Alkalinity	--		20000 F					Gold Book
2 Aluminum pH 6.5 - 9.0	7429905	750 G,I	87 G,I,L					53FR33178
3 Ammonia	7664417	FRESHWATER CRITERIA ARE pH, Temperature and Life-stage DEPENDENT -- SEE DOCUMENT D SALTWATER CRITERIA ARE pH AND TEMPERATURE DEPENDENT						EPA822-R-99-014 EPA440/5-88-004
4 Aesthetic Qualities	--	NARRATIVE STATEMENT-- SEE DOCUMENT						Gold Book
5 Bacteria	--	FOR PRIMARY RECREATION AND SHELLFISH USES -- SEE DOCUMENT						Gold Book
6 Barium	7440393					1,000 A		Gold Book
7 Boron	--	NARRATIVE STATEMENT-- SEE DOCUMENT						Gold Book
8 Chloride	16887006	860000 G	230000 G					53FR19028
9 Chlorine	7782505	19	11	13	7.5	C		Gold Book
10 Chlorophenoxy Herbicide (2,4,5,-TP)	93721					10 A		Gold Book
11 Chlorophenoxy Herbicide (2,4-D)	94757					100 A,C		Gold Book
12 Chloropyrifos	2921882	0.083 G	0.041 G	0.011 G	0.0056 G			Gold Book
13 Color	--	NARRATIVE STATEMENT-- SEE DOCUMENT F						Gold Book

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Non Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/Source
		CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
14 Demeton	8065483		0.1 F		0.1 F			Gold Book
15 Ether, Bis(Chloromethyl)	542881					0.00010 E, H	0.00029 E,H	65FR66443
16 Gases, Total Dissolved	--		NARRATIVE STATEMENT -- SEE DOCUMENT F					Gold Book
17 Guthion	86500		0.01 F		0.01 F			Gold Book
18 Hardness	--		NARRATIVE STATEMENT-- SEE DOCUMENT					Gold Book
19 Hexachlorocyclo-hexane-Technical	319868					0.0123	0.0414	Gold Book
20 Iron	7439896		1000 F			300 A		Gold Book
21 Malathion	121755		0.1 F		0.1 F			Gold Book
22 Manganese	7439965					50 A,O	100 A	Gold Book
23 Methoxychlor	72435		0.03 F		0.03 F	100 A,C		Gold Book
24 Mirex	2385855		0.001 F		0.001 F			Gold Book
25 Nitrates	14797558					10,000 A		Gold Book
26 Nitrosamines	--					0.0008	1.24	Gold Book
27 Dinitrophenols	25550587					69	5300	65FR66443
28 Nonylphenol	1044051	28	6.6	7.0	1.7			71FR9337
29 Nitrosodibutylamine,N	924163					0.0063 A,H	0.22 A,H	65FR66443
30 Nitrosodiethylamine,N	55185					0.0008 A,H	1.24 A,H	Gold Book

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR NON PRIORITY POLLUTANTS

Non Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/Source
		CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
31 Nitrosopyrrolidine,N	930552					0.016 H	34 H	65FR66443
32 Oil and Grease	--	NARRATIVE STATEMENT -- SEE DOCUMENT F						Gold Book
33 Oxygen, Dissolved Freshwater	7782447	WARMWATER AND COLDWATER MATRIX -- SEE DOCUMENT N						Gold Book
Oxygen, Dissolved Saltwater		SALTWATER -- SEE DOCUMENT						EPA-822R-00-012
34 Diazinon	333415	0.17	0.17	0.82	0.82			71FR9336
35 Parathion	56382	0.065 J	0.013 J					Gold Book
36 Pentachlorobenzene	608935					1.4 E	1.5 E	65FR66443
37 pH	--		6.5 - 9 F		6.5 - 8.5 F,K	5 - 9		Gold Book
38 Phosphorus Elemental	7723140				0.1 F,K			Gold Book
39 Nutrients	--	See EPA's Ecoregional criteria for Total Phosphorus, Total Nitrogen, Chlorophyll <i>a</i> and Water Clarity (Secchi depth for lakes; turbidity for streams and rivers) (& Level III Ecoregional criteria)						P
40 Solids Dissolved and Salinity	--					250,000 A		Gold Book
41 Solids Suspended and Turbidity	--	NARRATIVE STATEMENT -- SEE DOCUMENT F						Gold Book
42 Sulfide-Hydrogen Sulfide	7783064		2.0 F		2.0 F			Gold Book
43 Tainting Substances	--	NARRATIVE STATEMENT-- SEE DOCUMENT						Gold Book
44 Temperature	--	SPECIES DEPENDENT CRITERIA -- SEE DOCUMENT M						Gold Book

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	Non Priority Pollutant	CAS Number	Freshwater		Saltwater		Human Health For Consumption of:		FR Cite/Source
			CMC (Fg/L)	CCC (Fg/L)	CMC (Fg/L)	CCC (Fg/L)	Water + Organism (Fg/L)	Organism Only (Fg/L)	
45	Tetrachlorobenzene,1,2,4,5-	95943					0.97 E	1.1 E	65FR66443
46	Tributyltin (TBT)	--	0.46 Q	0.072 Q	0.42 Q	0.0074 Q			EPA 822-F-00-008
47	Trichlorophenol,2,4,5-	95954					1,800 B,E	3,600 B,E	65FR66443

Footnotes:

- A This human health criterion is the same as originally published in the Red Book which predates the 1980 methodology and did not utilize the fish ingestion BCF approach. This same criterion value is now published in the Gold Book.
- B The organoleptic effect criterion is more stringent than the value presented in the non priority pollutants table.
- C A more stringent Maximum Contaminant Level (MCL) has been issued by EPA under the Safe Drinking Water Act. Refer to drinking water regulations 40CFR141 or Safe Drinking Water Hotline (1-800-426-4791) for values.
- D According to the procedures described in the *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses*, except possibly where a very sensitive species is important at a site, freshwater aquatic life should be protected if both conditions specified in Appendix C to the Preamble- Calculation of Freshwater Ammonia Criterion are satisfied.
- E This criterion has been revised to reflect EPA's q1* or RfD, as contained in the Integrated Risk Information System (IRIS) as of May 17, 2002. The fish tissue bioconcentration factor (BCF) used to derive the original criterion was retained in each case.
- F The derivation of this value is presented in the Red Book (EPA 440/9-76-023, July, 1976).
- G This value is based on a 304(a) aquatic life criterion that was derived using the 1985 Guidelines (*Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses*, PB85-227049, January 1985) and was issued in one of the following criteria documents: Aluminum (EPA 440/5-86-008); Chloride (EPA 440/5-88-001); Chloropyrifos (EPA 440/5-86-005).
- H This criterion is based on carcinogenicity of 10⁻⁶ risk. Alternate risk levels may be obtained by moving the decimal point (e.g., for a risk level of 10⁻⁵, move the decimal point in the recommended criterion one place to the right).
- I This value for aluminum is expressed in terms of total recoverable metal in the water column.
- J This value is based on a 304(a) aquatic life criterion that was issued in the *1995 Updates: Water Quality Criteria Documents for the Protection of Aquatic Life in Ambient Water* (EPA-820-B-96-001). This value was derived using the GLI Guidelines (60FR15393-15399, March 23, 1995; 40CFR132 Appendix A); the differences between the 1985 Guidelines and the GLI Guidelines are explained on page iv of the 1995 Updates. No decision concerning this criterion was affected by any considerations that are specific to the Great Lakes.
- K According to page 181 of the Red Book:

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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 from the naturally occurring variation or 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 of some species, changes in pH should be avoided but in any case should not exceed the limits established for fresh water, i.e., 6.5-9.0.

- L There are three major reasons why the use of Water-Effect Ratios might be appropriate. (1) The value of 87 Fg/l is based on a toxicity test with the striped bass in water with pH= 6.5-6.6 and hardness <10 mg/L. Data in "Aluminum Water-Effect Ratio for the 3M Plant Effluent Discharge, Middleway, West Virginia" (May 1994) indicate that aluminum is substantially less toxic at higher pH and hardness, but the effects of pH and hardness are not well quantified at this time. (2) In tests with the brook trout at low pH and hardness, effects increased with increasing concentrations of total aluminum even though the concentration of dissolved aluminum was constant, indicating that total recoverable is a more appropriate measurement than dissolved, at least when particulate aluminum is primarily aluminum hydroxide particles. In surface waters, however, the total recoverable procedure might measure aluminum associated with clay particles, which might be less toxic than aluminum associated with aluminum hydroxide. (3) EPA is aware of field data indicating that many high quality waters in the U.S. contain more than 87 Fg aluminum/L, when either total recoverable or dissolved is measured.
- M U.S. EPA. 1973. Water Quality Criteria 1972. EPA-R3-73-033. National Technical Information Service, Springfield, VA.; U.S. EPA. 1977. Temperature Criteria for Freshwater Fish: Protocol and Procedures. EPA-600/3-77-061. National Technical Information Service, Springfield, VA.
- N U.S. EPA. 1986. Ambient Water Quality Criteria for Dissolved Oxygen. EPA 440/5-86-003. National Technical Information Service, Springfield, VA.
- O This criterion for manganese is not based on toxic effects, but rather is intended to minimize objectionable qualities such as laundry stains and objectionable tastes in beverages.
- P Lakes and Reservoirs in Nutrient Ecoregion: II EPA 822-B-00-007, III EPA 822-B-01-008, IV EPA 822-B-01-009, V EPA 822-B-01-010, VI EPA 822-B-00-008, VII EPA 822-B-00-009, VIII EPA 822-B-01-015, IX EPA 822-B-00-011, XI EPA 822-B-00-012, XII EPA 822-B-00-013, XIII EPA 822-B-00-014, XIV EPA 822-B-01-011; Rivers and Streams in Nutrient Ecoregion: I EPA 822-B-01-012, II EPA 822-B-00-015, III EPA 822-B-00-016, IV EPA 822-B-01-013, V EPA 822-B-01-014, VI EPA 822-B-00-017, VII EPA 822-B-00-018, VIII EPA 822-B-01-015, IX EPA 822-B-00-019, X EPA 822-B-01-016, XI EPA 822-B-00-020, XII EPA 822-B-00-021, XIV EPA 822-B-00-022; and Wetlands in Nutrient Ecoregion XIII EPA 822-B-00-023.
- Q EPA announced the availability of a draft updated tributyltin (TBT) document on August 7, 1997 (62FR42554). The Agency has reevaluated this document and anticipates releasing an updated document for public comment in the near future.

NATIONAL RECOMMENDED WATER QUALITY CRITERIA FOR ORGANOLEPTIC EFFECTS

	Pollutant	CAS Number	Organoleptic Effect Criteria (F g/L)	FR Cite/Source
1	Acenaphthene	83329	20	Gold Book
2	Monochlorobenzene	108907	20	Gold Book
3	3-Chlorophenol	--	0.1	Gold Book
4	4-Chlorophenol	106489	0.1	Gold Book
5	2,3-Dichlorophenol	--	0.04	Gold Book
6	2,5-Dichlorophenol	--	0.5	Gold Book
7	2,6-Dichlorophenol	--	0.2	Gold Book
8	3,4-Dichlorophenol	--	0.3	Gold Book
9	2,4,5-Trichlorophenol	95954	1	Gold Book
10	2,4,6-Trichlorophenol	88062	2	Gold Book
11	2,3,4,6-Tetrachlorophenol	--	1	Gold Book
12	2-Methyl-4-Chlorophenol	--	1800	Gold Book
13	3-Methyl-4-Chlorophenol	59507	3000	Gold Book
14	3-Methyl-6-Chlorophenol	--	20	Gold Book
15	2-Chlorophenol	95578	0.1	Gold Book
16	Copper	7440508	1000	Gold Book
17	2,4-Dichlorophenol	120832	0.3	Gold Book
18	2,4-Dimethylphenol	105679	400	Gold Book

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	Pollutant	CAS Number	Organoleptic Effect Criteria (Fg/L)	FR Cite/Source
19	Hexachlorocyclopentadiene	77474	1	Gold Book
20	Nitrobenzene	98953	30	Gold Book
21	Pentachlorophenol	87865	30	Gold Book
22	Phenol	108952	300	Gold Book
23	Zinc	7440666	5000	45 FR79341

General notes:

1. These criteria are based on organoleptic (taste and odor) effects. Because of variations in chemical nomenclature systems, this listing of pollutants does not duplicate the listing in Appendix A of 40 CFR Part 423. Also listed are the Chemical Abstracts Service (CAS) registry numbers, which provide a unique identification for each chemical.

NATIONAL RECOMMENDED WATER QUALITY CRITERIA

Additional Notes:

1. Criteria Maximum Concentration and Criterion Continuous Concentration

The Criteria Maximum Concentration (CMC) is an estimate of the highest concentration of a material in surface water to which an aquatic community can be exposed briefly without resulting in an unacceptable effect. The Criterion Continuous Concentration (CCC) is an estimate of the highest concentration of a material in surface water to which an aquatic community can be exposed indefinitely without resulting in an unacceptable effect. The CMC and CCC are just two of the six parts of an aquatic life criterion; the other four parts are the acute averaging period, chronic averaging period, acute frequency of allowed exceedence, and chronic frequency of allowed exceedence. Because 304(a) aquatic life criteria are national guidance, they are intended to be protective of the vast majority of the aquatic communities in the United States.

2. Criteria Recommendations for Priority Pollutants, Non Priority Pollutants and Organoleptic Effects

This compilation lists all priority toxic pollutants and some non priority toxic pollutants, and both human health effect and organoleptic effect criteria issued pursuant to CWA §304(a). Blank spaces indicate that EPA has no CWA §304(a) criteria recommendations. For a number of non-priority toxic pollutants not listed, CWA §304(a) "water + organism" human health criteria are not available, but EPA has published MCLs under the SDWA that may be used in establishing water quality standards to protect water supply designated uses. Because of variations in chemical nomenclature systems, this listing of toxic pollutants does not duplicate the listing in Appendix A of 40 CFR Part 423. Also listed are the Chemical Abstracts Service CAS registry numbers, which provide a unique identification for each chemical.

3. Human Health Risk

The human health criteria for the priority and non priority pollutants are based on carcinogenicity of 10^{-6} risk. Alternate risk levels may be obtained by moving the decimal point (e.g., for a risk level of 10^{-5} , move the decimal point in the recommended criterion one place to the right).

4. Water Quality Criteria published pursuant to Section 304(a) or Section 303(c) of the CWA

Many of the values in the compilation were published in the California Toxics Rule. Although such values were published pursuant to Section 303(c) of the CWA, they represent the Agency's most recent calculation of water quality criteria and are thus the Agency's 304(a) criteria.

5. Calculation of Dissolved Metals Criteria

The 304(a) criteria for metals, shown as dissolved metals, are calculated in one of two ways. For freshwater metals criteria that are hardness-dependent, the dissolved metal criteria were calculated using a hardness of 100 mg/l as CaCO_3 for illustrative purposes only. Saltwater and freshwater metals' criteria that are not hardness-dependent are calculated by multiplying the total recoverable criteria before rounding by the appropriate conversion factors. The final dissolved metals' criteria in the table are rounded to two significant figures. Information regarding the calculation of hardness dependent conversion factors are included in the footnotes.

6. Maximum Contaminant Levels

The compilation includes footnotes for pollutants with Maximum Contaminant Levels (MCLs) more stringent than the recommended water quality criteria in the compilation. MCLs for these pollutants are not included in the compilation, but can be found in the appropriate drinking water regulations (40 CFR 141.11-16 and 141.60-63), or can be accessed through the Safe Drinking Water Hotline (800-426-4791) or the Internet

(<http://www.epa.gov/waterscience/drinking/standards/dwstandards.pdf>).

7. Organoleptic Effects

The compilation contains 304(a) criteria for pollutants with toxicity-based criteria as well as non-toxicity based criteria. The basis for the non-toxicity based criteria are organoleptic effects (e.g., taste and odor) which would make water and edible aquatic life unpalatable but not toxic to humans. The table includes criteria for organoleptic effects for 23 pollutants. Pollutants with organoleptic effect criteria more stringent than the criteria based on toxicity (e.g., included in both the priority and non-priority pollutant tables) are footnoted as such.

8. Gold Book

The "Gold Book" is Quality Criteria for Water: 1986. EPA 440/5-86-001.

9. Correction of Chemical Abstract Services Number

The Chemical Abstract Services number (CAS) for Bis(2-Chlorisopropyl) Ether, has been revised in IRIS and in the table. The correct CAS number for this chemical is 108-60-1. The previous CAS number for this pollutant was 39638-32-9.

10. Contaminants with Blanks

EPA has not calculated criteria for contaminants with blanks. However, permit authorities should address these contaminants in NPDES permit actions using the States' existing narrative criteria for toxics.

11. Specific Chemical Calculations

A. Selenium

Aquatic Life

This compilation contains aquatic life criteria for selenium that are the same as those published in the proposed CTR. In the CTR, EPA proposed an acute criterion for selenium based on the criterion proposed for selenium in the Water Quality Guidance for the Great Lakes System (61 FR 58444). The GLI and CTR proposals take into account data showing that selenium's two prevalent oxidation states in water, selenite and selenate, present differing potentials for aquatic toxicity, as well as new data indicating that various forms of selenium are additive. The new approach produces a different selenium acute criterion concentration, or CMC, depending upon the relative proportions of selenite, selenate, and other forms of selenium that are present.

EPA is currently undertaking a reassessment of selenium, and expects the 304(a) criteria for selenium will be revised based on the final reassessment (63FR26186). However, until such time as revised water quality criteria for selenium are published by the Agency, the recommended water quality criteria in this compilation are EPA's current 304(a) criteria.

Appendices

Appendix A - Conversion Factors for Dissolved Metals

Metal	Conversion Factor freshwater CMC	Conversion Factor freshwater CCC	Conversion Factor saltwater CMC	Conversion Factor saltwater CCC ¹
Arsenic	1.000	1.000	1.000	1.000
Cadmium	$1.136672 - [(\ln \text{ hardness})(0.041838)]$	$1.101672 - [(\ln \text{ hardness})(0.041838)]$	0.994	0.994
Chromium III	0.316	0.860	--	--
Chromium VI	0.982	0.962	0.993	0.993
Copper	0.960	0.960	0.83	0.83
Lead	$1.46203 - [(\ln \text{ hardness})(0.145712)]$	$1.46203 - [(\ln \text{ hardness})(0.145712)]$	0.951	0.951
Mercury	0.85	0.85	0.85	0.85
Nickel	0.998	0.997	0.990	0.990
Selenium	--	--	0.998	0.998
Silver	0.85	--	0.85	--
Zinc	0.978	0.986	0.946	0.946

Appendix B - Parameters for Calculating Freshwater Dissolved Metals Criteria That Are Hardness-Dependent

Chemical	m _A	b _A	m _C	b _C	Freshwater Conversion Factors (CF)	
					CMC	CCC
Cadmium	1.0166	-3.924	0.7409	-4.719	$1.136672 - [(\ln \text{hardness})(0.041838)]$	$1.101672 - [(\ln \text{hardness})(0.041838)]$
Chromium III	0.8190	3.7256	0.8190	0.6848	0.316	0.860
Copper	0.9422	-1.700	0.8545	-1.702	0.960	0.960
Lead	1.273	-1.460	1.273	-4.705	$1.46203 - [(\ln \text{hardness})(0.145712)]$	$1.46203 - [(\ln \text{hardness})(0.145712)]$
Nickel	0.8460	2.255	0.8460	0.0584	0.998	0.997
Silver	1.72	-6.59	--	--	0.85	--
Zinc	0.8473	0.884	0.8473	0.884	0.978	0.986

Hardness-dependant metals' criteria may be calculated from the following:

CMC (dissolved) = $\exp\{m_A [\ln(\text{hardness})] + b_A\}$ (CF)

CCC (dissolved) = $\exp\{m_C [\ln(\text{hardness})] + b_C\}$ (CF)

Appendix C - Calculation of Freshwater Ammonia Criterion

1. The one-hour average concentration of total ammonia nitrogen (in mg N/L) does not exceed, more than once every three years on the average, the CMC (acute criterion) calculated using the following equations.

Where salmonid fish are present:

$$\text{CMC} = \frac{0.275}{\text{-----}} + \frac{39.0}{\text{-----}}$$

$$1 + 10^{7.204-\text{pH}} \quad 1 + 10^{\text{pH}-7.204}$$

Or where salmonid fish are not present:

$$\text{CMC} = \frac{0.411}{1 + 10^{7.204-\text{pH}}} + \frac{58.4}{1 + 10^{\text{pH}-7.204}}$$

2A. The thirty-day average concentration of total ammonia nitrogen (in mg N/L) does not exceed, more than once every three years on the average, the CCC (chronic criterion) calculated using the following equations.

When fish early life stages are present:

$$\text{CCC} = \% \frac{0.0577}{1 + 10^{7.688-\text{pH}}} + \frac{2.487}{1 + 10^{\text{pH}-7.688}} \quad \text{C} \quad \text{MIN} (2.85, 1.45 @ 0^{0.028 @ 25-\text{T}})$$

When fish early life stages are absent:

$$\text{CCC} = \% \frac{0.0577}{1 + 10^{7.688-\text{pH}}} + \frac{2.487}{1 + 10^{\text{pH}-7.688}} \quad \text{C} \quad 1.45 @ 0^{0.028 @ 25-\text{MAX} (\text{T}, 7)}$$

2B. In addition, the highest four-day average within the 30-day period should not exceed 2.5 times the CCC.

1. Substance List

2005 CERCLA Priority List of Hazardous Substances

2005 RANK	SUBSTANCE NAME	TOTAL POINTS	2003 RANK	CAS #
1	ARSENIC	1668.56	1	007440-38-2
2	LEAD	1534.54	2	007439-92-1
3	MERCURY	1507.31	3	007439-97-6
4	VINYL CHLORIDE	1389.02	4	000075-01-4
5	POLYCHLORINATED BIPHENYLS	1371.60	5	001336-36-3
6	BENZENE	1353.53	6	000071-43-2
7	POLYCYCLIC AROMATIC HYDROCARBONS	1321.72	8	130498-29-2
8	CADMIUM	1321.47	7	007440-43-9
9	BENZO(A)PYRENE	1307.76	9	000050-32-8
10	BENZO(B)FLUORANTHENE	1263.06	10	000205-99-2
11	CHLOROFORM	1224.22	11	000067-66-3
12	DDT, P,P'-	1194.95	12	000050-29-3
13	AROCLOR 1254	1182.53	13	011097-69-1
14	AROCLOR 1260	1179.51	14	011096-82-5
15	DIBENZO(A,H)ANTHRACENE	1165.46	15	000053-70-3
16	TRICHLOROETHYLENE	1158.15	16	000079-01-6
17	DIELDRIN	1153.23	18	000060-57-1
18	CHROMIUM, HEXAVALENT	1149.71	17	018540-29-9
19	PHOSPHORUS, WHITE	1144.69	19	007723-14-0
20	DDE, P,P'-	1135.78	21	000072-55-9
21	CHLORDANE	1133.31	20	000057-74-9
22	HEXACHLOROBUTADIENE	1130.66	22	000087-68-3
23	COAL TAR CREOSOTE	1124.08	23	008001-58-9
24	DDD, P,P'-	1121.42	24	000072-54-8
25	ALDRIN	1116.94	26	000309-00-2
26	BENZIDINE	1114.05	25	000092-87-5
27	AROCLOR 1248	1112.19	27	012672-29-6
28	CYANIDE	1098.75	28	000057-12-5
29	AROCLOR 1242	1092.87	29	053469-21-9
30	TOXAPHENE	1086.23	31	008001-35-2

31	TETRACHLORO ETHYLENE	1084.88	30	000127-18-4
32	HEXACHLOROCYCLOHEXANE, GAMMA-	1080.42	32	000058-89-9
33	HEPTACHLOR	1070.76	33	000076-44-8
34	1,2-DIBROMOETHANE	1064.58	34	000106-93-4
35	DISULFOTON	1058.74	36	000298-04-4
36	ACROLEIN	1057.72	71	000107-02-8
37	HEXACHLOROCYCLOHEXANE, BETA-	1056.45	37	000319-85-7
38	BENZO(A)ANTHRACENE	1055.63	35	000056-55-3
39	3,3'-DICHLOROBENZIDINE	1051.33	53	000091-94-1
40	BERYLLIUM	1046.48	38	007440-41-7
41	ENDRIN	1040.88	39	000072-20-8
42	HEXACHLOROCYCLOHEXANE, DELTA-	1038.14	40	000319-86-8
43	1,2-DIBROMO-3-CHLOROPROPANE	1035.87	41	000096-12-8
44	HEPTACHLOR EPOXIDE	1028.26	44	001024-57-3
45	PENTACHLOROPHENOL	1024.41	42	000087-86-5
46	CARBON TETRACHLORIDE	1022.74	43	000056-23-5
47	AROCLOR 1221	1018.20	45	011104-28-2
48	AROCLOR 1016	1014.83	46	012674-11-2
49	DDT, O,P'-	1014.65	47	000789-02-6
50	COBALT	1013.95	49	007440-48-4
51	CIS- CHLORDANE	1010.94	50	005103-71-9
52	DI-N-BUTYL PHTHALATE	1005.41	48	000084-74-2
53	ENDOSULFAN SULFATE	1004.89	52	001031-07-8
54	ENDOSULFAN	1004.26	54	000115-29-7
55	NICKEL	1003.95	51	007440-02-0
56	TRANS- CHLORDANE	1002.36	55	005103-74-2
57	DIAZINON	1001.89	114	000333-41-5
58	ENDOSULFAN, ALPHA	1000.76	57	000959-98-8
59	XYLENES, TOTAL	995.32	56	001330-20-7
60	DIBROMOCHLOROPROPANE	994.75	58	067708-83-2

61	METHOXYCHLO R	993.32	59	000072-43-5
62	AROCLOR	991.52	60	012767-79-2
63	BENZO(K)FLUO RANTHENE	980.61	61	000207-08-9
64	ENDRIN KETONE	978.86	62	053494-70-5
65	ENDOSULFAN, BETA	976.59	63	033213-65-9
66	CHROMIUM(VI) OXIDE	969.43	64	001333-82-0
67	METHANE	959.56	65	000074-82-8
68	AROCLOR 1232	955.38	67	011141-16-5
69	ENDRIN ALDEHYDE	954.50	66	007421-93-4
70	BENZOFUORA NTHENE	951.23	69	056832-73-6
71	TOLUENE	946.04	68	000108-88-3
72	2-HEXANONE	940.85	70	000591-78-6
73	2,3,7,8- TETRACHLORO DIBENZO-P- DIOXIN	937.04	72	001746-01-6
74	ZINC	930.42	73	007440-66-6
75	DIMETHYLARSI NIC ACID	921.93	74	000075-60-5
76	DI(2- ETHYLHEXYL)P HTHALATE	918.60	75	000117-81-7
77	CHROMIUM	905.03	76	007440-47-3
78	NAPHTHALENE	895.49	78	000091-20-3
79	1,1- DICHLOROETHE NE	894.91	77	000075-35-4
80	AROCLOR 1240	888.03	81	071328-89-7
81	METHYLENE CHLORIDE	886.69	80	000075-09-2
82	2,4,6- TRINITROTOLUE NE	879.06	82	000118-96-7
83	BROMODICHLO ROETHANE	869.91	86	000683-53-4
84	1,2- DICHLOROETHA NE	865.60	87	000107-06-2
85	HYDRAZINE	864.30	88	000302-01-2
86	2,4,6- TRICHLOROPHE NOL	863.10	83	000088-06-2
87	2,4- DINITROPHENO L	860.43	85	000051-28-5
88	BIS(2- CHLOROETHYL) ETHER	858.19	89	000111-44-4

89	THIOCYANATE	849.12	90	000302-04-5
90	ASBESTOS	842.16	92	001332-21-4
91	CYCLOTRIMETHYLENETRINITRAMINE (RDX)	840.72	93	000121-82-4
92	CHLORINE	840.04	94	007782-50-5
93	HEXACHLOROBENZENE	838.26	91	000118-74-1
94	RADIUM-226	835.89	98	013982-63-3
95	1,1,1-TRICHLOROETHANE	835.27	95	000071-55-6
96	2,4-DINITROTOLUENE	834.71	96	000121-14-2
97	ETHION	833.95	100	000563-12-2
98	URANIUM	833.16	97	007440-61-1
99	ETHYLBENZENE	830.62	99	000100-41-4
100	RADIUM	827.97	101	007440-14-4
101	THORIUM	825.03	102	007440-29-1
102	4,6-DINITRO-OCRESOL	822.35	103	000534-52-1
103	1,3,5-TRINITROBENZENE	819.11	106	000099-35-4
104	RADON	818.41	104	010043-92-2
105	CHLOROBENZENE	817.28	108	000108-90-7
106	RADIUM-228	816.58	107	015262-20-1
107	URANIUM-235	814.60	112	015117-96-1
107	THORIUM-230	814.60	109	014269-63-7
109	BARIUM	812.12	110	007440-39-3
110	URANIUM-234	812.01	113	013966-29-5
111	N-NITROSODI-N-PROPYLAMINE	811.01	111	000621-64-7
112	THORIUM-228	810.30	116	014274-82-9
113	FLUORANTHENE	810.29	115	000206-44-0
114	RADON-222	810.23	117	014859-67-7
115	MANGANESE	808.16	131	007439-96-5
116	HEXACHLOROCYCLOHEXANE, ALPHA-	807.72	118	000319-84-6
117	COAL TAR	807.03	124	008007-45-2
118	PLUTONIUM-239	806.68	122	015117-48-3
119	STRONTIUM-90	806.62	120	010098-97-2
119	CHRYSOTILE ASBESTOS	806.62	125	012001-29-5
121	METHYLMERCURY	806.47	119	022967-92-6
122	POLONIUM-210	806.34	120	013981-52-7
123	PLUTONIUM-238	805.93	123	013981-16-3
124	LEAD-210	805.86	126	014255-04-0

125	PLUTONIUM	805.19	128	007440-07-5
125	CHLORPYRIFOS	805.19	127	002921-88-2
127	RADON-220	804.56	129	022481-48-7
128	AMERICIUM-241	804.50	130	086954-36-1
129	AMOSITE ASBESTOS	804.02	176	012172-73-5
130	IODINE-131	803.48	132	010043-66-0
131	TRIBUTYL TIN	803.07	132	000688-73-3
132	HYDROGEN CYANIDE	803.03	134	000074-90-8
133	COPPER	802.60	141	007440-50-8
134	GUTHION	802.32	135	000086-50-0
135	NEPTUNIUM-237	802.11	136	013994-20-2
136	CHLORDEONE	801.63	137	000143-50-0
136	IODINE-129	801.63	137	015046-84-1
136	PLUTONIUM-240	801.63	137	014119-33-6
139	CHRYSENE	799.59	140	000218-01-9
140	S,S,S-TRIBUTYL PHOSPHOROTR ITHIOATE	797.81	142	000078-48-8
141	POLYBROMINAT ED BIPHENYLS	789.01	144	067774-32-7
142	BROMINE	789.01	143	007726-95-6
143	1,2,3- TRICHLOROBEN ZENE	787.73	151	000087-61-6
144	DICOFOL	787.52	145	000115-32-2
145	PARATHION	784.02	146	000056-38-2
146	1,1,2,2- TETRACHLORO ETHANE	778.29	148	000079-34-5
147	SELENIUM	777.65	147	007782-49-2
148	HEXACHLOROC YCLOHEXANE, TECHNICAL GRADE	774.60	149	000608-73-1
149	TRICHLOROFLU OROETHANE	770.66	152	027154-33-2
150	TRIFLURALIN	770.06	153	001582-09-8
151	DDD, O,P'-	768.62	154	000053-19-0
152	4,4'-METHYLENE BIS(2- CHLOROANILIN E)	766.59	155	000101-14-4
153	HEXACHLORODI BENZO-P- DIOXIN	760.07	156	034465-46-8
154	HEPTACHLORO DIBENZO-P- DIOXIN	754.08	157	037871-00-4
155	PENTACHLORO BENZENE	753.47	158	000608-93-5
156	AMMONIA	744.67	161	007664-41-7

157	2-METHYLNAPHTHALENE	743.90	159	000091-57-6
158	1,1-DICHLOROETHANE	737.82	162	000075-34-3
159	1,4-DICHLOROBENZENE	735.51	164	000106-46-7
160	ACENAPHTHENE	729.63	165	000083-32-9
161	1,2,3,4,6,7,8,9-OCTACHLORODIBENZOFURAN	725.79	167	039001-02-0
162	1,1,2-TRICHLOROETHANE	722.98	163	000079-00-5
163	TRICHLOROETHANE	722.85	166	025323-89-1
164	HEXACHLOROCYCLOPENTADIENE	718.71	168	000077-47-4
165	HEPTACHLORODIBENZOFURAN	718.25	169	038998-75-3
166	1,2-DIPHENYLHYDRAZINE	713.70	170	000122-66-7
167	2,3,4,7,8-PENTACHLORODIBENZOFURAN	710.58	171	057117-31-4
168	TETRACHLOROBIPHENYL	709.14	172	026914-33-0
169	CRESOL, PARA-	706.23	173	000106-44-5
170	OXYCHLORDANE	706.21	174	027304-13-8
171	1,2-DICHLOROBENZENE	703.53	182	000095-50-1
172	GAMMA-CHLORDENE	702.55	84	056641-38-4
173	TETRACHLOROPHENOL	702.38	181	025167-83-3
174	CARBON DISULFIDE	702.31	177	000075-15-0
175	URANIUM-233	701.59	246	013968-55-3
175	AMERICIUM	701.59	178	007440-35-9
177	PALLADIUM	700.60	185	007440-05-3
178	1,2-DICHLOROETHENE, TRANS-	700.56	175	000156-60-5
179	HEXACHLORODIBENZOFURAN	700.27	184	055684-94-1
180	INDENO(1,2,3-CD)PYRENE	698.45	183	000193-39-5
181	ACETONE	693.31	187	000067-64-1

182	CHLOROETHAN E	692.77	188	000075-00-3
183	PHENOL	692.66	186	000108-95-2
184	1,3- DICHLOROPRO PENE, CIS-	691.06	264	010061-01-5
185	P-XYLENE	689.26	190	000106-42-3
186	ALUMINUM	688.21	194	007429-90-5
187	DIBENZOFURAN	687.80	189	000132-64-9
188	CARBON MONOXIDE	684.41	193	000630-08-0
189	2,4- DIMETHYLPHEN OL	684.10	191	000105-67-9
190	TETRACHLORO ETHANE	678.08	205	025322-20-7
191	CHLOROMETHA NE	674.44	196	000074-87-3
192	PENTACHLORO DIBENZOFURAN	672.91	195	030402-15-4
193	HYDROGEN SULFIDE	672.87	197	007783-06-4
194	BIS(2- METHOXYETHY L) PHTHALATE	665.99	198	034006-76-3
195	BUTYL BENZYL PHTHALATE	657.37	200	000085-68-7
196	CRESOL, ORTHO-	657.19	199	000095-48-7
197	2,3,5,6- TETRACHLORO PHENOL	654.88	201	000935-95-5
198	VANADIUM	653.47	203	007440-62-2
199	HEXACHLORO ETHANE	652.73	202	000067-72-1
200	N- NITROSODIMET HYLAMINE	650.41	150	000062-75-9
201	1,3-BUTADIENE	647.46	204	000106-99-0
202	BROMOFORM	644.75	207	000075-25-2
203	1,2,4- TRICHLOROBEN ZENE	644.17	206	000120-82-1
204	TETRACHLORO DIBENZO-P- DIOXIN	635.43	208	041903-57-5
205	1,3- DICHLOROBENZ ENE	630.23	209	000541-73-1
206	1,2- DICHLOROETHY LENE	625.60	213	000540-59-0
207	PENTACHLORO DIBENZO-P- DIOXIN	624.88	212	036088-22-9

208	N- NITROSODIPHE NYLAMINE	623.80	211	000086-30-6
209	2-BUTANONE	622.18	214	000078-93-3
210	2,3,7,8- TETRACHLORO DIBENZOFURAN	621.90	216	051207-31-9
211	DICHLOROBENZ ENE	620.70	260	025321-22-6
212	2,4- DICHLOROPHEN OL	615.86	210	000120-83-2
213	SILVER	613.48	218	007440-22-4
214	FLUORINE	613.10	NEW	007782-41-4
215	1,4-DIOXANE	612.78	NEW	000123-91-1
216	NITRITE	612.49	220	014797-65-0
217	CESIUM-137	612.35	217	010045-97-3
218	CHROMIUM TRIOXIDE	610.71	221	007738-94-5
219	NITRATE	609.85	222	014797-55-8
220	POTASSIUM-40	608.80	224	013966-00-2
221	DINITROTOLUE NE	607.58	223	025321-14-6
222	ANTIMONY	606.30	230	007440-36-0
223	THORIUM-227	605.32	225	015623-47-9
224	COAL TAR PITCH	605.25	226	065996-93-2
225	2,4,5- TRICHLOROPHE NOL	604.44	228	000095-95-4
226	ARSENIC ACID	604.40	227	007778-39-4
227	ARSENIC TRIOXIDE	604.30	229	001327-53-3
228	PHORATE	603.07	231	000298-02-2
229	CRESOLS	603.02	250	001319-77-3
230	BENZOPYRENE	602.97	236	073467-76-2
231	CHLORDANE, TECHNICAL	602.60	NEW	012789-03-6
232	DIMETHOATE	602.59	233	000060-51-5
233	STROBANE	602.54	234	008001-50-1
233	ACTINIUM-227	602.54	234	014952-40-0
235	PYRETHRUM	602.49	236	008003-34-7
235	4- AMINOBIPHENY L	602.49	236	000092-67-1
237	ARSINE	602.39	239	007784-42-1
238	NALED	602.32	240	000300-76-5
239	ETHOPROP	602.11	241	013194-48-4
239	DIBENZOFURAN S, CHLORINATED	602.11	241	042934-53-2
241	ALPHA- CHLORDENE	601.91	243	056534-02-2

241	CARBOPHENOTHION	601.91	243	000786-19-6
243	DICHLORVOS	601.63	245	000062-73-7
244	SODIUM ARSENITE	601.43	NEW	007784-46-5
244	CALCIUM ARSENATE	601.43	246	007778-44-1
244	MERCURIC CHLORIDE	601.43	246	007487-94-7
247	FORMALDEHYDE	599.22	251	000050-00-0
248	2-CHLOROPHENOL	598.90	219	000095-57-8
249	PHENANTHRENE	595.25	249	000085-01-8
250	HYDROGEN FLUORIDE	587.88	253	007664-39-3
251	2,4-D ACID	584.13	252	000094-75-7
252	DIBROMOCHLOROMETHANE	580.41	255	000124-48-1
253	DIURON	579.09	NEW	000330-54-1
254	BUTYLATE	578.36	257	002008-41-5
255	DIMETHYL FORMAMIDE	578.04	258	000068-12-2
256	PYRENE	575.34	259	000129-00-0
257	ETHYL ETHER	572.10	261	000060-29-7
258	DICHLOROETHANE	570.47	262	001300-21-6
259	4-NITROPHENOL	566.05	263	000100-02-7
260	PHOSPHINE	559.64	265	007803-51-2
261	TRICHLOROBENZENE	558.13	266	012002-48-1
262	2,6-DINITROTOLUENE	554.50	267	000606-20-2
263	FLUORIDE ION	549.16	269	016984-48-8
264	1,2,3,4,6,7,8-HEPTACHLORODIBENZO-P-DIOXIN	547.70	270	035822-46-9
265	METHYL PARATHION	545.71	271	000298-00-0
266	PENTAERYTHRITOL TETRANITRATE	545.49	NEW	000078-11-5
267	1,3-DICHLOROPROPENE, TRANS-	545.07	268	010061-02-6
268	BIS(2-ETHYLHEXYL)ADIPATE	540.08	273	000103-23-1
269	CARBAZOLE	535.41	272	000086-74-8

270	1,2-DICHLOROETHENE, CIS-	532.34	NEW	000156-59-2
271	METHYL ISOBUTYL KETONE	531.89	274	000108-10-1
272	STYRENE	531.08	275	000100-42-5
273	CARBARYL	530.91	NEW	000063-25-2
274	1,2,3,4,6,7,8-HEPTACHLORO DIBENZOFURAN	529.28	NEW	067562-39-4
275	ACRYLONITRILE	528.09	NEW	000107-13-1

TOXICOLOGICAL PROFILE FOR
BORON AND COMPOUNDS

Agency for Toxic Substances and Disease Registry
U.S. Public Health Service

July 1992

DISCLAIMER

The use of company or product name(s) is for identification only and does not imply endorsement by the Agency for Toxic Substances and Disease Registry.

FOREWORD

The Superfund Amendments and Reauthorization Act (SARA) of 1986 (Public Law 99-499) extended and amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund). This public law directed the Agency for Toxic Substances and Disease Registry (ATSDR) to prepare toxicological profiles for hazardous substances which are most commonly found at facilities on the CERCLA National Priorities List and which pose the most significant potential threat to human health, as determined by ATSDR and the Environmental Protection Agency (EPA). The lists of the 250 most significant hazardous substances were published in the Federal Register on April 17, 1987; on October 20, 1988; on October 26, 1989; and on October 17, 1990. A revised list of 275 substances was published on October 17, 1991.

Section 104(i)(3) of CERCLA, as amended, directs the Administrator of ATSDR to prepare a toxicological profile for each substance on the lists. Each profile must include the following content:

(A) An examination, summary, and interpretation of available toxicological information and epidemiological evaluations on the hazardous substance in order to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects.

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure which present a significant risk to human health of acute, subacute, and chronic health effects.

(C) Where appropriate, an identification of toxicological testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

This toxicological profile is prepared in accordance with guidelines developed by ATSDR and EPA. The original guidelines were published in the Federal Register on April 17, 1987. Each profile will be revised and republished as necessary,

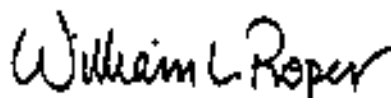
The ATSDR toxicological profile is intended to characterize succinctly the toxicological and adverse health effects information for the hazardous substance being described. Each profile identifies and reviews the key literature (that has been peer-reviewed) that describes a hazardous substance's toxicological properties. Other pertinent literature is also presented but described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

Foreword

Each toxicological profile begins with a public health statement, which describes in nontechnical language a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health will be identified by ATSDR, the National Toxicology Program (NTP) of the Public Health Service, and EPA. The focus of the profiles is on health and toxicological information; therefore, we have included this information in the beginning of the document.

The principal audiences for the toxicological profiles are health professionals at the federal, state, and local levels, interested private sector organizations and groups, and members of the public.

This profile reflects our assessment of all relevant toxicological testing and information that has been peer reviewed. It has been reviewed by scientists from ATSDR, the Centers for Disease Control, the NTP, and other federal agencies. It has also been reviewed by a panel of nongovernment peer reviewers. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.



William L. Roper, M.D., M.P.H.
Administrator
Agency for Toxic Substances and
Disease Registry

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1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about boron and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,177 sites on its National Priorities List (NPL). Boron has been found in at least 21 of these sites. However, we do not know how many of the 1,177 NPL sites have been evaluated for boron. As EPA evaluates more sites, the number of sites at which boron is found may change. This information is important for you to know because boron may cause harmful health effects and because these sites are potential or actual sources of human exposure to boron.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous chemical such as boron, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS BORON?

Boron is a solid substance that widely occurs in nature. It usually does not occur alone, but is often found in the environment combined with other substances to form compounds called borates. Common borate compounds include boric acid, salts of borates, and boron oxide. Boron and salts of borate have been found at hazardous waste sites. Boron alone does not dissolve in water nor does it evaporate easily, but it does stick to soil particles. No information was found on whether common forms of boron evaporate easily or stick to soil particles; however, these forms do dissolve in water.

Boron is present in air, water, and soil, but no information is available on how long it remains in these media. There is also no information available on the occurrence of borates in the environment or on how long they persist in the environment.

Borates are used mostly in the production of glass. They are also used in fire retardants, leather tanning and finishing industries, cosmetics, photographic materials, with certain metals, and for high-energy fuel. Pesticides for cockroach control and wood preservatives also contain borates.

1. PUBLIC HEALTH STATEMENT

More information on the properties and uses of boron and boron compounds and how they behave in the environment may be found in Chapters 3, 4, and 5.

1.2 HOW MIGHT I BE EXPOSED TO BORON?

Boron occurs mainly in the environment through release into air, water, or soil after natural weathering processes. It can also be released from glass manufacturing, coal-burning power plants, copper smelters, and through its use in agricultural fertilizer and pesticides. It is estimated that releases from these sources are less than through natural weathering processes.

You can be exposed to boron in food (mainly vegetables and fruits), water, air, and consumer products. Infants, in particular, can be exposed to borates in products used to control cockroaches. Since boron is taken up from the soil by plants, it can enter the food chain. Although boron has been found in animal tissue, it does not accumulate and it is not likely that eating fish or meat will increase the boron levels in your body. Boron has been found in groundwater at very low levels. Background levels of boron up to 5 parts of boron in 1 million parts (ppm) of surface water have been reported. However, in dry areas where there are natural boron-rich deposits, boron concentrations can be as high as 360 ppm. No data were found on the occurrence of boron compounds in surface or groundwater. While current drinking water surveys do not report any levels of boron, it has been found in tap water in the past. Levels reported in drinking water were less than 1-3 ppm. There is potential for exposure to boron through contact with soil, since boron sticks to soil particles. Background levels up to 300 ppm have been reported. Exposure to air contaminated with boron is not likely to occur in the general population; however, there is risk of exposure to borate dust in the workplace. Concentrations from 1-14 milligrams of boron dust per cubic meter of air (mg/m^3) have been reported in borax mining and refining plants and at sites where boric acid is manufactured. Exposure to boron may also occur from the use of consumer products, including cosmetics, topical medical preparations, and some laundry products. The average daily boron intake has been estimated to be 10-25 mg.

Further information on how you might be exposed to boron is given in Chapter 5.

1.3 HOW CAN BORON ENTER AND LEAVE MY BODY?

Boron can enter your body when you eat food (fruit and vegetables) breathe borate dust in the air, and when damaged skin comes in contact with it. Because very small amounts of boron are present in all drinking water, boron can enter your body when you drink water. When boron enters the body by mouth or when you breathe borate dust, it goes to the intestines where it is passed to various parts of the body including the liver, brain, and kidney. No information is available on what factors affect how fast boron enters the body. However, animal studies suggest boron readily enters the body after contact with damaged skin. Most of the boron leaves the body in urine

1. PUBLIC HEALTH STATEMENT

primarily from food eaten. Over half of the boron taken by mouth can be found in urine within 24 hours and the other half can be detected for up to 4 days. Boron compounds can be found in urine up to 23 days if you are accidentally exposed to very large amounts.

Further information on how boron enters and leaves the body is given in Chapter 2.

1.4 HOW CAN BORON AFFECT MY HEALTH?

If humans eat large amounts of boron (4,161 ppm) over short periods of time, it can affect the stomach, intestines, liver, kidney, and brain and can eventually lead to death. Irritation of the nose and throat or eyes can occur if small amounts of boron (4.1 mg/m³) are breathed in. Boron can irritate the eyes if it comes in contact with them for long periods of time. Animal studies indicate that the male reproductive organs, especially the testes, are affected if large amounts of boron are eaten or drunk for short or long periods of time. Studies in animals also indicate delayed development and structural defects in offspring, primarily in the rib cage, from maternal exposure to boron during pregnancy. These effects have not been seen in humans. Irritation of the nose can occur in animals if large amounts of boron are breathed in for long periods of time. These effects have not been seen in humans. No information is available on whether boron is likely to cause cancer in humans. There is no evidence of cancer in animals exposed to boron for long periods of time.

More information on the health effects of boron in humans and animals can be found in Chapter 2.

1.5 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO BORON?

There are reliable and accurate ways of measuring boron in the body. Blood and urine can be examined to determine if excessive exposure to boron has occurred. Boron and, to a limited extent, boron-related compounds can be measured in body fluids. However, special equipment is needed for detection and analysis. Tests are not routinely available in a doctor's office. It is not known whether boron levels measured in the body can be used to predict potential health effects.

Further information on how boron can be measured in exposed humans is presented in Chapters 2 and 6.

1.6 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government has set regulatory standards and guidelines to protect individuals from the effects that may occur if exposed to boron. The EPA has established tolerances for total boron of 30 ppm in or on cottonseed and 8 ppm in or on citrus fruits. The Food and Drug Administration has

1. PUBLIC HEALTH STATEMENT

designated that borax and boric acid are generally recognized as safe (GRAS) as indirect food additives in adhesive components, components of paper, paperboard, sizing and coatings. The Occupational Safety and Health Administration (OSHA) has set a permissible exposure limit of 10 mg/m³ for boron oxide and sodium tetraborate in the workplace air for 8 hour/day exposures over a 40-hour work week. Limits of 10 mg/m³ for boron tribromide and 3 mg/m³ for boron trifluoride have been set.

Additional information on governmental regulations regarding boron can be found in Chapter 7.

1.7 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns not covered here, please contact your state health or environmental department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. Such clinics specialize in recognizing, evaluating, and treating illnesses that result from exposure to hazardous substances.

2. HEALTH EFFECTS

2.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective of the toxicology of boron and compounds and a depiction of significant exposure levels associated with various adverse health effects. It contains descriptions and evaluations of studies and presents levels of significant exposure for boron based on toxicological studies and epidemiological investigations.

2.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure-- inhalation, oral, and dermal--and then by health effect--death, systemic, immunological, neurological, developmental, reproductive, genotoxic, and carcinogenic effects. These data are discussed in terms of three exposure periods--acute (less than 15 days), intermediate(15-364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies. LOAELs have been classified into "less serious" or "serious" effects. These distinctions are intended to help the users of the document identify the levels of exposure at which adverse health effects start to appear. They should also help to determine whether or not the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the tables and figures may differ depending on the user's perspective. For example, physicians concerned with the interpretation of clinical findings in exposed persons may be interested in levels of exposure associated with "serious" effects. Public health officials and project managers concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAEL) or exposure levels below which no adverse effects (NOAEL) have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels, MRLs) may be of interest to health professionals and citizens alike.

Estimates of exposure levels posing minimal risk to humans (MRLs) have been made, where data were believed reliable, for the most sensitive noncancer effect for each exposure duration. MRLs include adjustments to reflect human variability from laboratory animal data to humans.

Although methods have been established to derive these levels (Barnes et al. 1988; EPA 1989a), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the

2. HEALTH EFFECTS

application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs will be revised.

2.2.1 Inhalation Exposure

2.2.1.1 Death

No studies were located regarding death in humans or animals after inhalation exposure to boron.

2.2.1.2 Systemic Effects

No studies were located regarding cardiovascular, gastrointestinal, hematological, musculoskeletal, or renal effects in humans after inhalation exposure to boron. No studies were located regarding dermal/ocular effects after acute inhalation exposure in humans or animals for any duration category.

Information on respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, renal, and dermal/ocular effects is discussed below. The highest NOAEL values and all reliable LOAEL values for these systemic effects for each species and duration category are recorded in Table 2-1 and plotted in Figure 2-1.

Respiratory Effects. Boron (as boron oxide and boric acid dusts) has been shown to cause irritation of the upper respiratory tract in humans. Based on a medical questionnaire from 113 workers (96% males, 4% females) employed in the borax industry for an average of 11 years, mean exposures of 4.1 mg/m³ to boron oxide and boric acid dusts were associated with dryness of the mouth, nose, or throat, sore throat, and productive cough (Garabrant et al. 1984). While the authors reported differences between the test and control groups in age and numbers of smokers, no differences in symptoms were observed. Similarly, symptoms of acute respiratory irritation were related to exposures to borax dust at concentrations of 4 mg/m³ or more in a crosssectional study of 629 borax workers actively employed for 11.4 years (Garabrant et al. 1985). Decreases in the forced expiratory volume (FEV₁) were seen among smokers who had cumulative borax exposures of 80 mg/m³ or greater but were not seen among less exposed smokers or among nonsmokers. Radiographic abnormalities were not found. It was determined in a follow-up of the Garabrant et al. 1985 study that the cumulative borax exposure effect

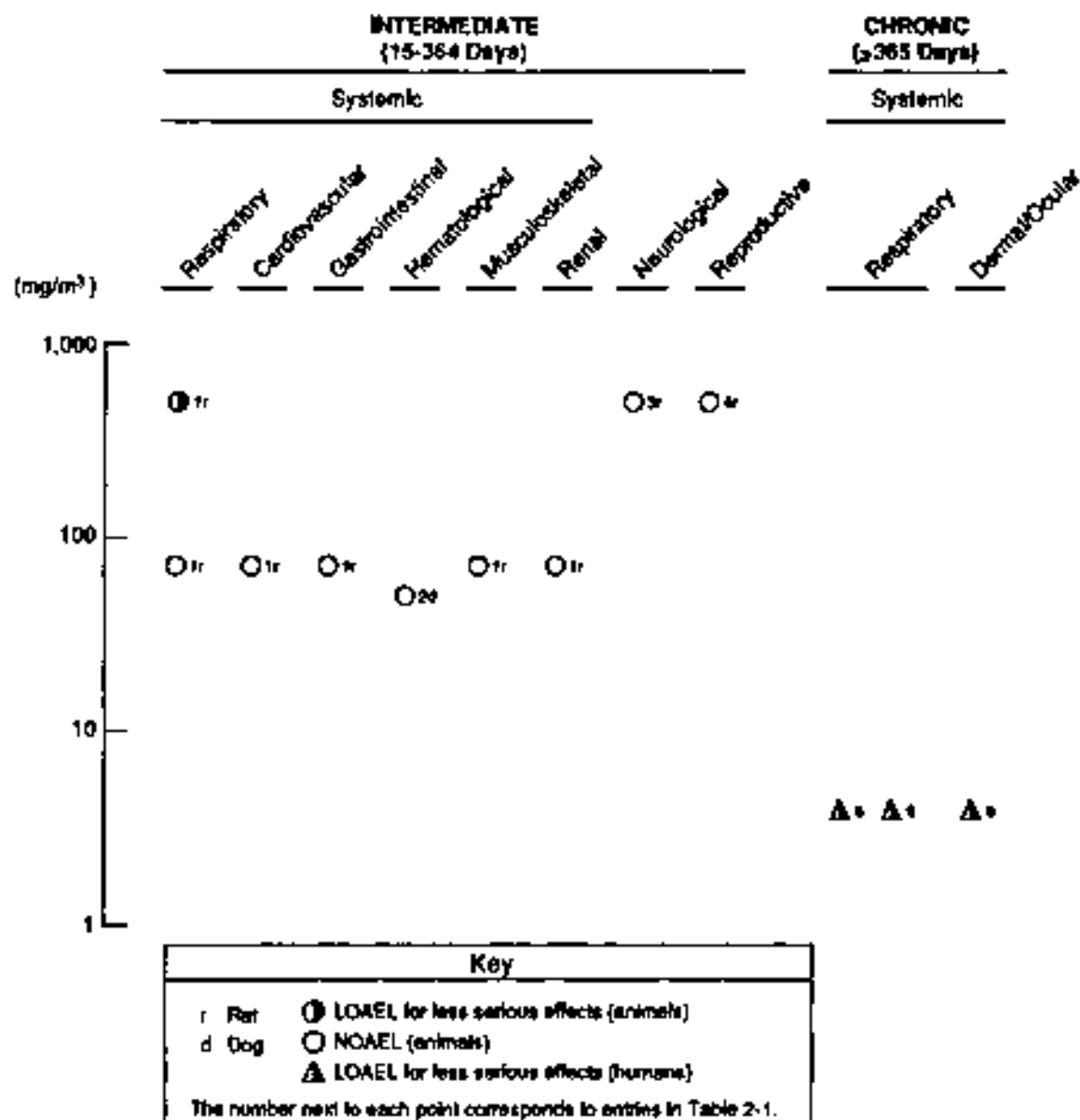
TABLE 2-1. Levels of Significant Exposure to Boron and Compounds - Inhalation

Key to Figure ^a	Species	Exposure frequency/duration	System	NOAEL (mg/m ³)	LOAEL (effect)		Reference	Form
					Less serious (mg/m ³)	Serious (mg/m ³)		
INTERMEDIATE EXPOSURE								
Systemic								
1	Rat	6-24 wk 5d/wk 6hr/d	Resp Cardio Musc/skel Renal Gastro	?? ?? ?? ?? ??	470 (respiratory irritation)		Wilding et al. 1959	BO
2	Dog	23 wk	Hemato	57			Wilding et al. 1959	BO
Neurological								
3	Rat	6-24 wk 5d/wk 6hr/d		470			Wilding et al. 1959	BO
Reproductive								
4	Rat	6-24 wk 5d/wk 6hr/d		470			Wilding et al. 1959	BO
CHRONIC EXPOSURE								
Systemic								
5	Human	11.4 yr (mean)	Resp		4.1 (respiratory irritation)		Casabrant et al. 1985	BX
6	Human	11.4 yr (mean)	Resp Derm/oc		4.1 (respiratory irritation) 4.1 (eye irritation)		Casabrant et al. 1984	BX, BA, BO

^aThe number corresponds to entries in Figure 2-1.

BA = boric acid; BO = boron oxide; BX = boron; Cardio = cardiovascular; d = day(s); Derm/oc = dermal/ocular; Gastro = gastrointestinal; Hemato = hematological; hr = hour(s); LOAEL = lowest-observed-adverse-effect level; Musc/skel = musculoskeletal; NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week(s); yr = year(s).

FIGURE 2-1. Levels of Significant Exposure to Boron and Compounds – Inhalation



2. HEALTH EFFECTS

found previously was probably due to smoking workers with longer boron work histories and who smoke disproportionately more than those with shorter work histories. There was no indication that borax exposure at the levels studied (up to 15 mg/m³) impaired pulmonary function (Wegman et al. 1991). Direct irritation to mucous membranes of the nose and throat was also studied by Wegman et al. (1991) using an irritation scoring system together with realtime measurements of borax exposure concentrations. The study concluded that borates 'are mild irritants. However, these effects are likely to occur at concentrations exceeding 10 mg/m³ (OSHA Permissible Exposure Limit).

Animal studies suggest that the respiratory tract is susceptible to boron toxicity. Rats exposed to 470 mg/m³ boron oxide aerosol for 10 weeks developed reddish exudates from their noses, but there were no deaths or signs of lung damage (Wilding et al. 1959). No changes were observed in rats in the 77 mg/m³ dose group after 24 weeks of exposure, or in dogs exposed to a concentration of 57 mg/m³ for 23 weeks (Wilding et al. 1959).

Cardiovascular Effects. Animal data are sparse. Rats exposed to aerosols of boron oxide at a concentration of 77 mg/m³ for 6 weeks showed no histopathological effects in the cardiovascular system (Wilding et al. 1959).

Gastrointestinal Effects. Animal data are sparse. No changes were seen in the gastrointestinal tract of rats exposed to aerosols of boron oxide at a concentration of 77 mg/m³ for 6 weeks (Wilding et al. 1959).

Hematological Effects. Little is known concerning the effects of boron in animals. Rats exposed to aerosols of boron oxide for 10-24 weeks (up to 470 mg/m³) and dogs for 23 weeks (57 mg/m³) showed no significant changes in total red and white blood cell count, hemoglobin, hematocrit, and differential count (Wilding et al. 1959).

Musculoskeletal Effects. Animal data are sparse. No histopathological effects of exposure were observed in the femur, rib, and muscle of rats exposed to aerosols of boron oxide at a concentration of 77 mg/m³ for 6 weeks (Wilding et al. 1959).

Renal Effects. Data on the effects of boron in animals are sparse. No renal effects were observed in rats exposed to aerosols of boron oxide at a concentration of 77 mg/m³ for 6 weeks (Wilding et al. 1959).

Dermal/Ocular Effects. Human occupational exposure to a mean concentration of 4.1 mg/m³ (as boron oxide and boric acid dust) produced eye irritation following chronic exposures in workers employed for an average of 11 years (Garabrant et al. 1984, 1985).

2.2.1.3 Immunological Effects

No studies were located regarding immunological effects in humans or animals after inhalation exposure to boron.

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2.2.1.4 Neurological Effects

No studies were located regarding neurological effects in humans after inhalation exposure to boron. Adverse effects were not found on the brain of rats exposed to aerosols of boron oxide at a concentration of 77 mg/m³ for 6 weeks (Wilding et al. 1959).

2.2.1.5 Developmental Effects

No studies were located regarding developmental effects in humans or animals after inhalation exposure to boron.

2.2.1.6 Reproductive Effects

Limited data were located regarding reproductive effects in humans after inhalation exposure to boron. One study was reported involving occupational exposure (10 years or greater) to boron aerosols (22-80 mg/m³) in males engaged in the production of boric acids (Tarasenko et al. 1972). The study group was small, consisting of 28 men. Low sperm counts, reduced sperm motility and elevated fructose content of seminal fluids were observed.

In animals, no effects were found on the ovary or testes of rats exposed to aerosols of boron oxide at a concentration of 77 mg/m³ for 6 weeks (Wilding et al. 1959).

2.2.1.7 Genotoxic Effects

No studies were located regarding the genotoxic effects in humans or animals after inhalation exposure to boron. Genotoxicity studies are discussed in Section 2.4.

2.2.1.8 Cancer

No studies were located regarding cancer in humans or animals after inhalation exposure to boron.

2.2.2 Oral Exposure

2.2.2.1 Death

Studies in humans, particularly infants, show that boron (as boric acid) can be lethal following ingestion. Infants who ingested formula accidentally prepared with 2.5% aqueous solution of boric acid died within 3 days after exposure (Wong et al. 1964). It was estimated that the amount of boric acid consumed ranged from 4.51 to 14 g. Although 5 of 11 infants died, the authors provided histopathological data and weights for only 2 infants who had ingested 9.25 g (505 mg boron/kg/day) and 14 g (765 mg boron/kg/day) (Wong et al. 1964). Infants became lethargic and developed vomiting and diarrhea.

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Degenerative changes were seen in the liver, kidney, and brain. Acute exposure to dose levels of 895 mg boron/kg as boric acid was not lethal in one adult (Linden et al. 1986).

In animals, boron (as boric acid and borax) is lethal following acute, intermediate, and chronic oral exposures. Estimates of oral LD₅₀ in rats were 898 and 642 mg boron/kg (as boric acid and borax, respectively) (Smyth et al. 1969) and 510 and 550 mg boron/kg as borax and boric acid (Weir and Fisher 1972). No deaths were reported in dogs exposed to 696 mg boron/kg as boric acid and 738 mg boron/kg as borax (Weir and Fisher 1972). In a 14-day repeated-dose feeding study in male mice, doses of 2,251 and 3,671 mg boron/kg/day (as boric acid) were lethal in 20% and 60% of males, respectively (NTP 1987). The mice were lethargic and the spleen, liver, and renal medullae were discolored. Hyperplasia and dysplasia of the forestomach were also observed (NTP 1987).

Survival was also reduced in mice following intermediate-duration exposure. Males (10%) died after exposure to a dose of 288 mg boron/kg/day (as boric acid) in the diet, while 80% of males and 60% of females died at 577 mg boron/kg/day (NTP 1987). Hyperkeratosis and/or acanthosis in the stomach and extramedullary hematopoiesis of the spleen in both sexes were observed at the highest dose tested (577 mg boron/kg/day). There was 100% mortality in rats fed 263 mg boron/kg/day for 90 days (Weir and Fisher 1972). Congestion of liver and kidneys, small gonads, and brain swelling were reported. When male mice consumed 48 and 96 mg boron/kg/day (as boric acid) for 103 weeks, mortality was 40% and 56%, respectively, compared to 18% in untreated controls (NTP 1987). No clinical signs were reported; however, boron caused increased incidence of testicular atrophy and interstitial hyperplasia. Mortality in female mice was 30% and 24% (48 and 96 mg boron/kg/day) compared to 34% in the untreated controls (NTP 1987).

The LD₅₀ values and the highest NOAEL values in animals and the lowest level at which death was reported in humans and the duration categories are recorded in Table 2-2 and plotted in Figure 2-2.

2.2.2.2 Systemic Effects

No studies were located regarding respiratory effects in animals or cardiovascular or musculoskeletal effects in humans or animals after oral exposure to boron.

Information on respiratory, gastrointestinal, hematological, hepatic, renal, and dermal/ocular effects is discussed below. The highest NOAEL values and all reliable LOAEL values for these systemic effects for each species and duration category are recorded in Table 2-2 and plotted in Figure 2-2.

Respiratory Effects. Widespread vascular congestion and hemorrhages in the lungs were reported in one infant who ingested 505 mg boron/kg/day (Wong et al. 1964).

TABLE 2-2. Levels of Significant Exposure to Boron and Compounds - Oral

Key to Figure ^a	Species	Route	Exposure Frequency/ duration	System	NOAEL (mg/kg/day)	LOAEL (effect)		Reference	Form
						Less serious (mg/kg/day)	Serious (mg/kg/day)		
ACUTE EXPOSURE									
Death									
1	Human	(F)	3-5 d				305 (increased mortality)	Wong et al. 1964	BA
2	Rat	(F)	1 d				550 (LD50)	Weir and Fisher 1972	BA
3	Rat	(M)	1 d				642 (LD50)	Smyth et al. 1969	BX
4	Rat	(F)	1 d				510 (LD50)	Weir and Fisher 1972	BX
5	Rat	(M)	1 d				898 (LD50)	Smyth et al. 1969	BA
6	Horse	(F)	14 d				2231 (increased mortality) [ty]	NTP 1987	BA
7	Dog	(C)	1 d		738			Weir and Fisher 1972	BX
8	Dog	(C)	1 d		698			Weir and Fisher 1972	BA
Systemic									
9	Human	(F)	3-5 d	Resp			305 (vascular congestion, hemorrhage in infants)	Wong et al. 1964	BA
				Hepatic			505 (parenchymatous degeneration, jaundice, fatty changes, congestion in infants)		
				Renal			765 (parenchymatous degeneration, reduced urine output, protein in urine in infants)		
				Dermatoc		505 (extensive shedding of skin)			

TABLE 2-1 (Continued)

Key to Figure*	Species	Route	Exposure frequency/duration	System	NOAEL (mg/kg/day)	LOAEL (effect)		Reference	Form
						Less serious (mg/kg/day)	Serious (mg/kg/day)		
10	Human	(F)	3-5 d	Gastro	104	(vomiting, diarrhea in infants)		Wong et al. 1964	BA
				Dermat	505	(erythema, desquamation in infants)			
11	Human	(F)	1 d	Gastro	241	(vomiting, diarrhea)		Linden et al. 1986	BA
12	Mouse	(F)	14 d	Gastro	2251	(gastric hyperplasia and dysplasia)		NTP 1987	BA
Neurological									
13	Human	(F)	3-5 d				505 (perivascular hemorrhage, congestion, thrombosis, edema in infants)	Wong et al. 1964	BA
INTERMEDIATE EXPOSURE									
Death									
14	Rat	(F)	90 d				263 (100% mortality)	Wair and Fisher 1972	BX
15	Rat	(F)	90 d				263 (100% mortality)	Wair and Fisher 1972	BA
16	Mouse	(F)	13 wk				144 (increased mortality)	NTP 1987	BA
Systemic									
17	Rat	(F)	90 d	Other	88			Wair and Fisher 1972	BX
18	Rat	(M)	1-14 wk	Hepatic	20.8			Sekimi et al. 1982	BX
19	Rat	(M)	70 d	Other		23.7 (decreased body and spleen weights)		Seal and Wuech 1980	BX

TABLE 2-2 (Continued)

Key to Figure*	Species	Route	Exposure frequency/ duration	System	NOAEL (mg/kg/day)	LOAEL (effect)		Reference	Form
						Less serious (mg/kg/day)	Serious (mg/kg/day)		
20	Rat	(F)	90 d	Other		55 (decreased body weight)		Walt and Fisher 1972	BA
21	Dog	(F)	90 d	Other	44			Walt and Fisher 1972	BA
22	Dog	(F)	90 d	Hemato	4.4	44 (decreased packed cell volume and hemoglobin)		Walt and Fisher 1972	BX
Neurological									
23	Rat	(M)	3-14 wk		20.8			Sattini et al. 1982	BX
Developmental									
24	Rat	(F)	20 d			13.6* (reduced fetal weight)	28.4 (rib cage defects, increased resorptions)	Reindel et al. 1991	BA
25	Mouse	(F)	17 d		43.4	79 (reduced fetal body weight)	135.3 (skeletal effects, increased resorptions)	Reindel et al. 1991	BA
Reproductive									
26	Rat	(F)	30-60 d		30		100 (testicular atrophy, decreased enzymes)	Lee et al. 1978	BX
27	Rat	(M)	90 d		0.6			Dixon et al. 1976	BX
28	Rat	(F)	90 d			26 (partial testicular atrophy)	88 (complete atrophy of testes)	Walt and Fisher 1972	BA
29	Rat	(F)	60 d		25	50 (reduced testicular enzymes, reduced testicular and epididymal weight)		Dixon et al. 1979	BX

TABLE 2-2 (Continued)

Key to figure ^a	Species	Route	Exposure frequency/duration	System	NOAEL (mg/kg/day)	LOAEL (effect)		Reference	Form
						Less serious (mg/kg/day)	Serious (mg/kg/day)		
30	Rat	(F)	90 d			26 (partial testicular atrophy)	88 (complete atrophy of testes)	Weir and Fisher 1972	BK
31	Rat	(W)	70 d			46.7 (impaired spermatogenesis)		Swai and Heath 1980	BK
32	Mouse	(F)	13 wk				288 (degeneration or atrophy of seminiferous tubules)	MTP 1987	BA
33	Mouse	(F)	27 wk		26.5	181 (impaired spermatogenesis, degeneration of seminiferous tubules)		NIHMS 1990	BA
34	Dog	(F)	38 wk			29 (testicular atrophy, spermatogenic arrest)		Weir and Fisher 1972	BK
35	Dog	(F)	90 d		4.4	44 (severe testicular atrophy)		Weir and Fisher 1972	BK
36	Dog	(F)	38 wk			29 (testicular atrophy, spermatogenic arrest)		Weir and Fisher 1972	BA
37	Dog	(F)	90 d		4.4	44 (severe testicular atrophy)		Weir and Fisher 1972	BA
CHRONIC EXPOSURE									
Death									
38	Mouse	(F)	103 wk				56 (40% mortality)	MTP 1987	BA
Reproductive									
39	Rat	(F)	2 yr		17.5		58.5 (atrophy of testes, decreased testis weight)	Weir and Fisher 1972	BK

TABLE 2-2 (Continued)

Key to figure*	Species	Route	Exposure frequency/ duration	System	NOAEL (mg/kg/day)	LOAEL (effect)		Reference	Form
						Less serious (mg/kg/day)	Serious (mg/kg/day)		
40	Rat	(F)	1 gen		17.5		58.5 (atrophy of testes, decreased ovulation)	Weir and Fisher 1972	BA
41	Rat	(F)	3 gen		17.5		58.5 (atrophy of testes, decreased ovulation)	Weir and Fisher 1972	BX
42	Rat	(F)	2 yr		17.5		58.5 (atrophy of seminiferous tubule epithelium, decreased tubule size, decreased testicular weight)	Weir and Fisher 1972	BA
43	Mouse	(F)	103 wk		48		96 (testicular atrophy, interstitial hyperplasia)	MTP 1987	BA
44	Dog	(F)	2 yr		8.75			Weir and Fisher 1972	BX
45	Dog	(F)	2 yr		8.75			Weir and Fisher 1972	BA

*The number corresponds to entries in Figure 2-2

*Used to derive an intermediate oral MRL of 0.01 mg/kg/day: dose divided by an uncertainty factor of 1000 (10 for use of a LOAEL, 10 for extrapolation from animals to humans, and 10 for human variability).

BA = boric acid; BX = borax; (C) = capsule; d = day(s); Derm/oc = dermal/ocular; (F) = feed; Gastro = gastrointestinal; gen = generation; LD50 = lethal dose, 50% kill; LOAEL = lowest-observed-adverse-effect level; NOAEL = no-observed-adverse-effect level; Resp = respiratory; (W) = water; wk = week(s); yr = year(s)

FIGURE 2-2. Levels of Significant Exposure to Boron and Compounds – Oral

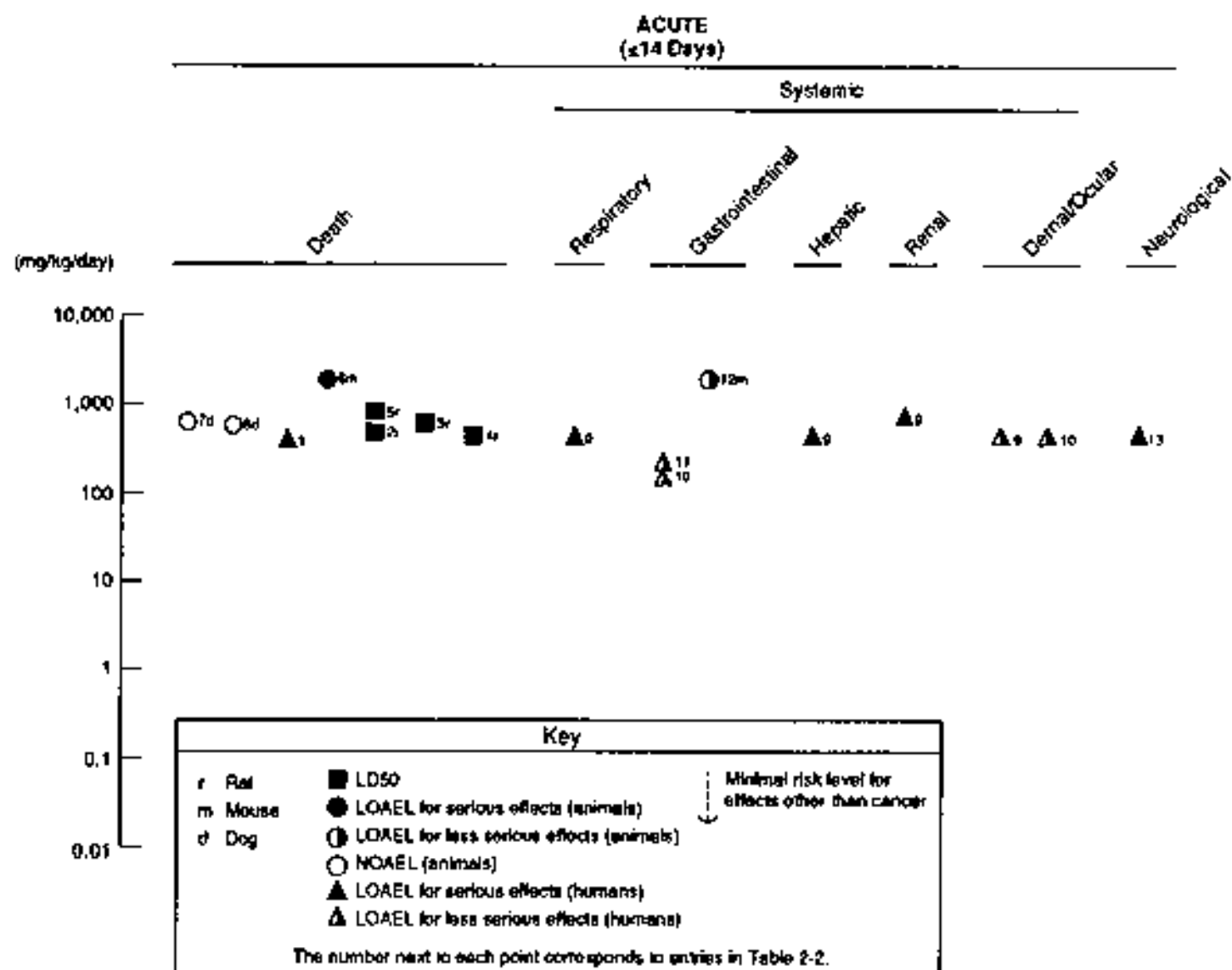


FIGURE 2-2 (Continued)

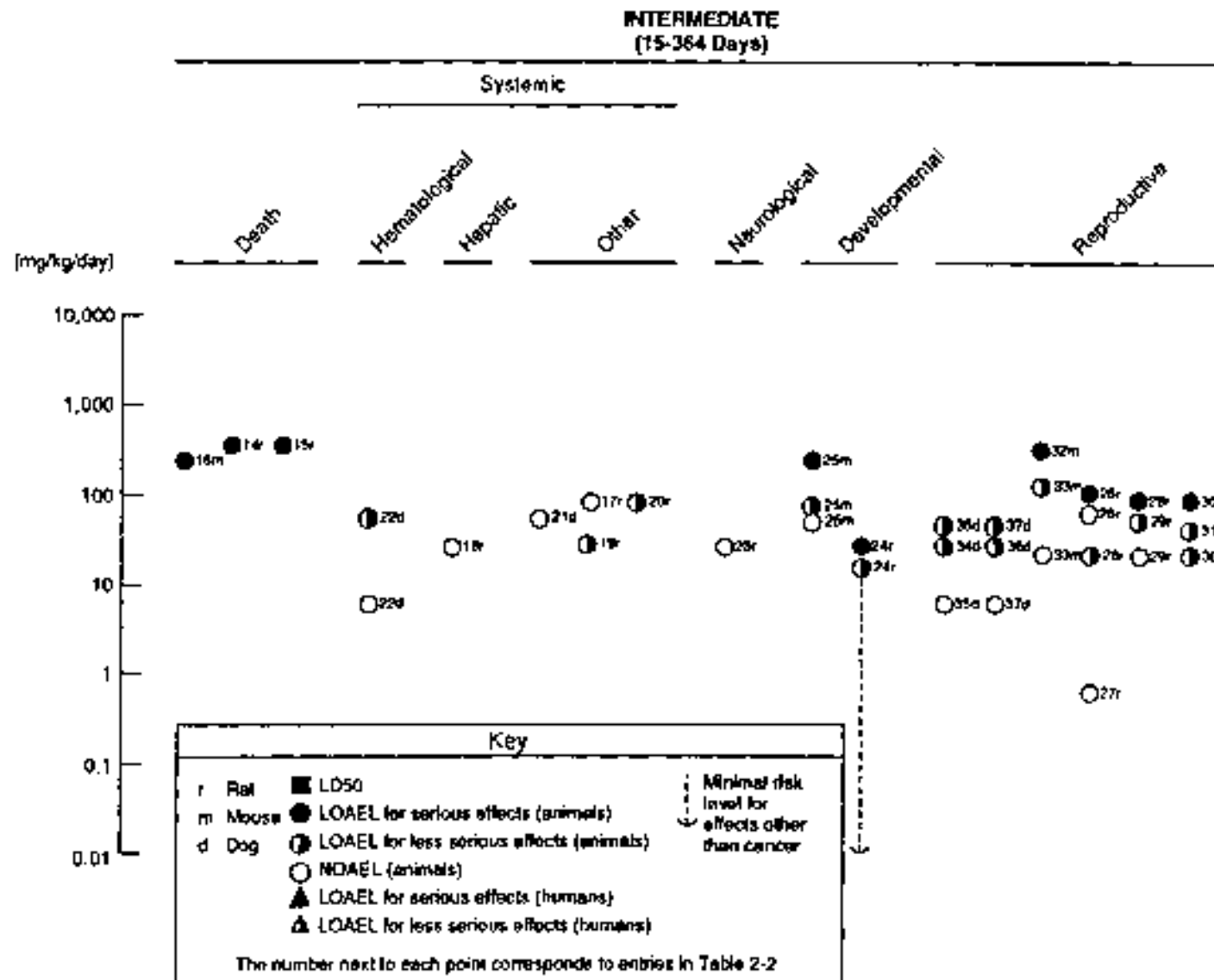
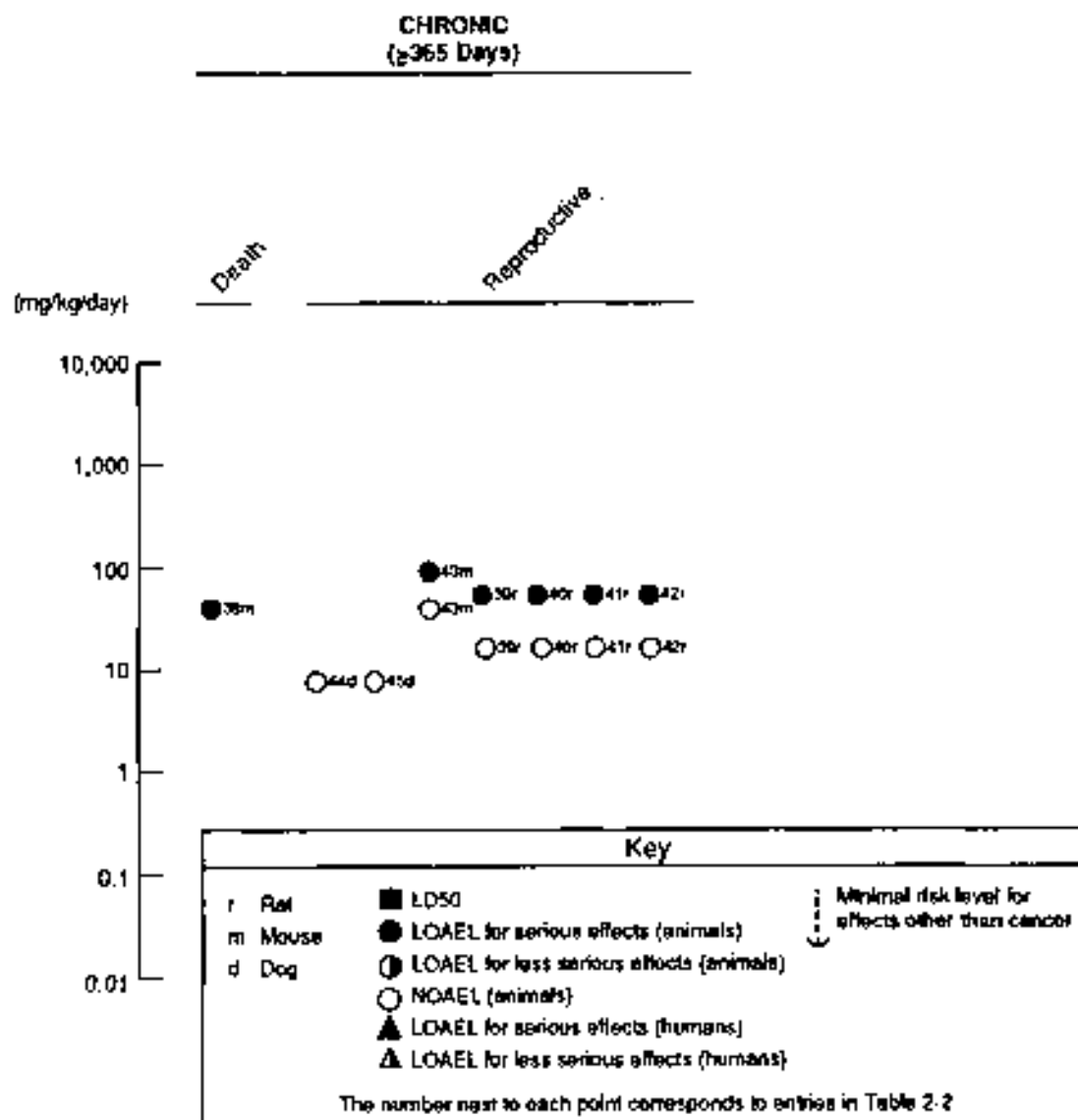


FIGURE 2-2 (Continued)



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Gastrointestinal Effects. Ingestion of boron in humans can cause gastrointestinal effects. Nausea, persistent vomiting, diarrhea, and colicky abdominal pain in infants were associated with acute ingestion of a total of 184 mg boron/kg/day or greater (based on 1.9 kg body weight) as boric acid which was accidentally incorporated in infant formula (Wong et al. 1964). Vomiting was the only sign-of boron toxicity in two adult females who had ingested 241 mg boron/kg/day as boric acid in a fungicide and 895 mg boron/kg of a boric acid-containing insecticide in a suicide attempt, The subjects were hospitalized for 24-96 hours and did not develop further symptoms following release (Linden et al. 1986).

Hematological Effects. Two male and three female dogs fed 44 mg boron/kg/day as borax had decreased packed cell volume and hemoglobin values. Erythrocyte count, total and differential leucocyte counts were comparable to control levels (Weir and Fisher 1972).

Hepatic Effects. Case reports in humans suggest that the liver is susceptible to boron toxicity at high dose levels (Wong et al. 1964). Jaundice has been reported, and there were mild alterations at histological examination in infants who ingested 505 or 765 mg boron/kg/day as boric acid (accidentally incorporated in infant formula) for 3-5 days (Wong et al. 1964). In the same incident, congestion and fatty changes were observed, and there was parenchymatous degeneration in newborn infants who ingested 505 or 765 mg boron/kg as boric acid for 3-5 days (Wong et al. 1964).

In rats given approximately 20.8 mg boron/kg/day as borax in drinking water, NADPH-cytochrome C reductase activity and cytochrome b, content decreased in the liver microsomal fraction after 10 and 14 weeks (Settimi et al. 1982). There was also a reduction in the cytochrome P-450 concentration detected at 14 weeks (Settimi et al. 1982).

Renal Effects. Human case reports involving high accidental ingestion levels show that boron can cause injury to the kidney. Degenerative changes in parenchymal cells with oliguria and albuminuria have been demonstrated in two newborn infants after ingestion of 505 and 765 mg boron/kg/day as boric acid in an evaporated milk formula over a period of 3-5 days (Wong et al. 1964).

Dermal/Ocular Effects. Skin effects can occur following ingestion of boron (as boric acid) in humans. Extensive exfoliative dermatitis began in infants as an erythema involving palms, soles, and buttocks. It eventually became generalized with subsequent bulbous formation, massive desquamation, and sloughing (Wong et al. 1964). These changes were associated with ingestion of 505 mg boron/kg/day; however, skin lesions were lacking following ingestion of 765 mg boron/kg/day. Similarly, extensive erythema with desquamation was observed in an adult who ingested boric acid powder (Schillinger et al. 1982). The exact amount ingested was not stated. However, 14 g (equivalent to 22.5 mg boron/kg based on 109 kg body weight) was measured as missing from a container from which the patient admitted consuming half its contents.

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In animals, rats fed 88 and 263 mg boron/kg/day as borax or boric acid had inflamed eyes and skin desquamations on the paws and tails (Weir and Fisher 1972).

2.2.2.3 Immunological Effects

No studies were located regarding immunological effects in humans or animals after oral exposure to boron.

2.2.2.4 Neurological Effects

Case reports in humans have indicated neurological effects after accidental ingestion of high levels of boron (as boric acid). Newborn infants who ingested 4.5-14 g boric acid showed central nervous system involvement manifested by headache, tremors, restlessness, and convulsions followed by weakness and coma (Wong et al. 1964). Histological examination of 2 of 11 infants revealed congestion and edema of brain and meninges with perivascular hemorrhage and intravascular thrombosis at a dose ≥ 505 mg boron/kg/day (Wong et al. 1964). Seizure disorders have been associated with boron exposures (as borax) in infants who ingested 4-30 g borax for 4-10 weeks (O'Sullivan and Taylor 1983) and 9-125 g borax for 5-12 weeks (Gordon et al. 1973). Estimates of boron consumption could not be determined since the authors did not provide data on kilogram body weights. Blood boron levels in patients who ingested borax ranged from 2.6 to 8.5 $\mu\text{g/mL}$ (O'Sullivan and Taylor 1983). In one infant with a seizure disorder who ingested borax for 3 months, the blood boron level was 1.64 mg/100 mL (Gordon et al. 1973).

In rats, exposure to approximately 20.8 mg boron/kg/day as borax (based on weight of 0.35 kg and average water consumption of 20.7 mL) in drinking water for up to 14 weeks caused increased cerebral succinate dehydrogenase activity after 10 and 14 weeks of exposure (Settimi et al. 1982). Increased RNA concentration and increased acid proteinase activity in brain occurred after 14 weeks (Settimi et al. 1982).

All LOAEL values for neurological effects in humans and animals are recorded in Table 2-2 and plotted in Figure 2-2.

2.2.2.5 Developmental Effects

No studies were located regarding developmental effects in humans after oral exposure to boron.

In animals, fetotoxicity was observed in rats and mice. The average fetal body weight per litter in rats was reduced in pups of dams administered 13.6 mg boron/kg/day or greater (78 mg/kg/day boric acid) on gestation days 0 to 20 (Heindel et al. 1991). Similarly, pups of mice administered 79 mg boron/kg/day (452 mg/kg/day boric acid) showed reduced body weight. Boron was also teratogenic in rats and mice. There was agenesis or shortening of rib XIII and the lateral ventricles of the brain were enlarged in rats at dose

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levels of 28.4 mg boron/kg/day (163 mg/kg/day boric acid) or greater (Heindel et al. 1991). Skeletal effects were reported at the highest dose tested (175.3 mg boron/kg/day or 1,003 mg/kg/day boric acid) in mice. No effects were observed in the 43.4 mg boron/kg/day (248 mg/kg/day boric acid) dose group (Heindel et al. 1991). Based on a value of 13.6 mg boron/kg/day, an intermediate oral MRL of 0.01 mg/kg/day was calculated as described in the footnote on Table 2-2.

2.2.2.6 Reproductive Effects

No studies were located regarding reproductive effects in humans after oral exposure to boron.

Animal studies demonstrated that boron can cause injury after intermediate and chronic exposure to the gonads in animals, especially the testes. Impaired spermatogenesis has been reported in rats administered 300 mg/boron/L as borax (44.7 mg boron/kg/day) in drinking water for 70 days (Seal and Weeth 1980), but no reproductive effects were evident in rats administered up to 6 mg boron/L of borax (0.6 mg boron/kg/day) in drinking water for 90 days (Dixon et al. 1976). While severe testicular atrophy was seen in dogs fed up to 44 mg boron/kg/day (1,750 ppm boron, as borax or boric acid) for 90 days (Weir and Fisher 1972), partial testicular atrophy in rats occurred at a dose of 26 mg boron/kg/day (525 ppm boron) (Weir and Fisher 1972). Degeneration or atrophy of the seminiferous tubules was demonstrated in mice fed 144 mg boron/kg/day as boric acid (5,000 ppm boric acid) (NTP 1987). In rats fed at least 50 mg boron/kg/day (as borax) up to 60 days, there were reduced testicular weight and germinal aplasia at 60 days (Dixon et al. 1979). In the same study, ≥ 50 mg boron/kg/day caused reduction in hyaluronidase, sorbitol dehydrogenase, and lactic acid dehydrogenase (isoenzyme-X) at 30 days and testicular and epididymal weights were reduced (Dixon et al. 1979).

In contrast, Lee et al. (1978) did not find significant adverse effects in male rats fed 50 mg boron/kg/day (as borax) for 30 and 60 days. Dogs were fed 29 mg boron/kg/day as borax and boric acid (1,170 ppm), respectively in the diet for 38 weeks (Weir and Fisher 1972). Testicular atrophy and spermatogenic arrest were reported. When dogs were administered 8.8 mg boron/kg/day (350 ppm borax or boric acid) for 2 years, no reproductive effects were observed (Weir and Fisher 1972). Reproductive effects were reported in rats following chronic exposure. In rats fed up to 58.5 mg boron/kg/day (as borax or boric acid) for several generations, there was a lack of viable sperm in atrophied testes and ovulation decreased in females (Weir and Fisher 1972). There were testicular atrophy and interstitial hyperplasia in mice that consumed lethal doses (48 and 96 mg boron/kg/day) over a period of 103 weeks. However, the authors did not specify cause of death (NTP 1987). In a 2-generation reproduction mouse study using continuous breeding protocol, there was degeneration of the seminiferous tubules and spermatogenesis was impaired at dose levels of 111 mg boron/kg/day (636 mg/kg/day boric acid) or greater. No effects were observed in the 27 mg boron/kg/day (152 mg/kg/day boric acid) dose group (NIEHS 1990).

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The highest NOAEL values and all reliable LOAEL values for reproductive effects in animals and duration category are recorded in Table 2-2 and plotted in Figure 2-2.

2.2.2.7 Genotoxic Effects

No studies were located regarding genotoxic effects in humans and animals after oral exposure to boron. Genotoxicity studies are discussed in Section 2.4.

2.2.2.8 Cancer

No studies were located regarding cancer in humans after oral exposure to boron.

In a life-time bioassay in which male and female B6C3F₁ mice consumed 48 mg boron/kg/day or 96 mg boron/kg/day as boric acid in the diet, there was no evidence of carcinogenicity (NTP 1987).

2.2.3 Dermal Exposure

2.2.3.1 Death

No studies were located regarding death in humans or animals after dermal exposure to boron.

2.2.3.2 Systemic Effects

No studies were located regarding hematological and dermal/ocular effects in humans and respiratory, cardiovascular, gastrointestinal, musculoskeletal, hepatic, or renal effects in humans or animals after dermal exposure to boron.

All reliable LOAEL values for systemic effects in each species and duration category are recorded in Table 2-3.

Hematological Effects. Data are sparse in animals. It was reported in Draize and Kelley (1959) that the application of 25-200 mg/kg/day boric acid in aqueous solution did not produce hematological changes when rubbed onto intact skin during a 90-day rabbit study. No quantitative data were provided; therefore, these results could not be evaluated.

Dermal/Ocular Effects. Animal studies show that boron oxide dust can affect the eye and skin. Instillation of boron oxide dust (50 mg) into the eyes of four rabbits produced conjunctivitis (Wilding et al. 1959). Application of 1 g boron oxide dust to a 25 cm² area of the skin of four rabbits produced erythema that lasted for 2-3 days (Wilding et al. 1959).

TABLE 2-3. Levels of Significant Exposure to Boron and Compounds - Dermal

Species	Exposure frequency/duration	System	NOAEL (mg/kg/day)	LOAEL (effect)		Reference	Form
				Less serious (mg/kg/day)	Serious (mg/kg/day)		
ACUTE EXPOSURE							
Systemic							
Rabbit	1 d	Derm/oc Derm/oc		13 (conjunctivitis) 1 ^a (eczema)		Wilding et al. 1959	BO

^aOriginal unit provided by author was 1 g/cm².

BO = boron oxide; d = day; Derm/oc = dermal/ocular; LOAEL = lowest-observed-adverse-effect level;
NOAEL = no-observed-adverse-effect level

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No studies were located regarding the following health effects in humans or animals after dermal exposure to boron:

2.2.3.3 Immunological Effects

2.2.3.4 Neurological Effects

2.2.3.5 Developmental Effects

2.2.3.6 Reproductive Effects

2.2.3.7 Genotoxic Effects

Genotoxicity studies are discussed in Section 2.4.

2.2.3.8 Cancer

No studies were located regarding cancer effects in humans or animals after dermal exposure to boron.

2.3 TOXICOKINETICS

2.3.1 Absorption

2.3.1.1 Inhalation Exposure

No quantitative studies were located regarding absorption in humans or animals after inhalation exposure to boron. Reports of upper respiratory tract symptoms following exposure to boron oxide and boric acid dusts suggest boron can deposit in the upper airway (Garabrant et al. 1984, 1985).

2.3.1.2 Oral Exposure

No quantitative studies were located regarding absorption in humans or animals after oral exposure to boron and compounds. Gastrointestinal absorption was indicated in humans as evident by the urinary recovery of 93.9% of the ingested dose of boric acid when urine samples were calculated over a 96 hour period (Jansen et al. 1984a). Neurological, kidney, and liver damage following ingestion further suggest that boron can be absorbed (Wong et al. 1964).

2.3.1.3 Dermal Exposure

No quantitative studies were located regarding boron absorption in humans or animals after dermal exposure. Urinary excretion studies in humans (Section 2.3.4.3) suggest there is very little absorption of boron through intact skin. Excretion studies (Section 2.3.4.3) in rabbits suggest that boron is readily absorbed following contact with damaged skin (Draize and Kelley 1959).

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2.3.2 Distribution

No quantitative studies were located regarding distribution in humans or animals after exposure to boron and compounds by the following routes:

2.3.2.1 Inhalation Exposure

2.3.2.2 Oral Exposure

2.3.2.3 Dermal Exposure

2.3.3 Metabolism

No studies were located regarding metabolism in humans or animals after exposure to boron or boron compounds by the following routes:

2.3.3.1 Inhalation Exposure

2.3.3.2 Oral Exposure

2.3.3.3 Dermal Exposure

2.3.4 Excretion

2.3.4.1 Inhalation Exposure

No studies were located regarding excretion in humans after inhalation exposure to boron. In rats that inhaled average concentrations of 77 mg/m³ boron oxide aerosols over a 22 week period, an average of 11.90 mg boron/kg/day was detected in the urine compared to 0.24 mg/kg/day in untreated control groups (Wilding et al. 1959).

2.3.4.2 Oral Exposure

Over 93% of the administered dose was excreted in the urine of six male human volunteers 96 hours after administration of a single oral dose of 1.9 mg boron/kg (as boric acid) (Jansen et al. 1984a). An analysis of nine cases involving boric acid poisoning revealed a mean half-life of 13.4 hours (4-27.8). There was no correlation between half-life and calculated serum boric acid level at t, (r=0.08, p=0.84) (Litovitz et al. 1988). Boric acid was detected in urine of patients 23 days after a single ingestion (Wang et al. 1964).

In rabbits, 50%-66% of the administered dose was recovered in urine after ingestion of 17.1-119.9 mg boron/kg/day as boric acid (Draize and Kelley 1959).

2.3.4.3 Dermal Exposure

Limited data in humans suggest that very little absorption of boron occurs through intact skin. There was no increase in the urinary excretion of boron in one human subject following the application of 15 g boric acid (37.5 mg boron/kg bw) on the forearm for 4 hours (Draize and Kelley 1959).

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Animal studies support human findings. Draize and Kelley (1959) applied 200 mg/kg as boric acid to intact, abraded or burnt, and partially denuded skin of rabbits. Net urinary excretion of boric acid per 24 hours during 4 consecutive days of compound treatment was 1.4, 7.6 and 21.4 mg/kg, respectively (0.25, 1.3, and 3.7 mg boron/kg, respectively).

2.3.4.4. Other Exposure

In eight adult volunteers administered a single dose of boric acid (562-611 mg) by intravenous infusion, 98.7% of the administered dose was recovered in urine 120 hours after injection (Jansen et al. 1984b). Renal blood clearance averaged 39.1 mL/min per 1.73 m² surface area in eight adult human subjects administered intravenous injections of 35 mg boron/kg (as sodium pentaborate). Urine boron concentrations on the day of administration averaged 1.19 mg/mL (Farr and Konikowski 1963).

2.4 RELEVANCE TO PUBLIC HEALTH

Estimates of levels of exposure to boron posing minimal risk to humans (MRLs) have been made. These are discussed in Section 2.2 and were based on data believed to be reliable for the most sensitive noncancer effect for each route and exposure duration. No data were located on effects of acute-duration inhalation exposure in humans or animals nor on intermediate-duration inhalation exposure to boron in humans. Available information on intermediate-duration inhalation exposure in animals and chronic-duration inhalation exposure in humans do not reliably identify the most sensitive target organ. No data on effects of acute-duration oral exposure to boron in humans or animals nor on intermediate exposure in humans were located. In animals, prenatal exposure of mice (79 mg boron/kg/day as boric acid) and rats (13.6 mg boron/kg/day as boric acid) during gestation days 0-17 and 0-20 caused developmental effects consisting of reduced fetal body weight or minor skeletal changes and possibly delay in maturation (Heindel et al. 1991). There was degeneration of the seminiferous tubules and impaired spermatogenesis in mice exposed to dose levels of 111 mg boron/kg/day as boric acid for 2 generations (NIEHS 1990). In other studies involving intermediate duration exposure, gonadal damage, primarily in the testes, was evident at dose levels from 26 to 288 mg/kg/day (NTP, 1987; Weir and Fisher 1972), but not at dose levels of 0.6 and 25 mg/kg/day (Dixon et al. 1976, 1979). Exposure of dogs to boron (as boric acid or borax) in the diet for 38 weeks caused testicular atrophy and spermatogenic arrest at dose levels of 29 mg boron/kg/day (Weir and Fisher 1972). Based on a LOAEL value of 13.6 mg boron/kg/day for developmental toxicity, an intermediate oral MRL of 0.01 mg boron/kg/day was derived using an uncertainty factor of 1,000 (10 for use of a LOAEL, 10 for extrapolation from animals to humans and 10 for human variability). However, testicular effects were reversible within 25 days after compound treatment ceased. No effects were observed in rats fed diets containing doses up to 8.75 mg boron/kg/day for 2 years (Weir and Fisher 1972). Because developmental toxicity occurred at dose levels less than those for reproductive toxicity, the intermediate MRL based on developmental toxicity should be protective against reproductive toxicity following chronic

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exposure. No data were located on effects of chronic-duration oral exposure in humans. A chronic MRL was not derived. Acute-duration, intermediateduration, and chronic-duration dermal MRLs were not derived for boron due to the lack of an appropriate methodology for the development of dermal MRLs.

No studies have been found regarding immunological effects of boron and compounds in humans or animals.

Death. Human studies have shown that boron can be lethal following short-term exposure. The minimal lethal dose of ingested boron (as boric acid) was reported to be 2-3 g in infants, 5-6 g in children and 15-20 g in adults (Locatelli et al. 1987; Wong et al. 1964). No data were found on the potential for boron to cause death in humans after intermediate and chronic inhalation and oral exposures. Liver, kidney, brain damage, and skin lesions have been found in lethal cases following ingestion of boron, but death has been attributable to respiratory failure. In other studies, chronic dermal exposure to boron in neonates was fatal (Litovitz et al. 1988). There appears to be a differential susceptibility with regard to death in adults. It has been postulated that increased competence of the adult kidney accounts for adult tolerance to boron. Based on these findings, lethality may be an area of concern following neonate exposure to boron.

Animal studies support human findings. Boron was lethal after ingestion for acute, intermediate, and chronic duration exposures (NTP 1987; Smyth et al. 1969; Weir and Fisher 1972).

Systemic Effects

Respiratory Effects. Symptoms of acute irritation of the upper airway were observed at borax and boric acid levels of 4 mg/m³ or greater (Garabrant et al. 1984, 1985). No adverse respiratory effects were observed in humans following intermediate inhalation exposures. Chronic inhalation exposure caused irritation of the upper respiratory tract (Garabrant et al. 1984, 1985). There were no changes in the FEV₁ and FVC in borax workers (Wegman et al. 1991). Intermediate inhalation exposure in animals caused irritation of the nose (Wilding et al. 1959).

Gastrointestinal Effects. Boron or boron compounds can result in gastrointestinal disorders in humans following acute and intermediate oral exposures. Most of the studies focused on clinical symptoms including vomiting and diarrhea. No data were found on biochemical changes and limited data were provided on histopathological effects. Infants appear to be particularly susceptible to boron toxicity, possibly due to the fact that their detoxifying enzyme systems are immature and there is greater gastrointestinal absorption.

No studies were located in animals regarding gastrointestinal effects following boron exposure.

2. HEALTH EFFECTS

Hepatic Effects. No adverse hepatic effects have been reported in humans or animals following inhalation or dermal exposure to boron or boron compounds. Acute oral exposure in humans caused congestion, fatty changes, and parenchymatous degeneration (Wong et al. 1964). No data were available on biochemical changes. It is not clear how boron affects the liver; however, limited animal data suggest impaired electron transfer and macrometabolism. In studies with rats, boron interfered with flavin metabolism in flavoprotein dependent pathways (Settimi et al. 1982). It is not clear if similar effects will occur in humans.

Renal Effects. No adverse renal effects have been reported in humans or animals following inhalation of boron oxide, boric acid dust, or boron oxide aerosol. Similarly, dermal exposure to boric acid in humans or animals did not adversely affect the kidneys. Renal tubular damage has been observed, and there was reduced urine output in infants who consumed 505 mg boron/kg in infant formula for 3-5 days (Wong et al. 1964). Since renal effects occurred in only a few cases and there is no confirming evidence in animals, the potential for boron to cause renal effects cannot be conclusively established.

Dermal/Ocular Effects. Human occupational exposure to boron oxide and boric acid dusts in workplace air irritated the eyes (Garabrant et al. 1984). Ingestion of large amounts of boron (505 mg boron/kg as boric acid) caused extensive exfoliative dermatitis in humans (Wong et al. 1964). The application of boric acid on the forearm of human subjects did not affect the skin (Draize and Kelley 1959). Rabbits developed erythema when boron oxide dust was applied to the skin and conjunctivitis was observed following contact with boron oxide dust (Wilding et al. 1959).

Immunological Effects. No studies were located regarding the effects of boron on the immune system in humans or animals after inhalation, oral, or dermal exposure. In the absence of effects on target organs and direct tests on immune function, the potential for boron to cause immunological effects in humans cannot be conclusively evaluated.

Neurological Effects. No adverse neurological effects have been observed in humans or animals following inhalation or dermal exposure. Acute and intermediate oral exposures to boron and boron compounds caused various neurological responses in humans. Degenerative changes in brain neurons which may have been an agonal effect were reported in one infant who consumed 505 mg boron/kg as boric acid for 3 days (Wong et al. 1964). At a higher dose (765 mg boron/kg), there was extensive vascular congestion, widespread perivascular hemorrhage, and intravascular thrombosis in another infant who ingested infant formula containing boric acid for 5 days (Wong et al. 1964). Biochemical changes have also been found. Cerebral succinate dehydrogenase activity was increased in rats that ingested borate in drinking water for 10-14 weeks, suggesting alteration in electron-transfer in the mitochondrial respiratory chain (Settimi et al. 1982). Increased RNA concentration and increased acid proteinase activity in the brain also occurred (Settimi et al. 1982). Altered metabolism and brain tissue redox state suggest changes in protein metabolism.

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Based on these considerations, neurological damage is an area of concern following exposure to boron at toxic levels.

Developmental Effects. Developmental changes in rats and mice have been observed in offspring of dams exposed to 28.4 mg boron/kg/day and 175.3 mg boron/kg/day, respectively (Heindel et al. 1991). These effects have been observed at dose levels in the same range as those producing changes in spermatogenesis. No epidemiological studies were located regarding the effects of boron on the developing fetus. Although human data are lacking and there are no direct quantitative studies regarding placental transfer of boron, positive responses in two animal species suggest that developmental toxicity may be an area of concern in humans following exposure to boron. The LOAEL value of 13.6 mg boron/kg/day (Heindel et al. 1991) was used to calculate an intermediate oral MRL of 0.01 mg/kg/day as described in the footnote in Table 2-2.

Reproductive Effects. A study of 28 male workers exposed to borate aerosols during the production of boric acid revealed low sperm counts in six of these workers (Tarasenko et al. 1972). The authors reported exposure concentrations ranging from 22 to 80 mg/m³. The overall reliability of these data is reduced due to the small study group. It should also be noted that low sperm count is a naturally occurring phenomenon. No studies were located regarding reproductive effects in humans after oral or dermal exposure.

In animals, boron affects gonads in dogs, rats, and mice. The testes are particularly susceptible after intermediate ingestion (44 and 29 mg boron/kg/day, respectively) (Seal and Weeth 1980; Weir and Fisher 1972). Following chronic oral exposure, no effects were observed at a dose of 8.75 mg boron/kg/day (Weir and Fisher 1972). In spite of the absence of reliable human data, limited evidence of reproductive effects in animals suggest that reproductive toxicity may be an area of concern following boron exposure in humans.

Genotoxic Effects. No studies were located regarding genotoxic effects of boron by inhalation, oral, or dermal exposure in humans and animals. Results were negative in bacterial assays and in the in vitro (Table 2-4) mammalian assays, including tests for chromosomal aberrations and gene mutation. Existing data suggest that genotoxicity is not an area of concern following exposure to boron in humans.

Cancer. No epidemiological studies were located associating cancer and boron exposure. In mice fed boron (as boric acid) for 103 weeks, the number of tumors observed did not differ significantly from untreated control levels (NTP 1987). In the absence of human data and studies from other animal species, and the lack of evidence of mutagenic activity, the carcinogenic potential of boron in humans cannot be determined conclusively.

TABLE 2-4. Genotoxicity of Boron In Vitro

Species (test system)	End point	Results		Reference
		With activation	Without activation	
Prokaryotic organisms:				
<u>Salmonella typhimurium</u>	Gene mutation	+	+	Reworth et al. 1983
<u>S. typhimurium</u>	Gene mutation	+	+	Benion et al. 1984
<u>Escherichia coli</u>	Gene mutation	+	+	Dewezet et al. 1981
<u>S. typhimurium</u>	Gene mutation	-	-	NTP 1987
Mammalian cells:				
Mouse lymphoma	Gene mutation	-	-	NTP 1987
Chinese hamster ovary	Chromosomal aberration	-	-	NTP 1987

- = negative result

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2.5 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989). A biomarker of exposure is a xenobiotic substance or its metabolite(s) or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s) or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time biologic samples can be taken. It may be difficult to identify individuals exposed to hazardous substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as copper, zinc, and selenium). Biomarkers of exposure to boron and compounds are discussed in Section 2.5.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are often not substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by boron and compounds are discussed in Section 2.5.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, biologically effective dose, or target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 2.7, "POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE."

2.5.1 Biomarkers Used to Identify and/or Quantify Exposure to Boron

Boron in blood and urine can be used as an indicator of exposure to boron. Normal dietary concentrations of boron in the blood of humans range from 0 to 1.25 pg/mL in children and infants (Fisher and Freimuth 1958; O'Sullivan and Taylor 1983). Boron blood levels (reported as borate) of

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20-150 µg/mL have been associated with adverse systemic effects in infants who ingested boric acid in infant formula (Wong et al. 1964). Boron concentrations, expressed as borate, reported in fatal cases vary from 200 to 1,600 µg/mL in infants (Wong et al. 1964). In adults, a serum boron level (as boric acid) of 2,320 µg/mL was not associated with significant toxicity (Linden et al. 1986).

Urinary excretion levels can also be useful indicators of elevated total body burden of boron. Concentrations of boron in the normal population range from 0.07 to 0.15 mg/100 mL (Vignec and Ellis 1954) and 0.004 to 0.66 mg/100 mL (Imbus et al, 1963). In one infant, the urine contained 13.9 mg boron/L as borax or 1.38 mg boron/mL of boric acid following ingestion of a borax and honey mixture over a period of 12 weeks (Gordon et al. 1973). Virtually complete urinary excretion was indicated by the recovery of 93.9% (over a 96-hour collection period) of a boric acid solution ingested by three human volunteers (Jansen et al. 1984a).

Neurological, dermal, gastrointestinal, liver, and kidney effects in humans have been associated with exposure to boron. Studies in animals have demonstrated gonadal injury. Various clinical and biochemical tests exist that may provide useful information on exposure. However, similar effects are caused by a variety of other substances and are, therefore, not specific for boron exposure.

2.5.2 Biomarkers Used to Characterize Effects Caused by Boron

Central nervous system injury, gastrointestinal effects, and skin damage are characteristic manifestations of boron toxicity in humans. Liver and kidneys in humans and testes in animals can also be affected. Various clinical and biochemical changes associated with these effects may be measured to detect the extent of exposure to boron. There is no single biological indicator of boron exposure; consequently, several parameters must be measured including boron levels in urine and blood and biochemical changes for systemic and neurological effects.

Neurological damage has been reported in humans. Neurological effects reported in humans have focused primarily on histopathological alterations. No data were provided on biochemical changes. In animals, testicular atrophy and reduced sperm production have been demonstrated following chronic boron exposure. There are clinical and biochemical tests to detect neurological and gonadal injury, but these are not specific for boron exposure. Sparse data in animals suggest some biochemical changes; for instance, cerebral succinate dehydrogenase was increased in rats after boron exposure. Animal data further demonstrate biochemical alterations following gonadal injury. Dose-dependent reduction in hyaluronidase, sorbitol dehydrogenase, and lactic acid dehydrogenase (isoenzyme-X) were observed in rats following boron exposure.

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2.6 INTERACTIONS WITH OTHER CHEMICALS

No studies were located regarding the influence of other chemicals on the toxicity of boron.

2.7 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

Neonatal children are unusually susceptible to boron exposure.

2.8 MITIGATION OF EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to boron. This section is intended to inform the public of existing clinical practice and the status of research concerning such methods. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to boron. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice.

Human exposure to boron may occur by inhalation, ingestion, or dermal contact (see Chapter 5). Boron in the form of boric acid or borate dust is an upper respiratory tract irritant following inhalation and may also irritate the eyes and skin. Ingestion of boron may cause gastrointestinal, neurological, hepatic, renal, and dermal effects (see Section 2.2). General recommendations for reducing absorption of boron following exposure have included removing the exposed individual from the contaminated area and removing the contaminated clothing. If the eyes and skin were exposed, they are flushed with water.

Nausea, vomiting, and diarrhea have been induced by ingestion of boron in humans. Some authors recommend reducing absorption of boron from the gastrointestinal tract by administration of emetics (e.g. syrup of ipecac) and cathartics (e.g. magnesium sulfate) (Stewart and McHugh 1990). Caution should be, however, taken not to induce further damage to the esophageal mucosa or to cause aspiration of the vomit into the lungs during emesis. There is disagreement regarding the efficiency of activated charcoal in preventing absorption of boron from the gastrointestinal tract following oral exposure (Ellenhorn and Barceloux 1988; Stewart and McHugh 1990). It has been suggested that activated charcoal be administered following gastric evacuation, but its effectiveness has not been established (Ellenhorn and Barceloux 1988). Administration of intravenous fluids may be required if severe dehydration or shock develop and local skin care may be necessary if skin desquamation occurs (Stewart and McHugh 1990). In addition, the treatment of boron poisoning may request a control for convulsions.

Elemental boron is not metabolized (see Section 2.3). Studies in human volunteers indicated that most of the administered dose is excreted in the urine within few days (Jansen et al. 1984a).

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Saline diuresis has been suggested to further enhance urinary excretion of boron (Goldfrank et al. 1990). Exchange transfusions, peritoneal dialysis, or hemodialysis may be employed to lower plasma boron levels following either acute or chronic intoxication. There are indications that hemodialysis is the most effective of these procedures (Goldfrank et al. 1990; Stewart and McHugh 1990). Additional details regarding treatment of boron intoxication may be found in the cited references.

2.9 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of boron is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of boron.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

2.9.1 Existing Information on Health Effects of Boron

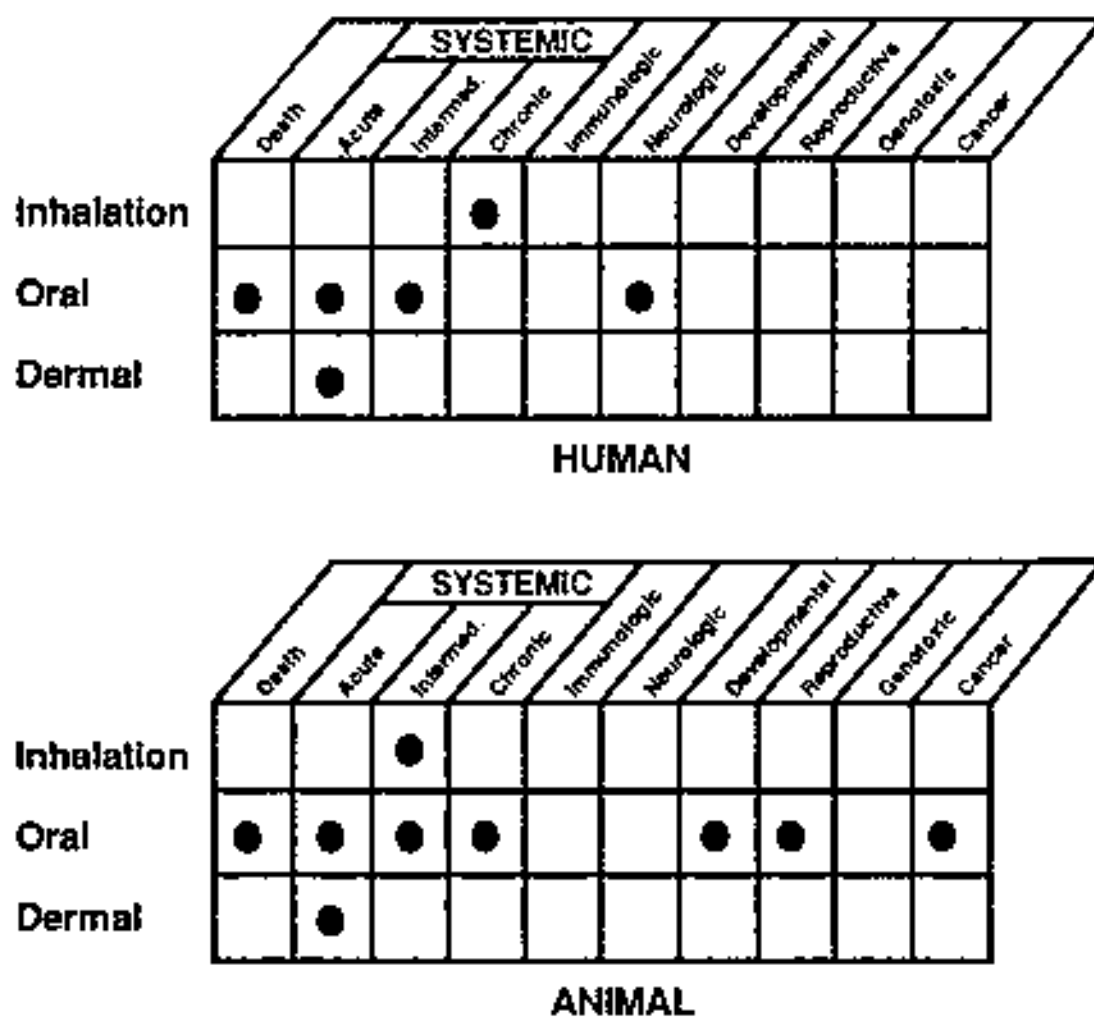
The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to boron are summarized in Figure 2-3. The purpose of this figure is to illustrate the existing information concerning the health effects of boron. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not imply anything about the quality of the study or studies. Gaps in this figure should not be interpreted as "data needs" information.

Most of the information concerning health effects of boron in humans is found in case reports of accidental acute and intermediate ingestion of boron. No information was found on effects after chronic ingestion. Those effects associated with inhalation occurred following chronic exposure in the workplace. No information was found on effects of boron after acute and intermediate inhalation exposures. Information on acute dermal exposure exist, but none was found on effects after intermediate and chronic exposures.

In animals, information exists on the acute, intermediate, and chronic ingestion of boron. Those effects associated with inhalation of boron occurred following intermediate exposures. No information was found on health effects of boron after acute and chronic inhalation exposures. Boron does cause health effects following acute dermal exposure. No information was found on health effects after intermediate and chronic dermal exposures.

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FIGURE 2-3. Existing Information on Health Effects of Boron



● Existing Studies

2. HEALTH EFFECTS

2.9.2 Data Needs

Acute-Duration Exposure. There are data indicating mild upper respiratory irritation in humans from acute inhalation of borate dusts (Wegman et al. 1991). Information on the effects of a single oral exposure to boron compounds in humans and animals have provided data on lethal effects, while injury to the lungs, brain, kidneys, and liver have been reported in infants (NTP 1987; Smyth et al. 1969; Weir and Fisher 1972; Wong et al. 1964). Many of the human data are derived from case reports involving toxic effects in infants. No adverse health effects have been demonstrated in humans after dermal exposure. However, dermal/ocular effects have been associated with dermal exposure in animals (Wilding et al. 1959). The irritation effects observed were probably due to the exothermic rehydration reaction of the anhydride boron oxide. While existing data are sufficient to identify target organs, additional oral and dermal studies may clarify dose-response relationships in target tissues and identify a threshold for systemic effects due to a single-dose exposure. Human and animal data were not sufficient to derive acute oral and inhalation MRLs. Existing data provide qualitative evidence of toxic effects; however, data gaps exist relative to concentration and effects in the target tissues.

Intermediate-Duration Exposure. No studies were located in humans after intermediate exposure to boron compounds by any route of exposure. Borates are not absorbed through intact skin (Draize and Kelley 1959). No studies were available on dermal or inhalation exposure in animals; however, lethal effects and injury to the gonads, particularly the testes, have been demonstrated after oral exposure (Dixon et al. 1979; Lee et al. 1978; NIEHS 1990b; NTP 1987; Seal and Weeth 1980; Weir and Fisher 1972). Data suggest differences in sensitivity to boron compounds among animal species, with dogs more sensitive than rats or mice (Weir and Fisher 1972). Developmental effects were reported in mice and rats after oral exposure (Heindel et al. 1991). Data are sufficient to develop an intermediate oral MKL. The MRL was based on developmental toxicity in rats (Heindel et al. 1991). Although the MEL value is lower than the average daily intake of boron, it should be noted that recommended daily allowance levels have not been established for boron. Further studies by other routes of exposure would be useful in confirming target tissues (e.g., testes) and effects on the fetus identified by the primary exposure route. Also, these data may be used to further assess the level of confidence in current NOAEL and LOAEL values. Additional data may also provide some insight into the basis for differential susceptibility among species which may be useful in assessing potential human risk.

Chronic-Duration Exposure and Cancer. Limited epidemiologic studies conducted in humans demonstrated that borate dust can affect the upper respiratory tract and cause eye irritation following inhalation (Gabarant et al. 1984, 1985). Data were not sufficient to derive a chronic-duration MEL. No studies were found on oral and dermal exposures in humans. Oral studies in animals demonstrated injury to the gonads and to the developing fetus (NIEHS 1990a; NTP 1987; Weir and Fisher 1972). Existing oral studies are sufficient to rule out effects on other organ systems or tissues (NTP

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1987; Weir and Fisher 1972). No studies were found on chronic dermal and inhalation data in animals. Additional studies are needed to identify critical effect levels. Although data are sufficient to develop a chronic oral MRL, a value was not derived. Because developmental toxicity occurred at dose levels less than those for reproductive effects, the intermediate MRL, which is based on developmental toxicity, should be protective against reproductive toxicity following chronic exposure. Additional studies would be useful in assessing the level of confidence in existing NOAEL and LOAEL values.

No epidemiologic studies have been conducted in humans regarding boron exposure and cancer. Well-designed and well-conducted case control or cohort studies would be useful in assessing risk to exposed humans. A long-term oral bioassay in mice was negative. No studies on chronic dermal or inhalation exposure evaluating carcinogenic potential in animals are available. The absence of effects in one species is not sufficient to rule out the potential to cause cancer. Additional chronic studies of other species and various doses would increase the level of confidence in results reported in existing studies.

Genotoxicity. No in vivo human data were located. Bacterial and limited mammalian assays were negative (Benson et al. 1984; Demerec et al. 1951; Haworth et al. 1983; NTP 1987). Considering the absence of mutagenic effects in bacterial and mammalian tests evaluating gene mutation and chromosomal aberrations, genotoxicity may not be an area of concern in humans. Based on existing data, additional studies are not needed at this time.

Reproductive Toxicity. No studies were found on the effects of boron compounds on the reproductive system in humans by any route of exposure. Oral studies in animals demonstrated injury to gonads, particularly the testes (Dixon et al. 1979; Lee et al. 1978; NIEHS 1990; Seal and Weeth 1980; Weir and Fisher 1972). No studies were found on chronic dermal and inhalation studies in animals. Sufficient data exist on the potential for boron compounds to affect male reproductive organs in animals (NIEHS 1990; NTP 1987; Weir and Fisher 1972). Data suggest that the severity of effects are species specific (Weir and Fisher 1972). Additional studies would be useful to clarify dose response relationships. Data suggest the female reproductive system is less susceptible and is affected only at very high dose levels (NIEHS 1990; NTP 1987; Weir and Fisher 1972). Additional studies evaluating reproductive effects in females may not be needed at this time.

Developmental Toxicity. No studies were found on the developmental effects of boron and compounds in humans following inhalation, oral, or dermal exposure. No data are available on the ability of boron to cross the placenta or accumulate in fetal tissue. Studies in rats and mice indicate delayed development and structural defects, primarily in the rib cage, following continuous oral exposure in the diet during pregnancy (Heindel et al. 1991). Existing animal data suggest additional testing would be useful in assessing potential risk to humans.

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Immunotoxicity. No studies were found in humans or animals on the effects of boron on the immune system by any route of exposure. Results of chronic studies do not suggest that the immune system is a potential target for boron toxicity. Additional studies are not needed at this time.

Neurotoxicity. Case reports in humans, primarily infants, indicate that neurological effects occur after ingestion of boron at high dose levels (Wong et al. 1964). Degenerative changes in brain cells, perivascular hemorrhage, and intravascular thrombosis have been reported in fatal case reports in infants, but neurochemical or neurophysiological changes have not been reported (Settimi et al. 1982; Wong et al. 1964). No studies are available on neurotoxic effects of boron following inhalation or dermal exposure in humans. Animal data are limited to increased brain enzyme activity (Settimi et al. 1982), but no histopathological data are available. Since data on effects are limited primarily to acute oral exposures at high dose levels, additional studies in animals evaluating other dose levels and exposure durations would be useful in evaluating potential risk to humans who may be exposed to low levels of boron compounds near hazardous waste sites.

Epidemiological and Human Dosimetry Studies. Information exists on the adverse health effects of boron compounds in humans. Studies of workers exposed to boron compounds demonstrated that boron can cause mild irritation of the eyes and respiratory tract (Garabrant et al. 1984, 1985). Other human studies involve case reports of accidental or intentional ingestion of large quantities of boron compounds (Litovitz et al. 1988; Locatelli et al. 1987). The studies identified key health effects (lung, kidney, brain, and liver) associated with boron exposure (Wong et al. 1984). Animal studies indicated the testes as a target tissue. Epidemiological studies of the birth rate of occupationally-exposed workers is currently underway at a major U.S. borate production facility (U.S. Borax and Chemical Corporation 1991).

Biomarkers of Exposure and Effect. Blood and urine borate concentrations are useful biomarkers of exposure (Jansen et al. 1984a; Litovitz et al. 1988). The gastrointestinal tract, skin, and brain are principal target organs following boron exposure in humans. Studies in animals demonstrate that boron compounds can also cause gonadal injury, particularly to the testes (Weir and Fisher 1972). Existing animal studies have established this effect as the most sensitive endpoint following oral exposure. Studies to determine other biomarkers would be useful in assessing the potential human health risk.

Absorption, Distribution, Metabolism, and Excretion. No quantitative information is available on the absorption, distribution, and metabolism of boron compounds; however, there are studies on the excretion of boron following oral (Jansen et al. 1984a; Litovitz et al. 1988) and inhalation (Wilding et al. 1959) exposures and after dermal exposure (Draize and Kelley 1959). Since data on toxicokinetics of boron are limited, additional studies are needed by all routes of exposure that will provide data on absorption rates, extent of conversion in the body and amount and rate of accumulation in various tissues. Limited data from oral and dermal studies suggest that boron

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is primarily excreted in urine. Since boron can deposit in the upper respiratory tract, additional excretion studies by this route would be useful in determining if excretion patterns are similar across all routes of exposure.

Comparative Toxicokinetics. Existing evidence from human and animal studies do not indicate whether or not boron compounds affect the same target tissues. Animal studies indicate the testes as a target tissue (Dixon et al. 1979; Lee et al. 1978; NIEHS 1990; Seal and Weeth 1980; Weir and Fisher 1972). Data suggest differences in species sensitivity, with dogs more sensitive than rats and mice (Weir and Fisher 1972). No data have been found on potential reproductive effects of boron and compounds in humans. Data exist on excretion of boron compounds. Based on excretion studies, boron compounds are absorbed by the gastrointestinal tract. There are no available quantitative toxicokinetics data on absorption, distribution, and metabolism. Additional toxicokinetics studies would be useful in assessing differences in species sensitivity, and provide a better basis for extrapolation of animal data to human exposure risk.

Mitigation of Effects. Methods for the mitigation of acute effects of boron poisoning include prevention of absorption of boron from the gastrointestinal tract and standard procedures used to prevent convulsions, severe dehydration or shock (Stewart and McHugh 1990). Saline diuresis, exchange transfusions, peritoneal dialysis, or hemodialysis may be employed to enhance removal of absorbed boron from the body (Goldfrank et al. 1990; Stewart and McHugh 1990). No additional information was located concerning mitigation of effects of lower-level or longer-term exposure to boron. Further information on techniques to mitigate such effects would be useful in determining the safety and effectiveness of possible methods for treating boron-exposed populations in the vicinity of hazardous waste sites.

2.9.3 On-going Studies

The National Institute of Environmental Health Sciences (J. Williams, investigator) is conducting a study on the disposition of boric acid in selected target and nontarget tissues. The potential of boric acid to cause in vivo riboflavin deficiency as a mechanism of the testicular toxicity is being investigated, as are the direct effects of boric acid applied to sertoli or leydig cells in primary culture from naive rats (CRISP 1990).

3. CHEMICAL AND PHYSICAL INFORMATION

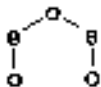
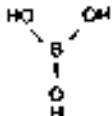
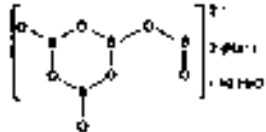
3.1 CHEMICAL IDENTITY

Table 3-1 lists common synonyms, trade names, and other pertinent information to identify boron and selected compounds.

3.2 PHYSICAL AND CHEMICAL PROPERTIES

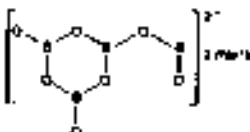
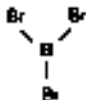

Table 3-2 lists important physical and chemical properties of boron and selected compounds.

TABLE 3-1. Chemical Identity of Boron and Compounds^a

Characteristic	Boron	Boron oxide	Boric acid	Borax
Synonyms	No data	Boric anhydride; diboron trioxide	Boracic acid; orthoboric acid	Borax decahydrate; tincal; polybor
Trade name	No data	No data	No data	No data
Chemical formula	B	B ₂ O ₃	H ₃ BO ₃	Na ₂ B ₄ O ₇ · 10H ₂ O
Chemical structure ^b	Not applicable			
Identification numbers:				
CAS Registry	7440-42-B ^c	1303-86-2	10043-35-3	1303-86-B ^c
NIOSH RTECS	ED7350000 ^d	ED7900000 ^d	ED6550000 ^d	U22275000 ^d
EPA Hazardous Waste	No data	No data	No data	No data
ORCA/TADS	7216607 ^e	No data	7216606 ^e	No data
DOT/UN/NA/IMCO Shipping	No data No data	No data No data	No data No data	No data No data
MSDS	1482 ^f	1609 ^f	1432 ^f	6328 ^f
PCI	No data	No data	No data	No data

3. CHEMICAL AND PHYSICAL INFORMATION

TABLE 3-1 (Continued)

Characteristic	Borax, anhydrous	Boron tribromide	Boron trifluoride
Synonyms	Borax, dehydrated; sodium tetraborate, anhydrous	Boron bromide	Boron fluoride
Trade name	No data	No data	No data
Chemical formula	$\text{Na}_2\text{B}_4\text{O}_7$	BBr_3	BF_3
Chemical structure ^{a,c}			
Identification numbers:			
CAS Registry	1303-96-4	10294-33-4	7432-07-2
NIOSH RTECS	VZ2275000 ^d	ED7400000 ^d	ED2275000 ^d
EPA Hazardous Waste	No data	No data	No data
ORDM/TADS	No data	No data	No data
DOT/UN/NA/IMCO Shipping	No data No data	UN2692 ^e No data	UN1005 ^e IMCO 2.3
HSDB	0328 ^f	0327 ^f	0325 ^f
MCI	No data	No data	No data

^aAll information obtained from Sax and Lewis 1987, except where noted.^bGrayson 1983^cMorrison and Boyd 1983^dEPA 1987b^eSittig 1985^fHSDB 1989

CAS = Chemical Abstracts Service

DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code

EPA = Environmental Protection Agency

HSDB = Hazardous Substances Data Bank

MCI = National Cancer Institute

NIOSH = National Institute for Occupational Safety and Health

ORDM/TADS = Oil and Hazardous Materials/Technical Assistance Data System

RTECS = Registry of Toxic Effects of Chemical Substances

TABLE 3-2. Physical and Chemical Properties of Boron and Compounds¹

Property	Boron	Boron oxide	Boric acid	Borax
Molecular weight	10.81	69.62	61.83	381.37
Color	Black or brown	Colorless	Colorless	Colorless
Physical state	Solid	Solid	Solid	Solid
Melting point	2300°C	450±2°C	147°C±1 tr to HBO ₃	73°C, -8H ₂ O, 50°C
Boiling point	2530°C	1300°C ^a	-10H ₂ O, 300°C	-10H ₂ O, 320°C
Density at 20°C	2.34	2.46	1.435 at 15°C	1.73
Odor	No data	No data	Odorless ^b	Odorless ^c
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Solubility:				
Water	Insoluble	Soluble in hot water; slightly soluble in cold water	63.5 g/L at 30°	20.1 g/L at 0°C
Organic solvents	Soluble in nitric and sulfuric acid ^d	Soluble in alcohol ^b	Soluble in alcohol, glycerol	Slightly soluble in alcohol, glycerol
Partition coefficients:				
Log octanol/water	No data	No data	No data	No data
Log K _{ow}	No data	No data	No data	No data
Vapor Pressure	1.56x10 ⁻³ mm at 2340°C ^e	No data	No data	No data
Henry's law constant	No data	No data	No data	No data
Autoignition temperature	No data	Noncombustible ^b	Noncombustible ^b	No data
Flashpoint	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data
Conversion Factors	No data	No data	No data	No data

TABLE 3-2 (Continued)

Property	Borax, anhydrous	Boron tribromide	Boron trifluoride
Molecular weight	201.22	250.52	67.81
Color	Colorless	Colorless	Colorless
Physical state	Solid	Liquid	Gas
Melting point	741°C	-46°C	-126.7°C
Boiling point	Decomposes at 1575°C	91.3±0.25°C	-99.9°C
Density at 20°C	2.37	1.69 at 15°C ^a	2.99 g/L
Odor	Odorless ^a	No data	Pungent ^a
Odor threshold:			
Water	No data	No data	No data
Air	No data	No data	1.1 mg/m ³
Solubility:			
Water	10.8 g/L at 0°C ^c 87.9 g/L at 40°C	Decomposes	1060 g/L at 20°C
Organic solvents	Insoluble in alcohol	Soluble in alcohol, CCl ₄	Soluble in sulfuric acid
Partition coefficients:			
Log octanol/water	No data	No data	No data
Log K _{ow}	No data	No data	No data
Vapor Pressure	No data	100 mmHg at 33.5°C ^c	40 mmHg at -131.0° (solid) 760 mmHg at -110.7°C (liquid) ^d
Henry's law constant	No data	No data	No data
Autoignition temperature	Noncombustible ^a	No data	Noncombustible ^a
Flashpoint	No data	No data	No data
Flammability limits	No data	No data	No data
Conversion factors	No data	No data	No data

^aAll information obtained from Meast 1983, except where noted.^bSan and Lewis 1997^cACGIH 1988^dRuth 1986^eWinkholz 1983^fHSDB 1989

4. PRODUCTION, IMPORT, USE, AND DISPOSAL

4.1 PRODUCTION

Boron is produced by the chemical reduction of boron compounds with reactive metals, either by nonaqueous electrolytic reduction or through thermal decomposition. Highly purified boron is produced by zone-refining or other thermal techniques (HSDB 1989; Stokinger 1981; U.S. Bureau of Mines 1989).

The United States produces most of the world's borates. Production figures for 1988 report 566,093 metric tons of boric oxide equivalent was produced from the mining of boron-containing minerals. Domestic production has remained relatively constant over the last 5 years ranging from a low of 570,629 metric tons in 1986 to a high of 625,061 metric tons in 1987 (Ferguson et al. 1982; U.S. Bureau of Mines 1989).

United States Borax 6 Chemical Corporation continues to be the primary world supplier of sodium borates. U.S Borax mines and processes crude and refined sodium borates, their anhydrous derivatives, and anhydrous boric acid at its plant, in Kern County, Boron, California. A second plant at Boron, California uses a proprietary process to produce technical-grade boric acid.

Kerr-McGee Chemical Corporation operates the Trona and Westend plants at Searles Lake, in San Bernardino County, to produce refined sodium borate compounds and boric acid from the mineral-rich lake brines.

4.2 IMPORT/EXPORT

The United States imported 59,875 metric tons of borax, boric acid, and the boron-containing minerals colemanite and ulexite in 1988 (U.S. Bureau of Mines 1988). As the world's largest producer of boron compounds, the United States exported 589,680 metric tons of boric acid and borates in 1988.

4.3 USE

Borates have diverse uses. Their principal uses (56%) are in the production of glass and glass products such as textiles and insulating fiberglass. It is also used to make the enamels and glazes used as coatings on household and industrial products. Borates are used in herbicides, insecticides, soaps and cleansers, cosmetics, antifreeze, and leather tanning. Borax and boric acid are used in atomic reactors as a neutron absorber (EPA 1986b; HSDB 1989; Stokinger 1981; U.S. Bureau of Mines 1989).

4.4 DISPOSAL

No federal regulations were located which control the disposal of borates including sodium borates and boric acid. No quantitative disposal data were located.

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Boron is a naturally-occurring element found combined with other elements throughout the environment. Boron is neither transformed nor degraded in the environment, although changes in the specific form of boron and its transport may occur, depending on environmental conditions. It is estimated that natural weathering is a significant source of environmental boron.

Ingestion of boron from food (primarily fruits and vegetables) and water is the most frequent route of human exposure, but occupational exposures to boron dusts may be significant. Boron is also a component of several consumer products, including cosmetics medicines and insecticides. Populations residing in areas of the western United States with natural boron-rich deposits may be exposed to higher-than-average levels of boron.

The EPA has identified 1,177 NPL sites. Boron, borate, and borax have been found at 21, 1, and 1, respectively, of the sites evaluated for these chemicals. However, we do not know how many of the 1,177 NPL sites have been evaluated for the presence of these chemicals. As more sites are evaluated by the EPA, these numbers may change (View 1989). The frequency of these sites within the United States can be seen in Figure 5-1.

5.2 RELEASES TO THE ENVIRONMENT

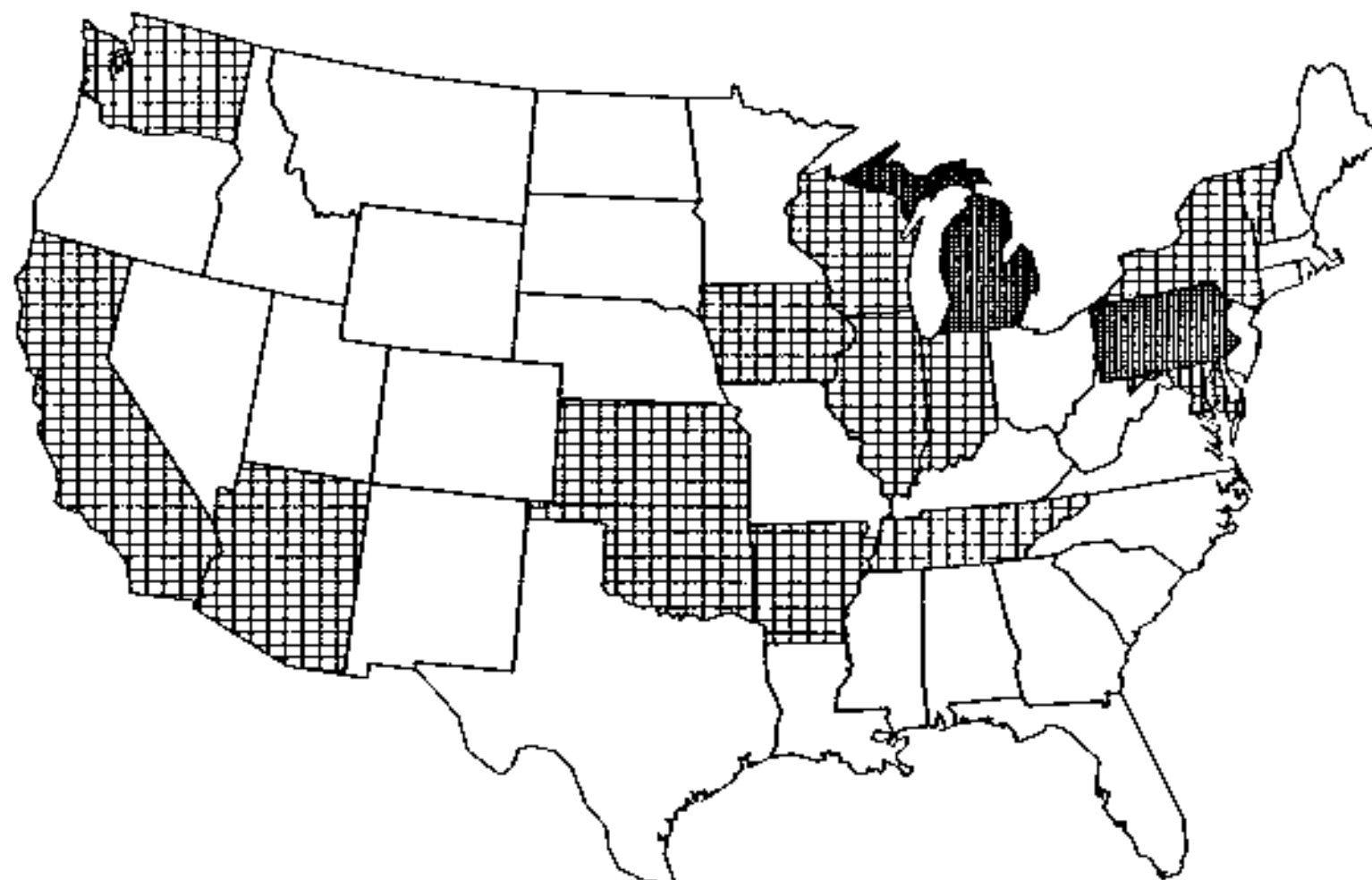
Borates are widespread, naturally-occurring substance found mainly as an inorganic compound in sediments and sedimentary rock. It is released to the environment slowly in low concentrations by weathering processes. Although few data are available quantifying boron releases from industrial sources, it is estimated that natural weathering releases more boron to the environment worldwide than do these industrial sources (Butterwick et al. 1989).

Releases of boron to the environment occur from the production and use of boron and boron-related compounds. However, neither boron nor boronrelated compounds are listed on the Section 313 toxic chemical list and, therefore, are not included in the Toxics Release Inventory (TRI).

5.2.1 Air

Borates are released to air from natural and industrial sources. Natural sources include oceans, volcanoes, and geothermal steam (Graedel 1978). Boron compounds are released from anthropogenic sources such as coalfired and geothermal steam power plants, chemical plants, and rockets as well as manufacturing facilities producing fiberglass and other products (EPA 1987c; Graedel 1978; Hollis et al. 1988; Lang et al. 1986; Rope et al. 1988; Stokinger 1981). No quantitative data regarding boron releases to air were located.

FIGURE 5-1. FREQUENCY OF NPL SITES WITH BORON CONTAMINATION *



FREQUENCY  1 SITE  2 SITES  3 SITES

* Derived from View 1989

5. POTENTIAL FOR HUMAN EXPOSURE

5.2.2 Water

Natural weathering of boron-containing rocks is a major source of boron compounds in water (Butterwick et al. 1989). The quantity of boron released varies widely with the geographic variations in boron-rich deposits. In the United States, the area richest in natural boron deposits is the Mojave Desert in California (Butterwick et al. 1989; Stokinger 1981).

Boron compounds are released to water in municipal sewage from perborates in detergents, and in waste waters from coal-burning power plants, copper smelters, and industries using boron. Borate levels above background may be present in runoff waters from areas where boron-containing fertilizers or herbicides were used (Butterwick et al. 1989; Nolte 1988; Waggott 1969). An average concentration of 1 mg boron/L was reported in sewage effluents in California (Butterwick et al. 1989). No other quantitative data regarding boron releases to water in the United States were located. However, Waggott (1969) reported that boron concentrations in municipal sewage in a treatment plant in England ranged from 2.5 to 6.5 mg/L, releasing between 130 and 240 kg boron/day.

Boron has been detected in surface water and groundwater at hazardous waste sites. Data from the Contract Laboratory Program (CLP) Statistical Database indicate that boron occurred at about 20% of the sites at a geometric mean concentration of 156 ppb (0.156 mg boron/L) in positive samples of groundwater and at about 5% of the sites at a geometric mean of 1,177 ppb (1.177 mg boron/L) in surface water (CLPSD 1989).

5.2.3 Soil

Boron is naturally released to soil and water by rainfall, weathering of boron-containing minerals, desorption from clays and by decomposition of boron-containing organic matter. Man-made sources include application of boron-containing fertilizers or herbicides, application of fly ash or sewage sludge as a soil amendment, the use of waste water for irrigation or land disposal of boron-containing industrial wastes (Butterwick et al. 1989; Hollis et al. 1988; Mumma et al. 1984; Nolte 1988; Rope et al. 1988).

No quantitative data were located regarding man-made releases of boron compounds to soil. However, Mumma et al. (1984) reported that the boron concentration in sewage sludges from 23 U.S. cities ranged from 7.1 to 53.3 mg/kg. Landfilling or land application is a common disposal method for these sludges.

Data from the CLP Statistical Database indicate boron was detected in soil at about 5% of hazardous waste sites at a geometric mean concentration of 8,055 ppm in positive samples (CLPSD 1989). However, earlier data from the CLPSD (1980-1983) indicate a geometric mean concentration of boron of 21 mg/kg and a maximum concentration of 320 mg/kg (Eckel and Langley 1988), essentially equivalent to reported background levels of boron in soil. Clarification of

5. POTENTIAL FOR HUMAN EXPOSURE

the discrepancy in the data is necessary in order to compare boron levels at hazardous waste sites to background levels.

5.3 ENVIRONMENTAL FATE

5.3.1 Transport and Partitioning

Boron is a nonvolatile metalloid that occurs in combination with most of the other elements known (Cotton and Wilkinson 1980). Atmospheric boron may be in the form of particulate matter or aerosols as borides, boron oxides, borates, boranes, organoboron compounds, trihalide boron compounds, or borazines. Borates are relatively soluble in water, and will probably be removed from the atmosphere by precipitation and dry deposition (EPA 1987c). The half-life of airborne particles is usually on the order of days, depending on the size of the particle and atmospheric conditions (Nriagu 1979). No specific information on the fate of atmospheric boron was located.

Boron readily hydrolyzes in water to form the electrically neutral, weak monobasic acid H_3BO_3 and the monovalent ion $B(OH)_3$. In concentrated solutions, boron may polymerize, leading to the formation of complex and diverse molecular arrangements. Rai et al. (1986) concluded that because most environmentally relevant boron minerals are highly soluble in water, it is unlikely that mineral equilibria will control the fate of boron in water. Waggott (1969), for example, noted that boron is not significantly removed during the conventional treatment of waste water. Boron may, however, be co-precipitated with aluminum, silicon, or iron to form hydroxyborate compounds on the surfaces of minerals (Biggar and Fireman 1960).

Water borne boron may be adsorbed by soils and sediments. Adsorption-desorption reactions are expected to be the only significant mechanism that will influence the fate of boron in water (Rai et al. 1986). The extent of boron adsorption depends on the pH of the water and the chemical composition of the soil. The greatest adsorption is generally observed at pH 7.5-9.0 (Keren et al. 1981; Keren and Mezuman 1981; Waggott 1969). Bingham et al. (1971) concluded that the single most important property of soil that will influence the mobility of boron is the abundance of amorphous aluminum oxide. The extent of boron adsorption has also been attributed to the levels of iron oxide (Sakata 1987), and to a lesser extent, the organic matter present in the soil (Parks and White 1952), although other studies (Mezuman and Keren 1981) found that the amount of organic matter present was not important.

The adsorption of boron may not be reversible in some soils. The lack of reversibility may be the result of solid-phase formation on mineral surfaces (Rai et al. 1986), and/or the slow release of boron by diffusion from the interior of clay minerals (Griffin and Bureau 1974).

Partition coefficients such as adsorption constants describe the tendency of a chemical to partition from water to solid phases. Adsorption constants for inorganic constituents such as boron cannot be predicted a priori, but must be measured for each soil-water combination. Compilations of

5. POTENTIAL FOR HUMAN EXPOSURE

available data for boron are given elsewhere (Rai et al. 1986). In general, boron adsorption will be most significant in soils that contain high concentrations of amorphous aluminum and iron oxides and hydroxides such as the reddish Ultisols in the southeastern United States.

It is unlikely that boron is bioconcentrated significantly by organisms from water. A bioconcentration factor (BCF) relates the concentration of a chemical in the tissues of aquatic and terrestrial animals or plants to the concentration of the chemical in water or soil. The BCFs of boron in marine and freshwater plants, fish, and invertebrates were estimated to be less than 100 (Thompson et al. 1972). Experimentally measured BCFs for fish have ranged from 52 to 198 (Tsui and McCart 1981). These BCFs suggest that boron is not significantly bioconcentrated. Boron in water is completely absorbed by the human system, but it does not accumulate in body tissues (Waggott 1969). No other experimentally measured BCFs were located. LD

5.3.2 Transformation and Degradation

5.3.2.1 Air

There is no information available that suggests that particulate boron compounds are transformed or degraded in the atmosphere.

5.3.2.2 Water

Elemental boron is inert in the presence of water. Boron compounds rapidly transform to borates, the naturally occurring form of boron, in the presence of water. No further degradation is possible. Borate and boric acid are in equilibrium depending only on the pH of the water. If dissolved in atmospheric water, the standard borate-boric acid equilibria are established.

5.3.2.3 Soil

Most boron compounds are transformed to borates in soil due to the presence of moisture. Borates themselves are not further degraded in soil. However, borates can exist in a variety of forms in soil (see Section 5.2.3). Borates are removed from soils by water leaching and by assimilation by plants.

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

5.4.1 Air

There are few studies made to estimate the concentration of boron-containing compounds in ambient air. This is partly due to difficulties of analysis at the low levels involved. Bertine and Goldberg (1971) estimated that approximately 11,600 tons of boron are injected into the atmosphere as a component of fly ash produced by coal combustion which was estimated to contain an average of about 75 mg/kg boron.

5. POTENTIAL FOR HUMAN EXPOSURE

5.4.2 Water

Boron is widely distributed in surface water and groundwater. Average surface water concentration in the United States is about 0.1 mg boron/L (Butterwick et al. 1989; EPA 1986b), but concentrations vary greatly, depending on boron content of local geologic formations and anthropogenic sources of boron (Butterwick et al. 1989). A survey of U.S. surface waters detected boron in 98% of 1,577 samples at concentrations ranging from 0.001 to 5 mg boron/L. Mean concentrations calculated for the 15 drainage basins in the continental United States ranged from 0.019 mg boron/L in the Western Great Lakes Basin to 0.289 mg boron/L in the Western Gulf Basin (Butterwick et al. 1989). The concentration of boron in sea water is about 4.5 mg/L (Butterwick et al. 1989; EPA 1986b).

Several studies have measured boron concentrations in water in those areas of California with boron-rich deposits. Reported high boron concentrations in surface waters ranged from 15 mg boron/L in coastal drainage waters to 360 mg boron/L in a boron-rich lake (Butterwick et al. 1989; Deverel and Millard 1988). Mean boron concentration in a California river ranged from 0.30 to 0.50 mg boron/L over a 20-year period (Butterwick et al. 1989). Reported boron concentrations in groundwater in the San Joaquin Valley ranged from 0.14 to 120 mg boron/L with a median concentration of about 4 mg boron/L (Butterwick et al. 1989; Deverel and Millard 1988). Waggott (1969) reports that groundwater boron concentrations greater than 100 mg/L are common in California.

Drinking water surveys generally do not report boron concentration. However, concentrations of boron in tap water have been reported in a range of 0.007-0.2 mg/L in the United States and England (Choi and Chen 1979; Waggott 1969), and the National Inorganics and Radionuclides Survey completed in 1987 reported relatively widespread occurrence of boron in 989 public water supplies (NIRS 1987). Boron concentrations ranged from less than 0.005 to greater than 2 mg/L, with concentrations of up to 0.4 mg/L in 90% of systems (NIRS 1987). A survey of 969 public water supply systems showed 99% contained boron at less than 1 mg/L. The maximum level measured was 3.28 mg/L (McCabe et al. 1970).

5.4.3 Soil

Background boron levels in U.S. soils were reported at a geometric mean concentration of 26 mg/kg with a maximum concentration of 300 mg/kg (Eckel and Langley 1988). Boron was detected in soils in Idaho at geometric mean concentrations of 4.6-9.8 mg/kg (Rope et al. 1988) and in sediments of Puget Sound (Malins et al. 1984).

Boron is an essential nutrient for plants. Boron soil concentrations for optimum plant growth reportedly range from 0.1 to 0.5 mg/kg for several plant species (Butterwick et al. 1989).

5. POTENTIAL FOR HUMAN EXPOSURE

5.4.4 Other Environmental Media

Boron is assimilated by plants from soil and is therefore a natural constituent of many foods, mainly fruits and vegetables. The amount of boron absorbed varies considerably among different plant species (Butterwick et al. 1989). The Food and Drug Administration (FDA) has set a tolerance limit of 8 ppm boron for citrus fruit (21 CFR 180.271).

Boron compounds are present in several consumer products. Sodium borate and boric acid are widely used in cosmetics. Over 600 cosmetic products, including makeup, skin and hair care preparations, and shaving creams, contain these compounds at concentrations of up to 5% (Beyer et al. 1983). These compounds have also been used in insecticide powders for roach control, in medicines applied to the skin at concentrations up to 5% (Beyer et al. 1983) and in some laundry products (Butterwick et al. 1989; Stokinger 1981; Waggott 1969).

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Human exposure to borates may occur through ingestion of food and water or insecticides used to control cockroaches, powders or dusts, inhalation of boron-containing or absorption of boron from cosmetics or medical preparations through mucous membranes or damaged skin. The most appreciable boron exposure to the general population is likely to be ingestion of food and to a lesser extent in water. Estimates of average daily boron ingestion by humans range from 10 to 25 mg (Beyer et al. 1983; Waggott 1969).

Occupational exposure to boron compounds may be higher. Workers in industries producing or using boron or boron compounds may be exposed by inhalation to boron-containing dusts or gaseous boron compounds due to process upsets or faulty equipment. Dermal absorption of boron may also occur if damaged skin is in contact with these materials, but this is considered a minor pathway (Stokinger 1981).

Borate dusts have been monitored in workplace air. Reported concentrations of borax dust in different areas of a large borax mining and refining plant ranged from 1.1 to 14.6 mg/m³ (Garabrant et al. 1985) and the mean boric acid/boron oxide dust concentration in a boric acid manufacturing plant was 4.1 mg/m³ (Garabrant et al. 1984). These values indicate that permissible exposure limits (PELs) set by OSHA, or threshold limit values (TLVs) recommended by the ACGIH, for boron-containing dusts in workplace air (Table 7-1) may, at times, be exceeded. Other industries include manufacture of fiberglass and other glass products, cleaning and laundry products, fertilizers, pesticides, and cosmetics (U.S. Borax and Chemical Corporation 1991; Stokinger 1981). Median normal values of boron in human blood (9.76 µg/100 g) and urine samples from these workers (720 µg boron/L) were reported (Stokinger 1981). Boron was not detected in a national survey of human adipose tissue (Stanley 1986). The National Institute for Occupational Safety and Health (NIOSH) estimated that the number of workers potentially exposed to boron increased from 6,500 in the early 1970s (NOHS 1989) to 35,600

5. POTENTIAL FOR HUMAN EXPOSURE

in the early 1980s (NOES 1989). Neither the NOHS nor the NOES databases contain information on the frequency, concentration, or duration of exposures of workers to any of the chemicals listed therein. These surveys provide only estimates of the number of workers potentially exposed to chemicals in the workplace. Sittig (1985) reports that NIOSH estimated the number of workers potentially exposed to borax at 2,490,000, to boron oxide at 21,000, and to boron trifluoride at 50,000.

5.6 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

The populations living in areas of California and other western states with boron-rich geological deposits have potentially high exposure to boron from drinking water and locally grown foods (Butterwick et al. 1989). Individuals using boron-containing cosmetics or medicines extensively, especially on damaged skin, may be exposed to higher-than-normal levels of boron (Beyer et al. 1983). Infants may be at risk in homes where boric acid containing roach powder on floor parameters is used to control cockroaches.

Workers in industries producing or using boron-containing materials also have potentially high exposure as noted above (Section 5.5). People living in the vicinity of waste sites are also at risk of higher-than-normal exposure levels.

5.7 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of boron is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of boron.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.7.1 Data Needs

Physical and Chemical Properties. The solubilities of many boron minerals are not known precisely, but this lack of detailed information may not be a major limitation, since it appears unlikely that mineral equilibria significantly influence the fate of boron in the environment.

5. POTENTIAL FOR HUMAN EXPOSURE

Production, Import/Export, Use, and Disposal. The production volume and uses of boron and boron compounds are well documented (Ferguson et al. 1982; HSDB 1989; U.S. Bureau of Mines 1989). However, data on disposal methods and volume would allow better estimation of human exposure to boron from this source.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1987, became available in May of 1989. However, neither boron nor boron-related compounds are currently listed in the database. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

Environmental Fate. The only quantifiable mechanism that influences the fate of boron is soil adsorption (Rai et al. 1986). Additional research with soils that do not have significant quantities of aluminum and iron oxide may provide a more comprehensive view of the mobility of boron in the environment.

Bioavailability from Environmental Media. Boron compounds can be absorbed following inhalation of contaminated workplace air, ingestion of contaminated food, or through damaged skin (Draize and Kelley 1959; Wong et al. 1964). The most significant routes of exposure near hazardous waste sites are likely to be through drinking boron-contaminated water and ingestion of locally grown food (Beyer et al. 1983; Butterwick et al. 1989; CLPSD 1989). While exposure can occur by these routes, quantitative data on amounts absorbed or are bioavailable would be useful in clarifying the toxic potential of boron in humans.

Food Chain Bioaccumulation. Only one study was located where boron bioconcentration was actually measured (Tsui and McCart 1981). Future research may be helpful, but it appears that boron is not significantly bioconcentrated. There are no data on the biomagnification of boron in the food chain, but it is not likely that bioaccumulation is a major environmental concern.

Exposure Levels in Environmental Media. Data on boron levels in surface water and soil are extensive (Butterwick et al. 1989; Eckel and Langley 1988; EPA 1986b), but additional data on air, food, and drinking water concentrations of boron would be useful in increasing the accuracy of human exposure estimates.

Exposure Levels in Humans. Normal levels of boron in human blood and urine have been reported (Stokinger 1981). Additional data on blood and/or urine concentrations in individuals with potentially high exposure to boron would be useful in assessing the magnitude of human exposure.

Exposure Registries. No exposure registries for boron were located. This compound is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The compound will be

5. POTENTIAL FOR HUMAN EXPOSURE

considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to the exposure to this compound.

5.7.2 On-going Studies

No information was located on any on-going studies on the fate, transport, or potential for human exposure for boron.

6. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting and/or measuring and monitoring boron in environmental media and in biological samples. The intent is not to provide an exhaustive list of analytical methods that could be used to detect and quantify boron. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used to detect boron in environmental samples are the methods approved by federal agencies such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that refine previously used methods to obtain lower detection limits, and/or to improve accuracy and precision.

6.1 BIOLOGICAL MATERIALS

Methods for the determination of boron in samples of toxicological interest have been summarized (Stokinger 1981; Van Ormer 1975). Usually total boron is determined, although in limited cases specific boron species can be determined as well. Boron is very poorly measured by atomic absorption analysis. High-temperature atomic spectrometric methods, especially inductively coupled plasma atomic emission spectrometry, including atomic emission spectrography, work well for boron. Colorimetry and prompt neutron activation analysis can also be used.

Methods for the determination of boron in biological materials are summarized in Table 6-1.

Normally, for determination in biological samples, the sample is digested or ashed, and the boron is measured by atomic spectrometric determination.

6.2 ENVIRONMENTAL SAMPLES

Methods for the determination of boron in environmental samples are summarized in Table 6-2.

Boron is readily measured in multielement analyses of air, water, and solid waste samples by inductively coupled plasma (ICP) atomic emission spectroscopy, the method of choice for the determination of boron in modern practice. Although not multielement procedures, calorimetric cucumin and calorimetric carmine methods are still reliable methods for the determination of boron in water, air and solid waste samples. These calorimetric procedures provide adequate methods when ICP instrumentation is not available.

TABLE 4-1. Analytical Methods for Determining Boron in Biological Materials

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Blood and urine	Ashed, dissolved in HCl	SA	5 µg/100g blood 40 µg/l urine	No data	Imbus et al. 1963
Blood	Ashed by oxygen in a Parr bomb, dissolved	Colorimetric carmine method	<0.1 µg/ml	84% at 3 µg/ml	Hill and Smith 1959
Serum (borate)	Deproteinized, allowed to react with reagent	Colorimetric carmine method	>endogenous levels which are <20 µg/l	92%-104%	Baselt 1988
Blood	Ashed, dissolved	Electrophoresis	No data	No data	Hill et al. 1957
Biological material ^a	Acid digestion	ICP/AES	5 µg/L ^b	No data	EPA 1988a

^aThis method is for water, sediments, and wastes.

^bMethod detection limit. Actual detection limits for boron in waste samples may be considerably higher.

ICP/AES = inductively coupled plasma atomic emission spectroscopy; HCl = hydrochloric acid; SA = atomic spectrographic analysis

TABLE 6-2. Analytical Methods for Determining Boron in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air	Collection on filter, workup in acid	ICP/AES	1 µg per sample	103% recovery	NIOSH 1984
Air for boron carbide	Collection on filter,ashed, suspended in 2-propanol, redeposited on silver membrane filter	x-ray powder diffraction	0.05 µg	No data	NIOSH 1985a
Water	Direct analysis	Colorimetric curcumin	0.2 µg	23% RSD	APHA 1985a
Water	Ash, dissolve in acid	Colorimetric curcumin	2 µg	36% RSD	APHA 1985b
Water	Acidify, inject	ICP/AES	0.3 µg/L	No data	APHA 1985c
Water	Direct analysis	Colorimetric curcumin	0.2 µg	23% RSD	EPA 1983
Water	Filter, acidify	ICP/AES aspiration	5 µg/L	No data	EPA 1982
Sediments, solid wastes, sludges	Acid digestion	ICP/AES	5 µg/L ^a	No data	EPA 1986a

^aMethod detection limit. Actual detection limits for boron in waste samples may be 1-3 orders of magnitude higher.

ICP/AES = inductively coupled plasma atomic emission spectroscopy; RSD = relative standard deviation.

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6.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of boron is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of boron.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.3.1 Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Boron can be determined sensitively and selectively by inductively coupled plasma atomic emission analysis (EPA 1986a; Imbus et al. 1963). This method of analysis requires that the analyte be placed in solution, which can be a problem with some of the more refractory boron species. With the exception of boron carbide (NIOSH 1985a), methods are lacking for the determination of specific boron compounds.

Methods for the determination of metabolites of boron in biological materials would be useful in studying the toxicity and metabolism of this element.

More specific methods for biomarkers of exposure would be helpful in toxicological studies of boron.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Inductively coupled plasma atomic emission spectrometry is the only satisfactory multielement method available for the determination of boron in water, air, and solid waste samples (APHA 1985c; EPA 1982, 1986a; NIOSH 1984). Calorimetric procedures are as sensitive and precise but are more labor intensive. Calorimetric procedures do provide adequate methods for those laboratories that do not have ICP instrumentation. There is a need for methods that require less expensive instrumentation, although such methods would be very difficult to develop.

Sampling methodologies for very low level elemental substances like boron continue to pose problems such as nonrepresentative samples, insufficient sample volumes, contamination, and labor-intensive, tedious extraction and purification procedures (Green and LePape 1987).

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6.3.2 On-going Studies

Examination of the literature does not suggest that major efforts are underway for the development of better methods for the determination of boron. This is due to the difficulties inherent in determining boron and the fact that an emphasis has not been placed on developing such methods because the element is relatively nontoxic.

7. REGULATIONS AND ADVISORIES

Because of its potential to cause adverse health effects in exposed people, a number of regulations and guidelines have been established for boron and its compounds by various national and state agencies. These values are summarized in Table 7-1.

7. REGULATIONS AND ADVISORIES

TABLE 7-1. Regulations and Guidelines Applicable to Boron and Compounds

Agency	Description	Information	Reference
<u>NATIONAL</u>			
Regulations:			
a. Air:			
OSHA	PEL TWA Boron oxide Total dust Respirable fraction Sodium tetraborates Ceiling Boron tribromide Boron trifluoride	10 mg/m ³ 5 mg/m ³ 10 mg/m ³ 10 mg/m ³ (1 ppm) 3 mg/m ³ (3 ppm)	OSHA 1989 (29 CFR 1910.1000) Table 2-1-A
b. Water:			
EPA OWQS	General permits under NPDES Boron, total	Yes	40 CFR 122, Appendix D, Table IV
c. Food:			
FDA	Food additive-modified hop extract Boron	310 ppm	21 CFR 172.360
d. Other:			
EPA OERR	Reportable quantity (proposed) Boron trichloride Boron trifluoride	100 lbs 100 lbs	EPA 1989b
	Extremely Hazardous Substance (PQ) Boron trichloride Boron trifluoride	300 lbs 300 lbs	EPA 1987a (40 CFR 355)
EPA OPP	Tolerances for pesticide chemicals on raw agricultural commodities Boron	8 to 10 ppm	40 CFR 180.271
Guidelines:			
a. Air:			
ACGIH	TLV TWA Sodium tetraborates Anhydrous and pentahydrate Decahydrate Boron oxide Ceiling Boron tribromide Boron trifluoride	1 mg/m ³ 3 mg/m ³ 10 mg/m ³ 1 ppm (10 mg/m ³) 1 ppm (7 mg/m ³)	ACGIH 1986
NIOSH	IDLH Boron trifluoride	100 ppm	NIOSH 1983b
b. Water:			
EPA OWQS	Ambient Water Quality Criteria Long-term irrigation on sensitive crops	730 µg/L	EPA 1986b
c. Other:			
EPA	Oral RfD Boron and Borates	9E-2 mg/kg/day	IRIS 1989

7. REGULATIONS AND ADVISORIES

TABLE 7-1 (Continued)

Agency	Description	Information	References
STATE			
Regulations and Guidelines:			
a. Air:	Acceptable ambient air concentrations		MATCHE 1980
Connecticut	Sodium tetraborates	20 $\mu\text{g}/\text{m}^3$ (8 hr) 100 $\mu\text{g}/\text{m}^3$ (8 hr)	
	Boron oxide	200 $\mu\text{g}/\text{m}^3$ (8 hr)	
	Boron tribromide	200 $\mu\text{g}/\text{m}^3$ (8 hr)	
	Boron trifluoride	0 $\mu\text{g}/\text{m}^3$ (8 hr)	
Nevada	Sodium tetraborates	2.4E-2 mg/m^3 (8 hr)	
	Boron oxide	2.38E-1 mg/m^3 (8 hr)	
	Boron tribromide	2.38E-1 mg/m^3 (8 hr)	
	Boron trifluoride	7.1E-2 mg/m^3 (8 hr)	
North Dakota	Sodium tetraborates	1.0E-2 mg/m^3 (8 hr) 5.0E-2 mg/m^3 (8 hr)	
	Boron oxide	1.0E-2 mg/m^3 (8 hr)	
	Boron tribromide	1.0E-1 mg/m^3 (8 hr)	
	Boron trifluoride	5.0E-2 mg/m^3 (8 hr)	
Virginia	Sodium tetraborates	16 $\mu\text{g}/\text{m}^3$ (24 hr)	
	Boron oxide	140 $\mu\text{g}/\text{m}^3$ (24 hr)	
	Boron tribromide	80 $\mu\text{g}/\text{m}^3$ (24 hr)	
	Boron trifluoride	25 $\mu\text{g}/\text{m}^3$ (24 hr)	

ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; IDLH = Immediately Dangerous to Life or Health Level; NIOSH = National Institute for Occupational Safety and Health; NPDES = National Pollutant Discharge Elimination System; OERR = Office of Emergency and Remedial Response; OPP = Office of Pesticide Products; OSHA = Occupational Safety and Health Administration; OWRS = Office of Water Regulations and Standards; PEL = Permissible Exposure Limit; TLV = Threshold Limit Value; TPQ = Threshold Planning Quantity; TWA = Time-Weighted Average

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9. GLOSSARY

Acute Exposure -- Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Adsorption Coefficient (K_{oc}) -- The ratio of the amount of a chemical adsorbed per unit weight of organic carbon in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (K_d) -- The amount of a chemical adsorbed by a sediment or soil (i.e., the solid phase) divided by the amount of chemical in the solution phase, which is in equilibrium with the solid phase, at a fixed solid/solution ratio. It is generally expressed in micrograms of chemical sorbed per gram of soil or sediment.

Bioconcentration Factor (BCF) -- The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the **same time** or during the same period.

Cancer Effect Level (CEL) -- The lowest dose of chemical in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Carcinogen -- A chemical capable of inducing cancer.

Ceiling Value -- A concentration of a substance that should not be exceeded, even instantaneously.

Chronic Exposure -- Exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Developmental Toxicity -- The occurrence of adverse effects on the developing organism that may result from exposure to a chemical prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Embryotoxicity and Fetotoxicity -- Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and in utero death.

EPA Health Advisory -- An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Immediately Dangerous to Life or Health (IDLH) -- The maximum environmental concentration of a contaminant from which one could escape within 30 min without any escape-impairing symptoms or irreversible health effects.

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Intermediate Exposure -- Exposure to a chemical for a duration of 15-364 days as specified in the Toxicological Profiles.

Immunologic Toxicity -- The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

In Vitro -- Isolated from the living organism and artificially maintained, as in a test tube.

In Vivo -- Occurring within the living organism.

Lethal Concentration_(LO) (LC_{LO}) -- The lowest concentration of a chemical in air which has been reported to have caused death in humans or animals.

Lethal Concentration ₍₅₀₎ (LC₅₀) -- A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

Lethal Dose _(LO) (LD_{LO}) -- The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals.

Lethal Dose₍₅₀₎ (LD₅₀) -- The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time₍₅₀₎ (LT₅₀) -- A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Lowest-Observed-Adverse-Effect Level (LOAEL) -- The lowest dose of chemical in a study or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Malformations -- Permanent structural changes that may adversely affect survival, development, or function.

Minimal Risk Level -- An estimate of daily human exposure to a chemical that is likely to be without an appreciable risk of deleterious effects (noncancerous) over a specified duration of exposure.

Mutagen -- A substance that causes mutations. A mutation is a change in the genetic material in a body cell. Mutations can lead to birth defects, miscarriages, or cancer.

Neurotoxicity -- The occurrence of adverse effects on the nervous system following exposure to chemical.

No-Observed-Adverse-Effect Level (NOAEL) -- The dose of chemical at which there were no statistically or biologically significant increases in frequency

9. GLOSSARY

or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Octanol-Water Partition Coefficient (K_{ow}) -- The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Permissible Exposure Limit (PEL) -- An allowable exposure level in workplace air averaged over an 8-hour shift.

q_1^* -- The upper-bound estimate of the low-dose slope of the dose-response curve as determined by the multistage procedure. The q_1^* can be used to calculate an estimate of carcinogenic potency, the incremental excess cancer risk per unit of exposure (usually $\mu\text{g/L}$ for water, mg/kg/day for food, and $\mu\text{g/m}^3$ for air).

Reference Dose (RfD) -- An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to nonthreshold effects such as cancer.

Reportable Quantity (RQ) -- The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are: (1) 1 lb or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity -- The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Short-Term Exposure Limit (STEL) -- The maximum concentration to which workers can be exposed for up to 15 min continually. No more than four excursions are allowed per day, and there must be at least 60 min between exposure periods. The daily TLV-TWA may not be exceeded.

Target Organ Toxicity -- This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

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Teratogen -- A chemical that causes structural defects that affect the development of an organism,

Threshold Limit Value (TLV) -- A concentration of a substance to which most workers can be exposed without adverse effect. The TLV may be expressed as a TWA, as a STEL, or as a CL.

Time-weighted Average (TWA) -- An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Toxic Dose (TD₅₀) -- A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Uncertainty Factor (UF) -- A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

APPENDIX A

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile *is* a health effects summary written in nontechnical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or substance release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the substance.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Tables and Figures for Levels of Significant Exposure (LSE)

Tables (2-1, 2-2, and 2-3) and figures (2-1 and 2-2) are used to summarize health effects by duration of exposure and endpoint and to illustrate graphically levels of exposure associated with those effects. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse-Effect Levels (NOAELs), Lowest-Observed- Adverse-Effect Levels (LOAELs) for Less Serious and Serious health effects, or Cancer Effect Levels (CELs). In addition, these tables and figures illustrate differences in response by species, Minimal Risk Levels (MRLs) to humans for noncancer endpoints, and EPA's estimated range associated with an upper-bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. The LSE tables and figures can be used for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text.

The legends presented below demonstrate the application of these tables and figures. A representative example of LSE Table 2-1 and Figure 2-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See LSE Table 2-1

- 1) Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exist,

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three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 2-1, 2-2, and 2-3, respectively). LSE figures are limited to the inhalation (LSE Figure 2-1) and oral (LSE Figure 2-2) routes.

- 2) Exposure Duration Three exposure periods: acute (14 days or less); intermediate (15 to 364 days); and chronic (365 days or more) are presented within each route of exposure. In this example, an inhalation study of intermediate duration exposure is reported.
- 3) Health Effect The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table.
- 4) Key to Figure Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to define a NOAEL and a Less Serious LOAEL (also see the two "18r" data points in Figure 2-1).
- 5) Species The test species, whether animal or human, are identified in this column.
- 6) Exposure Frequency/Duration The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to [substance x] via inhalation for 13 weeks, 5 days per week, for 6 hours per day.
- 7) System This column further defines the systemic effects. These systems include: respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, one systemic effect (respiratory) was investigated in this study.
- 8) NOAEL A No-Observed-Adverse-Effect Level (NOAEL) is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system which was used to derive an intermediate exposure, inhalation MRL of 0.005 ppm (see footnote "c").
- 9) LOAEL A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest exposure level used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to

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quantify the adverse effect accompanies the LOAEL. The "Less Serious" respiratory effect reported in key number 18 (hyperplasia) occurred at a LOAEL of 10 ppm.

- 10) Reference The complete reference citation is given in Chapter 8 of the profile.
- 11) CEL A Cancer Effect Level (CEL) is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiological studies. CELs are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses which did not cause a measurable increase in cancer.
- 12) Footnotes Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "c" indicates the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See LSE Figure 2-1

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure levels for particular exposure duration.

- 13) Exposure Duration The same exposure periods appear as in the LSE table. In this example, health effects observed within the intermediate and chronic exposure periods are illustrated.
- 14) Health Effect These are the categories of health effects for which reliable quantitative data exist. The same health effects appear in the LSE table.
- 15) Levels of Exposure Exposure levels for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure levels are reported on the log scale "y" axis. Inhalation exposure is reported in mg/m³ or ppm and oral exposure is reported in mg/kg/day.
- 16) NOAEL In this example, 18r NOAEL is the critical end point for which an intermediate inhalation exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates a NOAEL for the test species (rat). The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).
- 17) CEL Key number 38r is one of three studies for which Cancer Effect Levels (CELs) were derived. The diamond symbol refers to a CEL for the test species (rat). The number 38 corresponds to the entry in the LSE table.

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- 18) Estimated Upper-Bound Human Cancer Risk Levels This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1^*).
- 19) Key to LSE Figure The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 → TABLE 2-1. Levels of Significant Exposure to [Chemical x] - Inhalation

Key to figure ^a	Species	Exposure frequency/duration	System	NOAEL (ppm)	LOAEL (effect)		Reference
					Less serious (ppm)	Serious (ppm)	
2 →	INTERMEDIATE EXPOSURE						
3 →	Systemic	5	6	7	8	9	10
4 →	18	Rat	13 wk 5d/wk 6hr/d	Resp	3 ^b	10 (hyperplasia)	Nitschke et al. 1981

CHRONIC EXPOSURE							
Cancer							
38	Rat	18 mo 5d/wk 7hr/d				11 20 (CEL, multiple organs)	Wong et al. 1982
39	Rat	89-104 wk 5d/wk 6hr/d				10 (CEL, lung tumors, nasal tumors)	HTP 1982
40	Mouse	79-103 wk 5d/wk 6hr/d				10 (CEL, lung tumors, hemangiosarcomas)	HTP 1982

^a The number corresponds to entries in Figure 2-1.

12 → ^b Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5×10^{-3} ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

CEL = cancer effect level; d = day(s); hr = hour(s); LOAEL = lowest observed adverse-effect level; mo = month(s); NOAEL = no observed adverse-effect level; Resp = respiratory; wk = week(s)

SAMPLES

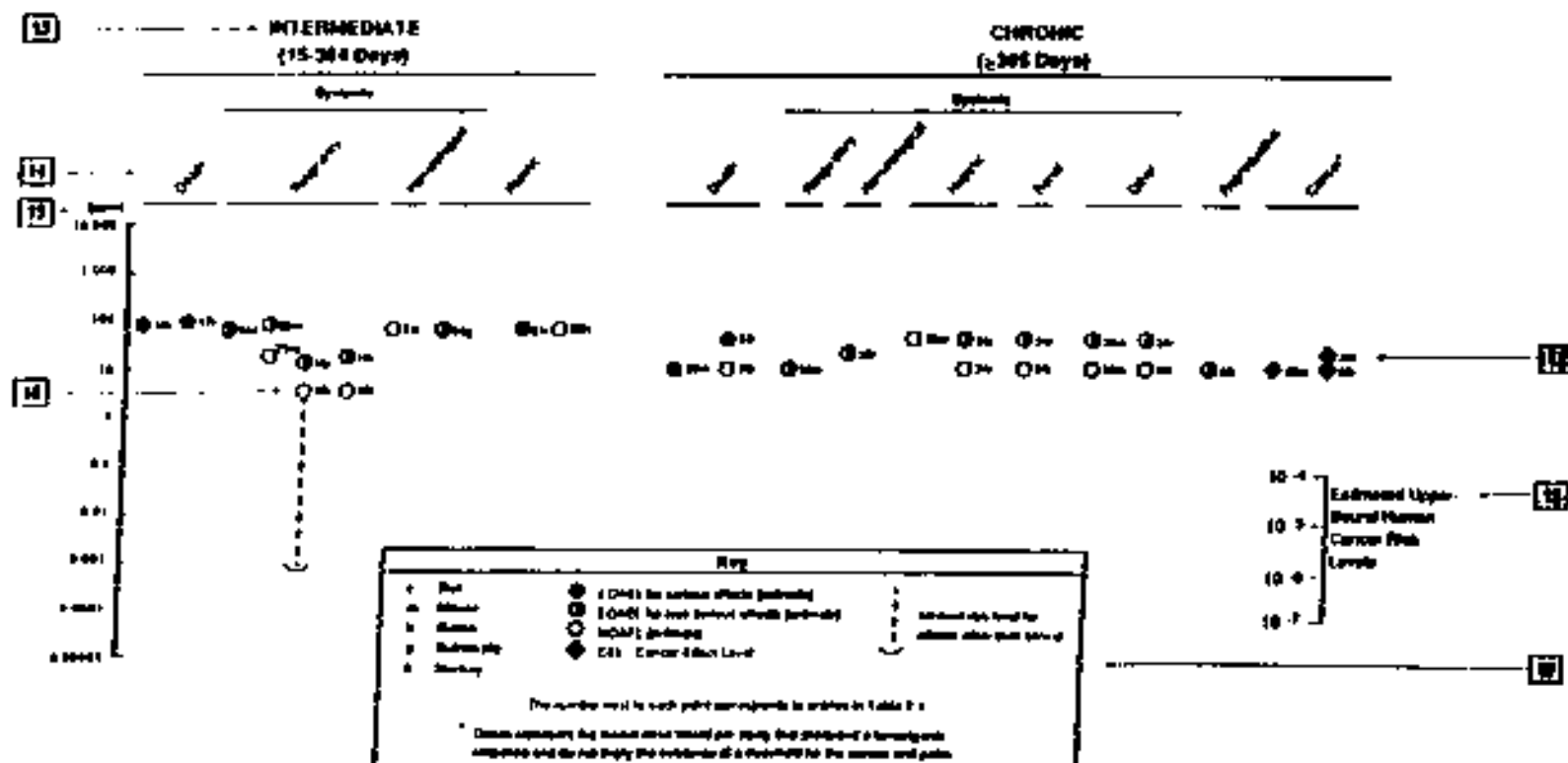


FIGURE 2-1. Levels of Significant Exposure to [Chemical X]-Inhalation

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Chapter 2 (Section 2.4)

Relevance to Public Health

The Relevance to Public Health section provides a health effects summary based on evaluations of existing toxicological, epidemiological, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?
2. What effects observed in animals are likely to be of concern to humans?
3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The section discusses health effects by end point. Human data are presented first, then animal data. Both are organized by route of exposure (inhalation, oral, and dermal) and by duration (acute, intermediate, and chronic). In vitro data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this section. If data are located in the scientific literature, a table of genotoxicity information is included.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. MRLs for noncancer end points if derived, and the end points from which they were derived are indicated and discussed in the appropriate section(s).

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Identification of Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information was available, MRLs were derived. MRLs are specific for route (inhalation or oral) and duration (acute, intermediate, or chronic) of exposure. Ideally, MRLs can be derived from all six exposure scenarios (e.g., Inhalation - acute, -intermediate, -chronic; Oral - acute, -intermediate, -chronic). These MRLs are not meant to support regulatory action, but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a substance emission, given the concentration of a contaminant in air or the estimated daily dose received via food or water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

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MRL users should be familiar with the toxicological information on which the number is based. Section 2.4, "Relevance to Public Health," contains basic information known about the substance. Other sections such as 2.6, "Interactions with Other Chemicals" and 2.7, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology used by the Environmental Protection Agency (EPA) (Barnes and Dourson, 1988; EPA 1989a) to derive reference doses (RfDs) for lifetime exposure.

To derive an MRL, ATSDR generally selects the end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential effects (e.g., systemic, neurological, and developmental). In order to compare NOAELs and LOAELs for specific end points, all inhalation exposure levels are adjusted for 24hr exposures and all intermittent exposures for inhalation and oral routes of intermediate and chronic duration are adjusted for continuous exposure (i.e., 7 days/week). If the information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. The NOAEL is the most suitable end point for deriving an MRL. When a NOAEL is not available, a Less Serious LOAEL can be used to derive an MRL, and an uncertainty factor (UF) of 10 is employed. MRLs are not derived from Serious LOAELs. Additional uncertainty factors of 10 each are used for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the adjusted inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables.

APPENDIX B

ACRONYMS, ABBREVIATIONS, AND SYMBOLS USED IN TEXT

ACGIH	American Conference of Governmental Industrial Hygienists
ADME	Absorption, Distribution, Metabolism, and Excretion
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BSC	Board of Scientific Counselors
CDC	Centers for Disease Control
CEL	Cancer Effect Level
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
CLP	Contract Laboratory Program
cm	centimeter
CNS	central nervous system
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DOL	Department of Labor
ECG	electrocardiogram
EEG	electroencephalogram
EPA	Environmental Protection Agency
EKG	see ECG
FAO	Food and Agricultural Organization of the United Nations
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
f ₁	first generation
fpm	feet per minute
ft	foot
FR	Federal Register
g	gram
GC	gas chromatography
HPLC	high performance liquid chromatography
hr	hour
IDLH	Immediately Dangerous to Life and Health
IARC	International Agency for Research on Cancer
ILO	International Labor Organization
in	inch
Kd	adsorption ratio
kg	kilogram
Koc	octanol-soil partition coefficient
Kow	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC _{Lo}	lethal concentration low
LC ₅₀	lethal concentration 50 percent kill
LD _{Lo}	lethal dose low
LD ₅₀	lethal dose 50 percent kill
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
m	meter

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mg	milligram
min	minute
mL	milliliter
mm	millimeters
mmol	millimole
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectroscopy
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
nm	nanometer
ng	nanogram
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPL	National Priorities List
NRC	National Research Council
NTIS	National Technical Information Service
NTP	National Toxicology Program
OSHA	Occupational Safety and Health Administration
PEL	permissible exposure limit
pg	picogram
pmol	picomole
PHS	Public Health Service
PMR	proportional mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
REL	recommended exposure limit
Rfd	Reference Dose
RTECS	Registry of Toxic Effects of Chemical Substances
sec	second
SCE	sister chromatid exchange
SIC	Standard Industrial Classification
SMR	standard mortality ratio
STEL	short-term exposure limit
STORET	<u>STORAGE</u> and <u>RETRIEVAL</u>
TLV	threshold limit value
TSCA	Toxic Substances Control Act
TRI	Toxic Release Inventory
TWA	time-weighted average
U.S.	United States
UF	uncertainty factor
WHO	World Health Organization
>	greater than
≥	greater than or equal to

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=	equal to
<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
δ	delta
γ	gamma
μm	micron
μg	microgram

APPENDIX C

PEER REVIEW

A peer review panel was assembled for boron. The panel consisted of the following members: Dr. Rajender Abraham, a private consultant; Dr. Hugh Evans, Associate Professor of Chemistry, Institute of Environmental Medicine, New York University Medical Center; Dr. Ernest Foulkes, Director, Department of Environmental Health, University of Cincinnati; and Dr. William Buck, Professor of Toxicology, College of Veterinary Medicine, University of Illinois. These experts collectively have knowledge of boron's physical and chemical properties, toxicokinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. A second panel of reviewers was assembled to review the sections on mitigation of effects. This panel consisted of: Dr. Brent Burton, Medical Director, Oregon Poison Center, Oregon Health Sciences University, Portland, Oregon; Dr. Alan Hall, Private Consultant, Evergreen, Colorado; and Dr. Alan Woolf, Director of Clinical Pharmacology and Toxicology, Massachusetts Poison Control System, The Children's Hospital, Boston, Massachusetts. All reviewers were selected in conformity with the conditions for peer review specified in the Comprehensive Environmental Response, Compensation, and Liability Act of 1986, Section 104.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers' comments and determined which comments will be included in the profile. A listing of the peer reviewers' comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.