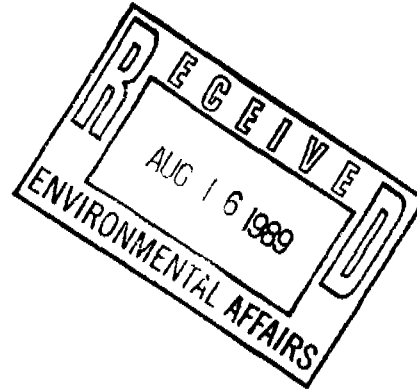




PPG Industries, Inc. Chemicals P.O. Box 1000 Lake Charles, Louisiana 70602

T. G. Brown
Works Manager

August 14, 1989



Ms. Maureen O'Neill
Assistant Secretary
Office of Water Resources
Dept. of Environmental Quality
P O Box 14091
Baton Rouge, LA 70804

Re: Comments on Proposed 1989 Louisiana Water Quality Standards

Dear Ms. O'Neill:

Enclosed is one copy of the Chem Risk™ report entitled:
Evaluation of the Proposed Louisiana Ambient Water Quality
Criteria, dated August 10, 1989. It is Attachment "A" to the PPG
comments which were faxed to your office and also mailed on
Friday, August 11, 1989. Please see that it gets to Mr. Dugan
Sabins and is filed with the PPG comments.

Respectfully,

W. J. Peard
Manager, Laboratory Services/Environmental Control

AJT/WJP/vc

Attachment

bcc: Ed McHamm, EPA Region VI
W. B. Graybill
File 301.2.10

SL 106015

EVALUATION OF PROPOSED LOUISIANA
AMBIENT WATER QUALITY CRITERIA

AUGUST 10, 1989



ChemRisk[™]
A McLaren Company

SL 106016

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Environmental Quality

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EXECUTIVE SUMMARY

This document provides an evaluation of the proposed State of Louisiana ambient water quality criteria for the protection of human health in public water supplies and recreational waters. The state criteria were introduced in the "Proposed Revision Louisiana Administrative Code, Title 33, Environmental Quality, Part IX. Water Quality Regulations, Chapter 11 Louisiana Surface Water Quality Standards" issued by the Louisiana Department of Environmental Quality, Office of Water Resources in June of 1989. These criteria revisions are in accordance with the Federal Clean Water Act (P.L. 92-500 as amended) and the Louisiana Water Control Law (R. S. 30:2071-2078).

The objectives of this criteria evaluation are to:

- provide a critical scientific review of the methods and data used by the DEQ to establish ambient water quality criteria for hexachlorobenzene, hexachlorobutadiene, 1,2-dichloroethane and 1,1,2,2-tetrachloroethane and other chlorinated chemicals of interest;
- review relevant scientific literature on the human health effects of the compounds;
- review and compile information related to the bioaccumulation of the identified compounds;
- evaluate the applicability of the USEPA and DEQ's assumptions and biological data used to develop ambient water quality criteria;

- identify key parameters necessary to more accurately quantify the exposure of humans to water borne contaminants through the consumption of drinking water and fish, and through recreational swimming;
- identify based on refined risk assessment procedures alternative criteria that are protective of human health.

This report addresses the criteria development of 17 chlorinated compounds, with special emphasis on hexachlorobenzene, hexachlorobutadiene, 1,2-dichloroethane and 1,1,2,2-tetrachloroethane. In addition this report provides information on the environmental fate, bioaccumulation and human health effects pertinent to the evaluation of the proposed criteria.

The following conclusions were made regarding the methods used to develop the proposed ambient water quality criteria:

- The equations used by the DEQ are consistent with those recommended by the USEPA except for DEQ's inclusion of an incidental water ingestion rate for swimming activities.
- The criteria were checked for accuracy and were found to be consistent with the methods reported by the DEQ.
- The cancer potency factors and BCF's used by the DEQ for calculating water quality criteria were confirmed for most of the values. However, the cancer potency factors most recently released by the USEPA in July 1989 are not in agreement with five of those cited by DEQ.

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- The carcinogenicity weight of evidence category cited by DEQ for bromoform (Group B2, probable human carcinogen) is not consistent with the USEPA designation (Group D, not classified as to carcinogenicity). The USEPA does not regulate bromoform as a carcinogen. The criteria proposed by DEQ for bromoform were calculated using the equations for carcinogens. This approach is inconsistent with federal policy.
- The RfD used by the DEQ for 1,1,1-trichloroethane is not consistent with that reported by the USEPA, and results in proposed criteria significantly more stringent than the USEPA guideline criteria.
- The criteria proposed by the DEQ were consistently more stringent than the USEPA guideline criteria for recreational waters and in most cases for public water supplies.
- The DEQ and USEPA criteria were consistently more stringent than the alternative criteria developed using refined risk assessment equations. This is due primarily to the screening level assessment approach used by agencies. A screening level risk assessment approach uses overly conservative assumptions and parameter values, which significantly overestimate human exposures.
- The drinking water consumption rate (2.0 L/day) used by the DEQ, however, is reported by the USEPA to be representative of the national 90th percentile water consumption rate rather than an average consumption rate.
- The use of an incidental water ingestion rate for swimming is inconsistent with USEPA methods. The rate used by the DEQ was 36 times greater than the incidental water ingestion rate suggested in the USEPA Exposure Factors Handbook.
- The assumption that 100% of the fish and water consumed by the people of Louisiana is obtained from contaminated water sources is not supported by national consumption survey data.
- The assumption of 100% gastrointestinal absorption efficiency is not justified for selected compounds. Evidence for HCB indicates that gastrointestinal absorption efficiency may be as low as 10% for some chemicals.
- The assumption of no loss of contaminants during food preparation is likely inaccurate. There is evidence that indicates that as much as 90% of the PCB's in fish may be lost during food preparation.

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- A 10^{-6} de minimus risk level was used by the DEQ, however, past federal and state regulatory practices indicate that a 10^{-5} level of risk may be as appropriate for the determination of water quality criteria.
- In general, it was concluded that the alternative criteria based on scientific data and USEPA recommended parameters are representative of levels safe for designated water uses, based on a realistic assessment of health risks.

1.0 INTRODUCTION

This report presents an evaluation of the ambient surface water quality criteria proposed by the state of Louisiana Department of Environmental Quality (DEQ) for 17 chemicals. These chemicals are identified in Table 1-1. The review focuses on hexachlorobenzene (HCB), hexachlorobutadiene (HCBd), 1,2-dichloroethane (or ethylene dichloride, EDC) and 1,1,2,2-tetrachloroethane. In addition, this document examines the criteria proposed for several other groups of chemicals including chlorinated ethanes, chlorinated ethylenes, trihalomethanes and other chlorinated compounds.

The revised criteria were proposed in June, 1989, under the authority of Section 2074 B(1) of the Louisiana Water Control Law (R.S. 30:2071-2078), Section 303(c) of the Federal Clean Water Act (P.L. 92-500 as amended) and 48 FR 51405, November 8, 1983. The stated purposes of the proposed Louisiana numerical criteria for surface waters are to:

- provide for the protection and preservation of the abundant natural resources of Louisiana's many and varied aquatic ecosystems;
- protect the public health and welfare that might otherwise be threatened by degradation of water quality;
- protect and enhance the quality of public waters for designated uses; and
- serve the objectives of the Louisiana Water Control Law and Federal Clean Water Act.

TABLE 1-1

LIST OF CHEMICALS EVALUATED FOR AMBIENT WATER QUALITY CRITERIA

CHEMICALS
<u>Primary</u>
Hexachlorobenzene
Hexachlorobutadiene
1,2-Dichloroethane
1,1,2,2-Tetrachloroethane
<u>Secondary</u>
Carbon Tetrachloride
Chloroform
Bromoform
Bromodichloromethane
Dibromochloromethane
1,1,1-Trichloroethane
1,1,2-Trichloroethane
1,1-Dichloroethylene
Trichloroethylene
Tetrachloroethylene
Vinyl Chloride
Methylene Chloride
Chloromethane

0802SMF.TAB

The water quality criterion for a substance is defined as:

"...the permissible level for that substance at which water quality will remain sufficient to support a designated use" (DEQ, 1989).

The DEQ proposed: 1) criteria protective of human health for both public water supplies and 2) criteria protective of human health and aquatic organisms for non-public water supplies. However, the apparent emphasis of the DEQ criteria development focuses on the protection of human health. In turn, the focus of this evaluation is with human health risk assessment and not aquatic toxicity which may also influence criteria development.

This criteria evaluation uses risk assessment methods to review the scientific basis for these proposed water quality criteria, and also provides bioaccumulation and toxicity data on HCB, HCB_D, EDC and 1,1,2,2-tetrachloroethane, as well as selected trihalomethanes, chlorinated ethanes, chloroethenes, and other chlorinated chemicals. In addition, refinements protective of human health and the environment, and based on good risk assessment practices and current scientific data are incorporated to calculate alternative criteria for selected chemicals.

1.1 Authorization

This assessment was prepared by ChemRisk for PPG Industries, Inc.

1.2 Objectives

Specific objectives of this evaluation were as follows:

- Identify and critique the data, methods, and assumptions used to establish the proposed Louisiana DEQ criteria.
- Evaluate the applicability of the U.S. Environmental Protection Agency's (USEPA) methods and assumptions and biological data used to develop ambient water quality criteria.
- Briefly examine the available data on the health effects and the environmental fate of the trihalomethanes (bromoform, bromodichloromethane, chloroform, dibromochloromethane), chloroethanes (1,1,1-trichloroethane, 1,1,2-trichloroethane), chloroethylenes (1,1-dichloroethylene, trichloroethylene, tetrachloroethylene), and other chlorinated chemicals (carbon tetrachloride, methylene chloride, methyl chloride, vinyl chloride) proposed for regulation by the DEQ.
- Review the scientific literature for the human health effects from exposure to HCD, HCBd, EDC and 1,1,2,2-tetrachloroethane.
- Review the bioaccumulation of the identified compounds.
- Review appropriate information on consumption of fish, ingestion of water and other possible recreational exposure pathways.
- Identify possible refinements to the exposure assumptions, and provide alternative criteria that are protective of human health and the environment for the compounds listed above.

1.3 Criteria Evaluation Approach

This assessment was based on current risk assessment methods, data available in the scientific literature and information on regulatory policy in regard to potential human health risks associated with the ingestion of contaminated fish and water, and exposure through recreational activities.

The criteria evaluation addresses current USEPA and State of Louisiana regulatory policy concerning human health risk assessments, as well as related issues such as the distribution of the compounds in water sediment, and fish tissues, and the pharmacodynamics and toxicity of the absorbed compounds. These factors are important in quantifying human exposures to chemically contaminated water and fish and are, therefore, critical to the evaluation of the proposed Title 33 water quality criteria for the identified chemicals.

1.4 Report Format

SECTION 1.0 INTRODUCTION - The conceptual approach, specific objectives and format of this report are presented.

SECTION 2.0 AMBIENT WATER QUALITY CRITERIA DEVELOPMENT - The methods employed by USEPA and the State of Louisiana DEQ to establish ambient

water quality criteria for carcinogens and non-carcinogens are discussed. Special emphasis is placed on the applicability of parameters and reasonableness of assumptions used to develop health protective criteria.

SECTION 3.0 HEXACHLOROBENZENE (HCB) - This section focuses on the important physical and chemical properties of HCB that determine its environmental and toxicological behavior. The proposed ambient water quality criterion for HCB are evaluated.

SECTION 4.0 HEXACHLOROBUTADIENE (HCBd) - The environmental and toxicological behavior of HCBd is discussed. Special emphasis is placed on the factors affecting the bioavailability of this compound to humans, and the importance of these characteristics in the establishment of ambient water quality criteria for HCBd.

SECTION 5.0 1,2-DICHLOROETHANE (EDC) - The environmental and toxicological behavior of EDC is discussed. Special emphasis is focused on the factors affecting the bioavailability of this compound to humans. The proposed DEQ ambient water quality criteria for EDC are critiqued.

SECTION 6.0 1,1,2,2-TETRACHLOROETHANE - The environmental and toxicological behavior of 1,1,2,2-tetrachlorethane is discussed with a special emphasis on the factors affecting 1,1,2,2-tetrachloroethane

bioavailability. The proposed ambient water quality criteria for 1,1,2,2-tetrachloroethane are evaluated.

SECTION 7.0 SUMMARY CRITERIA EVALUATIONS AND TOXICITY PROFILES FOR ADDITIONAL CHLORINATED COMPOUNDS - In this section the evaluations of the proposed DEQ criteria are summarized for four groups of chlorinated compounds including: trihalomethanes (THM), chlorinated ethanes, chlorinated ethenes and other chlorinated compounds. In addition, summary toxicity profiles are presented outlining the toxicity and fate characteristics of each of the compounds. Proposed DEQ criteria are evaluated.

SECTION 8.0 CONCLUSION

SECTION 9.0 REFERENCES

2.0 METHODS FOR AMBIENT WATER QUALITY CRITERIA DEVELOPMENT

The USEPA is responsible for developing and revising ambient water quality criteria for surface waters under Section 304(a)(1) of the Clean Water Action (33 U.S.C. 1314 (a) 1). The USEPA methods to establish ambient water quality criteria were first described in 1980 (45 FR 231). Numerical criteria have been established for more than 100 chemicals or chemical groups (USEPA, 1986). Numerical criteria for selected chemicals and the methods employed to establish criteria protective of human health have been revised as scientific knowledge has increased.

Ambient water quality criteria are provided by the USEPA to state agencies as scientific data and guidance to be used in the development of water quality standards or regulatory requirements that are protective of human health and the environment are not legally enforceable standards. Ambient water quality criteria are "...based solely on data and scientific judgments on the relationship between pollutant concentrations and environmental and human health effects", and do not consider technological feasibility or economic and social costs or benefits (USEPA, 1980d).

The state of Louisiana establishes and revises ambient water quality criteria under the authority of Section 2.0743(1) of the Louisiana Water Control Law (R.S. 30: 2071-2078) and Section 303(c) of the Federal Clean Water Act (P.L. (92-500 as amended)).

In this section, the methods used by USEPA to develop the numerical ambient water quality criteria for potential carcinogens and non-carcinogens, and the methods used by the state of Louisiana to establish regulatory water quality criteria are described and critiqued.

2.1 USEPA Methods of Ambient Water Quality Criteria Development

This section contains a description of the methods assumptions and calculations for the determination of a criteria level for chemicals in water based on the USEPA Office of Water Regulation and Standards guidelines. The USEPA procedures used to develop ambient water quality criteria were first published in the Federal Register on November 28, 1980 (45 FR 79347). Further direction was provided in the "Guidance for Establishment of Ambient Criteria to Limit Human Exposure to Contaminants in Fish and Shellfish," prepared by the Office of Water Regulations and Standards (USEPA, 1988).

Human health criteria for the consumption of chemically contaminated water and fish are intended as guidance for rules. As 45 FR 79319 describes,

"Section 304(a)(1) criteria are not rules and they have no regulatory impact. Rather, these criteria present scientific data and guidance on the environmental effect of pollutants which can be useful to derive regulatory requirements based on considerations of water quality impacts."

Federal water quality standards, toxic pollutant effluent standards, and state limits may, however, use these criteria as a basis for acceptable concentrations of contaminants in water.

USEPA criteria for ambient waters are developed using epidemiological data of two basic types; accidental human and terrestrial life exposure studies and controlled studies using test organisms (e.g. rats and mice). These data define the concentration of a contaminant or the dose (in mg/kg/day) that is estimated to cause adverse effects in the subject organism.

The effects measured by these studies are of two basis types: (1) noncarcinogenic effects which are first seen at a specific level of "threshold" and (2) carcinogenic effects where no threshold of effect is postulated and the effect (cancer) is assumed to be a direct function of the amount of exposure. The maximum dose level to avoid effects for non-carcinogens is called the reference dose (RfD). The dose response relationship for the carcinogens is described by the cancer potency slope factor.

The bioconcentration factor (BCF) provides the relating contaminant concentrations in the animal tissues to the water in which the animal resides. BCFs are a relative measure of the ability of a contaminant to be stored in lipid (fat) tissues and may be used to estimate accumulation

through the food chain. The higher the BCF, the greater the tendency for the contaminant to concentrate in animal tissues. The BCF, RfD and the potency factor are the basic coefficients used by the USEPA to develop water quality criteria. The USEPA generally uses a standard set of exposure assumptions in calculating criteria. However the USEPA (1988) indicates that criteria assumptions can be modified to accurately define the exposure pathways of a particular waterbody. For example, some areas may have higher or lower fish consumption rates than those used to develop national criteria.

The criteria for noncarcinogens are based on specific RfDs. The criteria for potential carcinogenic chemicals are calculated at various incremental risk levels, (10^{-4} , 10^{-5} , and 10^{-6}) in order to illustrate the concentration, or "maximum permissible" level associated with each risk level.

The USEPA estimates the maximum permissible concentration of a chemical carcinogen in surface water using a back calculation procedure associated with a specified level of risk. Risk is defined as the cancer potency factor times the lifetime average daily dose (LADD). The USEPA method for calculating ambient water quality criteria for human exposure via ingestion of chemically contaminated water and fish incorporates conservative (health protective) exposure assumptions. Key exposure parameters include the consumption rates of water and fish and the

bioconcentration factor (chemical concentration in fish tissue/chemical concentration in water). The following exposure assumptions have been identified:

- All drinking water and fish in the diet are assumed to come from the contaminated sources.
- 100% of the contaminant chemical is assumed to be absorbed in the gastrointestinal tract following ingestion
- The concentration of the chemical in water and fish are assumed to be constant over a lifetime of exposure (70 years).

Specific information on consumption patterns in the exposed population are not generally considered (although site-specific data may be incorporated to refine criteria). This conservative approach generally overstates the risk even to the maximally exposed individual and tends to generate overly conservative numerical criteria.

2.1.1 Human Health Criteria for Carcinogens

It is the policy of the USEPA to assume that a carcinogenic agent should be treated as if it does not display a threshold concentration for harmful biological impacts. For regulatory purposes, zero risk exists only at zero dose for carcinogens, i.e., for all doses, some risk is assumed to be present (Munro and Krewski, 1981). Since direct estimates of risk at low levels of exposure would require the testing of prohibitively large numbers of animals, it is important to recognize that models must be used

to predict tumor response at low levels. The purpose of so-called cancer models is to estimate a risk specific dose (RsD) based on the extrapolation of experimental results well below the dose range used in animal tests; usually three to four orders of magnitude below the no observed adverse effect level (NOAEL). The RsD represents the lifetime average daily dose for a specified level of risk. Statistical approaches to estimate the low-dose response involve mathematical models relating the probability of a specific response at very low doses. The uncertainty associated with this approach is quantified by using statistical bounds. Because of the statistical and biological problems inherent in the identification of a true no-effect level, most mathematical models for describing dose-response relationships for carcinogens have abandoned the concept of threshold, or a dose where no response would be expected.

The commonly used linearized multi-stage model is based on the assumption that the induction of irreversible self-replicating toxic effects such as carcinogenesis is the result of a number of different random biological events. The rate of occurrence of each event (response) is assumed to be in a strict linear relation to the dose rate. This linear dose-response curve is assumed to pass through the origin or point of zero dose-zero response (Crump et al., 1976).

2.1.2 Cancer Potency Factor

The cancer potency factor is defined by the USEPA as the upper 95% confidence limit on the slope of the dose-response curve. Cancer potency factors are estimated by the USEPA Carcinogen Assessment Group (CAG) based on the linearized multistage model developed by Crump (1980). In general, the cancer potency factor is developed from a chronic laboratory animal study or epidemiological study that provides the highest estimate of the lifetime carcinogenic risk (USEPA, 1980d). It is beyond the scope of this report to evaluate the use of this model in the development of cancer potency factors or to evaluate the data used by CAG to calculate carcinogenic potency for the specific chemicals of interest. All water quality criteria calculations presented in this report are based on the current cancer potency factors developed by the USEPA (USEPA, 1989a) except for those criteria proposed by DEQ that were calculated using values that are currently out of date.

2.1.3 Selection of the Risk Level for Carcinogens

The primary risk management decision that needs to be made by the State to adopt and implement human health criteria is the selection of the risk level for carcinogens. There is no USEPA policy on risk levels such a decision is to be made by individual States. The USEPA Office of Drinking

Water generally uses risk levels of 10^{-4} to 10^{-6} for deriving maximum contaminant levels for carcinogens.

2.1.4 Theoretical Incremental Cancer Risk Equation

The estimates of incremental cancer risk that follow are based on a fairly simple calculation using the chemical specific carcinogenic potency factor (as derived by the USEPA) and the "Lifetime Average Daily Dose" (LADD). Mathematically, this equation is expressed as follows:

$$R = PF \times LADD$$

Where:

- R - Theoretical Incremental Cancer Risk
- PF - Carcinogenic Potency Factor based on a surface area extrapolation of dose (mg/kg/day)⁻¹
- LADD - Lifetime Average Daily Dose (mg/kg/day)

2.1.4.1 Equation for Public Water Supplies

$$LADD = \frac{[(CR \times BCF) + WR] \times Cw}{BW}$$

Where:

- CR - Fish Consumption Rate = 0.0065 (kg/day)
- BCF - Bioconcentration Factor
- WR - Water Consumption Rate = (2.0 L/day)
- Cw - Concentration of the contaminant in the water (mg/L)

BW = Body Weight = 70 (kg)

These equations are then rearranged to solve for:

$$C_w = \frac{R \times BW}{PF[WR + (CR \times BCF)]}$$

2.1.4.2 Equation for Non-Public Water Supplies (Recreational Waters)

$$LADD = \frac{[(CR \times BCF) + WR] \times Cr}{BW}$$

Where:

CR = Fish Consumption Rate = 0.0065 (kg/day)

BCF = Bioconcentration Factor

Cr = Concentration of the contaminant in the water (mg/L)

BW = Body Weight = 70 (kg)

These equations are then rearranged to solve for:

$$Cr = \frac{R \times BW}{PF(CR \times BCF)}$$

2.1.4.3 Equations for non-carcinogens in Public Water Supplies and
Recreational Waters

Public Water Supplies (water and fish consumption)

$$C_w = \frac{RfD \times BLD}{WR + (CR \times BCF)}$$

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Recreational waters (fish consumption only)

$$C_r = \frac{RfD \times BW}{CR \times BCF}$$

Where:

C_w = Concentration of contaminant in the water (mg/L)

C_r = Concentration of contaminant in the water (mg/L)

RfD = Reference Dose (mg/kg/day)

BW = Adult body

CR = Fish consumption rate (kg/day)

BCF = Bioconcentration Factor

2.1.5 Parameter Description

By selection of an appropriate de minimis level of cancer risk, the above equation can be solved to estimate a maximum permissible concentration or ambient water quality criteria chemical based on the lifetime consumption of contaminated water and fish. The source and rationale for the parameters in the criterion are described below.

2.1.5.1 Carcinogenic Potency Factor

The carcinogenic potency factor is chemical specific. It is based on the dose-response relationship found in carcinogenicity bioassays. The USEPA

lists the potency factors for potential carcinogens in the Integrated Risk Information System (IRIS) data base. Current data was taken from Health Effects Assessment Summary Tables and Uses Guide (USEPA, 1989b). The potency factors for the chemicals of interest are presented in Table 2-1.

2.1.5.2 Fish Consumption Rate

The USEPA in developing ambient water quality criteria assumes that the average individual consumes 0.0065 kg/day (6.5 g/day) of fish. This estimate is based on a 1980 USEPA document entitled "Seafood Consumption Study 1973-1974" (NMFS, 1976). The rate is a national per capita estimate of the daily consumption of fish (including crustaceans and shellfish).

2.1.5.3 Water Consumption Rate

The USEPA presently recommends the use of a 2.0 L/day water consumption rate for adults. This rate includes all tap water consumed in the form of juices and other beverages, and is based on a historical figure set by the U.S. Army as the amount of water needed for each person in the field. Several studies outlined in the USEPA Exposure Factors Handbook (USEPA, 1989a) suggest that this rate significantly overestimates average water consumption. Data presented by the National Cancer Institute suggest that the 2.0 L per day rate approximates a 90th percentile water consumption rate (Cantor et al., 1987).

TABLE 2-1

RECENTLY PUBLISHED EPA CANCER POTENCY FACTORS AND CANCER POTENCY FACTORS
USE BY LADEQ FOR CALCULATING AMBIENT WATER QUALITY CRITERIA

Chemical	USEPA ^a July, 1989 Potency Factor (PF) or RfD	LADEQ Potency Factor (PF) or RfD	BCF	EPA Evidence Category Cited by ADEQ	EPA Evidence Category July, 1989
Hexachlorobenzene	1.70 ✓	1.688	8690	B2	B2
Hexachlorobutadiene	0.078	0.078	392	C	C
1,2-Dichloroethane	0.091	0.091	1.2	B2	B2
1,1,2,2-Tetrachloroethane	0.2	0.2	5	C	C
<u>Chlorinated Ethanes</u>					
1,1,1-Trichloroethane	0.9 ^b	0.09 ^b	3.75	B2	B2
1,1,2-trichloroethane	0.057	0.057	4.5	C	C
<u>Chlorinated Ethylenes</u>					
1,1-Dichloroethylene	0.6	0.6	5.6	C	B2
Tetrachloroethylene	0.051 ✓	0.039776	30.6	B2	B2
Trichloroethylene	0.11	0.011	10.6	B2	B2
<u>Trihalomethanes</u>					
Bromoform	0.20 ^b ✓	0.0061	8.3	B2	B2
Bromodichloromethane	0.13 ✓	0.0061	3.75	B2	B2
Chloroform	0.0061	0.0061	3.75	B2	B2
Dibromochloromethane	8.4 x 10 ⁻²	0.0061	3.75	B2	ND
<u>Other Chlorinated Compounds</u>					
Carbon Tetrachloride	0.13	0.13	18.75	B2	B2
Methyl Chloride	0.013	0.0061	3.75	B2	C
Methylene Chloride	0.0075	0.0075	0.91	B2	B2
Vinyl Chloride	2.3 ✓	0.0174	1.17	A	A

a) Health Effects Assessments Summary Tables and User's Guide (USEPA, 1989b).

b) Reference Dose (mg/kg/day)

ND = Not Determined

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2.1.5.4 Gastrointestinal Absorption

Once contaminated water or fish is ingested, only a fraction of the total concentration of the chemical contaminant is available for absorption from the gastrointestinal tract. The absorbed dose is determined by the bioavailability of the chemical in the gastrointestinal tract. The chemical may be adsorbed to undigestible particles or may be in gastric juices at concentrations in excess of what will penetrate the membrane of the gastrointestinal tract. Although few contaminants in water or food are completely assimilated, the USEPA assumes 100% absorption of chemicals in the gastrointestinal tract for the purposes of calculating ambient water quality criteria. This assumption is conservative as many organic chemicals, including hexachlorobenzene, are only fractionally absorbed from the gastrointestinal tract (Koss and Koransky, 1975; Zabik and Schemmel, 1980).

2.1.5.5 Fraction From the Contaminated Source

In developing ambient water quality criteria, the USEPA assumes that 100% of the water and fish consumed are from the contaminated source. This assumption is extremely conservative. Most consumers drink water from a number of relatively clean sources and a large portion of the total amount of fish consumed is purchased from uncontaminated commercial sources.

2.2 DEQ Methods for Ambient Water Quality Criteria Development

The USEPA ambient water quality criteria provide scientific data and guidance to the state agencies responsible for the development of regulatory criteria applicable to designated water uses. The USEPA states under Section 303 of the Clean Water Act,

"States may appropriately modify these values to reflect local conditions. In certain circumstances the criteria may not accurately reflect the toxicity of a pollutant because of the effect of local water quality characteristics or varying sensitivities of local populations." (USEPA, 1980d).

In general, state water quality standards consist of two components, numerical criteria and designated water uses. Numerical criteria are specific to the identified water use. Designated water uses include recreation; protection and propagation of fish and other aquatic life; agricultural and industrial uses; and public water supplies (USEPA, 1980d). When multiple designated uses are attributed to a single water resource, the most stringent criteria should be applied.

The state of Louisiana has developed water quality criteria for the protection of human health for both public water supplies and non-public water supplies (surface water uses other than the supply of drinking water). Criteria proposed for public water supplies are designed to be

protective of human health for the consumption of water from surface waterbodies designated as a public water supply and also for primary and secondary contact recreation and fish consumption. Criteria established for non-public water supplies are not intended to protect human health for water consumption, but do protect for primary and secondary recreational contact and fish consumption (LAC,1989), and will be referred to as recreational waters in this report.

The methods employed by the state of Louisiana's Department of Environmental Quality to develop ambient water quality criteria for potentially carcinogenic and non-carcinogenic chemicals are described in the following sections. The equations and parameters described below are presented in a report prepared by the Louisiana Office of Water Resources, Water Pollution Control Division entitled "Documentation of Numerical Criteria for Human Health Protection in the 1989 Water Quality Standards Revision" (June 1989) which is presented in Appendix A.

2.2.1 Equation for Public Water Supply Criteria

The equation used to develop criteria for carcinogens in a public water supply is a modified version of the criteria equation recommended by the USEPA described in Section 2.1.3.1, and is as follows:

$$C_w = \frac{(R \times BW)}{PF [IR + WR + (BCF \times CR)]}$$

Where:

- Cw = Criteria Concentration for public water supplies (mg/L)
- R = Theoretical Risk Level (10^{-6})
- BW = Adult Body Weight (70 kg)
- PF = Cancer Potency Slope (mg/kg/day)⁻¹
- IR = Incidental Water Consumption Rate (0.089 L/day)
- WR = Water Consumption Rate (2.0 L/day)
- BCF = Bioconcentration Factor (L/kg)
- CR = Fish Consumption Rate (0.02 kg/day)

2.2.2 Equation for Non-public Water Supply (Recreational Waters) Criteria

The equation used in the development of criteria for carcinogens in recreational waters is as follows:

$$Cr = \frac{(R \times BW)}{PF [IR + (BCF \times CR)]}$$

Where:

- Cr = Criteria concentration for recreational water (mg/L)
- R = Theoretical Risk Level (10^{-6})
- BW = Adult Body Weight (70 kg)
- PF = Cancer Potency Slope
- IR = Incidental Water Consumption Rate (0.089 L/day)
- BCF = Bioconcentration Factor (L/kg)

CR = Fish Consumption Rate (0.02 kg/day)

2.2.3 Equation for Non-Carcinogen Water Quality Criteria

$$C_w = \frac{RfD \times BW}{IR + WR + (BCF \times CR)}$$

$$C_r = \frac{RfD \times BW}{IR + (BCF \times CR)}$$

All parameters for the above equation are defined in Section 2.2.1 and 2.2.2 except for:

RfD = Reference Dose (mg/kg/day); chemical specific

2.2.4 Parameter Description

The source and rationale for each parameter presented by the state of Louisiana calculation methods are described below.

2.2.4.1 Acceptable Levels of Risk(R)

Theoretical incremental cancer risk values of 10^{-4} to 10^{-6} are often used to define risks that are negligible and below the level of regulatory concern. The selection of an appropriate risk value depends on several factors. In applying the de minimis concept and in setting other safety

standards, USEPA is often guided by the figure of 10^{-6} , or "one in a million" risk. The higher risk values (10^{-4} or 10^{-5}) are often considered appropriate by regulatory agencies for the protection of smaller populations. In the establishment of the proposed water quality criteria the state of Louisiana selected an incremental cancer risk level of 10^{-6} (DEQ 1989).

2.2.4.2 Cancer Potency Factor (PF) and Weight of Evidence

The cancer potency factor is chemical specific and is based on the dose-response relationship found in carcinogenicity bioassays. Cancer potency factors recommended by the USEPA for use in water quality criteria calculations and employed by the state of Louisiana for the chemicals addressed in this report are listed in Table 2-1. ~~Most of the cancer~~ potency factors used by the state of Louisiana were ~~estimated from the~~ USEPA's Integrated Risk Information System (IRIS) database as of Dec. 1, 1988, except for tetrachloroethylene, vinyl chloride, bromoform, bromodichloromethane, and hexchlorobenzene. DEQ indicated that the cancer potency for these chemicals were taken from the appropriate ambient water quality criteria documents. However, these documents were not specifically identified.

Based on the current reported USEPA potency factors and weight of evidence (Table 2-1), DEQ has incorrectly identified the weight of evidence for

1,1-dichloroethylene, bromoform and methyl chloride. This inconsistency is most evident in the cases of bromoform where the DEQ is regulating this chemical as a carcinogen and USEPA data indicates that the evidence of carcinogenesis is insufficient to support such a policy.

DEQ potency factors for tetrachloroethylene, bromodichloromethane, methyl chloride and vinyl chloride are also inconsistent with current USEPA values (Table 2-1).

2.2.4.3 Incidental Water Ingestion Rate (IR)

The incidental water ingestion rate is applied to those waters designated as swimmable, and is set at 0.089 L/day. The rate is based on the assumption that a person while swimming may ingest 250 ml of water per hour. The incidental ingestion rate was calculated as follows:

$$\text{IR (L/day)} = \frac{250 \text{ ml}}{\text{hr}} (\text{possible ingestion}) \times \frac{5 \text{ hrs}}{\text{wk}} (\text{swimming duration}) \times \frac{6 \text{ mos}}{12 \text{ mos}} (\text{swimming season}) \times \frac{1 \text{ wk}}{7 \text{ days}} = 89 \text{ ml/day} = 0.089 \text{ L/day}$$

The DEQ selected the DHH value of 250 ml/hr for possible ingestion of water while swimming from an unpublished in-house Louisiana Department of Health and Hospitals (DHH) report entitled, Health Hazards of Primary Contact Recreation at Bayou D'Inde. However, no scientific justification

was given for this value (Mr. Dugan Sabins, Louisiana DEQ, Water Pollution Control Division, pers. comm.).

The swimming exposure duration of 5 hours per week appears to have no scientific basis. However, it is the policy of the DEQ, Water Pollution Control Division to protect water quality to a level that is assumed to protect the right of the people of Louisiana to swim 5 hrs per week during the summer season at no significant risk, should they choose to do so (Mr. Dugan Sabins, Louisiana DEQ, Water Pollution Control Division, pers. comm.).

The incidental water ingestion rate 0.089 liters per day appears to significantly overestimate the volume of water ingested during swimming activities. The USEPA Superfund Exposure Assessment Manual (USEPA, 1988c) provides recommended parameters for the determination of incidental water ingestion during swimming, which results in an ingestion rate of 0.0025 liters per day. This parameter is discussed further in Section 2.3.2.9.

2.2.4.4 Bioconcentration Factor (BCF)

Bioconcentration factors used by DEQ to calculate water quality criteria were derived from the USEPA IRIS database, except for tetrachloroethylene, vinyl chloride, bromoform, bromodichloromethane and hexachlorobenzene. BCF's for these chemicals were taken from the appropriate ambient water

quality criteria documents and are listed in Table 2-1 (DEQ, 1989). The BCF for HCBd was provided by the USEPA ORD Laboratory in Duluth, Minnesota (USEPA, 1989d).

2.2.4.5 Fish Consumption Rate (CR)

The DEQ assumed a fish consumption rate of 0.02 kg/day (20 g/day) for fish and shellfish from marine, estuarine and fresh waters in Louisiana (DEQ, 1989). This consumption rate may represent an average for the U.S. population according to the U.S. Department of Agriculture's 1984 National Consumption Statistics.

The USEPA recommends that national average consumption rates should not be extrapolated to local consumption patterns unless local information is not available (USEPA, 1989c). However, fish consumption rates for potentially exposed populations in Louisiana are not adequately characterized for such an exposure assessment. Indications are that the Louisiana population has a high protein diet and may consume more fish and shellfish than the national population average. Reported average values for fish and shellfish consumption for the U.S. population generally range from 6.5 to 20 g/day (NMFS, 1976, 1984; SRI, 1980; USDA, 1984). A Michigan Department of Public Health study reported that fish eating populations near the Great Lakes consume 30 g/day (Humphrey et al., 1976). Only 0.1% of the U.S. population consumes 165 g/day of fish and shellfish

(Finch, 1973).

2.2.4.6 Water Consumption Rate

DEQ assumed a 2.0 L per day water consumption rate for adults. The selection of this water consumption rate by the DEQ is based on a policy that the people of Louisiana have the right to consume 2.0 liters of water per day at no significant risk should they choose to do so (Mr. Dugan Sabins, Louisiana, DEQ Water Pollution Control Division, pers. comm.).

The USEPA presently uses the quantity of 2.0 L/day for adults as the average amount consumed per person (USEPA, 1980d). The volume of 2.0 L/day is a historical figure set by the U.S. Army and is an overestimate for most people. The USEPA's Exposure Factors Handbook indicates that the average adult drinking water rate is 1.4 L/day (USEPA, 1989a).

2.2.4.7 Reference Dose

The RfD (mg/kg/day) employed in the above equations (Section 2.2.3) for 1,1,1-trichloroethane is reported by DEQ to be from the USEPA's Integrated Risk Information System (IRIS). However, the RfD for 1,1,1-

trichloroethane reported by the USEPA as July 1989 was a factor of 10 greater was presented in Table 2-1 (USEPA 1989a). 1,1,1-trichloroethane is the only compound of interest to which DEQ applied the noncarcinogen equations.

2.2.5 DEQ Assumptions

2.2.5.1 Gastrointestinal Absorption

Although few contaminants in water or food are completely assimilated, the DEQ consistent with the USEPA in developing ambient water quality criteria, assumes that 100% of the contaminant is absorbed in the gastrointestinal tract.

2.2.5.2 Fraction From the Contaminated Source

The fraction of the average daily weight of fish consumed that is attributable to the recreational catch from Louisiana waters is referred to as the "Fraction of the Recreational Catch" (FRC). DEQ assumes that 100% of the water and fish consumed is from the contaminated sources, even though most consumers eat a large portion of fish purchased from uncontaminated commercial sources and drink water from relatively clean

supplies. For the refined equations presented in Section 2.3, the FRC is assumed to be 10%. Justification of this value is presented in Section 2.3.2.7.

2.2.6 Summary of the DEQ Methods

The following is a summary of the assumptions and methods employed by the Louisiana DEQ in the development of ambient water quality criteria that are designed to be protective of human health for public water supplies and recreational waters.

- The cancer potency factor (cancer slope factor), reference dose (RfD) and BCF values were derived from USEPA documents or databases (DEQ 1989). However, the most current (July 1989) USEPA cancer potency factors are presented in Table 2-1.
- The determination of carcinogenic and non-carcinogenic chemicals was based on the USEPA weight of evidence designations. All of those chemicals placed in categories A, B, or C were designated as carcinogens and those in category D were designated non-carcinogens, which is consistent with USEPA recommendations. Three of the chemicals in Table 2-1 are not appropriately categorized by DEQ according to the current USEPA classification.
- The equations used to develop ambient water quality criteria for the protection of human health are consistent with those recommended by the USEPA except for the use of an incidental water ingestion rate of 0.089 L/day for swimming. Support for this value is based on a public right to swim rather than specific scientific data. Use of this incidental consumption rate appears to significantly overestimate water ingestion exposures.
- A fish consumption rate of 20 L/day was used in the calculation of water quality criteria which appears to be a reasonable figure based upon scientific literature and the extent of the recreational fishery in Louisiana.

- The DEQ employed a drinking water consumption rate consistent with USEPA methods, however, the average water consumption rate in the U.S. is significantly lower. The selection of the higher water consumption rate which approximates a 90th percentile rate is based on a personal rights rather than scientific basis.
- The DEQ calculations assume a 100% gastrointestinal absorption efficiency. The rated absorption should be determined on a chemical specific basis.
- The DEQ calculations assume that all fish and water consumed are derived from contaminated water sources, and that all swimming and recreational activities take place in contaminated water sources.
- The proposed water quality criteria calculated by the DEQ may have an associated risk level significantly less than the stated 10^{-6} level due to the repeated use of conservative assumptions.

2.3 Alternative Methods of Ambient Water Quality Criteria Development

The parameters and assumptions described in the previous sections indicate that the criteria set for the protection of human health for carcinogens and non-carcinogens have an associated risk level significantly lower than the established risk of 10^{-6} due to the repeated use of conservative assumptions. Ambient water quality criteria calculation methods employing USEPA values representing the 50th percentile for water consumption, and USEPA recommended parameters for incidental water ingestion during swimming activities; refinements to account for gastrointestinal absorption efficiency; and the portion of the fish diet attributable to the recreational catch are presented in the following sections.

2.3.1 Theoretical Cancer Risk Equation

The following theoretical cancer risk equation is modified to include factors that account for the gastrointestinal absorption efficiency and the fraction of the catch from contaminated sources.

2.3.1.1 Equation for Carcinogens in Public Water Supplies

$$R = PF \times LADD$$

LADD = Lifetime Acceptable Daily Dose

$$LADD = \frac{[(BCF \times CR \times FRC) + WR + IR] \times Cw \times BF}{BW}$$

Where:

R = Theoretical Risk Level (10^{-5})

PF = Cancer Potency Factor (mg/kg/day)⁻¹

BCF = Bioconcentration Factor

Cw = Water Quality Criteria (mg/L)

CR = Fish consumption rate (kg/day)

FRC = Fraction of Recreational Catch (0.1)

WR = Water consumption rate for the 50th percentile water consumer (1.4 L/day)

IR = Incidental water ingestion rate (0.0025 L/day)

BF = Gastrointestinal bioavailability factor

BW = Adult human body weight (70 kg)

These equations are arranged to solve for Cw as follows:

$$C_w = \frac{R \times BW}{PF [(BCF \times CR \times FRC) + WR + IR] \times BF}$$

2.3.1.2 Equation for Carcinogens in Recreational Waters

$$R = PF \times LADD$$

LADD = Lifetime Acceptable Daily Dose

$$LADD = \frac{[(BCF \times C \times FRC) + IR] \times Cr \times BF}{BW}$$

Where:

R = Theoretical Risk Level (10^{-5})

PF = Cancer Potency Factor (mg/kg/day)⁻¹

BCF = Bioconcentration Factor

Cr = Water Quality Criteria (mg/L)

FRC = Fraction of Recreational Catch (0.1)

CR = Fish consumption rate (0.02 kg/day)

IR = Incidental water ingestion rate (0.0025 L/day)

BF = Gastrointestinal bioavailability factor

BW = Adult human body weight (70 kg)

These equations are arranged to solve for Cr as follows:

$$Cr = \frac{R \times BW}{PF [(BCF \times CR \times FRC) + IR] \times BF}$$

2.3.1.3 Equations for the Water Quality Criteria for Non-carcinogens in
Public and Recreational Water Supplies.

The only difference in the equations for criteria for carcinogenic and non-carcinogenic compounds is the use of a "reference dose" (RfD) in place of the cancer potency factor (PF). The RfD in (mg/kg/day)⁻¹, is chemical specific and is an estimate of the maximum daily dose level which does not exceed the threshold at which toxic effects may occur in humans. The resulting equations for criteria for non-carcinogens in public water supplies and recreation waters are as follows:

Public Water Supplies:

$$C_w = \frac{RfD \times BW}{[(BCF \times CR \times FRC) + WR + IR] \times BF}$$

Recreational Waters:

$$C_r = \frac{RfD \times BW}{[(BCF \times CR \times FRC) + IR] \times BF}$$

All parameters for the above equations are defined above in Section 2.3.1.2 except for: RfD = Reference dose (mg/kg/day)

2.3.2 Parameter Description

The parameters used by the USEPA, DEQ and ChemRisk to calculate ambient water quality criteria are presented in Table 2-2. These parameters are discussed below.

2.3.2.1 Acceptable Levels of Risk

Theoretical incremental cancer risk values of 10^{-4} to 10^{-6} are often used to define risks that are negligible and below the level of regulatory concern.

Beginning in the late 1970's and early 1980's, regulatory agencies in the United States and abroad frequently adopted a cancer risk criteria of one in a million as a negligible (i.e., of no concern) risk when fairly large populations might be exposed to a suspect carcinogen. Unfortunately, theoretical increased cancer risks of one in a million are often incorrectly portrayed as a serious and genuine public health hazard. As recently discussed by Dr. Frank Young, (Young, 1987) the current commissioner of the FDA, this was not the intent of such estimates:

In applying the de minimis concept and in setting other safety standards, FDA has been guided by the figure of "one in a million." Other Federal agencies have also used a one in a million level, such as the Occupational Safety and Health Administration and the Environmental Protection Agency. Both agencies rely on the one in one million increased risk over a

TABLE 2-2

COMPARISON OF PARAMETERS FOR AMBIENT WATER QUALITY CRITERIA CALCULATION

Parameter	EPA Ambient Water Quality Criteria	LA DEQ	Refined Method
Risk level	10^{-6}	10^{-6}	10^{-5}
Incidental Ingestion Rate (Swimming)	NC	0.089 L/day	0.0025 L/Day
<u>Absorption Efficiency^a</u>			
HCB	100%	100%	10%
HCBD	100%	100%	10%
Water Consumption Rate (L/day)	2	2	1.4
Fish Consumption Rate (kg/day)	0.0065	0.02	0.02
Fraction of Fish from Contaminated Source	100%	100%	10%

a) All other chemical criteria were calculated at 100% absorption efficiency.

NC = Not Considered

lifetime as a reasonable criterion for separating high-risk problems warranting agency attention from negligible risk problems that do not.

The risk level of one in one million is often misunderstood by the public and the media. It is not an actual risk - i.e., we do not expect one out of every million people to get cancer if they drink decaffeinated coffee. Rather, it is a mathematical risk based on scientific assumptions used in risk assessment. FDA uses a conservative estimate to ensure that the risk is not understated. We interpret animal test results conservatively and we are extremely careful when we extrapolate risks to humans. When FDA uses the risk level of one in one million, it is confident that the risk to humans is virtually nonexistent.

In short, a "one in a million" cancer risk estimate, which is often tacitly assumed by some policy-makers to represent a trigger level for regulatory action, actually represents a level of risk that is so small as to be of negligible concern.

Another misperception within the risk assessment arena is that all occupational and environmental regulations have as their goal a theoretical maximum cancer risk of 1 in 1,000,000. Travis et al. (1987) recently conducted a retrospective examination of the level of risk which triggered regulatory action in 132 decisions. Three variables were considered: 1) individual risk (an upper-limit estimate of the probability that the most highly exposed individual in a population will develop cancer as a result of a lifetime exposure), 2) population risk (an upper-limit estimate of the number of additional incidences of cancer in the exposed population), and 3) population size. The findings of Travis et al. (1987) can be summarized as follows:

1. Every chemical with an individual lifetime risk above 4×10^{-3} received regulation. Those with values below 1×10^{-6} remained unregulated.
2. For small populations, regulatory action never resulted for individual risks below 1×10^{-4} .
3. For effects resulting from exposures to the entire U.S. population, a risk level below 1×10^{-6} never triggered action; above 3×10^{-4} it always triggered action.

In short, regulatory agencies have found risks far in excess of 1 in 1,000,000 acceptable if experienced by small populations, not only have regulatory agencies taken exception to the unilateral application of 1 in 1,000,000 risk, but many common human activities entail risks greatly in excess of 1 in 1,000,000.

The USEPA ambient water quality criteria for potential carcinogenic are presented for a 10^{-4} to 10^{-6} range of incremental risk. California (Title 22 California Code of Regulations Section 1271 and other states are regulating carcinogens at a no significant risk level of 10^{-5} . This level of risk is appropriate for criteria calculations.

2.3.2.2 Cancer Potency Factor

The cancer potency factors employed in the equations described above are those developed by the USEPA and reported in the USEPA Health Effects

Assessments Summary Tables and User's Guide (USEPA, 1986b). The USEPA calculated potency factors represent an upperbound value, and are therefore, very conservative (health protective) estimates.

2.3.2.3 Reference Dose

The RfD's (mg/kg/day) employed in the equations described above are those developed by the USEPA and reported in the USEPA Health Effects Assessments Summary Tables and User's Guide (USEPA, 1986b).

2.3.2.4 Bioconcentration Factor

The BCF's employed in the equations described above are those recommended by the USEPA and employed by the DEQ in the development of the proposed Louisiana ambient water quality criteria for both carcinogens and non-carcinogens.

2.3.2.5 Water Quality Criteria (mg/L)

The water quality criteria developed using the above equations are chemical specific and represent the chemical concentration in public water supplies (Cw) and recreational waters (Cr) associated with a specific level of risk for carcinogens and the expected threshold level for non-carcinogens.

2.3.2.6 Fish Consumption Rate

Average values for fish and shellfish consumption for the U.S. population generally range from 6.5 to 20 g/day (NMFS, 1976, 1984; SRI, 1980; USDA, 1984). A Michigan Department of Public Health study reported that fish eating populations near the Great Lakes consume 30 g/day (Humphrey et al., 1976). Only 0.1% of the U.S. population consumes 165 g/day of fish and shellfish (Finch, 1973). There are inadequate data on consumption rates specific to populations in Louisiana. However, based on the above data, it may be assumed that an average individual in the state of Louisiana may consume 20 g fish/day. This assumption is consistent with that used by the DEQ.

2.3.2.7 Fraction of the Recreational Catch

It is assumed that the consumption of contaminated fish is primarily associated with recreational fishing. The chemical content of commercially caught fish which are sold interstate are regulated by the U.S. Food and Drug Administration (FDA). The commercial catch is largely from marine resources and is assumed to be uncontaminated.

National survey data indicate that freshwater finfish constitute only 10% of the total fish consumption by the average U.S. Consumer (USDA, 1985).

It is only freshwater aquatic species that are likely to inhabit surface waters near sources of contaminant release. The small fraction of freshwater fish which may come from contaminated sources are likely largely a recreational catch. This would suggest that the recreational fisherman may be at some risk of exposure, but that the average individual is likely at negligible risk.

The assumption of a 10% fraction from contaminated sources is likely very conservative as that would represent the entire fraction of freshwater fish in the average diet.

2.3.2.8 Water Consumption Rate

The USEPA currently uses a water consumption rate of 2.0 L/day. However, noted in Section 2.1.4.3 the USEPA has stated that the actual average water consumption rate for the U.S. population is 1.4 L/day and that a 2.0 L/day value approximates the 90th percentile water consumption rate (USEPA, 1989a). The 1.4 L/day water consumption rate is used in the calculation of water quality criteria for public water supplies.

2.3.2.9 Incidental Water Ingestion Rate

The incidental water ingestion rate for swimming activities is estimated to be 0.0025 L/day. This rate is based on USEPA recommended parameters

for incidental water ingestion presented in the Superfund Exposure Assessment Manual (USEPA, 1989d), and was calculated in the following manner:

$$IR = WI \times SD \times EF \times 1 \text{ yr}/365 \text{ days} \times 1 \text{ L}/1000 \text{ ml}$$

Where:

IR = Incidental water ingestion rate for swimming (L/day).

WI = Water ingestion rate for swimming (50 ml/hr). (Versar est. 1987)

SD = Swimming duration (2.6 hr). (USDOI 1973)

EF = Exposure frequency (USDOI 1986)

$$[25 \text{ days/yr.} \times \frac{20(\text{years swimming})}{70(\text{yrs. in lifetime})} = 7 \text{ days/yr.}]$$

Conversion factors = 1 yr/365 days; 1 L/1000ml.

$$IR = (50 \text{ ml/hr}) \times (2.6 \text{ hr}) \times (7 \text{ day/yr}) \times (1/365) \times 1 \text{ L}/1000\text{ml}$$

$$= 0.0025 \text{ L/day}$$

2.3.2.10 Gastrointestinal Bioavailability Factor

In order to quantitatively and accurately describe the dose of the chemical contaminant that is absorbed from food and water, the absorption efficiency must be defined based on the best available data from laboratory and chemical studies. Assuming 100% absorption where sufficient contrary evidence is available adds unnecessary conservatism to regulatory criteria.

Data discussed in Section 3.4.1.1 indicate that less than 100% of ingested HCB is absorbed. HCB absorption is dependent on the solvent used during administration. Thus, when HCB was administered in olive oil, approximately 80% of the dose was absorbed, but when HCB was administered in an aqueous solution, only 6% of the dose was absorbed (Koss and Koransky, 1975). A higher percentage of HCB was absorbed in rats given a high-fat diet than in rats given a high-carbohydrate diet (Zabik and Schemmel, 1980). These data suggest that a representative gastrointestinal bioavailability factor associated with human consumption of HCB contaminated fish is 10% (BF = 0.1). Little is known about the gastrointestinal absorption efficiencies of many chemicals and where no substantial information is available the absorption efficiency was assumed to be 100%.

2.3.2.11 Other Considerations

Contaminated fish can potentially pose a problem to consumers. However, there is some evidence that food preparation reduces the available levels of chemicals in fish. The loss of lipids from fish during cooking has been correlated with a reduction in residue concentrations. Once fish is cooked, the heat "melts" the lipid portion and associated chemicals, leaving only a small portion for consumption. The PCB concentrations in cooked fish were reported by Cordle et al. (1982) to be 2 to 10 times less

than concentrations in raw tissues. The lipid portion of fish is located in between the layers of tissues (3%) and also in and around the internal organs (97%). Since consumption of raw tissue and/or consumption of the internal organs of fish is not a standard practice, the risk associated with eating fish from a contaminated source is significantly reduced. Thus, the risk of ingesting slightly contaminated fish is likely to be small considering that the fish consumed are cooked and are free of internal organs. Contaminant loss due to cleaning (gutting) and cooking of fish is not taken into consideration in the above calculations, thus adding additional conservatism to the calculations.

3.0 HEXACHLOROBENZENE

3.1 Environmental Fate of HCB

The environmental fate of HCB is determined by the cumulative interaction of transport and transformation processes. In turn, the environmental fate strongly influences the availability of HCB to fish and ultimately to humans.

Once released into an aquatic system, this compound may partition within various environmental compartments, including water, biota, sediment, and/or ambient air. The transport processes which define the movement of HCB between these compartments are highly dependent on the physio-chemical properties of both the chemical and the media. Transformation processes change the chemical structure of the compound and usually result in the degradation of the "parent" compound. Transformation processes include photolysis, hydrolysis, oxidation, reduction and microbial degradation. In the following sections, the physical and chemical properties of HCB and the environmental fate and persistence of this compound are discussed.

3.1.1 Physical and Chemical Properties of HCB

An understanding of physical and chemical properties of HCB helps determine how this chemical will be partitioned in aquatic systems and aids in predicting its overall behavior in the environment. Several of the important physical and chemical properties which govern this process are presented in Table 3-1.

The water solubility of HCB (0.006 mg/L) indicates that it is relatively insoluble in water. HCB is less soluble than most chlorinated pesticides, with the possible exception of DDT (0.006 mg/L). The hydrophobic character of HCB is also reflected in its relatively high soil-organic carbon partition coefficient (Koc). This parameter is indicative of a compound's affinity for the organic matter in soil and sediment. Chemicals with Koc's greater than 10,000 are generally tightly bound to sediment and not readily mobilized. HCB's limited water solubility and high soil-organic carbon partition coefficient suggest that most HCB found in an aquatic system will be strongly sorbed to sediment.

The vapor pressure and Henry's Law Constant values listed in Table 3-1 indicate that HCB in water is moderately volatile. Callahan et al. (1979) calculated an evaporative half-life from a one meter column of water to be approximately 8 hours. However, under equilibrium conditions little

TABLE 3-1

PHYSICAL & CHEMICAL PROPERTIES
OF CHEMICALS OF INTEREST

Chemical	Molecular Weight (g/mole)	Water Solubility at 20°C (mg/L)	Soil Sorption Constant (K _{oc})	Vapor Pressure (mmHg at 20°C)	Henry's Law Constant at 20°C (atm·m ³ /mole)	Octanol-Water Partition Coefficient (K _{ow})	Bioconcentration Factor (BCF)
Hexachlorobenzene	285 ⁱ	0.0062 ^{a,e}	3,900 ^j 3,914 ^f 28,000 ^{b,j} 10,000 ^g 100,000 ^e	1.0 x 10 ^{-5j} 1.1 x 10 ^{-5j}	6.81 x 10 ^{-4aj}	169,924 ^j 204,174 ^e	8,690 ^j 8,600 ^f 18,500 ^k 44,437 ^g
Hexachlorobutadiene	261 ⁱ	2.0 ⁱ 2.55 ^e	5,181 ^{b,e}	1.5 x 10 ^{-1j}	4.57 ^{a,j}	60,256 ^j 63,000 ^b 79,433 ^e	2.8 ^j 2,300 ^g 5,800 ^h 17,000 ^e
1,2-Dichloroethane	98.96 ^l	8,690 ^l	14 - 19 ^l	61 ^l	4.5 x 10 ^{-2l}	28 - 30 ^l	1.2 ^m
1,1,2,2-Tetrachloroethane	167.85 ^l	2,870 ^l	46 ^l	5.95 ^{a,l}	3.8 x 10 ^{-4l}	245 ^l	
Chloroform	119.38 ^r	8,200 ^s	---	159.6 ^r	---	93 ^r	5 ^m
Carbon Tetrachloride	153.8 ^l	785 ^l	110 ^l	91.3 ^l	2.41 x 10 ^{-2l}	437 ^l	3.75 ^m
1,1,1-Trichloroethane	133.41 ^s	480-4,400 ^s	---	96 ^s	---	148 ^s	18.75 ^m
1,1,2-Trichloroethane	133.41 ^l	4,400 ^l	11-309 ^{l,n}	22.49 ^{a,l}	9.1 x 10 ^{-4l}	263 ^l	5.6 ^m
1,1-Dichloroethylene	96.95 ^l	0.25 ^{l,o}	65 ^l	500 ^l	0.19 ^l	135 ^l	4.5 ^m
Trichloroethylene	131.40 ^l	1,366 ^{a,l}	---	59 ^l	0.02 ^l	263 ^l	5.6 ^m
Tetrachloroethylene	165.83 ^d	150 ^{a,b}	---	14 ^d	0.82	759 ^d	10.6 ^m
Vinyl Chloride	62.5 ^l	1,100-2,763 ^{a,l}	---	2660 ^l	1.2 ^l	23 ^l	30.6 ^m
Bromoform	252.77 ^l	3,190 ^{p,q}	---	5.6 ^{a,p}	---	200 ^s	1.17 ^m
Bromodichloromethane	163.83 ^l	4,500 ^l	63 ^l	50 ^l	2.41 x 10 ^{-3l}	126 ^l	8.3 ^m
Methylene Chloride	84.93 ^d	20,000 ^d	8.8 ^d	349 ^{a,d}	2.03 x 10 ^{3d}	20 ^d	3.75 ^m
Methyl Chloride	50.59 ^s	6,450-7,250 ^s	---	3,765 ^s	---	8 ^s	0.91 ^m
Dibromochloromethane	208.3 ^p	---	---	3,800 ^p	---	123 ^s	3.75 ^m

a) At 20°C

b) Calculated Value

d) ATSDR, 1987

e) Howard, 1989

f) Kenaga, 1980

g) Laseter *et al.*,
1976a,b

i) USEPA, 1985a

j) USEPA, 1986

k) Veith, 1979

l) ATSDR, 1988

m) USEPA IRIS, 1988

n) estimated

o) % by weight

p) Verschueren, 1983

q) At 30°C

r) USEPA, 1985

s) USEPA, 1979

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HCB is in solution to evaporate. HCB is a lipophilic compound with a relatively high octanol-water partitioning coefficient (Kow). Consequently, it is expected to be accumulated by aquatic organisms.

3.1.2 Fate of HCB in Aquatic Systems

In this section, the transport and transformation of HCB in water and sediment are described. Aquatic transport studies of chlorinated benzenes have shown that the more chlorinated benzenes (such as HCB) are primarily sequestered in sediment. Using models, Falco et al. (1982), predicted that 62% to 95% of pentachlorinated benzene (which is similar in structure to HCB) would be adsorbed to sediment. It may be assumed from this study that HCB will also sorb strongly to sediment. Laseter et al. (1976a) performed a water to soil partitioning study. After 24 hours, measured concentrations of HCB were 40 times higher in the soil than in the water. No movement of HCB in soil was observed in laboratory studies employing thin layer chromatography (Griffin and Chou, 1981). Ausmus et al. (1979) reported that approximately 0.01% of HCB in soil was released as a result of leaching studies. The rate of desorption of HCB from sediment is therefore likely to be negligible.

HCB is an environmentally stable chemical (USEPA, 1985), and although limited data are available, HCB does not appear to be significantly transformed by hydrolysis, photolysis or oxidation reactions (Callahan et

al., 1979). Laboratory studies indicate that microbial degradation of HCB does not occur to a significant extent (Tabak et al., 1981; Korte and Klein, 1982). McConnell et al. (1975) concluded that microbial degradation does not contribute substantially to the mineralization of HCB.

3.1.3 Fate Summary

The following summarizes the environmental fate of HCB:

- HCB is highly insoluble in water.
- HCB is highly absorbed to sediment and the rate of desorption is likely negligible.
- HCB adsorbed to sediment is expected to persist based on its resistance to chemical and biological degradation.
- HCB is lipophilic and readily bioaccumulated.

3.2 Bioaccumulation

HCB released to aquatic systems may be accumulated by aquatic organisms. If this chemical is accumulated to levels exceeding toxicity thresholds, aquatic organisms may be adversely affected. Furthermore, the consumption of HCB contaminated fish and shellfish species provides a potential exposure route to humans. In this section, the bioaccumulation of HCB is described.

3.2.1 Bioaccumulation

Bioaccumulation is the uptake of chemicals by organisms via water and food. Bioaccumulative chemicals preferentially partition from the environment into organisms resulting in higher concentrations in organisms than in the surrounding environment. Bioaccumulation occurs only when the rate of uptake in an organism exceeds its rate of elimination. The uptake rate is a function of various dermal, respiratory, and dietary vectors.

When applied to aquatic organisms, there are two components to bioaccumulation: bioconcentration and biomagnification. Bioconcentration is the direct uptake of a substance from water by an exposed organism. In contrast, biomagnification is the direct uptake of a substance by an organism via ingestion of food. This distinction is particularly important for carnivorous fish species that can accumulate high levels of chemicals through their diet. Bioaccumulation is the aggregate uptake for the chemical via exposures to both food and water. The bioconcentration factor (BCF) is the ratio of the chemical concentration in an organism to the chemical concentration in water. In general, BCF values are developed from laboratory studies of chemical uptake, though they may be calculated using equations based on correlations of octanol-water partitioning or chemical solubility in water and residue levels in fish (Ernst, 1985).

Not surprisingly, the tendency for an organic chemical to accumulate in aquatic organisms can be correlated with its lipophilicity (Branson et al., 1975). A lipophilic chemical will be preferentially distributed into the octanol portion of an octanol-water mixture. Similarly, upon ingestion, a lipophilic chemical will partition into the lipid portion of the organism. However, the differences between the simple unstructured interface of an octanol-water mixture, and the structured, highly complex arrangement of lipids in a cell, create some problems with the direct extrapolation from octanol-water partitioning (Kow) values to the levels of chemical accumulation expected to occur in organisms. This fact will be discussed in greater detail later in this section.

The transport of a chemical across biological membranes is aided if the chemical is highly lipophilic (Wagner, 1961). Lipophilic organic chemicals are associated with an organism's lipid tissues, and their metabolism and clearance is directly related with the mobilization and turnover of tissue lipids (de Freitas and Norstrom, 1974). The turnover of the lipid pool is related to lipid pool size and factors that control metabolic processes such as body size, temperature, growth, physiologic status, and reproductive condition (Roberts et al., 1977).

The lipid phase in fish consists primarily of biological membranes in which the molecules are predominantly arranged in bilayers (Gobas et al.,

1987). Unlike beef, fish do not exhibit widespread distribution of lipids throughout muscle tissue. Instead, lipids in fish congregate in membranes, skin, or internal organs. There are significant differences in the lipid concentrations among various fish species (Henderson and Tocher, 1987; Personal Communication James McKim, USEPA, Duluth, MN, and James Moore, USEPA, Gulf Breeze, FL). Roberts et al. (1977) reported that bioaccumulation of lipophilic chemicals was better expressed in terms of neutral lipids rather than total lipids since phospholipids do not appear to form part of the lipid pool involved in the storage and release of synthetic organic chemicals.

3.2.1.1 Bioconcentration Factors

A number of researchers have presented evidence that indicates the partition coefficients of organic compounds in lipid-water systems (Kow) are directly correlated with fish bioconcentration factors (Neely et al., 1974; Veith et al., 1979; Kenaga, 1980; Chiou, 1985). Some deviation from the Kow - BCF relationship has been noted for highly lipophilic chemicals (Kow > 100,000) such as HCB. For chemicals like HCB, chemical equilibrium between the aquatic system and the organism is seldom demonstrated within the duration of bioaccumulation studies (Oliver and Niimi, 1983), due in part to the size of the molecule and its limited ability to penetrate biological membranes as well as the very slow rate of metabolism of these chemicals once assimilated into lipid tissue. In addition, the octanol

test solution does not fully represent the lipid structure of the fish, and therefore, octanol-water partitioning does not directly mimic actual tissue accumulation even under equilibrium conditions (Gobas et al., 1989).

The Kow values and corresponding BCF values for HCB are presented in Table 3-1. The BCF's for HCB is less than those measured for PCB's (194,00) and DDT (61,600).

The variation in reported BCF values for the same species reflects differences in the dose and duration of exposure (since HCB requires a long period of time before it reaches equilibrium between fish and water). Oliver and Niimi (1983) reported that even after 119 days of continuous exposure, HCB had not reached an equilibrium in rainbow trout (Salmo gairdneri). Because an HCB equilibrium was not established, the projected BCF value based on the Kow is far larger than the measured concentration. The non-equilibrium BCF for HCB was proven to be a poor predictor of the actual concentration in Lake Ontario fish. Since the mean concentration of HCB in Lake Ontario fish was 30 times greater than that predicted from the measured BCF (Oliver and Niimi, 1983). In this case it was hypothesized that the rate of uptake exceeded the capacity of the fish to eliminate HCB, and concentrations would continue to increase with a longer duration of exposure (Oliver and Niimi, 1983).

Konemann and Van Leeuwen (1980) demonstrated that the extent of chlorination affects the rate of depuration of chlorobenzenes. The rate of elimination of HCB from fish was very slow. It has been suggested that the estimated half-life of HCB in fish could be in excess of 7 months (Niimi and Cho, 1981). In contrast, the half-life for HCB in fish is estimated to be approximately 6 days (McConnell et al., 1975).

Laseter et al. (1976a,b) performed extensive ecological studies on HCB. In short-term studies with large mouth black bass (Micropterus Salmoides), HCB accumulated in fish tissues to a level in excess of 44,000 times the amount found in the water. The bioconcentration factor of the gut tissues exceeded 100,000. Following thirteen days of depuration, 8.6% to 26.9% of the HCB remained in the whole body extracts of fish (Laseter et al., 1976a).

The laboratory data strongly suggest that HCB accumulates to high concentrations in fish, although the accumulation process is not well described.

3.3 Human Health Effects Associated With HCB Exposure

There is considerable information available concerning the toxicity of HCB to mammalian species. In this section, a brief account of human exposure to HCB is presented and the pharmacodynamics are discussed to provide a basis for understanding the relationship between the administered dose and the eventual dose to the target tissue, and quantitative dose-response relationships are developed for carcinogenic effects. The carcinogenic endpoint provides the focus for this analysis of possible effects associated with human consumption of chemically contaminated water and fish.

3.3.1 Pharmacodynamics

To understand the relationship between the administered dose and the dose to the target tissue, a discussion of pharmacodynamics is provided below. Pharmacodynamics is the study of the fate of a chemical within the body. The discussion of pharmacodynamics is divided into four parts: gastrointestinal absorption, distribution, metabolism, and excretion.

3.3.1.1 Gastrointestinal Absorption

An understanding of gastrointestinal absorption efficiency is necessary to quantify the dose of HCB that is assimilated as the result of ingestion of contaminated water and fish. Absorption of HCB in the gastrointestinal tract in animals has been fairly well characterized. HCB is absorbed more readily when administered in lipid solutions such as oils, than in aqueous solutions. Koss and Koransky (1975) compared absorption in rats of HCB administered in olive oil and in an aqueous solution. About 80% of the HCB in olive oil was absorbed, compared with only 6% in the aqueous solution. Similarly, Zabik and Schemmel (1980) found that high-fat diets resulted in more HCB absorption and less excretion than high-carbohydrate diets. Approximately 2% of the HCB in the high-carbohydrate diet was absorbed by rats while approximately 7% was absorbed from the high-fat diet. These data suggest that the absorption of ingested HCB in the diet should not exceed 10% if the rat is an adequate model for humans.

3.3.1.2 Distribution

Since HCB is a lipophilic chemical, it generally distributes according to a tissue's lipid content. Adipose tissue accumulates high concentrations of HCB, as does the bone marrow and skin (USEPA, 1985b). Significant amounts of HCB have been shown to collect in the adrenal cortex of rhesus monkeys (USEPA, 1985b), as well as in the lymph nodes of rats (USEPA,

1980). Other organs such as the liver also accumulate HCB (USEPA, 1985b). In addition, HCB can cross placental membranes (USEPA, 1985a). Rapid weight loss may result in redistribution of these chemicals within the body, as fat reserves are depleted. Under normal conditions, tissues with high lipid content will accordingly accumulate higher levels of HCB than tissues with less fat content.

3.3.1.3 Metabolism

HCB may be metabolically transformed within the body. In mammals, HCB is slowly metabolized into less-substituted benzenes, chlorinated phenols and other minor metabolites. Glucuronide and glutathione conjugates of these metabolites have been measured in tissues of exposed animals (Renner, 1981). Evidence indicated that lipid bound HCB is relatively resistant to metabolism, as most HCB residues in adipose tissue remain unmetabolized (USEPA, 1985b).

HCB at low doses apparently enhances the ability of some animals to metabolize other xenobiotics (USEPA, 1980).

3.3.1.4 Excretion

Excretion is the removal of a toxicant and its metabolites from the body. HCB appears to be excreted in two phases: an initial, relatively rapid phase, and a later, slower phase. Storage in fat retards excretion in the latter phase, since lipid-bound HCB is not readily released or metabolized. Small amounts of HCB are excreted in the feces, while the water soluble metabolites are excreted in urine (Ingebrigtsen et al., 1981).

3.3.2 Carcinogenicity of HCB

The critical human health endpoint for HCB is cancer. Animal dose-response data for carcinogenic effects are used to assess the human health risks associated with the consumption of water and fish contaminated with HCB. The carcinogenic potential and carcinogenic potency of HCB in animals is described in the following section.

Exposure to HCB has been shown to induce tumors in hamsters, mice and rats. The primary target organ appears to be the liver in all three species. The incidence of liver hemangioendotheliomas in hamsters and hepatocellular carcinomas in rats were significantly higher in HCB-treated animals than in untreated animals (USEPA, 1985b). The USEPA Carcinogen Assessment Group (CAG) calculated the potency of HCB on the basis of

hepatocellular carcinomas in female rats (USEPA, 1985b). This particular bioassay was selected for calculating the carcinogenic potency because the exposure regime resulted in malignant tumors in the primary target organ, giving the highest potency estimate. The USEPA classifies HCB as a Group B-2 compound, a probable human carcinogen, based on sufficient evidence of carcinogenicity in animals, but inadequate evidence of carcinogenicity in humans. In the USEPA Health Assessment Document for chlorinated Benzenes, it was concluded that available epidemiologic studies of occupationally exposed individuals were inadequate to establish a relationship between HCB exposures and cancer in humans (USEPA, 1985b).

Fourteen data sets showing significant tumor incidence were used to calculate the oral carcinogenic potency of HCB using the multistage model for low-dose extrapolation (USEPA, 1985b). These potencies provide a range of estimates that reflect the uncertainties associated with differences in species, tumor sites and influences of other exposures. The USEPA selected the highest potency estimate (1.7 (mg/kg/day)⁻¹) for risk assessment (USEPA, 1985b). This potency estimate is published in the 1989 Health Effects Assessment Summary Tables and Users Guide (USEPA, 1989).

3.3.3 Human Health Effects Summary

The following factors were identified as keys to understanding the adverse health effects which may result from HCB and HCB_D exposures:

- When ingested HCB is absorbed from the gastrointestinal tract. The efficiency of absorption is related to the matrix in which the chemical are ingested. Approximately 10% ingested HCB in food is absorbed.
- Assimilated HCB is primarily bound to adipose (lipid) tissue.
- Lipid-bound HCB is slowly metabolized.
- HCB is classified as a Group B-2, probable human carcinogen, with USEPA carcinogenic potency factor of $1.7 \text{ (mg/kg/day)}^{-1}$.

3.4 Critique of Proposed Louisiana Ambient Water Quality Criteria for Hexachlorobenzene

The proposed DEQ criteria for HCB is 0.24 ng/L for a incremental cancer risk level of 10^{-6} for both public water supplies and recreational waters (Table 3-2). The criteria values for the two designated uses are the same because of the high BCF for HCB which makes fish consumption the dominant contributor to chemical uptake in both scenarios.

TABLE 3-2

COMPARISON OF PUBLIC WATER SUPPLY (PWS) AND RECREATIONAL (REC) CRITERIA CALCULATED BY
EPA, DEQ AT THE 10^{-6} RISK LEVEL AND ALTERNATIVE METHOD AT THE 10^{-5} RISK LEVEL

Chemical	EPA PWS ^a	EPA REC ^b	DEQ ^d PWS	DEQ ^c REC	Refined Criteria ^d for PWS	Refined Criteria for REC ^c
Hexachlorobenzene	0.72 ng/L	0.74 ng/L	0.24 ng/L	0.24 ng/L	219 ng/L	237 ng/L
Hexachlorobutadiene	0.45 µg/L	50 µg/L	0.09 µg/L	0.09 µg/L	41.0 µg/L	114 µg/L
1,2-Dichloroethane	0.94 µg/L	243 µg/L	0.36 µg/L	6.8 µg/L	5.47 µg/L	1,570 µg/L
1,1,2,2-Tetrachloroethane	0.17 µg/L	10.7 µg/L	0.16 µg/L	1.8 µg/L	2.48 µg/L	280 µg/L

a) Fish and Water Ingestion

b) Fish Ingestion Only

c) Fish and Incidental Water Ingestion

d) Fish, Water, and Incidental Water Ingestion

efficiency and the small fraction of contaminated fish likely eaten in proportion to the total quantity of fish consumed.

The refined risk assessment method employed a gastrointestinal absorption efficiency of 10% based on scientific data for HCB (see Section 3.1.1). It was assumed, based on consumer data, that the fraction of fish consumed from contaminated sources is 10% of the total amount of fish consumed by the average person. The alternative criteria were calculated at a de minimus risk level of 10^{-5} . These factors result in an appropriate criteria concentration of 240 ng/L (Table 3-2) for both public water supplies and recreational waters.

3.4.3.1 Fraction From the Contaminated Source

Another of the conservative assumptions employed by the DEQ is that all of the drinking water ingested, aquatic organisms consumed and water ingested during swimming activities is obtained from a contaminated source. This is highly unlikely. For example, the national market that exists in the United States provides consumers with food resources (fish) from a variety of regions and in effect dilutes the potential for the consumption of a particular contaminant. National survey data indicate that freshwater finfish constitute only approximately 10% of the total fish consumption in the United States (USDA, 1985). Most of the fish consumed in the United States is from marine resources and is therefore

likely uncontaminated by localized chemical releases into freshwater. In addition, it is likely that only a small fraction of the freshwater fish in the human diet is caught from contaminated sources. That small fraction of catch is likely largely limited to recreational fisherman. To assume that 100% of the fish consumed by the people of Louisiana is from contaminated sources is unreasonable. The assumption of a 10% fraction from contaminated sources is likely very conservative as that would represent the entire fraction of freshwater finfish in the average diet.

3.4.3.2 Gastrointestinal Absorption Efficiency

The DEQ and the USEPA assume the gastrointestinal absorption efficiency is 100%. Based on the evidence presented in Section 3.2.1.1 this assumption is very conservative and likely significantly overestimates the absorbed dose of HCB that a human may receive from ingestion of contaminated water or fish. The gastrointestinal absorption efficiency of ingested HCB has been estimated in animal studies. The estimates vary with matrix, but when administered in aqueous solution or food, less than 10% of HCB was absorbed. It is reasonable to assume that humans will absorb approximately 10% of the HCB from ingested water and fish.

3.4.3.3 Acceptable Risk

Based on studies by Travis et al. (1987), it was determined that potentially carcinogenic chemicals with lifetime risks above 4×10^{-3} received regulation and those with risks below 1×10^{-6} remained unregulated. In addition, for effects resulting from exposures to the entire U.S. population, a risk level below 1×10^{-6} never triggered action but a level above 3×10^{-4} always triggered action. For smaller populations regulatory action never resulted for individuals below a level of risk of 1×10^{-4} .

These trends in regulatory policy have led to a number of states, such as California, to select 1×10^{-5} to constitute the no significant risk level for potentially carcinogenic chemicals (Title 22 California Code of Regulations Section 12711). A risk level of 1×10^{-5} represents the mid-range reported by the USEPA. This risk level is appropriate for the calculations of ambient water quality criteria.

3.4.4 Summary

The important points regarding the development of ambient water quality criteria for HCB in public water supplies and recreational waters are listed below.

- HCB is considered a probable human carcinogen.

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- The criteria concentrations are driven by the high bioconcentration factor, because of HCB's high degree of lipophilicity.
- HCB adsorbs readily to sediments, is very insoluble in water and is environmentally persistent.
- HCB gastrointestinal absorption efficiency is assumed to be 100% by the USEPA and DEQ, but scientific data suggests that HCB absorption is approximately 10%.
- The USEPA and DEQ assume that 100% of the fish and water consumed is derived from a contaminated source. This assumption significantly overestimates the fraction of the environmental concentration that is received by a human. Only a small fraction, perhaps 10%, of the fish consumed in Louisiana, the fraction from recreational catch in freshwater near sources of chemicals, may be significantly contaminated.
- Significant amounts of HCB may be lost during food preparation primarily through the loss of oils during cooking. Therefore, concentrations of HCB in cooked fish are likely substantially less than predicted by DEQ model based on the solubility concentration and BCF.
- The alternative criteria concentrations calculated for HCB using refined, scientifically based parameter values at the 10^{-5} de minimus risk level are a factor of 1000 greater than the DEQ criteria and are still health protective.

4.0 HEXACHLOROBUTADIENE

4.1 Environmental Fate of HCB

Transport and transformation processes determine the environmental fate of HCB. In turn, the environmental fate strongly influences the availability of HCB to fish and ultimately to humans.

Once released into an aquatic system, HCB may partition within various compartments: water, biota, sediment, or they may escape to the atmosphere. The transport processes which define the movement of the chemical between these compartments highly depends on the physico-chemical properties of both the chemical and the media. Transformation, a change in the chemical structure of the compound, usually results in the degradation of the "parent" compound. Transformation processes include photolysis, hydrolysis, oxidation, reduction and microbial degradation. In the following sections, the persistence, physical and chemical properties, and environmental fate of HCB are discussed.

4.1.1 Physical and Chemical Properties of HCB

Some of the important physical and chemical properties of HCB are presented in Table 3-1. An understanding of the physical and chemical

properties of HCBd helps determine how the chemical will be partitioned in aquatic systems and aids in predicting the overall behavior of HCBd in the environment.

HCBd is soluble in water at approximately 2.5 mg/L and is approximately 1000 times more soluble in water than HCB. As a result, HCBd is expected to be found in higher concentrations in the water than HCB, but in lower concentrations in sediment.

The vapor pressure and Henry's Law Constant values are listed in Table 3-1. Because of HCBd's greater water solubility and Henry's Law constant, volatilization represents a more important mechanism of HCBd loss from water than for HCB.

Although HCBd is water-soluble, it is a lipophilic chemical with a relatively high octanol-water partitioning coefficient (K_{ow}) value. Consequently, HCBd is expected to be accumulated by aquatic organisms. The bioaccumulation of this chemical is addressed in Section 4.2.

4.1.2 Fate of HCBd in Aquatic Systems

In this section, the transport and transformation of HCBd in water and sediment are described.

HCBD has a relatively high vapor pressure and Henry's Law constant which suggest rapid volatilization from water. The Koc value (5,181) calculated for HCBD suggests that it should strongly adsorb to sediment. Experimental values support this assertion. Laseter et al. (1976b), using a laboratory model, observed that sediment contained over 60 times more HCBD than water after four days of exposure. Furthermore, almost 70% of the HCBD was present four days after clean water was exchanged for contaminated water in the test system. However, in contrast to HCB, the physico-chemical properties of HCBD indicate that it is more water soluble and less strongly adsorbed to sediment than HCB.

Tabak et al. (1981) observed that HCBD biodegrades in aerobic batch cultures, but not in anaerobic cultures. Based on monitoring data, estimated half-lives for HCBD degradation are 3-30 days in river water, and 30-300 days in lake water and groundwater (Zoeteman et al., 1980). The role of hydrolysis in the abiotic degradation of HCBD in water is considered minor (Howard, 1989). Photochemical reactions with HCBD have been shown to occur in the atmosphere, but have not been observed in water (Howard, 1989).

HCBD is more mobile and less resistant to degradation than HCB in aquatic systems.

4.1.3 Fate Summary

The following summarizes the environmental fate of HCBd:

- HCBd is relatively soluble in water and is more readily degraded than HCB.
- HCBd is lipophilic and readily bioaccumulated.
- HCBd is relatively long lived in the atmosphere and may be widely dispersed.

4.2 Bioaccumulation of HCBd

HCBd released to aquatic systems may be accumulated by aquatic organisms. If the chemical is accumulated to levels exceeding toxicity thresholds, aquatic organisms may be adversely affected. Furthermore, the consumption of HCBd contaminated water, fish and shellfish species provides a potential exposure route to humans. In this section, bioaccumulation and of HCBd is described.

4.2.1 Bioaccumulation

Bioaccumulation is the uptake of chemicals by organisms via water and food and important aspects of bioaccumulation were discussed in Section 3.2.1. HCBd is a lipophilic chemical and may bioaccumulate in fish and shellfish.

4.2.1.1 Bioconcentration Factors

Measured bioconcentration factors for HCB_D in selected fish species are presented in Table 3-1. Oliver and Niimi (1983) reported that after 119 days of continuous exposure to HCB_D, rainbow trout (Salmo gairdneri) attained equilibrium by day 69 of exposure. Following the establishment of equilibrium, the rate of HCB_D accumulation was directly proportional to the rate of fish growth.

Oliver and Niimi (1983) reported that since the experimentally measured bioconcentration factor for HCB_D reached equilibrium, it adequately predicted measured concentrations in Lake Ontario fish. However, the non-equilibrium BCF for HCB discussed in Section 3.2.1.1, was proven to be a poor predictor of the actual concentration in Lake Ontario fish, as the mean concentration of HCB in Lake Ontario fish was 30 times greater than predicted (Oliver and Niimi, 1983). In this case it was hypothesized that the rate of uptake exceeded the capacity of the fish to eliminate HCB, and concentrations would continue to increase with a longer duration of exposure (Oliver and Niimi, 1983).

Konemann and Van Leeuwen (1980) demonstrated that the extent of chlorination affects that rate of depuration of chlorobenzenes. The half-life of HCB_D in fish is estimated to be approximately 6 days (McConnell et al., 1975).

As mentioned earlier, Laseter et al. (1976a,b) performed extensive ecological studies on HCB. In short-term studies with large mouth black bass (Micropterus salmoides), concentration factors for HCB remained relatively low, reaching a maximum body burden of 112 times greater than the concentration in water (Laseter et al., 1976b). Furthermore, uptake was irregular with respect to the concentration of HCB in water, and bass accumulated more from water than from food (Laseter et al., 1976b).

The laboratory data suggest that HCB is bioaccumulated but to a lesser extent than HCB.

4.3 Human Health Effects Associated with HCB Exposure

There is limited literature on the toxicity of HCB. In this section, a brief review of human exposure to HCB is presented. In addition, the pharmacodynamics of the compound are discussed to provide a basis for understanding the relationship between the administered dose and the eventual dose to the target tissue, and quantitative dose-response relationships are developed for carcinogenic effects. The carcinogenic endpoint provides the focus for this analysis of possible effects associated with human consumption of chemically contaminated water and fish.

4.3.1 Pharmacodynamics

To understand the relationship between the administered dose and the dose to the target tissue, a discussion of pharmacodynamics is provided below. Pharmacodynamics is the study of the fate of a chemical within the body. The discussion of pharmacodynamics is divided into four parts: gastrointestinal absorption, distribution, metabolism, and excretion.

4.3.2 Gastrointestinal Absorption

Information on the gastrointestinal absorption of HCB_D is not well characterized (USEPA, 1984). The limited evidence suggests that HCB_D is absorbed in the gastrointestinal tract, though the amount of absorption has not been quantified in humans or animals (USEPA, 1980b).

4.3.3 Distribution

Since HCB_D is a lipophilic chemical, it will distribute according to a tissues' lipid content. HCB_D may accumulate in the adipose tissue of selected organs (USEPA, 1980b). However, in rats administered a mixture of chlorinated hydrocarbons, HCB_D did not show a strong tendency to accumulate in tissues with high lipid concentrations (USEPA, 1988b).

Rapid weight loss may result in redistribution of these chemicals within the body, as fat reserves are depleted.

Under normal conditions, tissues with high lipid content will accordingly accumulate higher levels of HCBd than tissues with less fat content.

4.3.4 Metabolism

HCBd may be metabolically transformed within the body. Although no substantive information was found on the metabolism of HCBd in test organisms or humans.

4.3.5 Excretion

There are no definitive data on the excretion of HCBd in humans or animals.

4.3.6 Carcinogenicity of HCBd

The critical human health endpoint for HCBd is cancer. Animal dose-response data for carcinogenic effects are used to assess the human health

risks associated with the consumption of water and fish contaminated with HCB_D. The carcinogenic potency of HCB_D in animals is described in this section.

HCB_D produced kidney, and to a lesser extent, lung tumors in a two year rat bioassay at an oral dose of 20 mg/kg/day (Kociba *et al.*, 1977a,b). HCB_D was reported as mutagenic in the Ames assay (IARC, 1979b). Nonetheless, in its 1980 Health Effects Assessment, the USEPA concluded that carcinogenic data for HCB_D in animals was limited, and that there were no pertinent data on the carcinogenicity of this compound in humans. Based on the weight of evidence, the USEPA has designated HCB_D a Group C compound, a possible human carcinogen (USEPA, 1980b).

The USEPA, using the linear multistage model, calculated a potency factor for oral HCB_D exposures of 7.75×10^{-2} (mg/kg/day)⁻¹ which is published in the USEPA's 1989 IRIS data base.

4.3.7 Human Health Effects Summary

The following factors were identified as keys to understanding the adverse health effects which may result from HCB_D exposures:

- The efficiency of gastrointestinal absorption is unknown, but is probably related to the matrix in which the chemical is ingested.

- HCB₁₂D is classified as a possible human carcinogen (Group C) with an USEPA carcinogenic potency factor of 7.75×10^{-2} (mg/kg/day)⁻¹.

4.4 Critique of Proposed Louisiana Ambient Water Quality Criteria for Hexachlorobutadiene.

The proposed DEQ criteria for HCB₁₂D is 0.09 µg/L for an incremental cancer risk level of 10^{-6} for both public water supplies and recreational waters (Table 3-2). The criteria values are the same because of HCB₁₂D's relatively high BCF which makes fish consumption the dominant contributor to chemical uptake in both scenarios.

4.4.1 Accuracy Check

The proposed criteria are consistent with the described DEQ methods and were accurately calculated. The potency factor and BCF used are up to date values as of July 1989 (USEPA, 1989b).

4.4.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed DEQ criteria for HCB are significantly more stringent than the USEPA guideline criteria (Table 3-2). The USEPA criteria were calculated using a smaller fish consumption rate than used by DEQ (6.5

g/day versus 20 g/day). Secondly, the DEQ criteria calculation included incidental ingestion of water during swimming which was not considered by the USEPA.

4.4.3 Alternative HCB Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for HCB. The refined criteria are significantly less stringent than the proposed DEQ criteria. They are, however, still protective of human health from chemical uptake associated with the consumption of drinking water, ingestion of fish and incidental ingestion of water during swimming activities. Both USEPA and DEQ methods fail to take in to account scientific data on gastrointestinal absorption efficiency and the fraction of contaminated fish likely eaten in proportion to the total quantity of fish consumed.

The refined method employed a gastrointestinal absorption efficiency of 10% based on scientific data for lipophilic chemicals such as HCB (see Section 3.1.1). It was assumed, based on consumer data, that the fraction of fish consumed from recreational sources is 10% of the total amount of fish consumed by the average person. The alternative criteria were calculated at a de minimis risk level of 10^{-5} . These factors result in an appropriate criteria concentrations of 41 $\mu\text{g/L}$ and 114 $\mu\text{g/L}$ for public water supplies and recreational waters, respectively (Table 3-2).

4.4.3.1 Fraction From the Contaminated Source

Another of the conservative assumptions employed by the DEQ is that all of the drinking water ingested, aquatic organisms consumed and water incidentally ingested during swimming activities is obtained from a contaminated source. This is highly unlikely. For example in the case of fish, the national market that exists in the United States provides consumers with food resources from a variety of regions and in effect dilutes the potential for the consumption of a particular contaminant. National survey data indicate that freshwater finfish constitute only approximately 10% of the total fish consumption the United States (USDA, 1985). Most of the fish consumed in the United States is from marine resources, and is therefore, likely uncontaminated by localized chemical releases into freshwater. In addition, it is likely that only a small fraction of the freshwater fish in the human diet is caught from contaminated sources. That small fraction of catch is likely largely limited to recreational fisherman. To assume that 100% of the fish consumed by the people of Louisiana is from contaminated sources is unreasonable. The assumption of a 10% fraction from contaminated sources is likely very conservative as that would represent the entire fraction of freshwater finfish in the average diet.

4.4.3.2 Gastrointestinal Absorption Efficiency

The DEQ and the USEPA assume the gastrointestinal absorption efficiency is 100%. Based on the evidence presented in Section 3.2.1.1 this assumption is very conservative and likely overestimates the absorbed dose of HCB_D that a human may receive from ingestion of contaminated water or fish. The gastrointestinal absorption efficiency of ingested HCB_D has not been determined for humans. However, as previously stated, the absorption of similar lipophilic chemicals such as HCB is only approximately 10% for water and food matrices.

4.4.3.3 Acceptable Risk

Based on studies by Travis et al. (1987), it was determined that potentially carcinogenic chemicals with lifetime risks above 4×10^{-3} received regulation and those with risks below 1×10^{-6} remained unregulated. In addition, for effects resulting from exposures to the entire U.S. population, a risk level below 1×10^{-6} never triggered action but a level above 3×10^{-4} always triggered action. For smaller populations, regulatory action never resulted for individuals below a level of risk of 1×10^{-4} .

These trends in regulatory policy have led to a number of states, such as California, to select 1×10^{-5} to constitute the no significant risk level for potentially carcinogenic chemicals (Title 22 California Code of Regulations (CCR) Section 12711). This risk level is appropriate for the calculations of ambient water quality criteria.

4.4.4 Summary

The important points regarding the development of ambient water quality criteria for HCBd in public water supplies and recreational waters are listed below.

- HCBd is considered a possible human carcinogen.
- HCBd is lipophilic and bioaccumulates in aquatic organisms.
- HCBd is relatively soluble in water and is environmentally persistent.
- The gastrointestinal absorption efficiency for HCBd is assumed to be 100% by the USEPA and DEQ, but scientific data for HCB suggests that HCBd absorption is approximately 10%.
- The USEPA and DEQ assume that 100% of the fish and water consumed is derived from a contaminated source. This assumption significantly overestimates the fraction of the environmental concentration that is received by a human. Only a small fraction, perhaps 10%, of the fish consumed in Louisiana, the fraction from recreational catch in freshwater near sources of chemicals, may be significantly contaminated.
- Significant amounts of HCBd may be lost during food preparation primarily through the loss of oils during cooking. Therefore, concentrations of HCBd in cooked fish are likely substantially less than predicted by DEQ model based on the solubility concentration and BCF.

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- The alternative criteria concentrations calculated for HCB_D using refined, scientifically based parameter values at the 10^{-5} de minimis risk level are a factor of 500 and 1000 greater than the DEQ criteria for public water supplies and recreational waters, respectively.

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5.0 1,2-DICHLOROETHANE (Ethylene Dichloride (EDC))

5.1 Environmental Fate of 1,2-Dichloroethane

The environmental fate of a compound once released into the aquatic environment is regulated by the cumulative interaction of transport and transformation processes as discussed in Section 3.1. In the following sections the persistence, physical and chemical properties, and environmental fate of EDC are discussed.

5.1.1 Physical and Chemical Properties of EDC

Some of the important physical and chemical properties of EDC, and related compounds are presented in Table 3-1.

EDC is highly soluble in water at approximately 8 g/L. However, the Henry's Law Constant and vapor pressure for EDC indicate that the compound is readily volatilized. The log octanol-water coefficient (K_{ow}) is relatively low indicating that EDC is not highly lipophilic and is not likely to be accumulated in the tissues of aquatic organisms to a significant degree, but EDC may be found in nonaqueous-phase liquids (Clement, 1985). The soil adsorption coefficient (K_{oc}) indicates that EDC does not readily partition to organic carbon sources or adhere to sediments. These factors are important as they regulate the movement of

EDC between the various media phases in the environment. It is reported that the dominant dispersion route for EDC in the environment is through volatilization (Clement, 1985).

Pearson and McConnell (1975) suggest that the presence of halogenated aliphatic compounds including EDC in ambient waters is due primarily to absorption from the atmosphere and deposition by precipitation from the atmosphere.

5.1.2 Fate of 1,2-Dichloroethane in Aquatic Systems

EDC is highly water soluble (8 g/L at 20°C) but there is no evidence that it accumulates in aquatic organisms or is adsorbed to sediments (USEPA, 1979c). Due to the high vapor pressure of EDC, it is expected to disperse rapidly in the atmosphere. Several environmental factors influence the evaporative half-life including the wind speed and the mixing conditions of the receiving waters. Evaporative half-lives for EDC determined under laboratory conditions by Dilling et al. (1975) and Dilling (1977) were 28 and 29 minutes in a stirred, open water column at a concentration of 1 mg/L. However, the evaporative half-life determined for a eutrophic lake by the EXAMS model was 10 days (ATSDR, 1988c). The ATSDR report (1988c) for 1,2-dichloroethane indicated that volatilization from large volume spills may be very limited. EDC released to subsurface soils where volatilization is limited and the organic content of the soil is low, will

readily move through porous soil formations to the groundwater (Wilson et al., 1981). Another factor reported by Dilling et al. (1975) reported that presence of sodium chloride also influenced EDC's volatilization. A sodium chloride concentration of 3 percent, which is similar to sea water, resulted in a 10 percent decrease in the volatilization rate of EDC.

Information on the transformation and degradation of EDC in the aquatic environment is limited. It is believed that EDC is lost to the atmosphere before significant chemical or biological degradation occurs. The hydrolytic half-life for EDC is thought to be similar to the half-life of the structural analog chloroethane which Radding et al. (1977) reported to be ≤ 40 days. Dilling et al. (1975) reported that direct oxidation and photolysis of analogous compounds (dichloromethane, trichloromethane and 1,1,1-trichloroethane) in surface water was not accelerated by sunlight, and therefore, these processes are not expected to be important in the transformation of EDC. EDC appears to undergo little or no biodegradation in the aquatic environment (ATSDR, 1988c) and is reported to be resistant to biodegradation under both aerobic and anaerobic conditions (Tabak et al., 1981; Jafvert and Wolfe, 1981 and Wilson et al., 1983). The lifetime of EDC in groundwater is expected to be on the order of months to years (USEPA, 1985a).

5.1.3 Fate Summary

The following summarizes the environmental fate of EDC:

- EDC is relatively soluble in water, but readily partitions to the atmosphere.
- EDC is not expected to accumulate in aquatic organisms or to adsorb to sediments.
- EDC does not undergo significant physical or biological transformation or degradation in the aquatic environment, but is photooxidized in the atmosphere with an expected lifetime of 2 to 4 months.

5.2 Bioaccumulation of 1,2-Dichloroethane

EDC is not prone to accumulation by aquatic organisms, and therefore, the consumption of aquatic organisms is not likely to be a significant route of EDC exposure for humans. In this section the bioaccumulation and EDC is described.

5.2.1 Bioaccumulation

Bioaccumulation is the uptake of chemicals by organisms via water and food and important aspects of bioaccumulation were discussed in Section 3.2.1. Although there is some evidence suggesting that EDC may partition in

nonaqueous-phase liquids, this is not substantial evidence that it bioaccumulates in aquatic organisms (Clement 1985).

5.2.1.1 Bioconcentration Factor

The bioconcentration factor is the ratio of the concentration measured in the tissues of a test organism to the concentration in the water under steady state conditions. It represents the tendency of a chemical to partition from the water and accumulate in the lipid tissues of aquatic organisms. The USEPA ambient water quality criteria document for chlorinated ethanes (USEPA, 1980c) states that the BCF's associated with chlorinated ethanes increase with increased levels of chlorination. For example, the BCF reported for 1,2-dichloroethane is 2 and that for hexachloroethane is 139. Bioconcentration in aquatic organisms of chemicals with BCF's less than 100 is considered to be insignificant. Only 0.4% of the human exposure to waterborne EDC is expected to occur through the consumption of aquatic organisms while 99.6% of exposure is expected to be associated with drinking water consumption (USEPA, 1980c).

5.2.1.2 Tissue Residues

No information on tissue residues of EDC were available. This is undoubtedly due to EDCs low bioaccumulation potential.

3.4.1 Accuracy Check

The proposed criteria are consistent with the described DEQ methods and were accurately calculated. The potency factor and BCF used are up to date values reported by USEPA as of July 1989.

3.4.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed DEQ criteria for HCB are significantly more stringent than the USEPA guideline criteria (Table 3-2). The USEPA criteria were calculated using a smaller fish consumption rate than used by DEQ (6.5 g/day versus 20 g/day). Secondly, the DEQ criteria calculation included incidental ingestion of water during swimming which was not considered by the USEPA.

3.4.3 Alternative HCB Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for HCB. The refined criteria are significantly less stringent than the proposed DEQ criteria. They are, however, still protective of human health from chemical uptake associated with the consumption of drinking water, ingestion of fish and incidental ingestion of water during swimming activities. Both USEPA and DEQ methods fail to take in to account scientific data on gastrointestinal absorption

5.2.3 Summary

Outlined below are the important points concerning the bioaccumulation and aquatic toxicity of EDC.

- EDC has a low bioconcentration factor and does not accumulate in aquatic organisms to a significant degree.
- Only 0.4% of human exposure to water borne EDC is expected to occur through the consumption of aquatic organisms.

5.3 Human Health Effects Associated with 1,2-Dichloroethane Exposure

5.3.1 Pharmacokinetics

Several researchers have studied the pharmacokinetics of 1,2-dichloroethane (ethylene dichloride) in animals, although little quantitative information has been gathered on absorption, distribution, metabolism, and excretion of EDC in human subjects. However, the physiological behavior of EDC can be predicted on the basis of some chemical properties. For example, EDC is very soluble in water (8 g/L) and possesses a high vapor pressure (64 torr at room temperature) (USEPA, 1985a). Consequently, these properties suggest rapid and extensive absorption of EDC from both the lungs and the gastrointestinal tract. EDC is neutral and lipophilic and transmucosal passage is expected to occur readily (USEPA, 1985).

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5.3.1.1 Gastrointestinal Absorption

No studies regarding absorption in humans following oral exposure to EDC were located. However, case studies of acute human exposure suggest that EDC can be rapidly absorbed to the systemic circulation.

In experimental animals, EDC is readily absorbed. Spreafico (1978, 1979, 1980) found absorption in rats to be dose dependent. In corn oil, half of the lowest dose administered was absorbed in 3.3 minutes, while at the highest dose it took 6.4 minutes for the rats to absorb half of the dose (Spreafico, 1978, 1979, 1980). EDC administered in water is absorbed faster and to a greater extent than the dose in corn oil (Withey et al., 1982).

On average, Spreafico (1978,1979,1980) found that 78% of the administered dose was absorbed by rats, and that if metabolism (first pass effects in the liver) and elimination (through the lung) were taken into account, close to 100% absorption would be observed. In support, Reitz et al. (1980,1982)) completely recovered 14 C-EDC in expired air, urine, and carcass, reflecting the complete absorption of EDC.

5.3.1.2 Distribution

Researchers studying the anesthetic properties of EDC, found that it passes freely across the blood-brain, and placental barriers which indicates that EDC can circulate throughout the body (USEPA, 1985a). However, little information exists on the distribution in human tissues, and there are few controlled animal studies. Spreafico (1978,1979,1980) studied EDC concentrations in the blood, liver, lung, and epididymal tissue of rats upon oral and inhalatory doses. For orally administrated doses, accumulation occurred most rapidly in the liver and adipose tissue. However, the concentrations measured in the tissues were less than the concentration found in the blood. All tissues paralleled the first order decline in blood concentration once exposure ceased (Spreafico, 1978, 1979, 1980).

5.3.1.3 Metabolism

The metabolism of EDC in the mouse and rat has been fairly well characterized within the past decade or so (USEPA, 1985a). Yllner (1971) determined that 55-86% of EDC is biotransformed in the mouse, and that this appeared to follow a dose-dependent metabolism that is limited by saturated enzymes. In this study, approximately 13% of the dose mineralized to CO₂, 73% was converted to metabolites found in urine, and 11% was exhaled.

EDC is conjugated by the glutathione pathway. USEPA (1985a) listed 14 different metabolites that various researchers have identified. Other substances may effect EDC metabolism as USEPA (1985a) noted that ethanol may inhibit or enhance the metabolism depending on the tissue concentration.

5.3.1.4 Excretion

The excretion of EDC in man has not been characterized. In animals, the principal route of excretion of unmetabolized EDC is in expired air. Yllner (1971a) recovered 45% of an intravenous dose of EDC in exhaled air of mice, and found when the dose increased, the amount of unchanged EDC increased also. In fact, the percentage of parent compound recovered in expired air increases exponentially with dose which reflects the saturable metabolic processes.

In support, Reitz et al. (1980, 1982) found 29% of the oral dose was recoverable in expired air; however, only 1.8% of the lowest dose of the inhalation dose was recovered. Excretion via routes other than pulmonary excretion are not typical (USEPA, 1985a), so this low recover may indicate metabolic processes.

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5.3.1.5 Human Health Effects

EDC appears to be readily absorbed via ingestion and inhalation, because of its physical properties. EDC's relatively high water solubility allows for rapid gastrointestinal absorption of aqueous solutions, and its high vapor pressure and lipophilicity allow for pulmonary absorption. Once absorbed, the highest concentration appears in the blood. Distribution in humans is not well characterized, but animal studies have provided adequate data. While the liver may accumulate significant amounts, cessation of exposure results in the parallel decline of EDC in all tissues. Absorbed EDC is excreted rapidly from the lungs, but EDC has been shown to be metabolized into 14 different compounds which may be excreted through expired air (CO₂) or urination (glutathione conjugates).

5.3.2 Carcinogenicity

The USEPA considers EDC a group B2 compound--a "probable" human carcinogen. The USEPA deduced this from the four following lines of evidence: 1) multiple tumor types in an oral rat bioassay and an oral mouse bioassay conducted by the National Cancer Institute (NCI, 1978a), 2) suggestive evidence in two other animal bioassays, 3) demonstrated evidence of reactive metabolites of EDC and formation of a DNA adduct, and 4) evidence that EDC is also a mutagen.

Note that the only inhalation study (Maltoni et al., 1980) available to the USEPA in its health assessment of EDC did not indicate any significant carcinogenic effects in either rats or mice. Several factors may account for the differences between the NCI and Maltoni studies including: the use of different rodent strains; routes of exposure and exposure durations.

5.4 Critique of Proposed Louisiana Ambient Water Quality Criteria for 1,2-Dichloroethane

The ambient water quality criteria proposed by the Louisiana DEQ for EDC in public water supplies and recreational waters are 0.36 $\mu\text{g/L}$ and 6.8 $\mu\text{g/L}$, respectively (Table 3-2). The primary route of human exposure to water borne EDC is through water consumption, therefore, the risk assessment is primarily driven by the water consumption rate.

5.4.1 Accuracy Check

The proposed criteria are consistent with described DEQ methods and were accurately calculated. The potency factor and BCF used by DEQ in their calculation are up to date values reported by the USEPA as of July 1989 (USEPA, 1989b).

5.4.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed EDC criteria are essentially equivalent to the USEPA criteria of EDC in public water supplies (Table 3-2). However, the DEQ criterion for EDC in recreational waters is significantly more stringent than the USEPA criterion. The difference in the recreational criteria is due to the fact that the DEQ used a 20 g/day fish consumption rate versus the 6.5 g/day rate used by the USEPA, and DEQ included an incidental water ingestion rate for swimming of 0.089 L/day. The combined influence of these two factors was sufficient to significantly reduce the value of the proposed DEQ recreational water criterion for EDC.

5.4.3 Alternative 1,2-Dichloroethane Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for EDC. The refined criteria are significantly less stringent than the DEQ criteria for both public water supplies and recreational waters (Table 3-2). These criteria, however, are still protective of human health from chemical uptake associated with the consumption of drinking water, ingestion of fish and incidental ingestion of water during swimming activities. The water consumption rates are the controlling factors, and both the USEPA and DEQ criteria use a water consumption rate of 2.0 L/day which approximates the national, 90th percentile drinking water consumption rate. The refined method used an

average water consumption rate of 1.4 L/day suggested by the USEPA. Secondly, DEQ used an incidental water ingestion rate that is 36 times greater than the incidental ingestion rate calculated using USEPA suggested parameters (USEPA, 1988e) and used in the refined equations.

The refined method employed a gastrointestinal absorption efficiency of 100% based on scientific data. It is assumed, based on consumer data, that the fraction of fish consumed from recreational sources is 10% of the total amount of fish consumed by the average person (USDA, 1985). The criteria are calculated at a de minimus risk level of 10^{-5} based on trends in regulatory action (Travis et al., 1987). These factors result in an appropriate EDC criteria concentrations of 5.47 $\mu\text{g/L}$ and 1,578 $\mu\text{g/L}$ for public water supplies and recreational waters, respectively. The values are approximately 16 and 200 times the proposed Louisiana Criteria for the two scenarios, respectively.

5.4.3.1 Fraction From the Contaminated Source

The DEQ and USEPA assume that all of the water, aquatic organisms and water ingested during swimming activities originated from a contaminated source. This assumption is highly unlikely. For example, in the case of fish consumption, a national market exists in the United States that provides consumers with food resources from a variety of regions and in effect dilutes the potential for the consumption of a particular

contaminant. National survey data indicate that freshwater finfish constitute only approximately 10% of total fish consumption in the United States (USDA, 1985). Most of the fish consumed in the United States is from marine resources, and therefore, likely uncontaminated by localized chemical releases into freshwater. In addition it is likely that only a small fraction of the freshwater fish in the human diet is caught from contaminated waters. That small fraction of catch is likely limited to recreational fishermen and not the general population.

To assume that 100% of the fish consumed by the people of Louisiana are from contaminated sources is unreasonable. The assumption of a 10% fraction from contaminated sources is likely very conservative as that would represent the entire fraction of freshwater finfish in the average diet.

5.4.3.2 Gastrointestinal Absorption Efficiency

The DEQ and the USEPA assume the gastrointestinal absorption efficiency is 100%. Based on scientific data this assumption appears to be valid. However, it should be noted that this compound is volatile and is not prone to bioaccumulate, therefore, the concentration likely to be received by a human through ingestion is very small.

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- EDC is water soluble, but is readily lost from aquatic systems via volatilization.
- EDC does not bioaccumulate significantly in aquatic organisms and only 1.6% of waterborne EDC exposure to humans is attributable to the consumption of aquatic organisms.
- The gastrointestinal absorption efficiency is expected to be nearly 100%.
- The USEPA and DEQ assume that 100% of the fish and water consumed is derived from a contaminated source, which may significantly overestimates the fraction of the environmental concentration that is received by a consumer. Only a small fraction, perhaps 10%, of the fish consumed in Louisiana, the fraction from recreational catch in freshwater near sources of chemicals, may be significantly contaminated.
- The criteria concentrations calculated for EDC in public water supplies and recreational waters using refined, scientifically based parameter values at the 10^{-5} de minimus risk level are significantly less stringent than the DEQ criteria. The difference are factors of approximately 16 and 200 for public water supplies and recreational waters, respectively.

5.4.3.3 Acceptable Risk

Based on studies by Travis et. al. (1987), it was determined that for carcinogenic chemicals with lifetime risks above 4×10^{-3} received regulation and those with risks below 1×10^{-6} remained unregulated. In addition, for effects resulting from exposures to the entire U. S. population, a risk level below 1×10^{-6} never trigger action, but a level above 3×10^{-4} always triggered action. For smaller populations regulatory action never resulted for individuals at an incremental risk level below 1×10^{-4} .

These trends in regulatory policy have led a number of states, such as California, to select 1×10^{-5} to constitute the no significant risk level for potentially carcinogenic chemicals (22 CCR Section 12711). This risk level is appropriate for the calculation of ambient water quality criteria.

5.4.4 Summary

The important points regarding the development of ambient water quality criteria for EDC in public water supplies and recreational waters are listed below.

- EDC is considered a probable human carcinogen.

6.0 1,1,2,2-TETRACHLOROETHANE

6.1 Environmental Fate of 1,1,2,2-Tetrachloroethane

6.1.1 Physical and Chemical Properties of 1,1,2,2-Tetrachloroethane

Some of the important physical and chemical properties of 1,1,2,2-tetrachloroethane are presented in Table 3-1. An understanding of physical and chemical properties of a chemical is essential in determining how the chemical will be partitioned in aquatic systems.

A solubility in water of 2,870 mg/L (Riddick et al., 1986) indicates that 1,1,2,2,-tetrachloroethane is very soluble in water. The hydrophilic character of the chemical is also reflected in its low soil-organic carbon partition coefficient. Tetrachloroethane's high water solubility and low soil-organic carbon coefficient indicate that the chemical will primarily dissolve into the water and very little will be sequestered in the sediment of aquatic systems.

The vapor pressure and Henry's Law Constant values listed in Table 3-1 indicate that 1,1,2,2-tetrachloroethane in water is moderately volatile. The volatilization half-life of the chemical in a model river 1 m deep flowing 1 m/sec with a wind of 3 m/sec was estimated by Lyman et al. (1982) to be 6.8 hours. In such cases, both the current and wind speed,

as well as the soil-organic carbon partition coefficient, may effect the rate of volatilization; thus, the half-life in a lake or pond may be much longer.

1,1,2,2-tetrachloroethane has a relatively low octanol-water partitioning coefficient (K_{ow}) and is not expected to be accumulated by aquatic organisms. The bioaccumulation of this chemical is addressed in Section 6.2.

6.1.2 Fate of 1,1,2,2-Tetrachloroethane in Aquatic Systems

In this section, the transport and transformation of 1,1,2,2-tetrachloroethane in water and sediment are described.

1,1,2,2-tetrachloroethane does not readily adsorb to sediment, as indicated by its K_{oc} value, and would be expected to readily volatilize from surface water.

1,1,2,2-tetrachloroethane undergoes base-catalyzed hydrolysis in water at environmental pHs to form trichloroethane (Cooper et al., 1987; Haag and Mill, 1988). Half-lives ranged from 6.6 hours to 102 days depending on pH and ionic strength (Cooper et al., 1987; Haag and Mill, 1988). At ppm levels, 1,1,2,2-tetrachloroethane was found to undergo hydrolytic dehydrodehalogenation to trichloroethylene (25% in 28 days) in a sterile,

anaerobic solution at pH 7 (Klecka and Gonsior, 1983). Under simulated anaerobic conditions, there was a 97% removal rate of the chemical after four months (Bouwer and McCarty, 1983). This indicates that the half-life of 1,1,2,2-tetrachloroethane is variable depending upon environmental conditions such as pH, organic matter content and dissolved oxygen concentration.

6.1.3 Fate Summary

- 1,1,2,2-tetrachloroethane is highly soluble in water.
- 1,1,2,2-tetrachloroethane does not adsorb readily to sediment.
- 1,1,2,2-tetrachloroethane in water and adsorbed to sediment is not expected to persist based upon its susceptibility to chemical degradation.
- 1,1,2,2-tetrachloroethane is hydrophilic and not readily bioaccumulated.

6.2 Bioaccumulation of 1,1,2,2-Tetrachloroethane

6.2.1 Bioaccumulation

Bioaccumulation is the uptake of chemicals by organisms via water and food. Bioaccumulation occurs only when the rate of uptake in an organism exceeds its rate of elimination.

6.2.1.1 Bioconcentration factor

The bioconcentration factor (BCF) is the aggregate uptake for the chemical via exposures to both food and water. The reported values for BCF of 1,1,2,2-tetrachloroethane in fish are 5 (USEPA, 1980c), 8 (Barrows et al., 1980) and 10 (Kawasaki, 1980). Bioconcentration in fish is only considered to be significant when chemicals have BCF values greater than 100. Therefore, these results indicate that there is little tendency for 1,1,2,2-tetrachloroethane to bioaccumulate in fish and aquatic organisms.

6.2.1.2 Tissue Residues

The half-life of 1,1,2,2-tetrachloroethane in fish tissue was reported as less than 1 day. (Barrows et al., 1980).

6.2.3 Summary

The following summarizes the bioaccumulation and aquatic toxicity of 1,1,2,2-tetrachloroethane:

- Bioaccumulation of 1,2,2-Tetrachloroethane in aquatic organisms is not expected to be a significant route of 1,1,2,2-tetrachloroethane exposure to humans.
- 1,1,2,2-tetrachloroethane does not readily bioaccumulate

- 1,1,2,2-Tetrachloroethane is rapidly eliminated from the tissues of fish upon removal of the fish from the contamination source.

6.3 Human Health Effects Associated with 1,1,2,2-Tetrachloroethane

Exposure

6.3.1 Pharmacodynamics

Pharmacodynamics is the study of the fate of a chemical within the body. The discussion of pharmacodynamics is divided into four parts: gastrointestinal absorption, distribution, metabolism, and excretion.

6.3.1.1 Gastrointestinal Absorption

The gastrointestinal absorption of 1,1,2,2-tetrachloroethane in humans has not been quantified (ATSDR, 1988a). Rats and mice that received the chemical orally, absorbed most of the dose (no details reported) (Milman et al., 1984). In another study, rats and mice given 1,1,2,2-tetrachloroethane orally, metabolized 70% of the dose within 48 hours, indicating absorption of at least 70% (Mitoma et al., 1985).

6.3.1.2 Distribution

There is a high level of hepatic protein binding in mice and rats after an oral dose of 1,1,2,2-tetrachloroethane (Mitoma et al., 1985). Since the liver is the primary target organ for 1,1,2,2-tetrachloroethane, it is reasonable to assume that the liver receives a significant portion of the absorbed dose (ATSDR, 1988a).

6.3.1.3 Metabolism

There is no information regarding the metabolism of 1,1,2,2-tetrachloroethane in humans (ATSDR, 1988a). Approximately 80% of the administered dose is metabolized and excreted in 48-72 hours following oral or intraperitoneal administration in both rats and mice (Mitoma et al., 1985; Yllner, 1971). 1,1,2,2-tetrachloroethane is metabolized to trichloroethanol, trichloroacetic acid, and dichloroacetic acid, which is then broken down to glyoxylic acid and oxalic acid (Yllner, 1971; Mitoma et al., 1985; Ikeda and Ohtsuji, 1972). Non-enzymatic degradation of the chemical was thought to occur via dehydrochlorination by alkali to form trichloroethylene and tetrachloroethylene (Yllner 1971). There are data from an in vitro study which suggest that the formation of these compounds from 1,1,2,2-tetrachloroethane may be both enzymatic and non-enzymatic (Koizumi et al., 1982).

6.3.1.4 Excretion

Following inhalation exposure by human volunteers, 3% of the inhaled 1,1,2,2-tetrachloroethane was excreted in exhaled air. The measured urinary excretion rate was 0.015%/min (Morgan et al., 1970).

The most comprehensive study on the metabolism and excretion of 1,1,2,2-tetrachloroethane was an intraperitoneal study in mice. In this study, approximately 4% of the dose was expired unchanged in the breath, 47% was expired as CO₂, 28% was excreted in the urine, 1% was in the feces, and 16% remained in the carcass (Yllner, 1971).

6.3.2 Carcinogenicity of 1,1,2,2-Tetrachloroethane

A weak correlation between exposure to 1,1,2,2-tetrachloroethane and the subsequent development of genital tumors and leukemia was shown following an epidemiological study by Norman et al. (1981). However, the authors believed that no definite conclusions could be drawn from the study due to other uncontrolled factors.

1,1,2,2-tetrachloroethane was shown to cause liver tumors (hepatocellular carcinomas) in both male and female B6C3F1 hybrid mice following oral exposure of 142 mg/kg/day for 78 weeks. In a similar study on Osborne-Mendel rats, no significant increase in tumors for any tumor type in

either sex was reported. However, two male rats at the high dose level (108 mg/kg/day) developed with hepatocellular carcinomas (NCI 1978b).

Since the evidence for carcinogenicity in animals is restricted to mice, and since the information for humans is inconclusive, the USEPA has classified 1,1,2,2-tetrachloroethane as a Group C compound, a possible human carcinogen (USEPA, 1988b). The International Agency for Research on Cancer (IARC) has stated that 1,1,2,2-tetrachloroethane is not classifiable as to carcinogenicity in humans (IARC 1987).

The induction of hepatocellular carcinomas in female B6C3F1 mice (NCI, 1978b) was the basis for the carcinogenic potency calculation for 1,1,2,2-tetrachloroethane. The carcinogenic potency factor for oral exposure to 1,1,2,2-tetrachloroethane reported by the USEPA is 0.2 (mg/kg/day)⁻¹. This value was published in the USEPA's Ambient Water Quality Criteria for Chlorinated Ethanes (USEPA, 1980c) and is reported in the current (July, 1989) Health Effects Assessment Summary Tables and User's Guide (USEPA, 1989b).

6.3.3 Human Health Effects Summary

The following factors were identified as keys to understanding the adverse health effects which may result from 1,1,2,2-tetrachloroethane exposures:

- This chemical is readily absorbed from the gastrointestinal tract following ingestion.
- 1,1,2,2-tetrachloroethane is classified as Group C, possible human carcinogen with an USEPA carcinogenic potency factor of 0.2 (mg/kg/day)⁻¹.

6.4 Critique of the Proposed Louisiana Ambient Water Quality Criteria for 1,1,2,2-Tetrachloroethane.

The ambient water quality criteria proposed by the Louisiana DEQ for 1,1,2,2-tetrachloroethane in public water supplies and recreational waters are 0.16 g/L and 1.8 g/L, respectively (Table 3-2). The primary route of human exposure to waterborne 1,1,2,2-tetrachloroethane is through water consumption, therefore, the risk assessment is primarily driven by the water consumption rate.

6.4.1 Accuracy Check

The proposed criteria are consistent with described DEQ methods and were accurately calculated. The potency factor and BCF used by DEQ in their calculation are up to date values reported by the USEPA as of July 1989 (USEPA, 1989b).

6.4.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed EDC criteria are essentially equivalent to the USEPA criteria for EDC in public water supplies (Table 3-1). However, the DEQ criterion for 1,1,2,2-tetrachlorethane in recreational waters is significantly more stringent than the USEPA criterion. The difference in the recreational criteria is due to the fact that the DEQ used a 20 g/day fish consumption rate versus the 6.5 g/day rate used by the USEPA, and DEQ included an incidental water ingestion rate of 0.089 L/day for swimming. The combined influence of these two factors was sufficient to significantly reduce the value of the proposed DEQ recreational water criterion for 1,1,2,2-tetrachloroethane.

6.4.3 Alternative 1,1,2,2-Tetrachloroethane Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for 1,1,2,2-tetrachloroethane. The refined criteria are significantly less stringent than the DEQ criteria for both public water supplies and recreational waters (Table 3-2). These criteria, however, are still protective of human health from chemical uptake associated with the consumption of drinking water, ingestion of fish and incidental ingestion of water during swimming activities. The water consumption rates are the controlling factors, and both the USEPA and DEQ criteria use a water consumption rate of 2.0 L/day which

approximates the national, 90th percentile drinking water consumption rate. The refined method used an average water consumption rate of 1.4 L/day reported by the USEPA (1989a). Secondly, DEQ used an incidental water ingestion rate for swimming that is 36 times greater than the incidental ingestion rate calculated using parameters reported by the USEPA (1988).

The refined method employed a gastrointestinal absorption efficiency of 100% based on scientific data. It is assumed, based on consumer data, that the fraction of fish consumed from recreational sources is 10% of the total amount of fish consumed by the average person (USDA, 1985). The criteria are calculated at a de minimus risk level of 10^{-5} based on trends in regulatory action (Travis *et al.*, 1987). These factors result in appropriate 1,1,2,2,-tetrachlorethane criteria concentrations of 2.48 g/L and 280 g/L for public water supplies and recreational water, respectively (Table 3-2). The values are approximately 15 and 155 times the proposed Louisiana Criteria for the two scenarios, respectively.

6.4.3.1 Fraction From the Contaminated Source

The DEQ and USEPA assume that all of the water, aquatic organisms and water ingested during swimming activities originate from a contaminated source. This assumption is highly unlikely. For example, in the case of fish consumption, a national market exists in the United States that

provides consumers with food resources from a variety of regions and in effect dilutes the potential for the consumption of a particular contaminant. National survey data indicate that freshwater finfish constitute only approximately 10% of total fish consumption in the United States (USDA, 1985). Most of the fish consumed in the United States is from marine resources, and therefore, uncontaminated by localized chemical releases into freshwater. In addition it is likely that only a small fraction of the freshwater fish in the human diet is caught from contaminated waters. That small fraction of catch is likely limited to recreational fishermen and not the general population.

To assume that 100% of the fish consumed by the people of Louisiana are from contaminated sources is unreasonable. The assumption of a 10% fraction from contaminated sources is still very conservative and represent the entire fraction of freshwater finfish consumed in the average diet.

6.4.3.2 Gastrointestinal Absorption Efficiency

The DEQ and the USEPA assume the gastrointestinal absorption efficiency is 100%. Based on scientific data, this assumption appears to be valid. However, it should be noted that this compound is volatile and is not prone to bioaccumulate, therefore, the actual concentration likely to be absorbed by a human through ingestion is very small.

6.4.3.3 Acceptable Risk

Based on studies by Travis et. al. (1987), it was determined that for carcinogenic chemicals with lifetime risks above 4×10^{-3} received regulation and those with risks below 1×10^{-6} remained unregulated. In addition, for effects resulting from exposures to the entire U. S. population, a risk level below 1×10^{-6} never trigger action, but a level above 3×10^{-4} always triggered action. For smaller populations regulatory action never resulted for individuals at an incremental risk level below 1×10^{-4} .

These trends in regulatory policy have led a number of states, such as California, to select 1×10^{-5} to constitute the no significant risk level for potentially carcinogenic chemicals (22 CCR Section 12711). This risk level represents the median level calculated by the USEPA and is appropriate for the calculation of ambient water quality criteria.

6.4.4 Summary

The important points regarding the development of ambient water quality criteria for 1,1,2,2-tetrachloroethane in public water supplies and recreational waters are listed below.

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- 1,1,2,2-tetrachloroethane is considered a probable human carcinogen.
- 1,1,2,2-tetrachloroethane is water soluble, but is readily lost from aquatic system via volatilization.
- 1,1,2,2-tetrachloroethane does not bioaccumulate significantly in aquatic organisms and only 1.6% of waterborne exposure to humans is attributable to the consumption of aquatic organisms.
- The gastrointestinal absorption efficiency is expected to be nearly 100%.
- The USEPA and DEQ assume that 100% of the fish and water consumed is derived from a contaminated source, which may significantly overestimates the fraction of the environmental concentration that is received by a consumer. Only a small fraction, perhaps 10%, of the fish consumed in Louisiana, the fraction from recreational catch in freshwater near sources of chemicals, may be significantly contaminated.
- The criteria concentrations calculated for 1,1,2,2-tetrachloroethane in public water supplies and recreational waters using refined, scientifically based parameter values at the 10^{-5} de minimus risk level are significantly less stringent than the DEQ criteria. The difference are factors of approximately 15 and 155 for public water supplies and recreational waters, respectively.

7.0 SUMMARY CRITERIA EVALUATION AND TOXICITY PROFILES FOR ADDITIONAL
CHLORINATED COMPOUNDS

This section briefly describes the environmental fate, transport, and toxicity of other chlorinated compounds of interest, with the primary focus on the proposed DEQ water quality criteria.

7.1 Chlorinated Ethanes

In this section, the characteristics and proposed DEQ criteria for 1,1,1-trichloromethane (1,1,1-TCA) and 1,1,2-trichloromethane (1,1,2-TCA) are examined. Sorption to sediment, bioaccumulation and biodegradation are of minor importance to the fate of the chlorinated ethenes in aquatic systems. Instead, the high vapor pressure of these chlorinated ethanes tend to allow for extensive volatilization. Once airborne, these compounds are degraded by photo-oxidation.

As a group, the chlorinated ethanes are considered a low acute hazard to animals based on the results of acute bioassays with 1,1,1-TCA and 1,1,2-TCA. There is no evidence that 1,1,2-TCA is carcinogenic in humans, but there is limited evidence that it is carcinogenic in animals. In contrast, there is no evidence that 1,1,1-TCA is carcinogenic in either humans or animals.

The next section reviews the methods and assumptions developed in the DEQ criteria and suggests refinements based on the available scientific information. Section 7.1.2 provides a toxicological profile for 1,1,1-TCA and 1,1,2-TCA.

7.1.1 Criteria Evaluation for Chlorinated Ethanes

The DEQ proposed ambient water quality criteria for four chlorinated ethanes for both public water supplies and recreational waters. EDC (1,2,-dichloroethane) and 1,1,2,2-tetrachloroethane were discussed in Sections 5.0 and 6.0, respectively. Comments on the criteria proposed for 1,1,1-trichloromethane (1,1,1-TCA) and 1,1,2-trichloromethane (1,1,2-TCA) are summarized below. The criteria proposed for 1,1,1-TCA and 1,1,2-TCA for public water supplies and recreational waters are presented in Table 7-1. The criteria for the two designated uses are significantly different due to the fact that greater than 90% of the waterborne exposure to humans for chlorinated ethanes is likely to occur through water consumption.

It should be noted that 1,1,1-TCA is not considered a carcinogen, and therefore, the equations for non-carcinogens apply.

TABLE 7-1

COMPARISON OF PUBLIC WATER SUPPLY (PWS) AND RECREATIONAL CRITERIA CALCULATED BY
EPA, LA DEQ AT THE 10^{-6} RISK LEVEL, AND REFINED METHOD AT THE 10^{-5} RISK LEVEL

	EPA PWS ^a	USEPA Rec. ^b	LA DEQ PWS ^a	LA DEQ Rec. ^c	Refined Criteria PWS ^d	Refined Criteria Rec. ^c
Chloroform	5.67 µg/L	471 µg/L	5.3 µg/L	70.0 µg/L	81.4 µg/L	1.15 mg/L
Carbon Tetrachloride	0.25 µg/L	4.42 µg/L	0.22 µg/L	1.2 µg/L	3.74 µg/L	135 µg/L
1,1,1-Trichloroethane	30.9 mg/L	1730 mg/L	2.86 mg/L	31.34 mg/L	44.5 mg/L	550 mg/L
1,1,2-Trichloroethane	0.6 µg/L	41.8 µg/L	0.56 µg/L	6.9 µg/L	8.70 µg/L	1,068 µg/L
1,1-Dichloroethylene	0.033 µg/L	1.85 µg/L	0.05 µg/L	0.58 µg/L	0.82 µg/L	85 µg/L
Trichloroethylene	2.7 µg/L	80.7 µg/L	2.8 µg/L	21 µg/L	44.7 µg/L	2,685 µg/L
Tetrachloroethylene	0.8 µg/L	8.85 µg/L	0.65 µg/L	2.5 µg/L	9.38 µg/L	215 µg/L
Vinyl Chloride	0.015 µg/L	4.0 µg/L	1.9 µg/L	35.8 µg/L	0.22 µg/L	62.9 µg/L
Bromoform	6,800 µg/L	250 mg/L	5.1 µg/L	45 µg/L	8.89 mg/L	80 mg/L
Bromodichloromethane	0.26 µg/L	22.1 µg/L	5.3 µg/L	70.0 µg/L	3.8 µg/L	538 µg/L
Methylene Chloride	4.6 µg/L	1,578 µg/L	4.4 µg/L	87 µg/L	66.0 µg/L	21.6 mg/L
Methyl Chloride	2.6 µg/L	221 µg/L	5.3 µg/L	70.0 µg/L	38.2 µg/L	5385 µg/L
Dibromochloromethane	0.41 µg/L	34.2 mg/L	5.3 µg/L	70.0 µg/L	5.9 µg/L	8.33 mg/L

a) Fish and water ingestion

b) Fish ingestion only

c) Fish and incidental water ingestion

d) Fish, water, and incidental water ingestion

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7.1.1.1 Accuracy Check

The proposed criteria are consistent with the DEQ methods presented in Section 2.2. The potency factor and BCF used by DEQ for 1,1,2-TCA are the same as currently reported by USEPA as of July 1989 (USEPA, 1986). However, the RfD used by DEQ for 1,1,1-TCA calculations is 0.09 (mg/kg/day), and the RfD reported by the USEPA in July 1989 is 0.9 (mg/kg/day) (USEPA, 1986). The use of DEQ's figure produces significantly more stringent criteria.

7.1.1.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed criteria for chlorinated ethanes are significantly more stringent than the USEPA guideline criteria for 1,1,1-TCA (Table 7-1). The DEQ criteria for 1,1,2-TCA were not significantly different from the USEPA criteria for public water supplies, but were significantly more stringent than those for recreational waters. The USEPA criteria were calculated using a smaller fish consumption rate of 6.5 g/day versus 20 g/day. Secondly, the USEPA criteria calculation does not consider incidental ingestion during swimming.

7.1.1.3 Alternative Chlorinated Ethane Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for the chlorinated ethanes. The refined criteria are significantly less stringent than the proposed DEQ criteria, while still protective of human health from chemical uptake from the consumption of drinking water, ingestion of fish and incidental ingestion of water during swimming activities. Both USEPA and DEQ methods fail to take in to account scientific data on gastrointestinal absorption efficiency and the small fraction of contaminated fish in proportion to the total quantity of fish likely consumed.

The refined method employed a gastrointestinal absorption efficiency of 100% based on scientific data. It is assumed, based on consumer data, that the fraction of fish consumed from recreational sources is 10% of the total amount of fish consumed by the average person. The criteria are calculated at a de minimus risk level of 10^{-5} based on regulatory policy. The criteria for chlorinated ethanes is driven by the water consumption rates, which were based on the national average water consumption rate of 1.4 L/day, and the incidental ingestion rate for swimming activities of 0.0025 L/day. These factors result in appropriate 1,1,1-TCA criteria concentrations of 44.5 mg/L and 550 mg/L for public water supplies and

recreational waters, respectively. The criteria concentrations for 1,1,2-TCA in public water supplies and recreational waters are 8.70 $\mu\text{g/L}$ and 1068 $\mu\text{g/L}$, respectively.

7.1.1.4 Fraction From the Contaminated Source

One of the conservative assumptions employed by the DEQ is that all of the water, aquatic organisms and water ingested during swimming activities is obtained from a contaminated source. This is highly unlikely, for example in the case of fish, the national market that exists in the United States provides consumers with food resources from a variety of regions and in effect dilutes the potential for the consumption of a particular contaminant. Secondly, contamination at levels that may pose a health hazard are generally isolated water sources, and the likelihood that a person would derive all of the fish consumed from such a water source is very remote.

7.1.1.5 Gastrointestinal Absorption Efficiency

The DEQ and the USEPA assume the gastrointestinal absorption efficiency is 100%. Based on scientific data this assumption appears to be valid.

7.1.1.6 Summary

The important points regarding the development of ambient water quality criteria for chlorinated ethanes in public water supplies and recreational waters are listed below.

- The criteria concentrations are driven by the water consumption rates due to the solubility and low BCF of the compounds. However, the fact that volatilization is not considered causes the equations to significantly overestimate the dose that a human may receive.
- 1,1,2-TCA is considered a probable human carcinogen, and 1,1,1-TCA is considered a non-carcinogen for water quality criteria calculation purposes.
- Chlorinated ethanes are soluble in water and are expected to volatilize rapidly.
- Gastrointestinal absorption efficiency for chlorinated ethanes is reported to be nearly 100%.
- The USEPA and DEQ assume that 100% of the fish and water consumed is derived from a contaminated source. This assumption significantly overestimates the fraction of the environmental concentration that is received by a human. Only a small fraction of the fish consumed in Louisiana, the fraction from recreational catch, may be significantly contaminated.
- The criteria concentrations calculated for 1,1,1-TCA using refined scientifically based parameter values are greater than the DEQ criteria by a factor of approximately 17.
- The criteria concentrations calculated for 1,1,2-TCA using refined, scientifically based parameter values at the 10^{-5} de minimus risk level are approximately a factor of 100 greater than the DEQ criteria.

7.1.2 Toxicological Profiles for Chlorinated Ethanes

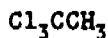
7.1.2.1 1,1,1-Trichloroethane

SYNONYMS:

Methyl chloroform

TCA

STRUCTURE:



TOXICITY SUMMARY:

TCA is relatively non-toxic. At vapor exposures of 250 ppm and more, irritation of the respiratory tract, narcosis, and mild organ pathology are the major toxic effects seen. When pathology is mild, the effects are reversible. A retrospective epidemiological study among workers manufacturing TCA found no association between exposure and any health effect (Krammer et al., 1978).

ACUTE TOXICITY:

At high exposures, TCA acts as a narcotic on the nervous system. Mild eye irritation occurs at 1000-1100 ppm, throat irritation at 1900-2000 ppm,

lightheadedness at 2600 ppm and inability to stand at 2650 ppm. TCA is very low in toxicity having an oral LD₅₀ of 6 to 12 g/kg in rabbits, mice, rats and guinea pigs.

CHRONIC TOXICITY:

No toxicity was observed in rats dosed at 1750 ppm for 6 hours per day and 5 days per week (Rampy et al., 1978). Another inhalation study resulted in no adverse effects in mice exposed to 1500 ppm for 6 hours per day and 5 days per week for two years (Quast et al., 1985). No valid chronic bioassays are available by the oral route for TCA. However, little difference in toxicity between the oral and inhalation routes of exposure have been observed.

CARCINOGENICITY:

Maltoni et al. (1986) administered 500 mg/kg/day of TCA in olive oil at a single dose 4-5 days per week for 104 weeks. TCA had no effect on mortality or body weight, however Maltoni et al. (1986) reported excess leukemia in the rats exposed to TCA (13/100 compared to 4/80). This result is in contradiction to the National Cancer Institutes results stating that excess cancers due to TCA are not expected.

MUTAGENICITY:

Most mutagenicity tests of TCA have been negative. However, a few studies using the Ames/Salmonella assay have been weakly positive (Byard, 1987).

DEVELOPMENTAL TOXICITY:

Teratogenic effects were not found in rodents exposed to TCA. No malformations were produced by TCA at exposures up to 30 ppm in drinking water (George et al., 1987).

REPRODUCTIVE TOXICITY:

In general, no reproductive effects were found in studies except for a slight decrease in fetal weight and delayed fetal development in rats exposed in utero to 2100 ppm TCA (York et al., 1982). There was an observed increase in cardiac abnormalities and other visceral abnormalities in rats exposed in utero to 10 ppm TCA in drinking water.

IMMUNOLOGIC TOXICITY:

No information is available for review.

NEUROLOGIC TOXICITY:

No information is available for review.

ENVIRONMENTAL FATE:

Volatilization is the major transport process for removal of 1,1,1-TCA from aquatic systems. In the troposphere, 1,1,1-TCA reacts with hydroxyl radicals, where the principal tropospheric photooxidation product of 1,1,1-TCA is trichloroacetaldehyde, which is oxidized to trichloroacetic acid. It does not appear that either oxidation or hydrolysis are important fate

processes of 1,1,1-TCA in the aquatic environment. Evidence regarding the importance of adsorption, bioaccumulation, and biodegradation is not definitive (USEPA, 1979b).

STANDARDS:

USEPA RfD: 0.9 mg/kg/day chronic oral
 0.30 mg/kg/day chronic inhalation

7.1.2.2 1,1,2-Trichloroethane

STRUCTURE:



ACUTE TOXICITY:

The following toxic doses are available in the literature (ATSDR, 1988d):

• inhalation rat NOEL	- 890 ppm/2 hour
• oral rat LD ₅₀	- 837 mg/kg gavage undiluted
• oral mice LD ₅₀	- 378 mg/kg gavage in water
• inhalation mice LC ₅₀	- 416 ppm
• inhalation rat LC ₅₀	- 1654 ppm
• intraperitoneal rat LD ₅₀	- 938 mg/kg
• intraperitoneal mice LD ₅₀	- 505 mg/kg

CHRONIC TOXICITY:

Adverse effects were observed on livers of rats, mice, guinea pigs and dogs when administered orally or by inhalation. The effects included necrosis, elevated SGPT and SGOT levels, and reduced liver glycogen

content. Kidney damage was reported, but there is only one reliable report available they can be used for evaluation. Wright and Schaffer (1932) found cloudy swelling and congestion in the kidneys of treated dogs.

CARCINOGENICITY:

There is no evidence for carcinogenicity of 1,1,2-TCA in humans. In mice, 1,1,2-TCA is carcinogenic but not in rats. In a study by the National Cancer Institute (NCI) this compound produced significant increases in the incidence of hepatocellular carcinomas and adrenal pheochromocytomas in mice. From the limited evidence in mice, USEPA has classified 1,1,2-TCA in Group C as a possible human carcinogen (ATSDR, 1988d).

MUTAGENICITY:

Mutagenicity assays were negative in *Salmonella typhimurium* and positive for *Saccharomyces cerevisiae*. Although there are negative and positive results, it is evident that this compound does have some genetic effects both in vitro and in vivo. The significance of the effects to humans is not clear, especially since results of in vivo mammalian assays showed species variability (ATSDR, 1988d).

DEVELOPMENTAL TOXICITY:

Adequate studies were unavailable in the literature for review of the developmental toxicity of 1,1,2-TCA in humans or animals following all routes of exposure (ATSDR, 1988d).

REPRODUCTIVE TOXICITY:

Adequate studies were unavailable in the literature for review of the reproductive toxicity of 1,1,2-TCA in humans or animals following inhalation exposure (ATSDR, 1988d).

IMMUNOLOGICAL TOXICITY:

A study by Sanders et al. (1985) reported that significant effects on mouse immune function were found at doses as low as 44 to 46 mg/kg/day in a 90 day study. Humoral immune function and functional activity of the fixed macrophages of the reticuloendothelial system, and macrophage phagocytic activity were all affected. There was a distinct sex difference in mice where males showed a depressed ability to phagocytize sheep red blood cells, and females showed reduced spleen lymphocyte response to lipopolysaccharide and vascular clearance. There is no data regarding the immunotoxicity of 1,1,2-TCA to humans, but data suggest that 1,1,2-TCA may interfere with immune function in animals (ATSDR, 1988d).

NEUROLOGIC TOXICITY:

Anesthesia has been produced in animals through the oral, inhalation and intraperitoneal routes. Central nervous system depression was reported by De Ceaurriz et al. (1981) following inhalation exposure in mice. No data on neurological effects of 1,1,2-TCA in humans were located, but the evidence in animals suggests that this compound may have central nervous depressant effects in humans as well (ATSDR, 1988d).

ENVIRONMENTAL FATE:

Volatilization and subsequent photo-oxidation in the troposphere are probably the primary transport and fate processes for 1,1,2-TCA. Some sorption, bioaccumulation, and biodegradation may occur, but these processes are probably not very important processes in the transport and fate of 1,1,2-TCA (USEPA, 1979b).

STANDARDS:

USEPA P.F.: $5.70 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$ oral/inhalation

7.2 Chlorinated Ethylenes

This section examines the characteristics and proposed DEQ criteria for 1,1-dichloroethylene, tetrachloroethylene, and trichloroethylene. Like the chlorinated ethanes, the chlorinated ethylenes are highly volatile and undergo extensive photodegradation. Trichloroethylene can adsorb to soil and does bioaccumulate to some degree.

Although both tetrachloroethylene and trichloroethylene exhibit low acute toxicity, they have both exhibited positive results in some cancer bioassays. There is little information on the acute toxicity of 1,1-dichloroethylene, but based on limited evidence from animal studies, it is considered a human carcinogen.

A criteria review for 1,1-dichloroethylene, tetrachloroethylene and trichloroethylene is discussed in the next section. The toxicity of these three compounds is summarized in Section 7.2.2.

7.2.1 Criteria Evaluation for Chlorinated Ethylenes

The proposed ambient water quality criteria for 1,1-dichloroethylene, trichloroethylene, and tetrachloroethylene are presented in Table 7-1. The criteria for the two designated uses are significantly different due to the fact that greater than 90% of the water borne exposure to humans for chlorinated ethylenes is likely to occur through water consumption.

7.2.1.1 Accuracy Check

The proposed criteria are consistent with the methods outlined by DEQ.

7.2.1.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed criteria for this group are slightly more stringent than the USEPA guideline criteria (Table 7-1) except for 1,1-dichloroethylene in which the USEPA criteria is 10 times smaller. The USEPA criteria were

calculated using a smaller fish consumption rate of 6.5 kg/day versus 20 kg/day. Secondly, the USEPA criteria calculation does not consider incidental ingestion during swimming.

7.2.1.3 Alternative Chlorinated Ethylene Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for the chlorinated ethylenes which are still protective of human health from chemical exposure due to ingestion of drinking water, fish and incidental water ingestion during swimming.

- A fraction of the total consumption rate was used since it provided a more realistic consumption rate.
- A gastrointestinal absorption rate of 100 % was assumed for the chlorinated ethylenes.
- Both USEPA and DEQ methods fail to take in to account the small fraction of contaminated fish in proportion to the total quantity of fish likely consumed. It is assumed, based on consumer data, that the fraction of fish consumed from recreational sources is 10% of the total amount of fish consumed by the average person.
- The calculated 10^{-5} risk level yields the following refined criteria concentrations:

Public water supplies

1,1-dichloroethylene,	0.82 $\mu\text{g/L}$
trichloroethylene,	44.7 $\mu\text{g/L}$
tetrachloroethylene,	9.38 $\mu\text{g/L}$

Recreational uses

1,1-dichloroethylene,	85 $\mu\text{g/L}$
trichloroethylene,	2685 $\mu\text{g/L}$
tetrachloroethylene,	215 $\mu\text{g/L}$

SUMMARY:

The salient features regarding the development of ambient water quality criteria for the chlorinated ethylenes in public water supplies and recreational waters are listed below.

- The criteria concentrations are driven by the water consumption rates. Volatilization is the primary environmental fate process which is not considered in the criteria calculations.
- Trichloroethylene and tetrachloroethylene are probable human carcinogens, while 1,1-dichloroethylene is a possible human carcinogen.
- The chlorinated ethylenes are very volatile and degrade primarily via photolysis processes. They do not adsorb readily to sediment.
- The gastrointestinal absorption efficiency of this group is assumed to be 100%.
- The criteria concentrations calculated for the chlorinated ethylenes using refined, scientifically-based parameter values are significantly larger than the DEQ values.

7.2.2 Toxicological Profiles for Chlorinated Ethylenes

7.2.2.1 1,1-Dichloroethylene

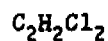
SYNONYMS:

Vinylidene chloride

1,1-Dichloroethene

DCE

STRUCTURE:



ACUTE TOXICITY:

Anaesthesia, hepatotoxicity, and nephrotoxicity are commonly measured indices of acute exposure in experimental animals. The no-effect-levels for experimental animals were estimated at 1000 ppm for up to 1 hour and 200 ppm for up to 8 hours. These values indicate that the 4 hour LC_{50} for rats is 6350 ppm. The target organs are the kidneys and liver regardless of the route of administration (USEPA, 1985c).

CHRONIC TOXICITY:

No relation has been found between chronic occupational exposure and the occurrence of angiosarcoma in rubber plant workers.

CARCINOGENICITY:

There has been no relationship found between occupational exposure and cancer among workers. However, this may be due to the lack of adequate studies available for review. DCE is regarded as an animal carcinogen and a possible human carcinogen. Based on the limited evidence from animal studies, supporting evidence from mutagenicity studies, and related biochemical and toxicity considerations, USEPA recommends that DCE be considered a "possible" carcinogen for humans, or a Group C agent.

Male and female mice exposed by inhalation to DCE for 4 hours/day and 4-5 days/week at concentrations of 0, 10 or 25 ppm for 1 year were observed until spontaneous death occurred. An increase in both malignant and non-malignant tumors were observed. In females, carcinomas of the mammary gland were increased, and in males lung tumors were increased at 10 ppm and in both males and females at 25 ppm. There was also an increase in malignant mammary tumors and leukemia in rats exposed to 100 ppm of DCE, by inhalation 7 hours/day at 5 days/week (Maltoni et al., 1985).

MUTAGENICITY:

A number of studies show that DCE is mutagenic to bacteria, where this activity is dependent on microsomal activity. DCE was found to alkylate the DNA of mice exposed through inhalation and may have caused unscheduled DNA synthesis in the kidneys of similarly exposed mice. USEPA has classed DCE as having insufficient data on either mutagenicity or interaction with germ cells to classify the evidence as either sufficient or suggestive of potential germ-cell mutagenicity (ATSDR, 1988e; USEPA, 1985c).

DEVELOPMENTAL TOXICITY:

DCE has demonstrated weak teratogenic effects (ATSDR, 1988e; USEPA, 1985c). Developmental toxicity was often observed at doses of DCE that also induce maternal toxicity in animals.

The sensitivity of pregnant rats was greater by inhalation than by ingestion. After inhalation exposure at 80 and 160 ppm for 7 hours/day on gestation days 6-18, DCE produced maternal toxicity and increased resorption and skeletal alterations (ATSDR, 1988e).

REPRODUCTIVE TOXICITY:

Rats administered 28 mg/kg/day DCE in drinking water for three generations developed mild dose related hepatotoxic effects. No dose related changes were seen in reproduction or neonatal development (Nitschke et al., 1985).

Reproductive toxicity studies via the inhalation route were inadequate to establish a dose-response relationship due to the limited numbers of animals used (ATSDR, 1988e).

IMMUNOLOGIC TOXICITY:

No studies are available for review regarding immunotoxicity of DCE by either the inhalation, oral or dermal routes (ATSDR, 1988e).

NEUROLOGIC TOXICITY:

Central nervous system toxicity has been observed in humans acutely exposed to high concentrations (approximately 4000 ppm) of inhaled DCE (Tierney et al., 1979). Symptoms include inebriation which may progress to convulsions, spasms, and unconsciousness. Recovery is complete if exposure is not prolonged.

ENVIRONMENTAL FATE:

Little information regarding environmental fate is available for DCE. Upon entering the atmosphere, DCE may interact with oxygen, hydroxyl, and $RO_2^{\cdot}$ radicals and with ozone already present in the atmosphere. The most significant oxidation reaction of DCE in the atmosphere is its reaction with hydroxyl radicals, with the estimated half-life for this reaction being 2 days. The transport of DCE from aquatic media to the atmosphere through volatilization appears to be the primary transport process. In soils, it is predicted through the low log K_{oc}, and high solubility and vapor pressure that volatilization and leaching play a significant role in DCEs fate in soils (USEPA, 1979b).

STANDARDS:

USEPA PF: 0.60 (mg/kg/day)⁻¹ oral
 1.20 (mg/kg/day)⁻¹ inhalation

7.2.2.2 TETRACHLOROETHYLENE

SYNONYMS:

PERCHLOROETHYLENE

TETRACHLOROETHANE

1,1,2,2-TETRACHLOROETHYLENE

STRUCTURE:



ACUTE TOXICITY:

Limited acute toxicity data indicate that tetrachloroethylene is relatively nontoxic by the inhalation and oral routes. A 4-hour LC_{50} of 5200 ppm for mice (Friberg et al., 1953) and a single dose oral LD_{50} of 3005 mg/kg for female rats (Hayes et al., 1986) have been reported.

CHRONIC TOXICITY:

The central nervous system (CNS) and liver appear to be the most sensitive target organs for tetrachloroethylene toxicity. Continuous-inhalation exposure studies have shown that hepatic effects in mice occurred at concentrations as low as 9 ppm (Kellstrand et al., 1984); CNS effects in gerbils occurred at concentrations as low as 60 ppm (Rosengren et al., 1986).

CARCINOGENICITY:

Epidemiological studies suggest a possible association between chronic tetrachloroethylene exposure and increased cancer risk (Blair et al., 1979; Kaplan 1980; Katz and Jowett 1981; Duh and Asal 1984; Brown and Kaplan 1985). However, confounding factors and study limitations make the association inconclusive. Chronic inhalation exposure to tetrachloroethylene produces increased incidences of mononuclear cell

leukemia in rats and hepatocellular adenomas and carcinomas in mice (NTP 1986). Chronic oral exposure to tetrachloroethylene produced increased incidences of hepatocellular carcinomas in mice (NCI 1977).

Tetrachloroethylene has been classified as a possible human carcinogen, Group B2, by the International Agency for Research on Cancer (IARC 1987).

MUTAGENICITY:

The genotoxicity of tetrachloroethylene has been studied using a variety of assays in both in vivo and in vitro test systems. The results of these tests were largely negative. The few positive results were from studies where there were problems related to test compound purity and study design (ATSDR 1987a).

REPRODUCTIVE TOXICITY:

There is little information concerning reproductive effects of tetrachloroethylene. An inhalation study in mice suggests that tetrachloroethylene may cause sperm abnormalities (Beliles et al., 1980).

DEVELOPMENTAL TOXICITY:

Fetotoxic effects, including skeletal ossification anomalies, occurred in mice and rats exposed to greater than or equal to 300 ppm tetrachloroethylene by inhalation, but teratogenic effects were not observed (Schwetz et al., 1975).

IMMUNOLOGIC TOXICITY:

The results of an inhalation study in mice suggest that inhalation of tetrachloroethylene may increase susceptibility of respiratory infection (Aranyi *et al.*, 1986).

ENVIRONMENTAL FATE:

Tetrachloroethylene rapidly volatilizes from water or soil surfaces into the atmosphere where it degrades by reacting with hydroxyl radicals to form phosgene and chloroacetylchlorides (Singh *et al.*, 1975). A relatively long half-life in the atmosphere (~96 days) permits long-range global atmospheric transport of tetrachloroethylene. Tetrachloroethylene is moderately to highly mobile in soil (Swann *et al.*, 1983) and susceptible to significant leaching (Giger *et al.*, 1983; Piet *et al.*, 1981). In subsurface regions where volatilization cannot occur, tetrachloroethylene is only slowly degraded and may be relatively persistent (Bouwer and McCarty 1984, Wilson *et al.*, 1983b).

STANDARDS:

OSHA PEL:	100 ppm
USEPA PF:	$5.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$ oral
	$3.3 \times 10^{-3} \text{ (mg/kg/day)}^{-1}$ inhalation

7.2.2.3 TRICHLOROETHYLENE

SYNONYMS:

TCE

STRUCTURE:



ACUTE TOXICITY:

Trichloroethylene has low acute toxicity; the acute oral LD₅₀ value in several species ranged from 6,000 to 7,000 mg/kg (ATSDR, 1988f).

CHRONIC TOXICITY:

Trichloroethylene has been shown to cause renal toxicity, hepatotoxicity, neurotoxicity, and dermatological reactions in animals following chronic exposure to levels greater than 2,000 mg/m³ for 6 months (ATSDR, 1988f).

CARCINOGENICITY:

Trichloroethylene is carcinogenic to mice after oral administration, producing hepatocellular carcinomas (NCI 1976; NTP 1982). Based on the mouse-liver tumor response, the USEPA has classified TCE as a B2 carcinogen, a probable human carcinogen (USEPA, 1985d).

MUTAGENICITY:

Percocco and Prodi (1981) found positive results for unscheduled DNA synthesis both with and without metabolic activation in human lymphocytes in vivo. USEPA (1985) stated that this study was inconclusive due to the lack of matched controls and the possibility that the incidence of hypodiploid cells was due to preparation of the chromosomes (USEPA, 1985d).

The available genotoxicity data provide suggestive evidence that TCE may be a weakly active indirect mutagen causing effects in a number of different test systems representing a wide evolutionary range of organisms, including humans. The observation that TCE causes adverse effects in the testes of mice also suggests that TCE may cause adverse testicular effects in man (ATSDR, 1988f).

REPRODUCTIVE TOXICITY:

Trichloroethylene does not appear to cause reproductive effects. However, the USEPA reported that a study performed by Land et al., (1979) provided suggestive evidence that high concentrations of TCE can damage early spermatocytes.

DEVELOPMENTAL TOXICITY:

Trichloroethylene does not appear to cause teratogenic effects. Exposure of rats, mice, and rabbits during gestation to levels (300 ppm or 1614

mg/m³) greatly in excess of those generally found in the environment, has not been observed to result in any teratogenic effects (USEPA, 1985d). There have been reports of increased incidences of miscarriages among nurses exposed to various anesthetics including trichloroethylene in the operating room, but specific association of TCE with miscarriages or developmental effects was not established (Corbett et al., 1973, 1974).

IMMUNOLOGIC TOXICITY:

No significant immunologic changes were reported in the studies cited in: USEPA (1985).

ENVIRONMENTAL FATE:

Trichloroethylene rapidly volatilizes into the atmosphere where it reacts with hydroxyl radicals to produce hydrochloric acid, carbon dioxide, carbon monoxide, and carboxylic acid. This is probably the most important transport and fate process for trichloroethylene in aquatic systems and in the upper layer of soil. TCE adsorbs to organic materials and can be bioaccumulated to some degree. However, it is unclear whether trichloroethylene bound to organic material can be degraded by microorganisms or must be desorbed to be destroyed. There is some evidence that higher organisms can metabolize TCE. Trichloroethylene leaches into groundwater fairly readily, and is a common contaminant of groundwater around hazardous waste sites (USEPA, 1979b).

STANDARDS:

USEPA PF: 0.017 (mg/kg/day)⁻¹ inhalation
 0.011 (mg/kg/day)⁻¹ oral

7.3 Trihalomethanes

This section examines the characteristics and proposed DEQ criteria for bromoform, bromodichloromethane, chloroform, and dibromochloromethane. There was too little information available on the toxicity and environmental fate of dibromochloromethane to draw any conclusions about its impact on humans or the environment. Therefore, the following generalizations are based on information from the remaining three halomethanes.

The primary transport of bromodichloromethane and chloroform in the environment is through volatilization. Subsequently, degradation of these compounds is driven by photo-catalyzed reactions. Aquatic species appear to be adversely affected at fairly low concentrations.

Acute toxicity studies indicate that the trihalomethanes are moderately toxic. All three compounds are considered probable human carcinogens on the basis of animal cancer studies. Epidemiological evidence for carcinogenicity in humans is inadequate.

The next section contains a review of the criteria for bromoform, bromodichloromethane, chloroform and dibromochloromethane. Section 7.3.2 contains toxicological profiles for each trihalomethane.

7.3.1 Criteria Evaluation for Trihalomethanes

The DEQ proposed ambient water quality criteria for four trihalomethanes for both public water supplies and recreational waters. In this section, comments on the criteria proposed for bromoform, bromodichloromethane, dibromochloromethane and chloroform are summarized. The criteria proposed for the compounds for public water supplies and recreational waters are presented in Table 7-1. The criteria for the two designated uses are significantly different due to the fact that the majority of the water borne exposure to humans for chlorinated ethanes is likely to occur through water consumption (not considering dermal or inhalation exposure). Bromodichloromethane, dibromochloromethane and chloroform are reported by the USEPA to be probable human carcinogens (B2) and bromoform is considered a non-carcinogen.

7.3.1.1 Accuracy Check

The proposed criteria are consistent with the DEQ methods presented in Section 2.2. The potency factor and BCF used by DEQ for chloroform are the same as currently reported by USEPA as of July 1989 (USEPA, 1989b).

However, the cancer potency factor and BCF used by DEQ for all of the THM's is the same and implies that the chloroform values were used for all four chemicals. The USEPA reported (USEPA, 1989b) cancer potency factors and for bromodichloromethane, and dibromochloromethane (chlorodibromomethane) and late RfD for bromoform are presented in Table 2-1. These are significantly different from those reported by the DEQ. Bromoform has a D designation indicating that it should not be considered a carcinogen. DEQ's regulation of bromoform as a carcinogen makes a considerable difference the calculated are criteria concentration. Bromodichloromethane has a potency factor of 1.3×10^{-1} , which provides a significantly more stringent criterion than proposed by the DEQ. There is some confusion regarding the weight of evidence designation and factors associated with chlorodibromomethane due to apparent typographical errors in the USEPA documents. Based on the information in the July 1989 Health Effects Assessments Summary Tables and User's Guide (USEPA, 1989b) this compound is a B2 level carcinogen and has a potency factor of 8.4×10^{-2} , which provides criteria concentrations significantly more stringent than those proposed by the DEQ.

7.3.1.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed criteria for trihalomethanes are significantly more stringent than the USEPA guideline criteria for chloroform in recreational waters, but are nearly equivalent for public water supplies (Table 7-1). The DEQ

criteria for bromoform were significantly more stringent than the USEPA criteria for both public water supplies and recreational waters due the fact that the DEQ designated bromoform as a B2 carcinogen. The USEPA criteria are more stringent than the proposed DEQ criteria for bromodichloromethane and dibromochloromethane due to updated cancer potency factors for these two compounds.

Two methodological differences may have contributed to the differences in the two agencies's criteria. First, a smaller fish consumption rate of 6.5 g/day was used by the USEPA. Secondly, the USEPA criteria calculation does not consider incidental ingestion during swimming.

7.3.1.3 Alternative Trihalomethane Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for these compounds using appropriate potency factors and RfD's. The refined criteria for chloroform and bromoform are significantly less stringent than the proposed DEQ criteria, while still protective of human health. The criteria for bromodichloromethane and dibromochloromethane are significantly greater than the proposed DEQ criteria for recreational waters, and the criteria for public water supplies are not significantly different, based on the updated cancer potency factors.

The refined method employed a gastrointestinal absorption efficiency of 100% based on scientific data. It is assumed, based on consumer data, that the fraction of fish consumed from recreational sources is 10% of the total amount of fish consumed by the average person. The criteria are calculated at a de minimus risk level of 10^{-5} based on regulatory policy. The criteria for trihalomethanes are driven by the water consumption rates, which were based on the national average water consumption rate of 1.4 L/day, and the incidental ingestion rate for swimming activities of 0.0025 L/day. These factors result in an appropriate criteria concentrations for public water supplies and recreational waters for the above trihalomethanes (Table 7-1).

7.3.1.4 Fraction From the Contaminated Source

One of the conservative assumptions employed by the DEQ is that all of the water, aquatic organisms and water ingested during swimming activities is obtained from a contaminated source. This is highly unlikely, for example in the case of fish, the national market that exists in the United States provides consumers with food resources from a variety of regions and in effect dilutes the potential for the consumption of a particular contaminant. Secondly, contamination at levels that may pose a health hazard are generally isolated water sources, and the likelihood that a person would derive all of the fish consumed from such a water source is very remote.

7.3.1.5 Gastrointestinal Absorption Efficiency

The DEQ and the USEPA assume the gastrointestinal absorption efficiency is 100%. Based on scientific data, this assumption appears to be valid.

7.3.1.6 Summary

The important points regarding the development of ambient water quality criteria for the above trihalomethanes in public water supplies and recreational waters are listed below.

- The criteria concentrations are driven by the water consumption rates due to the solubility and low BCF of the compounds.
- Chloroform, bromodichloromethane and dibromochloromethane are considered probable human carcinogens, and bromoform is considered a non-carcinogen.
- The USEPA and refined criteria for three of the four trihalomethanes are significantly different from the DEQ criteria due to the use of updated cancer potency factors and RfD's.
- Gastrointestinal absorption efficiency for trihalomethanes is assumed to be 100%.
- The USEPA and DEQ assume that 100% of the fish and water consumed is derived from a contaminated source. This assumption significantly overestimates the fraction of the environmental concentration that is received by a human. Only a small fraction of the fish consumed in Louisiana, the fraction from recreational catch, may be significantly contaminated.
- The refined chloroform criteria for recreational waters are greater than the DEQ criteria by a factor of less than 100.

- The refined bromoform criteria for both public water supplies and recreational waters exceed the DEQ criteria by greater than a factor of 1000.
- The refined criteria for bromodichloromethane and dibromochloromethane in recreational waters exceed the DEQ criteria by a factor of approximately 10 and the criteria for public water supplies are nearly equivalent.

7.3.2 Toxicological Profiles for Trihalomethanes

7.3.2.1 Bromoform

SYNONYMS:

Tribromomethane

STRUCTURE:



TOXICITY SUMMARY:

There was very little data on bromoform in the literature that was available for review. For further understanding of the toxicity of trihalomethanes, please refer to the Chloroform Toxicological Profile in Section 7.3.2.3.

ACUTE TOXICITY:

Exposure to bromoform vapor causes irritation of the respiratory tract, pharynx and larynx, as well as lacrimation and salivation (Von Oettingen, 1955). It may be absorbed through the lungs, from the gastrointestinal tract and, to a certain extent, through the skin (Leuze, 1922). Bromoform has been shown to cause liver damage (Graham, 1915). In dogs, exposure to 29,000 ppm caused a deep narcosis after 8 minutes and death after 1 hour (Sax, 1979). The acute subcutaneous LD₅₀ in mice has been reported as 1820 mg/kg (NIOSH, 1977).

ENVIRONMENTAL FATE:

The currently reviewed literature contains insufficient environmental fate information to indicate the aquatic fate of bromoform (USEPA, 1979b).

Bioaccumulation of bromoform in animals is possible due to its partition coefficient. However, there is no information available to verify its occurrence in the environment (USEPA, 1980d).

STANDARDS:

U.S. EPA RfD: 0.2 mg/kg/day chronic oral

7.3.2.2 Bromodichloromethane

STRUCTURE:

CHBrCl_2

TOXICITY SUMMARY:

The liver, kidney and intestine are the principal target tissues of bromodichloromethane. Effects on the central nervous system are the result of large doses. Humans with pre-existing liver and/or kidney conditions may be particularly susceptible.

ACUTE TOXICITY:

Most estimates of acute oral LD_{50} values for bromodichloromethane in rodents range between 400 and 1000 mg/kg. Rats administered a single dose of 390 mg/kg displayed reduced hemoglobin and hematocrit. Bromodichloromethane is carcinogenic in animals studies, and has been found to be mutagenic in some assays.

CHRONIC TOXICITY:

Exposure to 213 mg/kg/day for 90 days caused no effect on lymphocyte levels in the rat. However, after the 90 days, the females showed a slight reduction in lymphocyte count. Rats administered 130 mg/kg/day in the diet for 24 months showed hematological changes compared to the controls (Tobe et al., 1982).

Studies indicate that bromodichloromethane causes liver damage, where typical signs include increased liver weight, pale discoloration, increased levels of hepatic tissue enzymes in serum, decreased levels of secreted hepatic proteins in blood and focal areas of inflammation or degeneration. Subchronic studies have noted effects on the liver at doses as low as 37 mg/kg/day and 50 mg/kg/day on mice and rats. Effects are more pronounced at 125 to 300 mg/kg/day (Condie et al., 1983).

The kidney is susceptible to injury at doses comparable to those that cause liver injury. A chronic exposure of 50 to 100 mg/kg/day may cause increased renal weight, necrosis and cytomegaly of the kidney.

CARCINOGENICITY:

Bromodichloromethane causes the widest spectrum of neoplasms in rats and mice and is the only trihalomethane to cause intestinal tumors (observed in rats, not mice).

Rats exposed to 150 mg/kg/day of bromodichloromethane for 180 weeks showed an increased frequency of liver tumors in females and kidney tumors in males and females administered 100 mg/kg/day (NTP, 1987; Dunnick et al., 1987; Tumasonis et al., 1985).

MUTAGENICITY:

Mice dosed with 50 or 100 mg/kg/day orally exhibited a statistically significant increase in sister chromatic exchanges (Morimoto and Koizumi 1983).

DEVELOPMENTAL TOXICITY:

In a study by Ruddick et al., (1983), there was a reported increase in the incidence of sternebrae and interparietal anomalies in fetuses from rats that had been exposed to doses of 50 mg/kg/day or higher on days 6 to 15 of gestation. Body weight loss of 40% was observed in the mothers demonstrating maternal toxicity.

REPRODUCTIVE TOXICITY:

No studies were available for review regarding the reproductive toxicity of bromodichloromethane in humans or animals following oral or inhalation exposure.

IMMUNOLOGIC TOXICITY:

Although the effects of bromodichloromethane on the immune system have not been thoroughly studied, a study by Munson et al., (1982) found that mice administered doses of 125 to 150 mg/kg/day for 14 days resulted in a decrease in the number of antibody forming cells in the spleen and a decrease in the hemagglutination titer.

NEUROLOGICAL TOXICITY:

Rats and mice often display signs of central nervous system depression when administered oral doses of 150 to 600 mg/kg. The CNS depression tends to be a reversible effect after a couple hours (NTP 1987; Aida et al., 1987; Balster and Borzelleca 1982; Chu et al., 1980). Data suggest that partial tolerance is developed, and that the effects observed are not progressive (Balster and Borzelleca, 1982).

ENVIRONMENTAL FATE:

Bromodichloromethane is a volatile compound that readily evaporates to the air from water. Once in the air, bromodichloromethane undergoes slow oxidation with a half-life of 2-3 months. Volatilization is the major transport mechanism for the chemical. It does not adsorb to soils or sediments very strongly ($\log K_{ow} = 2.1$), and in soil or water may be degraded by microbes (USEPA, 1979b).

STANDARDS:

No current standards are listed by OSHA or ACGIH.

USEPA P.F.: $5.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$ oral

7.3.2.3 Chloroform

SYNONYMS:

Trichloromethane

STRUCTURE:

CHCl_3

ACUTE TOXICITY:

Acute inhalation experiments revealed that a single exposure of 100 ppm was sufficient to produce mild hepatic effects in mice. The exposure level that would produce renal effects is unknown (USEPA, 1985e).

CHRONIC TOXICITY:

Chronic administration by gavage is reported to produce a dose related increase in the incidence of kidney epithelial tumors in rats and a dose related increase in the incidence of hepatocellular carcinomas in mice. No controlled studies have been performed to define dose-response thresholds for neurological or cardiac effects of ingested or inhaled chloroform (USEPA, 1985e).

CARCINOGENICITY:

Animal data from carcinogenicity bioassays show that there are statistically significant increases in renal epithelial tumors in male rats, hepatocellular carcinomas in male and female mice, kidney tumors in male mice, and hepatomas in female mice. Chloroform has been shown to

promote growth and metastasis of murine tumors. In these cancer studies the carcinogenicity of chloroform is organ specific. The primary target organs of acute chloroform toxicity are the liver and kidney, (USEPA, 1985e).

USEPA has classed chloroform as having sufficient animal evidence and inadequate epidemiological evidence, with an overall weight of evidence classification of B2--probably carcinogenic in humans. IARC also classes chloroform as being a probable human carcinogen, with an IARC classification of 2B.

MUTAGENICITY:

No definitive conclusion can be reached concerning the mutagenicity of chloroform. The potential for metabolically activated chloroform to bind to DNA cannot be determined from the available studies, but, if binding to DNA does occur, it would be at a very low level. Evidence from studies measuring binding to macromolecules, DNA damage, and mitotic arrest suggest that chloroform may be mutagenic (USEPA, 1985e).

REPRODUCTIVE TOXICITY:

Chloroform has a much greater effect on developmental toxicity than reproductive toxicity as can be seen by the adverse effects on development in the conceptus versus the maternal toxicity (Schwetz et al., 1974; Murray et al., 1979).

DEVELOPMENTAL TOXICITY:

Chloroform is a potential developmental toxicant. Chloroform has no significant adverse behavioral effect on the fetus and produces embryotoxic effects only at maternally toxic levels (Schwetz et al., 1974; Murray et al., 1979).

IMMUNOLOGIC TOXICITY:

Immunologic toxicity information on chloroform was unavailable for review.

ENVIRONMENTAL FATE:

Volatilization into the atmosphere is the major transport process for removal of chloroform from aquatic systems. Once in the troposphere, chloroform is attacked by hydroxyl radicals with the subsequent formation of phosgene and possibly chlorine oxide radicals. Phosgene is readily hydrolyzed to hydrochloric acid and carbon dioxide. Reaction with hydroxyl radicals is thought to be the primary environmental fate of chloroform. Photolysis, hydrolysis and sorption do not appear to be significant environmental fate processes for chloroform.

STANDARDS:

USEPA PF: 0.081 (mg/kg/day)⁻¹ inhalation
 0.0061 (mg/kg/day)⁻¹ oral

7.3.2.4 Dibromochloromethane

TOXICITY:

There was no information available in the literature.

CARCINOGENICITY:

The USEPA has classed dibromochloromethane as a B2 carcinogen meaning that there is sufficient evidence of carcinogenicity in animals with inadequate evidence in humans (USEPA, 1989b).

ENVIRONMENTAL FATE:

Very little information on physical properties or reaction of dibromochloromethane were found in the literature. Thus, it is not possible to determine the environmental fate of dibromochloromethane (USEPA, 1979a).

STANDARDS:

USEPA PF: $0.084 \text{ (mg/kg/day)}^{-1}$ oral

No current standards are listed by OSHA or ACGIH

7.4 Other Chlorinated Compounds

This section examines the characteristics and proposed DEQ criteria for the remaining chlorinated compounds: carbon tetrachloride, methyl

chloride, methylene chloride, and vinyl chloride. On the basis of their similar physical and chemical properties, and for the sake of convenience, these compounds will be grouped together as "chlorinated compounds".

These chlorinated compounds readily volatilize from soil and surface water. This route represents the primary mode of transport. Degradation processes such as oxidation, hydrolysis, and biodegradation are of minor importance when compared to the attack of photo-catalyzed radicals in the atmosphere. Additionally, the high water solubility and low Koc values typical of this group allow them to leach through soil into groundwater.

Toxicity studies on these compounds have been conducted primarily to assess the hazard from occupational exposure. As a result, most studies have considered only the toxicity of pulmonary absorption, and only a few studies have examined the effects of oral ingestion of these chlorinated compounds. Nonetheless, vinyl chloride is considered a known human carcinogen, while carbon tetrachloride, methyl chloride and methylene chloride are considered probable human carcinogens.

The criteria review for these chlorinated compounds is discussed in the next section. Toxicological profiles are provided in Section 7.4.3.

7.4.1 Criteria Evaluation for other Chlorinated Compounds

In this section comments on the criteria proposed for carbon tetrachloride, vinyl chloride, methylene chloride and methyl chloride are summarized. The criteria proposed for the compounds for public water supplies and recreational waters are presented in Table 7-1. The criteria for the two designated uses are significantly different due to the fact that the majority of the water borne exposure to humans for these chlorinated compounds is likely to occur through water consumption (not considering dermal or inhalation exposure). All four compounds are designated as carcinogens.

7.4.1.1 Accuracy Check

The proposed criteria are consistent with the DEQ methods presented in Section 2.2. The potency factors used by DEQ for carbon tetrachloride and methylene chloride are the same as currently reported by USEPA as of July 1989. However, the cancer potency factors used by DEQ for vinyl chloride and methyl chloride are smaller than those currently reported by the USEPA as of July 1989 (Table 2-1)(USEPA, 1989b).

7.4.1.2 Comparison to USEPA Ambient Water Quality Criteria

The proposed criteria for these compounds vary significantly in comparison to the USEPA guideline criteria for these compounds in public water supplies and recreational waters (Table 7-1). The DEQ criteria for carbon tetrachloride were significantly more stringent than the USEPA criteria for recreational waters and nearly equivalent for public water supplies. For vinyl chloride, the DEQ criteria were significantly less stringent due to the use of the updated USEPA cancer potency factor, which reduced the USEPA criteria concentrations markedly from previously reported values. The methylene chloride criteria for public water supplies are nearly equal, but the USEPA criteria for recreational waters was greater than the DEQ criteria by a factor of 18. The DEQ criteria for methyl chloride is greater than the USEPA criteria for public water supplies, and is significantly more stringent for recreational waters. These differences in criteria comparison are created by the complex relationship each compound exhibits between its cancer potency factor and BCF. Although these chemicals have similar environmental physical and chemical characteristics, the manner in which criteria are derived causes these chemicals to have vastly different criteria concentrations for both public water supplies and recreational waters.

Methodological differences may also contribute to the differences in the two agencies criteria. First, a smaller fish consumption rate of 6.5

g/day was used by the USEPA. Secondly, the USEPA criteria calculation does not consider incidental water ingestion during swimming.

7.4.1.3 Alternative Chlorinated Compound Criteria

Based on the refined methods described in Section 2.3, alternative criteria were calculated for these compounds using the current potency factors. The refined criteria for all four compounds are less stringent than the proposed DEQ criteria for public water supplies and recreational waters, except for vinyl chloride. The DEQ criteria for vinyl chloride in public water supplies exceeds the refined criteria value by a factor of approximately 8 due to the use of the updated cancer potency factor.

The refined method employed a gastrointestinal absorption efficiency of 100% based on scientific data. It is assumed, based on consumer data, that the fraction of fish consumed from recreational sources is 10% of the total amount of fish consumed by the average person. The criteria are calculated at a de minimus risk level of 10^{-5} based on regulatory policy. The criteria for trihalomethanes is driven by the water consumption rates, which were based on the national average water consumption rate of 1.4 L/day, and the incidental ingestion rate for swimming activities of 0.0025 L/day. These factors result in appropriate criteria concentrations for public water supplies and recreational waters for the above chlorinated compounds (Table 7-1).

7.4.1.4 Fraction From the Contaminated Source

One of the conservative assumptions employed by the DEQ is that all of the water, aquatic organisms and water ingested during swimming activities is obtained from a contaminated source. This is highly unlikely. For example in the case of fish, the national market that exists in the United States provides consumers with food resources from a variety of regions and in effect dilutes the potential for the consumption of a particular contaminant. Secondly, contamination at levels that may pose a health hazard are generally associated with local water sources, and the likelihood that a person would derive all of the fish consumed from such a water source is very remote.

7.4.1.5 Gastrointestinal Absorption Efficiency

The DEQ and the USEPA assume the gastrointestinal absorption efficiency is 100%. Based on scientific data, this assumption appears to be valid.

7.4.1.6 Summary

The important points regarding the development of ambient water quality criteria for the above chlorinated compounds in public water supplies and recreational waters are listed below.

- The criteria concentrations are driven by the water consumption rates due to the solubility and low BCF of the compounds. Although the atmospheric and aquatic half-lives of these compounds are varied, volatilization is the primary environmental fate process for all four compounds.
- All of these compounds are considered carcinogens, and vinyl chloride is considered a known human carcinogen.
- The USEPA criteria for vinyl chloride and methyl chloride are different from previously reported values due to the use of updated cancer potency factors. The DEQ criteria were calculated with the out of date potency factors which for both designated water uses were less stringent.
- Gastrointestinal absorption efficiency for these compounds is assumed to be nearly 100%.
- The USEPA and DEQ assume that 100% of the fish and water consumed is derived from a contaminated source. This assumption significantly overestimates the fraction of the environmental concentration that is received by a human. Only a small fraction of the fish consumed in Louisiana, the fraction from recreational catch, may be significantly contaminated.
- The refined recreational criteria are greater than the DEQ criteria by factors ranging from 2 to 250.
- The refined criteria for carbon tetrachloride, methylene chloride and methyl chloride in public water supplies exceed the DEQ criteria by factors ranging from 7 to 17.
- The DEQ criteria for vinyl chloride in public water supplies exceed the refined criteria by a factor of approximately 8.

7.4.2 Toxicological Profiles for Other Chlorinated Compounds

7.4.2.1 Carbon Tetrachloride

STRUCTURE:



ACUTE TOXICITY:

In mice, the estimated LC_{50} for an eight hour exposure is 9500 ppm. An alcoholic exposed to 250 ppm for 15 minutes died, whereas non-alcoholics exposed at the same level for 4 hours only suffered slight headaches.

Pulmonary edema is common among humans exposed to high levels of carbon tetrachloride in the air. However, animals do not demonstrate this effect. Some inhalation studies do not detect any injury to the liver and/or kidney where changes in blood pressure or heart rate are sometimes observed. These effects are probably secondary to either renal effects on fluid and electrolyte retention or to central nervous system effects on the heart or blood vessels (ATSDR, 1988g).

Dermal contact with carbon tetrachloride results in a burning sensation on the skin with mild erythema (Stewart and Dodd, 1964).

CHRONIC TOXICITY:

Nearly all studies of carbon tetrachloride toxicity reveal liver injury. The symptoms of liver injury include clinical signs (jaundice and a

swollen and tender liver), biochemical alterations, or histological examination. In addition, kidney injury is observed in almost all carbon tetrachloride toxicity studies. Symptoms of kidney injury include oliguria or anuria resulting in azotemia and edema which leads to hypertension and pulmonary edema. Other tissues affected by carbon tetrachloride exposure include the adrenals, pancreas, testes, pituitary, spleen, and thyroid (ATSDR, 1988g).

CARCINOGENICITY:

Numerous rodent studies of oral exposure to carbon tetrachloride have resulted in hepatic tumors. IARC (1979a) and USEPA (1988d) have concluded that there is sufficient evidence that carbon tetrachloride is carcinogenic in experimental animals (ATSDR, 1988g). Not enough information is available to establish a cause and effect relationship of carbon tetrachloride and cancer in humans.

MUTAGENICITY:

Most studies of mutagenicity have proved negative in prokaryotic and eukaryotic systems (ATSDR, 1988g). However, genotoxicity was noted in one study in yeast, but only at concentrations of carbon tetrachloride (34mM) considerably above the solubility of carbon tetrachloride in water (5mM) (Callen et al., 1980).

REPRODUCTIVE TOXICITY:

There is no information available for reproductive effects in humans exposed to carbon tetrachloride. No effects were observed in a five generation study where rats were exposed to carbon tetrachloride in food (Alumot et al., 1976).

DEVELOPMENTAL TOXICITY:

There is no information available for developmental effects in humans exposed to carbon tetrachloride by the oral route. In animals, ingestion of carbon tetrachloride at doses of 1400 mg/kg/day during gestation caused maternal toxicity but no teratogenic effects or other adverse effects were observed in the surviving litters (Wilson, 1954).

IMMUNOLOGIC TOXICITY:

No studies are available for review regarding the immunologic toxicity of carbon tetrachloride by either the oral, inhalation or dermal routes (ATSDR, 1988g).

NEUROLOGIC TOXICITY:

Effects to the central nervous system are rapid by the oral and inhalation routes. Characteristics of CNS effects were dyspnea, headache, vertigo, confusion, lethargy and stupor. High concentrations of exposure may cause respiratory depression and cardiac output (ATSDR, 1988g).

ENVIRONMENTAL FATE:

Carbon tetrachloride degrades slowly in the environment, and consequently accumulates. The fate and transport of carbon tetrachloride rests in volatilization followed by photodecomposition in the stratosphere. The rate of oxidation is so slow that the tropospheric half-life is over 330 years. No studies were available for carbon tetrachloride in soils (USEPA, 1979b).

STANDARDS:

USEPA P.F.: 1.30 (mg/kg/day)⁻¹ oral/inhalation

7.4.2.2 Methyl Chloride

SYNONYMS:

Chloromethane

TOXICITY:

There is no available information in the literature.

ENVIRONMENTAL FATE:

Volatilization is the major transport process for removal of methyl chloride from aquatic systems. Once in the troposphere methyl chloride

is attacked by hydroxyl radicals with the subsequent formation of formyl chloride as the principal initial reaction product. Any unreacted methyl chloride reaching the stratosphere will undergo photodissociation.

Oxidation, hydrolysis, and biodegradation are not important fate processes of methyl chloride in the aquatic environment. No information was found indicating that adsorption and bioaccumulation are important environmental transport processes for methyl chloride (USEPA, 1979).

STANDARDS:

USEPA PF: 0.0075 (mg/kg/day) oral
 0.0014 (mg/kg/day) inhalation

7.4.2.3 Methylene Chloride

SYNONYMS:

Dichloromethane

STRUCTURE:



ACUTE TOXICITY:

Neural and behavioral toxicity are commonly measured indices of acute exposure in humans. Decreases in eye and hand coordination have been

observed following a four-hour exposure at 200 ppm (Putz et al., 1976), the lowest concentration reported to have affected behavior (USEPA, 1985f).

CHRONIC TOXICITY:

Toxic encephalosis due to occupational exposure to methylene chloride was diagnosed in a chemist (Weiss, 1967). An individual exposed to methylene chloride for three years developed bilateral temporal lobe degeneration (Barrowcliff, 1978; Barrowcliff and Knell, 1979).

CARCINOGENICITY:

There has been no relationship found between occupational exposure and cancer among workers. However, the available epidemiological studies are not sufficient to classify methylene chloride as a human carcinogen. In an animal oncogenicity study, male rats exposed by inhalation to 1500 or 3000 ppm methylene chloride for two years appeared to have a significant increase in salivary gland sarcomas (Dow Chemical Company, 1980). In a second animal oncogenicity study, male mice exposed to doses ranging from 125 to 250 mg/kg/day in water appeared to have a borderline significant increase in hepatocellular adenomas and/or carcinomas (National Coffee Association, 1983).

Methylene chloride has been classified as a possible human carcinogen, Group 2B, by the International Agency for Research on Cancer (IARC, 1987).

In comparison, USEPA considers methylene chloride to be a probable human carcinogen (USEPA, 1989d).

MUTAGENICITY:

Methylene chloride has been evaluated in a variety of short-term mammalian systems in vitro and in vivo to assess its potential to induce gene mutation and cause chromosomal aberrations and DNA damage and repair (Illing and Shillaker, 1985; CEFIC, 1986). Methylene chloride has been shown to cause mutations in in vitro test systems employing bacteria or yeast. Results were generally negative in vivo.

DEVELOPMENTAL TOXICITY:

Inhalation of methylene chloride at a dose level of 1250 ppm produced a minor skeletal variant in mice (Schwetz et al., 1975). Fetus weight was reduced, and behavioral changes occurred in rat pups following exposure of dams to 4500 ppm methylene chloride (Harding and Manson, 1980). Since these findings were observed at maternally toxic doses and there was only one dose level in each of these studies, there is insufficient evidence to conclusively characterize the developmentally toxic potential of methylene chloride (ATSDR 1987b).

REPRODUCTIVE TOXICITY:

Methylene chloride did not adversely affect reproduction in rats exposed to 100-1500 ppm via inhalation for two generations (Nitschke et al., 1985, unpublished).

IMMUNOLOGIC TOXICITY:

No studies are available for review regarding immunotoxicity of methylene chloride by either the inhalation, oral, or dermal routes (ATSDR 1987b).

NEUROLOGIC TOXICITY:

The major manifestation in human of short-term exposure to methylene chloride is the impairment in the functioning of the central nervous system (CNS). Decreased visual and auditory functions were observed in human volunteers exposed via inhalation to methylene chloride concentrations of 300 ppm or above (Winneke, 1974). CNS impairment was observed in two of three subjects who inhaled methylene chloride vapors (1000 ppm) for 1 to 2 hours (Stewart et al., 1972).

ENVIRONMENTAL FATE:

Volatilization of methylene chloride will occur from all sources and in all environmental media. Accumulation of methylene chloride in the atmosphere is not expected due to degradation by hydroxyl radicals, photolysis, and intermedia transfer via rainout. Volitization is the most

important fate process in surface waters. An estimated half-life of 18 months at 25°C has been reported by the USEPA (USEPA, 1985f). Methylene chloride is moderately soluble in water and leaching from soil to groundwater and surface water are important fate processes.

Biodegradation may be an important fate process, but bioaccumulation and bioconcentration are not considered to be significant factors in aquatic, sediment, and wastewater treatment systems (ATSDR, 1987b).

STANDARDS:

USEPA PF: 7.50×10^{-3} (mg/kg/day)⁻¹ oral
 1.40×10^{-2} (mg/kg/day)⁻¹ inhalation

7.4.2.4 Vinyl Chloride

STRUCTURE:



ACUTE TOXICITY:

The liver and the central nervous system are the important target organs for vinyl chloride exposure. The following toxicity doses were found in the literature (Clement, 1985; Sax, 1984);

- mice LC_{50} - 117 to 500 ppm 2 hour
- rabbits LC_{50} - 230 to 800 ppm 2 hour
- rats LD_{50} oral - 500 mg/kg

After exposure to 1000 ppm for 5 to 9 days, mice demonstrated acute lethality associated with toxic hepatitis and tubular necrosis of the renal cortex (Lee et al., 1977, 1978).

CHRONIC TOXICITY:

People occupationally exposed to high levels of vinyl chloride acquire a syndrome called vinyl chloride disease, which displays signs of toxicity involving the liver, central nervous system (CNS), peripheral circulation and nerves.

A decrease in longevity was seen in mice and rats exposed to 0, 50, 250, or 1000 ppm 6 hour/day, 5 day/week and for 6 months (mice) and 10 months

(rats). After the exposure duration there was a twelve month observation period where a concentration and duration of exposure related decrease in longevity was observed in both species at all exposure concentrations, which was attributed to a combination of systemic toxicity and tumor development (Hong et al., 1981).

CARCINOGENICITY:

There is sufficient evidence to classify vinyl chloride as a known human carcinogen. The incidence of liver cancer, in particular angiosarcoma, provides the most convincing evidence for carcinogenicity of vinyl chloride because the expected background level is extremely low.

Subsequently, the IARC has classed vinyl chloride as a Group 1, and USEPA has classed vinyl chloride in Carcinogen Assessment Group A (ATSDR, 1988b). Both classifications reflect the designation of vinyl chloride as a known human carcinogen.

MUTAGENICITY:

Vinyl chloride causes chromosomal aberrations in lymphocytes in occupationally exposed workers. Positive results were obtained in microorganisms in non-human systems. Such as the recessive lethal test in *Drosophila* and other mammalian test systems.

DEVELOPMENTAL TOXICITY:

An increased incidence of fetal loss has been associated with occupational exposure to vinyl chloride. However, exposures have not been quantified (Infante et al., 1976; Waxweiler et al., 1977).

REPRODUCTIVE TOXICITY:

Insufficient data are available regarding the reproductive toxicity of vinyl chloride. Occupational studies associate effects on sexual and

endocrinological function in men and women and on gynecological health in women with exposure to vinyl chloride. Animal data are limited to one year with intermittent exposures (ATSDR, 1988b).

IMMUNOLOGIC TOXICITY:

No information is available regarding the immunologic toxicity of vinyl chloride to animals and or humans (ATSDR, 1988).

NEUROLOGIC TOXICITY:

Vinyl chloride was once used as an inhalation anesthetic. Acute exposures of 0.8 to 2.0% have been associated with dizziness, giddiness, euphoria, ataxia, headache, and narcosis (Nicholson et al., 1975; Lester et al., 1963).

ENVIRONMENTAL FATE:

When released into the atmosphere, vinyl chloride is expected to be removed by reaction with photochemically generated hydroxyl radicals (half-life = 1.2 to 1.8 days). This reaction produces HCl, formaldehyde, formyl chloride, acetylene, chloroacetaldehyde, chloroacetylchloranil, and chloroethylene. In photochemical smog situations, vinyl chloride has a half-life of 3 to 7 hours. When released to water, volatilization is expected to be the primary fate process (half-life = 8.7 to 43.3 hours). In waters containing photosensitizers, such as humic materials, sensitized

photodegradation may also be important. When released to soil, vinyl chloride will either volatilize rapidly from soil surfaces, or leach readily through soil, ultimately entering groundwater (USEPA, 1979b).

It does not appear that oxidation, hydrolysis, and biodegradation are important fate processes for vinyl chloride in the aquatic environment, nor do sorption and bioaccumulation appear to be important transport processes (USEPA, 1979b).

STANDARDS:

USEPA PF: 0.295 (mg/kg/day)⁻¹ inhalation
 2.30 (mg/kg/day)⁻¹ oral

8.0 CONCLUSIONS

This evaluation of the proposed Louisiana ambient water quality criteria was based on an analysis of the methods, physical and biological data and the assumptions upon which the criteria are founded. Conclusions for the specific chemicals are found at the end of the respective sections. Listed below are the major findings regarding the methods and assumptions employed by the DEQ.

- The equations used by the DEQ are consistent with those recommended by the USEPA, except for DEQs inclusion of an incidental water ingestion rate for swimming activities.
- The criteria were checked for accuracy and were found to be consistent with the methods reported by the DEQ.
- The cancer potency factors and BCF's used by the DEQ for calculating water quality criteria were confirmed for most of the values. However, the cancer potency factors most recently released by the USEPA in July 1989 are not in agreement with five cited by DEQ (see Table 2-1).
- The carcinogenicity weight of evidence category cited by DEQ for bromoform (Group B2, probably human carcinogen) is not consistent with the USEPA designation (Group D, not classified as to carcinogenicity). The USEPA does not regulate bromoform as a carcinogen. The criteria proposed by DEQ for bromoform were calculated using the equations for carcinogens. This approach is inconsistent with federal policy.
- The RfD used by the DEQ for 1,1,1-trichloroethane is not consistent with that reported by the USEPA, and results in proposed criteria significantly more stringent than the USEPA guideline criteria.

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- The criteria proposed by the DEQ for the selected chemicals were consistently more stringent than the USEPA guideline criteria for recreational waters and in many cases public water supplies.
- The DEQ and USEPA criteria were consistently more stringent than the alternative criteria developed using refined risk assessment equations. This is due primarily to the screening level risk assessment approach used by agencies. A screening level approach uses overly conservative assumptions and parameter values which significantly overestimate human exposures.
- The drinking water consumption rate (2.0 L/day) used by the DEQ is reported by the USEPA to be representative of the national 90th percentile water consumption rate rather than an average consumption rate.
- The use of an incidental water ingestion rate for swimming is inconsistent with USEPA methods. The rate used by the DEQ was 36 times greater than the incidental water ingestion rate suggested in USEPA Exposure Factors Handbook.
- The assumption that 100% of the fish and water consumed by the people of Louisiana is obtained from contaminated water sources is not supported by national consumption survey data.
- The assumption of 100% gastrointestinal absorption efficiency is not justified for selected compounds. Evidence for HCB indicates that gastrointestinal absorption efficiency may be as low as 10% for some chemicals.
- The assumption of no loss of contaminants during food preparation is likely inaccurate. There is evidence that indicates that as much as 90% of the PCB's in fish may be lost during food preparation.
- A 10^{-6} de minimus risk level was used by the DEQ, however, past federal and state regulatory practices indicate that a 10^{-5} level of risk may be as appropriate for the determination of water quality criteria.

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- In general, it was concluded that the alternative criteria based on scientific data and USEPA recommended parameters are representative of levels safe for designated water uses, based on a realistic assessment for health risks.

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APPENDIX A

**CRITERIA DOCUMENTATION PREPARED BY THE
LOUISIANA DEPARTMENT OF ENVIRONMENTAL QUALITY**

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LOUISIANA DEPARTMENT OF ENVIRONMENTAL QUALITY
OFFICE OF WATER RESOURCES
WATER POLLUTION CONTROL DIVISION
JUNE, 1989

LIST OF REFERENCES REVIEWED FOR THE 1989 LOUISIANA
WATER QUALITY STANDARDS REVISION

EPA Guidance Documents

1. Water Quality Criteria. 1972. National Academy of Sciences, National Academy of Engineering. Environmental Protection Agency, Ecological Research Series. EPA R3.73:033. Washington D.C. U.S. Government Printing Office. 1974.
2. Quality Criteria for Water. 1976. U.S. EPA. Washington D.C.
3. Ambient Water Quality Criteria. 1980, 1984, 1985. U.S. EPA Series No. 440/5-80-84-85. Washington D.C.
4. Water Quality Standards Handbook. 1983. U.S. EPA. Office of Water.
5. Technical Support Manual: Waterbody Surveys and Assessments for Conducting Use Attainability Analysis. 1983. Office of Water Regulations and Standards.
6. Technical Support Document for Water Quality-based Toxic Control. 1985. U.S. EPA. Office of Water.
7. Questions and Answers On: Antidegradation. 1985. U.S. EPA. Office of Water Regulations and Standards.
8. Quality Criteria for Water. 1986. U.S. EPA. Office of Water Regulations and Standards. EPA 440/5-86-001 with updates.
9. The Risk Assessment Guidelines of 1986. 1987. U.S. EPA. Office of Health and Environmental Assessment. EPA 600/8-87/045.
10. Guidance Manual for Assessing Human Health Risks from Chemical Contaminated Fish and Shellfish. U.S. EPA. Office of Marine and Estuarine Protection: Draft. December 1987.
11. Methods for Aquatic Toxicity Identification Evaluations. 1988. U.S. EPA. Environmental Research Laboratory. Duluth, MN. EPA/600/3-88/034.
12. Introduction to Water Quality Standards. 1988. U.S. EPA. Office of Water Regulations and Standards. EPA 440/5 88-089.
13. Establishment of Ambient Criteria to Limit Human Exposure to Contaminants in Fish and Shellfish. U.S. EPA. Office of Water Regulations and Standards. Draft. January 5, 1989.

14. Integrated Risk Information System (IRIS). U.S. EPA. Office of Health and Environmental Assessment. Office of Research and Development.
15. Integrated Risk Information System (IRIS). U.S. EPA. Office of Health and Environmental Assessment. Office of Research and Development. Chemical Files for Selected Louisiana Toxic Substances.

EPA Regulations, Rules and Notices

1. 45 FR 79318, November 28, 1980. Water Quality Criteria Documents; Availability.
2. 48 FR 51405, November 8, 1983. Water Quality Standards Regulation.
3. 49 FR 9016, March 9, 1984. Development of Water Quality-based Permit Limitations for Toxic Pollutants; National Policy.
4. 51 FR 33992, September 24, 1986. Risk Assessment Guidelines.
5. 52 FR 42522, November 5, 1987. Organic Chemicals and Plastics and Synthetic Fibers Category Effluent Limitations Guidelines, Pretreatment Standards, and New Source Performance Standards.
6. Clean Water Act as amended by the Water Quality Act of 1987, PL 100-4. Section 303.

EPA and Other Miscellaneous Documents

1. Variances in Water Quality Standards. U.S. EPA. Office of Water. Memo to Water Directors. Office of Water Regulations and Standards. March 15, 1985.
2. Draft Framework for the Water Quality Standards Program. U.S. EPA. Office of Water. Office of Water Regulations and Standards. Draft. November, 1988.
3. State Adoption/Proposal of Numeric Criteria for Priority Pollutants as of August 1988. U.S. EPA. Office of Water Regulations and Standards. EPA 440/5 89-002.
4. Agricultural Statistics, 1984. U.S. Department of Agriculture. U.S. Government Printing Office. Table 697, p. 506. Consumption and Family Living.
5. Health Hazards of Primary Contact Recreation at Bayou D' Inde. Louisiana Department of Health and Hospitals.
6. BCF for Hexachlorobutadiene (HCBD). 1989. U.S. Environmental Protection Agency. Memorandum from Nelson A. Thomas ERL-Duluth to Robert B. Elliott. Region VI EPA. April 11, 1989.

LOUISIANA DEPARTMENT OF ENVIRONMENTAL QUALITY
OFFICE OF WATER RESOURCES
WATER POLLUTION CONTROL DIVISION

JUNE, 1989

DOCUMENTATION OF NUMERICAL CRITERIA FOR HUMAN HEALTH PROTECTION
IN THE 1989 WATER QUALITY STANDARDS REVISION

The development of numerical criteria for human health protection follows guidelines established by the Environmental Protection Agency. The basic formulas, as illustrated below, are obtained from 45 FR 79318, November 28, 1980. Further explanation and description of these guidelines can be found in the recently released EPA document, "Establishment of Ambient Criteria to Limit Human Exposure to Contaminants in Fish and Shellfish". Guidance in appropriate application of the guidelines were obtained from both Headquarters and Region VI EPA.

The 1980 federal register notice established the use of 2 liters for average water consumption and the use of 70 kg for an average adult body weight, both of which are utilized in the formulas. The bioconcentration factors (BCF) used in the formulas are obtained from the 1980, 1984 or 1985 EPA Ambient Water Quality Criteria Series for each toxic substance. Both the Cancer Potency Slopes (SF) and Non-carcinogenic Reference Dose (RfD) are obtained from the EPA's Integrated Risk Information System (IRIS). A fish consumption rate of 20 grams per day is used in the formulas and is obtained from the U. S. Department of Agriculture's 1984 National Consumption Statistics as reported in the EPA document, Guidance Manual for Assessing Human health Risks from Chemically Contaminated Fish and Shellfish. As a final step in the formula, a 10^{-6} , one in a million, health risk level has been used for those toxic substances designated as carcinogens and an incidental ingestion consideration is made to provide a basic level of protection while swimming.

The following further clarifies the methodology used for calculating human health criteria.

Toxic Substances are classified as carcinogenic or non-carcinogenic. Those with an EPA cancer classification of A, B1, B2, or C are considered to be carcinogens. This is consistent with EPA human health guidance. Cancer class designations were obtained from the EPA Integrated Risk Information System (IRIS). For waters designated as public water supplies 2 liters is included in the calculation. Two liters is the amount of water an average adult consumes per day and has been standard in EPA human health calculations. For water designated as swimmable, incidental ingestion is included. Incidental ingestion is equal to 0.089 liters/day and based upon the following assumptions modified from the Louisiana Department of Health and Hospitals:

$$\frac{250 \text{ ml (possible ingestion)}}{\text{hr}} \times \frac{5 \text{ hrs (swimming duration)}}{\text{wk}} \times \frac{6 \text{ mos (swimming season)}}{12 \text{ mos}}$$

$$\frac{\times 1 \text{ week}}{7 \text{ days}} = 89 \text{ ml/day} = 0.089 \text{ liters/day incidental ingestion.}$$

The equation used for a carcinogen in waters designated for public water supply is as follows:

$$\text{Criteria (P)} = \frac{(10^{-6})(70 \text{ kg})}{\text{mg/l} \quad \text{SF} [0.089 + 2 \text{ L} + (\text{BCF})(.02 \text{ kg/day})]}$$

where 10^{-6} = risk level
 70 kg = average adult male body weight
 BCF = bioconcentration factor (L/kg)
 0.02 kg/day = national average amount of fish/shellfish consumed daily in kilograms (=20g/day)
 SF = Cancer Potency Slope (mg/kg/day) $^{-1}$ - has been equated to q^1 *

The equation for a carcinogen in waters not designated as public water supplies is as follows:

$$\text{Criteria (N)} = \frac{(10^{-6})(70 \text{ kg})}{\text{mg/l} \quad \text{SF} [0.089 + (\text{BCF})(.02)]}$$

The following equation is for a non-carcinogenic chemical in waterbodies designated as public water supplies:

$$\text{Criteria (P)} = \frac{\text{RfD} \times 70 \text{ kg}}{0.089 + 2 \text{ L} + (\text{BCF})(.02)}$$

where RfD = reference dose (mg/kg/day)

The equation for a non-carcinogen in waters not designated as public water supplies is as follows:

$$\text{Criteria (N)} = \frac{\text{RfD} \times 70 \text{ kg}}{0.089 + (\text{BCF})(.02)}$$

All the cancer potency slopes and reference doses were extracted from the IRIS database except for lindane, PCBs, tetrachloroethylene, vinyl chloride, bromoform, bromodichloromethane, and hexachlorobenzene. These chemicals have not been assessed for IRIS as of Dec. 1, 1988. The parameters necessary for calculating human health criteria for these chemicals were taken from the appropriate ambient water quality criteria documents. Bioconcentration factors used were also extracted from the same criteria documents with the exception of hexachlorobutadiene. A new BCF for hexachlorobutadiene was used as provided by the EPA ORD Laboratory in Duluth.

The following table contains all the necessary information to calculate human health criteria:

HUMAN HEALTH CRITERIA FOR PROPOSED LOUISIANA CHEMICALS

<u>Chemical</u>	<u>BCF</u>	<u>SF or (RfD)</u>	<u>Criteria(P)1</u>	<u>Criteria(N)1</u>	<u>Cancer Group</u>
Aldrin	4,670	17	0.04 ng/l	0.04 ng/l	B2
Chlordane	14,000	1.3	0.19 ng/l	0.19 ng/l	B2
DDT	53,600	0.34	0.19 ng/l	0.19 ng/l	B2
Dieldrin	4,670	16	0.05 ng/l	0.05 ng/l	B2
Endosulfan	270	(0.00005)	0.47 ug/l	0.64 ug/l	--
Endrin	3,970	(0.00003)	0.26 ug/l	0.26 ug/l	--
Heptachlor	11,200	4.5	0.07 ng/l	0.07 ng/l	B2
Lindane	130	1.3	0.011 ug/l	0.02 ug/l	B2
PCBs	31,200	4.3396	0.03 ng/l	0.03 ng/l	B2
Toxaphene	13,100	1.1	0.24 ng/l	0.24 ng/l	B2
Benzene	5.2	0.029	1.1 ug/l	12.5 ug/l	A
Carbon Tetra- chloride	18.75	0.13	0.22 ug/l	1.2 ug/l	B2
Chloroform	3.75	0.0061	5.3 ug/l	70.0 ug/l	B2
Ethylbenzene	37.5	(0.097)	2.39 mg/l	8.1 mg/l	D
1,2,-Dichloro- ethane	1.2	0.091	0.36 ug/l	6.8 ug/l	B2
1,1,1-Trichloro- ethane	5.6	(0.09)	**2.86 mg/l	21.34 mg/l	D
1,1,2-Trichloro- ethane	4.5	0.057	0.56 ug/l	6.9 ug/l	C
1,1,2,2-Tetra- chloroethane	5	0.2	0.16 ug/l	1.8 ug/l	C
1,1-Dichloro- ethylene	5.6	0.6	0.05 ug/l	0.58 ug/l	C

** MCL is lower at 0.2 mg/l

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<u>Chemical</u>	<u>BCF</u>	<u>SF or (RfD)</u>	<u>Criteria(P)1</u>	<u>Criteria(N)1</u>	<u>Cancer Group</u>
Trichloro- ethylene	10.6	0.011	2.8 ug/l	21 ug/l	B2
Tetrachloro- ethylene	30.6	0.039776	0.65 ug/l	2.5 ug/l	B2
Toluene	10.7	(0.3)	9.1 mg/l	69.3 mg/l	D
Vinyl Chloride	1.17	0.0174	1.9 ug/l	35.8 ug/l	A
Bromoform	8.3	0.0061	5.1 ug/l	45 ug/l	B2
Bromodichloro- methane	3.75	0.0061	5.3 ug/l	70.0 ug/l	B2
Methylene Chloride	0.91	0.0075	4.4 ug/l	87 ug/l	B2
Methyl Chloride	3.75	0.0061	5.3 ug/l	70.0 ug/l	B2
Dibromochloro- methane	3.75	0.0061	5.3 ug/l	70.0 ug/l	B2
1,3-Dichloro- propene	2	0.18	0.18 ug/l	3.0 ug/l	B2
2-Chlorophenol	134	(0.005)	73.4 ug/l	126.4 ug/l	--
2,4-Dichloro- phenol	40.7	(0.003)	72.3 ug/l	232.6 ug/l	--
Benzidine	87.5	230	0.08 ng/l	0.17 ng/l	A
Hexachloro- benzene	8,690	1.688	0.24 ng/l	0.24 ng/l	B2
Hexachloro- butadiene	392	0.078	0.09 ug/l	0.11 ug/l	C

(1) Criteria (P) are applicable to waters designated for fishing, swimming, and public water supply. Criteria (N) are applicable to waters not designated for public water supply but designated for fishing and swimming.

LOUISIANA DEPARTMENT OF ENVIRONMENTAL QUALITY
OFFICE OF WATER RESOURCES
WATER POLLUTION CONTROL DIVISION
JUNE, 1989

DOCUMENTATION OF NUMERICAL CRITERIA FOR ACUTE AND CHRONIC
AQUATIC LIFE PROTECTION IN THE 1989 WATER QUALITY STANDARDS REVISION

Numerical criteria for fresh water and marine water aquatic life protection as listed in Table 1 of the proposed 1989 Water Quality Standards revision were derived from criteria documents of the Environmental Protection Agency. Aquatic life criteria for the following toxic substances were taken directly from those recommended in the EPA document Quality Criteria for Water 1986:

- | | |
|--|--|
| 1. Aldrin | 2. Chlordane |
| 3. DDT | 6. Dieldrin |
| 7. Endosulfan | 8. Endrin |
| 9. Heptachlor | 10. Hexachlorocyclohexane (gamma BHC, Lindane) |
| 11. Polychlorinated Biphenyls, Total (PCB's) | 12. Toxaphene |
| 13. 2, 4-Dichlorophenoxyacetic acid (2, 4-D) | 14. 2-(2, 4, 5-Trichlorophenoxy) propionic acid (2, 4, 5-TP, Silvex) |
| 46. Arsenic | 47. Chromium III (Tri) - Freshwater Acute and Chronic only |
| 48. Chromium VI (Hex) | |
| 49. Zinc | |

Numerical criteria for aquatic life protection for the remaining toxic substances were not directly available from EPA and were derived from LC50 data for each toxic substance as presented in the following EPA documents; (1) Ambient Water Quality Criteria, 1980. EPA Series 440/5-80 and (2) Ambient Water Quality Criteria, 1984. EPA Series 440/5-84-85. To derive a criterion value, an application factor was multiplied by the lowest reported LC50 value for a representative Louisiana species as listed in Table 1 of the EPA criteria documents. Application factors used were those recommended in the EPA Water Quality Criteria 1972 (p. 123) and Quality Criteria for Water 1976 (p. 2, 3). This approach was developed in cooperation with Region VI EPA. For nonpersistent or noncumulative toxic substances, an application factor of 0.1 was used for acute protection and 0.05 was used for chronic protection. For persistent or cumulative toxic substances, an application factor of 0.05 was used for acute protection and 0.01 was used for chronic protection. The use of application factors provides a safety consideration to protect all life stages of a test species as well as to protect associated species that have not been tested and may be more sensitive to the tested toxic substance.

The following is a listing of the lowest reported LC50 values and representative Louisiana species utilized to derive numerical criteria.

Toxic Substance	Class ¹	Species ²	LC50 ³
4. TDE (DDD)	P	Scud Oyster	0.6 25
5. DDE	P	Planarian Oyster	1,050 14
15. Benzene	NP	Bluegill <u>P. pugio</u> ⁴	22,490 27,000
16. Carbon Tetrachloride	NP	Bluegill T. Silverside	27,300 150,000
17. Chloroform	NP	<u>Daphnia m.</u> <u>Pink Shrimp</u>	28,900 81,500
18. Ethylbenzene	NP	Bluegill <u>M. bahia</u> ⁵	32,000 87,600
19. 1, 2-Dichloroethane (EDC)	NP	Fathead minnow <u>M. bahia</u>	118,000 113,000
20. 1, 1, 1-Trichloroethane	NP	Fathead minnow <u>M. bahia</u>	52,800 31,200
21. 1, 1, 2-Trichloroethane	NP	<u>Daphnia m.</u> No data for Marine Water Species	18,000
22. 1, 1, 2, 2-Tetrachloroethane	NP	<u>Daphnia m.</u> <u>M. bahia</u>	9,230 9,020
23. 1, 1-Dichloroethylene	NP	<u>Daphnia m.</u> <u>M. bahia</u>	11,600 224,000
24. Trichloroethylene	NP	<u>Daphnia p.</u> <u>P. pugio</u>	39,000 2,000
25. Tetrachloroethylene	NP	<u>Daphnia m.</u> <u>P. pugio</u>	8,500 1,300
26. Toluene	NP	Bluegill <u>P. pugio</u>	12,700 9,500
27. Vinyl Chloride	No Aquatic Toxicity Data Reported		
28. Bromoform	NP	Bluegill Sheepshead minnow	29,300 17,900
29. Bromodichloromethane	No Aquatic Toxicity Data Reported		

Toxic Substance	Class ¹	Species ²	LC50 ³
30. Methylene Chloride	NP	Fathead minnow <u>M. bahia</u>	193,000 256,000
31. Methyl Chloride	NP	Bluegill T. Silverside	550,000 270,000
32. Dibromochloromethane	No Aquatic Toxicity Data Reported		
33. 1, 3-Dichloropropene	NP	Bluegill <u>M. bahia</u>	6,060 790
34. 2-Chlorophenol	NP	<u>Daphnia m.</u> No Data for Marine Water Species	2,580
35. 3-Chlorophenol	No Aquatic Toxicity Data Reported		
36. 4-Chlorophenol	NP	Bluegill Sheepshead minnow	3,830 5,350
37. 2, 3-Dichlorophenol	No Aquatic Toxicity Data Reported		
38. 2, 4-Dichlorophenol	NP	Bluegill No Data for Marine Species	2,020
39. 2, 5-Dichlorophenol	No Aquatic Toxicity Data Reported		
40. 2, 6-Dichlorophenol	No Aquatic Toxicity Data Reported		
41. 3, 4-Dichlorophenol	No Aquatic Toxicity Data Reported		
42. Phenol (total)	NP	<u>Daphnia m.</u> <u>P. pugio</u>	7,000 5,800
43. Benzidine	NP	Red Shiner No Data for Marine Water Species	2,500
44. Hexachlorobenzene	No Aquatic Toxicity Data Reported		
45. Hexachlorobutadiene	P	Fathead Minnow <u>P. pugio</u>	102 32
47. Chromium III	P	- Oyster	10,300

1. P - persistent; application factors - 0.05 (acute), 0.01 (chronic)
NP - nonpersistent; application factors - 0.10 (acute), 0.05 (chronic)
2. First listed species for Freshwater
Second listed species for Marine Water
3. LC 50's reported in ug/L, parts per billion
4. Grass shrimp, Palaemonetes pugio
5. Mysid shrimp, Mysidopsis bahia

4 ppb, and is acutely toxic to fish at 10 ppb. Assume here for simplicity that no upstream sources of this substance are anticipated. Critical flow in the Mississippi River is 111,000 cubic feet per second (cfs). The numerical criteria of 5 ppb and 4 ppb apply outside the mixing zone, and therefore the 4 ppb value, being more stringent, will be used in the effluent limitation calculation. The following calculations then illustrate the limit calculation:

STREAM NAME: MISSISSIPPI RIVER			
CRITICAL STREAM FLOW (CFS):	111000		
FRACTION OF FLOW IN MZ:	0.1	FLOW IN MZ=	11100
FRACTION OF FLOW IN ZID:	0.01	FLOW IN ZID=	1110
UPSTREAM CONCENTRATION (PPB):	0		
EFFLUENT FLOW (CFS):	1110		

NUMERICAL CRITERION (PPB): 4 CHRONIC
 10 ACUTE

EFFLUENT LIMIT (PPB): 44 CHRONIC
 20 ACUTE

— 20 FINAL EFFLUENT LIMIT (PPB)

Note that the acute criterion in this case results in a limit of 20 ppb, and the chronic criterion results in a limit of 44 ppb. In this case, therefore, the effluent limitation is controlled by the more stringent acute aquatic toxicity criteria which must be met at all points outside the zone of initial dilution. In general any of the three numerical criteria cited above may be the controlling factor in determination of effluent limitations.