United States
Environmental Protection
Agency

FINAL DRAFT
ECAO-CIN-G023
November, 1987

Research and Development

HEALTH AND ENVIRONMENTAL EFFECTS DOCUMENT FOR P-CHLOROBENZOIC ACID

Prepared for

OFFICE OF SOLID WASTE AND
EMERGENCY RESPONSE

Prepared by

Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
U.S. Environmental Protection Agency
Cincinnati, OH 45268

DRAFT: DO NOT CITE OR QUOTE

NOTICE

This document is a preliminary draft. It has not been formally released by the U.S. Environmental Protection Agency and should not at this stage be construed to represent Agency policy. It is being circulated for comments on its technical accuracy

DISCLAIMER

This report is an external draft for review purposes only and does not constitute Agency policy. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Documents (HEEDs) are prepared for the Office of Solid Waste and Emergency Response (OSWER). This document series is intended to support listings under the Resource Conservation and Recovery Act (RCRA) as well as to provide health-related limits and goals for emergency and remedial actions under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency Program Office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched for in this document and the dates searched are included in "Appendix: Literature Searched." Literature search material is current up to 8 months previous to the final draft date listed on the front cover. Final draft document dates (front cover) reflect the date the document is sent to the Program Officer (OSWER).

Several quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic and subchronic exposures for both the inhalation and oral exposures. The subchronic or partial lifetime RfD, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval, for example, one that does not constitute a significant portion of the lifespan. This type of exposure estimate has not been extensively used, or rigorously defined as previous risk assessment efforts have focused primarily on lifetime exposure scenarios. Animal data used for subchronic estimates generally reflect exposure durations of 30-90 days. The general methodology for estimating subchronic RfDs is the same as traditionally employed for chronic estimates, except that subchronic data are utilized when available.

In the case of suspected carcinogens, RfDs are not estimated. A carcinogenic potency factor, or q_1^* (U.S. EPA, 1980), is provided instead. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under the CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity, and acute mammalian toxicity). Chemical-specific RQs reflect the lowest of these six primary criteria. The methodology for chronic toxicity and cancer-based RQs are defined in U.S. EPA, 1984 and 1986a, respectively.

EXECUTIVE SUMMARY

p-Chlorobenzoic acid (74-11-3) is a crystalline solid at room temperature (Windholz, 1983). It is soluble in ethanol and ethyl ether but is sparingly soluble in water (Windholz, 1983; Perry and Green, 1984). It may undergo reactions involving the aromatic nucleus or the chlorine substituent, as well as reactions typical of the carboxyl group (Muir, 1963). This compound may be prepared by the oxidation of p-chlorotoluene (Gelfand, 1979; Muir, 1963). The 1987 OPD Chemical Buyers Directory lists eight suppliers of p-chlorobenzoic acid; however, The Directory of Chemical Producers (SRI, 1986) and recent U.S. International Trade Commission publications on chemical production and sales (USITC, 1985, 1986) contain no production data for this compound. The limited amount of production data on p-chlorobenzoic acid suggests that this compound may be imported or produced on a specialty chemical basis in the United States. p-Chlorobenzoic acid has been used in plasticizers, as a dye carrier, and as a fungicide and dye intermediate; its esters have potential for use as insecticides and plant growth regulators (Williams, 1978).

p-Chlorobenzoic acid is an acidic compound that is expected to ionize significantly under environmental conditions (pH 5-9) and to form salts that are much more water soluble than the parent compound. Variations in the behavior of p-chlorobenzoic acid may result from ionization. Because of the lack of pertinent data, the fate of this compound in the atmosphere cannot be predicted. Data by Korte and Klein (1982) indicate that direct photolysis may not be a dominant removal mechanism in the atmosphere. p-Chlorobenzoic vapor reacting with photochemically generated hydroxyl radicals in the atmosphere has an estimated half-life of ~7 days (U.S. EPA, 1987). This

compound is not expected to react significantly with atmospheric ozone (U.S. EPA, 1987). If released to water, biodegradation of p-chlorobenzoic acid under aerobic conditions is expected to be the dominant fate process. Screening studies suggest that the aerobic biodegradation half-life of this compound in natural water systems should range from 2 weeks to <1 month (Horvath, 1973; Haller, 1978; DiGeronimo et al., 1979; Freitag et al., 1985). 4-Dihydroxybenzoate, procatechuate and cis-dihydrodiol have been identified as products of p-chlorobenzoic acid metabolism (Marks et al., 1984; Karasevich and Zaitsev, 1984; Gibson, 1977). Chemical hydrolysis, reaction with photochemically generated hydroxyl radicals in water ($t_{1/2}$ of 1.1 years) (Anbar et al., 1966; Mill et al., 1980), photolysis (Crosby and Leitis, 1969), bioconcentration in aquatic organisms (Freitag et al., 1985), adsorption to suspended solids and sediments, volatilization and anaerobic biodegradation (Gibson and Suflita, 1986; Horowitz et al., 1982) are not expected to be significant fate processes. If released to soil, aerobic biodegradation may be an important fate process. Results of one biodegradation screening study indicated that p-chlorobenzoic acid at an initial concentration of 25 mg/ℓ was completely degraded by a soil inoculum in 64 days (Alexander and Lustigman, 1966). p-Chlorobenzoic acid has the potential to leach into groundwater; mobility may increase with increasing pH because of greater ionization. Volatilization from moist soil surfaces is not expected to be significant. p-Chlorobenzoic acid is predicted to be resistant to biodegradation in soil under anaerobic conditions.

p-Chlorobenzoic acid has been positively identified in drinking water samples from Poplarville, MS, and Cincinnati, OH, and tentatively identified in drinking water samples from New Orleans, LA, and Philadelphia, PA (Lucas, 1984). The occurrence of p-chlorobenzoic acid in drinking water and wastewater samples may be the result of chlorination of humic and fulvic acids.

Quimby et al. (1980) have tentatively identified this compound as a chlorination product of humic and fulvic acids in aqueous solution. Monitoring data regarding exposure to p-chlorobenzoic acid by inhalation, dermal contact or ingestion of food could not be located in the available literature as cited in Appendix A.

Information regarding the toxicity of p-chlorobenzoic acid to aquatic organisms was limited. Trabalka and Burch (1978) reported that no mortality occurred in Daphnia pulex exposed to p-chlorobenzoic acid at concentrations of $\leq$100 mg/ℓ for 96 hours under static conditions. Casida (1955) reported an LC_{50} of 8.7 mM (1362 mg/ℓ) for mosquito, Aedes aegyptii, larvae exposed for an unspecified period.

The only pharmacokinetic study of p-chlorobenzoic acid showed that nearly all the radioactivity of a 50 mg dose of neutralized labeled p-chlorobenzoic acid was recovered in the urine within 24 hours (Lang and Lang, 1956) Small amounts of radioactivity were recovered in the feces and insignificant levels remained in the tissues. Kieckebusch et al. (1960) state that the main metabolite of p-chlorobenzoic acid is p-chlorohippuric acid.

Rats fed p-chlorobenzoic acid in the diet at 0.1 or 0.2% did not develop toxic effects, and no differences in the number of litters or the development of the young were noted (Kieckebusch et al., 1960). D'eng et al. (1983) observed that a single oral LD_{50} dose produced a transient stimulation of protein synthesis whereas daily dosing with 1/10 LD_{50} and 1/50 LD_{50} resulted in inhibition of protein synthesis in the liver of rats. Kaulla (1962) reported that p-chlorobenzoic acid has in vitro fibrinolytic activity. The rat intraperitoneal LD_{50} for p-chlorobenzoic acid is 100 mg/kg (NIOSH, 1986).

Pertinent data regarding the carcinogenicity, mutagenicity, teratogenicity and toxicity of p-chlorobenzoic acid following subchronic and chronic inhalation exposure and chronic oral exposure could not be located in the available literature as cited in Appendix A.

A subchronic RfD for oral exposure of 121 mg/day was derived from the NOAEL of 0.2% of the diet (173 mg/kg/day) in the 5-month rat dietary study by application of an uncertainty factor of 100 (Kieckebusch et al., 1960). An RfD of 12 mg/day for chronic oral exposure was derived by application of an additional uncertainty factor of 10. Confidence in the chronic RfD is very low. The RfD is based on a freestanding NOAEL found in a subchronic oral study (Kieckebusch et al., 1960), and no supporting studies are available. Data were insufficient for derivation of RfDs for inhalation exposure or for derivation of a toxicity-based RQ. p-Chlorobenzoic acid was assigned to EPA Group D. Data were insufficient for quantitative estimation of cancer potency or hazard ranking based on carcinogenicity.

TABLE OF CONTENTS

		Page
1.	INTRODUCTION	1
1.1.	STRUCTURE AND CAS NUMBER	1
1.2.	PHYSICAL AND CHEMICAL PROPERTIES	1
1.3.	PRODUCTION DATA	2
1.4.	USE DATA	2
1.5.	SUMMARY	2
2.	ENVIRONMENTAL FATE AND TRANSPORT	4
2.1.	AIR	4
2.1.1.	Reaction with Hydroxyl Radicals	4
2.1.2.	Reaction with Ozone	4
2.1.3.	Photolysis	5
2.2.	WATER	5
2.2.1.	Hydrolysis	5
2.2.2.	Oxidation	5
2.2.3.	Photolysis	5
2.2.4.	Microbial Degradation	5
2.2.5.	Bioconcentration	7
2.2.6.	Adsorption	7
2.2.7.	Volatilization	7
2.3.	SOIL	7
2.3.1.	Microbial Degradation	7
2.3.2.	Leaching	8
2.3.3.	Volatilization	9
2.4.	SUMMARY	9
3.	EXPOSURE	11
3.1.	WATER	11
3.2.	SUMMARY	11
4.	AQUATIC TOXICITY	13
4.1.	ACUTE TOXICITY	13
4.2.	CHRONIC EFFECTS	13
4.3.	PLANT EFFECTS	13
4.4.	SUMMARY	13

TABLE OF CONTENTS (cont.)

		Page
5.	PHARMACOKINETCS	14
5.1.	ABSORPTION	14
5.2.	DISTRIBUTION	14
5.3.	METABOLISM	14
5.4.	EXCRETION	14
5.5.	SUMMARY	14
6.	EFFECTS	15
6.1.	SYSTEMIC TOXICITY	15
6.1.1.	Inhalation Exposures	15
6.1.2.	Oral Exposures	15
6.1.3.	Other Relevant Information	15
6.2.	CARCINOGENICITY	16
6.3.	MUTAGENICITY	16
6.4.	TERATOGENICITY	16
6.5.	OTHER REPRODUCTIVE EFFECTS	16
6.6.	SUMMARY	16
7.	EXISTING GUIDELINES AND STANDARDS	18
7.1.	HUMAN	18
7.2.	AQUATIC	18
8.	RISK ASSESSMENT	19
8.1.	CARCINOGENICITY	19
8.1.1.	Weight of Evidence	19
8.1.2.	Quantitative Risk Estimates	19
8.2.	SYSTEMIC TOXICITY	19
8.2.1.	Inhalation Exposure	19
8.2.2.	Oral Exposure	19
9.	REPORTABLE QUANTITIES	21
9.1.	BASED ON SYSTEMIC TOXICITY	21
9.2.	BASED ON CARCINOGENICITY	21
10.	REFERENCES	23
APPENDIX A:	LITERATURE SEARCHED	30
APPENDIX B:	SUMMARY TABLE FOR p-CHLOROBENZOIC ACID	33

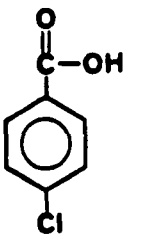
LIST OF ABBREVIATIONS

BCF	Bioconcentration factor
K_{oc}	Soil sorption coefficient standardized with respect to organic carbon
K_{ow}	Octanol/water partition coefficient
LC_{50}	Concentration lethal to 50% of recipients (and all other subscripted dose levels)
LD_{50}	Dose lethal to 50% of recipients
NOAEL	No-observed-adverse-effect level
RfD	Reference dose
RQ	Reportable quantity

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

p-Chlorobenzoic acid is also known as 4-chlorobenzoic acid, chlorodracylic acid and p-carboxychlorobenzene (NIOSH, 1987). The structure, molecular weight, empirical formula and CAS Registry number are as follows:



Molecular weight: 156.57

Empirical formula: $C_7H_5ClO_2$

CAS Registry number: 74-11-3

1.2. PHYSICAL AND CHEMICAL PROPERTIES

p-Chlorobenzoic acid is a crystalline solid at room temperature (Windholz, 1983). This compound may undergo reactions involving the aromatic nucleus or the chlorine substituent, as well as reactions typical of the carboxyl group (Muir, 1963). It is freely soluble in ethanol and ethyl ether (Windholz, 1983). Relevant physical properties are as follows:

Melting point (°C):	243	Muir, 1963
Boiling point (°C):	sublimes	Muir, 1963
Vapor pressure:	not available	
Water solubility (25°C):	80 mg/ℓ	Perry and Green, 1984
Log K_{ow}:	2.65	Hansch and Leo, 1985
pKa (25°C):	3.98	Muir, 1963
Specific gravity:	1.541 (24/4)	Muir, 1963

1.3. PRODUCTION DATA

p-Chlorobenzoic acid is prepared by the oxidation of p-chlorotoluene (Gelfand, 1979; Muir, 1963). The public portion of the U.S. EPA TSCA Production File (U.S. EPA, 1977) reports that one company manufactured p-chlorobenzoic acid in 1977 in the United States; however, the identity of the company and its production volume are confidential. U.S. EPA (1977) indicated that Tenneco Chemicals Inc. and Riches-Nelson Inc. imported/manufactured this compound before 1977. The 1987 OPD Chemical Buyers Directory (Van, 1986) lists Aldrich Chemical Co., Chugai International, Conray Chemicals, Maypro Industries, Miki Sangyo (USA), Mobay Corp., Niagara Technology and Wall Chemical Corp. as current suppliers of this compound. The Directory of Chemical Producers (SRI, 1986) and recent U.S. International Trade Commission publications on chemical production and sales (USITC, 1985, 1986) contain no production data for p-chlorobenzoic acid. The lack of data regarding current domestic production of this compound suggest that it may be imported or produced on a specialty chemical basis in the United States.

1.4. USE DATA

p-Chlorobenzoic acid has been used in plasticizers, as a dye carrier, and as a fungicide and dye intermediate (Williams, 1978). Its esters have potential for use as insecticides and plant growth regulators (Williams, 1978).

1.5. SUMMARY

p-Chlorobenzoic acid (74-11-3) is a crystalline solid at room temperature (Windholz, 1983). It is soluble in ethanol and ethyl ether but is sparingly soluble in water (Windholz, 1983; Perry and Green, 1984). It may

undergo reactions involving the aromatic nucleus or the chlorine substituent, as well as reactions typical of the carboxyl group (Muir, 1963). This compound may be prepared by the oxidation of p-chlorotoluene (Gelfand, 1979; Muir, 1963). The 1987 OPD Chemical Buyers Directory lists eight suppliers of p-chlorobenzoic acid; however, The Directory of Chemical Producers (SRI, 1986) and recent U.S. International Trade Commission publications on chemical production and sales (USITC, 1985, 1986) contain no production data for this compound. The limited amount of production data on p-chlorobenzoic acid suggest that this compound may be imported or produced on a specialty chemical basis in the United States. p-Chlorobenzoic acid has been used in plasticizers, as a dye carrier, and as a fungicide and dye intermediate; its esters have potential for use as insecticides and plant growth regulators (Williams, 1978).

2. ENVIRONMENTAL FATE AND TRANSPORT

Limited data pertaining to the environmental fate and transport of p-chlorobenzoic acid could be located in the available literature as cited in Appendix A. When possible, information concerning the fate and transport of this compound was derived from its physical properties or its molecular structure.

Based on a pKa of 3.98 (Muir, 1963), p-chlorobenzoic acid is expected to ionize significantly under environmental conditions (pH 5-9) and to form salts, that are much more water soluble than the parent compound. Ionization can alter the behavior of a compound in water or soil (e.g., by decreasing volatilization or by increasing or decreasing adsorption to soil, sediments or suspended solids in water).

2.1. AIR

2.1.1. Reaction with Hydroxyl Radicals. The estimated half-life for p-chlorobenzoic acid vapor reacting with photochemically generated hydroxyl radicals in the atmosphere is ~7 days, using an estimated reaction rate constant of 1.5×10^{-12} cm^3/molecule-sec at 25°C and an average hydroxyl radical concentration of 8.0×10^5 molecules/cm^3 in a typical atmosphere (U.S. EPA, 1987). This reaction rate, however, will be different if p-chlorobenzoic acid is present in particulate phase in the atmosphere. The physical state of a compound (solid, vapor, etc.) in the atmosphere can be predicted from its equilibrium vapor pressure (Eisenreich et al., 1981). Since the vapor pressure value for p-chlorobenzoic acid is not known, its physical state in the atmosphere cannot be predicted.

2.1.2. Reaction with Ozone. p-Chlorobenzoic acid is not susceptible to oxidation by ozone in the atmosphere (U.S. EPA, 1987).

2.1.3. Photolysis. When irradiated with light from a photoreactor at wavelengths >290 nm for 17 hours (Korte and Klein, 1982), 50 ppb [^{14}C]-p-chlorobenzoic acid adsorbed onto silica gel underwent 6.3% photomineralization. Korte and Klein (1982) were attempting to relate photomineralization to atmospheric degradation. Their results, however, indicate that direct photolysis will not be a dominant removal mechanism in the atmosphere.

2.2. WATER

2.2.1. Hydrolysis. Halogenated aromatic compounds and carboxylic acids are generally resistant to hydrolysis under environmental conditions (Lyman et al., 1982). Therefore, p-chlorobenzoic acid is not expected to be susceptible to chemical hydrolysis.

2.2.2. Oxidation. The estimated half-life for the reaction of p-chlorobenzoic acid with photochemically generated hydroxyl radicals in water is ~1.1 years using an experimentally determined reaction rate constant of 1.92×10^{9} ℓ/mol-sec (Anbar et al., 1966) and an ambient hydroxyl radical concentration of 1×10^{-17} mol/ℓ (Mill et al., 1980). Experimental data concerning the reaction of p-chlorobenzoic acid with alkylperoxy radicals or singlet oxygen were not available.

2.2.3. Photolysis. p-Chlorobenzoic acid appeared to be unaffected when an aqueous solution of its sodium salt was irradiated with outdoor sunlight in a period of $\leq$14 days (Crosby and Leitis, 1969).

2.2.4. Microbial Degradation. When 47 and 0.47 mg/ℓ radiolabeled p-chlorobenzoic acid (ring U-^{14}C) was incubated in stream water samples at 29°C for 12 days, the loss of initial ^{14}C as $^{14}CO_2$ was found to be 5.9 and 79%, respectively (Boethling and Alexander, 1979). No measurable loss of ^{14}C was observed when 4.7×10^{-3} mg/ℓ p-chlorobenzoic acid was incubated in autoclaved samples of stream water for 2 days (Boethling and

Alexander, 1979). It was concluded from this investigation that biodegradation occurs more readily at low concentrations of substrate. Results of biodegradation screening studies using activated sludge as inoculum indicate that this compound is likely to biodegrade under aerobic conditions by mixed microbial populations found in natural water systems (Horvath, 1973; Haller, 1978; DiGeronimo et al., 1979; Freitag et al., 1985). It appears that the rate of biodegradation may be enhanced by microbial adaptation or by the presence of a co-substrate (Thom and Agg, 1975; Haller, 1978; Horvath, 1973). For example, 25 mg/ℓ p-chlorobenzoate incubated with an activated sludge inoculum was decomposed after an initial 4-day lag period and was 50% degraded after 24 days (Horvath, 1973). In the presence of a co-substrate (glucose), the lag period was essentially eliminated; 50% degradation occurred in 12 days and 100% degradation took place in 28 days (Horvath, 1973). Metabolism of p-chlorobenzoic acid by an isolated culture of Alcaligenes eutrophus under aerobic conditions resulted in the formation of cis-dihydrodiol (Gibson, 1977). Further degradation of dihydrodiols has been found to involve dehydration to ortho-dihydroxy derivatives (catechols) before ring cleavage (Gibson, 1977). Other microbial isolates have been found to metabolize p-chlorobenzoate by initial dehalogenation to p-hydroxybenzoate followed by further degradation to protocatechuate (Marks et al., 1984; Karasevich and Zaitsev, 1984).

When p-chlorobenzoic acid was incubated under anaerobic conditions with digester sludge, anoxic pond sediment, microflora from a methogenic aquifer and microflora from a sulfate-reducing aquifer, loss of initial substrate was 17, 6, 14 and 11%, respectively, after 3 months (Gibson and Suflita, 1986). This loss, however, was not attributed to biodegradation since substrate disappearance did not exceed loss observed in sterile controls.

No mineralization was observed when this compound was incubated under anaerobic conditions for 65 weeks in an aqueous solution inoculated with anoxic sediment taken from a hypoeutrophic lake (Horowitz et al., 1982). Likewise, no mineralization was observed when this compound was incubated for 8 weeks in aqueous solution amended with sludge taken from an anaerobic digester (Horowitz et al., 1982).

2.2.5. Bioconcentration. The BCF of p-chlorobenzoic acid was determined to be 63 in green alga, Chlorella fusca, and 3.4 in the golden orfe, Leuciscus idus melanotus (Freitag et al., 1985). These BCF values suggest that bioconcentration of p-chlorobenzoic acid in aquatic organisms would not be significant.

2.2.6. Adsorption. Based on the low K_{oc} values (Section 2.3.2.), p-chlorobenzoic acid is not expected to adsorb significantly to sediments or suspended solids in water.

2.2.7. Volatilization. Volatilization from water surfaces is not expected to be significant because p-chlorobenzoic acid is present predominantly in the ionic state under most environmental conditions.

2.3. SOIL

2.3.1. Microbial Degradation. The degradation period for a 25 mg/ℓ p-chlorobenzoic acid inoculated with a mixed culture of microorganisms obtained from soil was found to be 64 days (Alexander and Lustigman, 1966). No significant loss in p-chlorobenzoic acid, at an initial concentration of 16 mg/ℓ, was observed after 25 days incubation with a soil inoculum (Haller, 1978). Microorganisms isolated from soil samples taken from a landfill site (previously used to dispose of chlorinated organics) showed growth when incubated with p-chlorobenzoic acid for 72 hours at 25°C, which was attributed to the presence of plasmids in the isolated cells that were

capable of transferring the trait to nonadopted cells (Vandenbergh et al., 1981). These results suggest that p-chlorobenzoic acid will biodegrade in soil after a lag period. Data pertaining to the biodegradation of p-chlorobenzoic acid in aqueous media suggest that aerobic biodegradation may be an important fate process in soil as well. Cells of one Pseudomonas sp. and one Bacillus sp. isolated from soil and grown on benzoate were found to cooxidize p-chlorobenzoate, but other microorganisms isolated from soil and grown on benzoate (two Pseudomonas sp., three Nocardia sp. and one Achromobacter sp.) did not cooxidize p-chlorobenzoate (Spokes and Walker, 1974). A Nocardia sp. isolated from soil metabolized p-chlorobenzoic acid to p-hydroxybenzoic acid (Klages and Lingens, 1979). Based on available data pertaining to the anaerobic biodegradation of p-chlorobenzoic acid in aqueous media, this compound is expected to resist biodegradation in soil under anaerobic conditions.

2.3.2. Leaching. A K_{oc} of 390-660 has been estimated for p-chlorobenzoic acid using a water solubility of 80 mg/ℓ at 25°C (Perry and Green, 1984) and the following linear regression equation (Lyman et al., 1982):

$$\log K_{oc} = -0.55 \log S + 3.64 \qquad (2\text{-}1)$$

This K_{oc} value suggests that p-chlorobenzoic acid would be moderately mobile in soil (Swann et al., 1983); however, ionization can have a significant effect on the behavior of this compound in soil. In general, neutral species of a compound adsorb much more strongly than its ionic species when the sorption mechanisms depend on organic carbon content of soil (Lyman et al., 1982). Based on a pKa of 3.98 (Muir, 1963), the ratio of ionized to un-ionized p-chlorobenzoic acid in natural waters with relatively low ionic strength has been estimated to be 10:1 at pH 5, 105:1 at pH 6, 1050:1 at pH 7 and so forth. This information suggests that p-chlorobenzoic acid may

have a greater tendency to leach through soil than would be predicted from its K_{OC} value and that the mobility of this compound would increase with increasing pH if such sorptive mechanisms are operative.

2.3.3. Volatilization. Volatilization from moist soil surfaces is not expected to be significant because p-chlorobenzoic acid is ionized under environmental conditions.

2.4. SUMMARY

p-Chlorobenzoic acid is an acidic compound that is expected to ionize significantly under environmental conditions (pH 5-9) and form salts that are much more water soluble than the parent compound. Variations in the behavior of p-chlorobenzoic acid may result from ionization. Lack of pertinent data precludes determination of the fate of this compound in the atmosphere. Data by Korte and Klein (1982) indicate that direct photolysis may not be a dominant removal mechanism in the atmosphere. p-Chlorobenzoic vapor reacting with photochemically-generated hydroxyl radicals in the atmosphere has an estimated half-life of ~7 days (U.S. EPA, 1987). This compound is not expected to react significantly with atmospheric ozone (U.S. EPA, 1987). If released to water, biodegradation of p-chlorobenzoic acid under aerobic conditions is expected to be the dominant fate process. Screening studies suggest that the aerobic biodegradation half-life of this compound in natural water systems should range from 2 weeks to <1 month (Horvath, 1973; Haller, 1978; DiGeronimo et al., 1979; Freitag et al., 1985). 4-Dihydroxybenzoate, procatechuate and cis-dihydrodiol have been identified as products of p-chlorobenzoic acid metabolism (Marks et al., 1984; Karasevich and Zaitsev, 1984; Gibson, 1977). Chemical hydrolysis, reaction with photochemically generated hydroxyl radicals in water ($t_{1/2}$ of 1.1 years) (Anbar et al., 1966; Mill et al., 1980), photolysis (Crosby

and Leitis, 1969), bioconcentration in aquatic organisms (Freitag et al., 1985), adsorption to suspended solids and sediments, volatilization and anaerobic biodegradation (Gibson and Suflita, 1986; Horowitz et al., 1982) are not expected to be significant fate processes. If released to soil, aerobic biodegradation may be an important fate process. Results of one biodegradation screening study indicate that p-chlorobenzoic acid at an initial concentration of 25 mg/ℓ was completely degraded by a soil inoculum in 64 days (Alexander and Lustigman, 1966). p-Chlorobenzoic acid has the potential to leach into groundwater; mobility may increase with increasing pH because of greater ionization. Volatilization from moist soil surfaces is not expected to be significant. p-Chlorobenzoic acid is predicted to be resistant to biodegradation in soil under anaerobic conditions.

3. EXPOSURE

Pertinent monitoring data regarding exposure to 4-chlorobenzoic acid by inhalation, dermal contact or ingestion of food could not be located in the available literature as cited in Appendix A.

3.1. WATER

p-Chlorobenzoic acid has been positively identified in drinking water obtained from Poplarville, MS, during 1979 and Cincinnati, OH, during 1978, and it has been tentatively identified in drinking water obtained from New Orleans, LA, and Philadelphia, PA, during during 1976 (Lucas, 1984). p-Chlorobenzoic acid has been positively identified in samples of advanced waste treatment water taken from facilities in Lake Tahoe during 1974, Pomona, CA, during 1975, Escondido, CA, during 1975 and Dallas, TX, during 1974 (Lucas, 1984). The occurrence of p-chlorobenzoic acid in drinking water and wastewater samples may be the result of chlorination of humic and fulvic acids. Quimby et al. (1980) have tentatively identified this compound as a chlorination product of humic and fulvic acids in aqueous solution.

3.2. SUMMARY

p-Chlorobenzoic acid has been positively identified in drinking water samples from Poplarville, MS, and Cincinnati, OH, and tentatively identified in drinking water samples from New Orleans, LA, and Philadelphia, PA (Lucas, 1984). The occurrence of p-chlorobenzoic acid in drinking water and wastewater samples may be the result of chlorination of humic and fulvic acids. Quimby et al. (1980) have tentatively identified this compound as a chlorination product of humic and fulvic acids in aqueous solution.

Monitoring data regarding exposure to p-chlorobenzoic acid by inhalation, dermal contact or ingestion of food could not be located in the available literature as cited in Appendix A.

4. AQUATIC TOXICITY

4.1. ACUTE TOXICITY

Information regarding the toxicity of p-chlorobenzoic acid to aquatic organisms was limited. Trabalka and Burch (1978) reported that no mortality occurred in Daphnia pulex exposed to p-chlorobenzoic acid at concentrations of ≤100 mg/ℓ for 96 hours under static conditions. Casida (1955) reported an LC_{50} of 8.7 mM (1362 mg/ℓ) for mosquito, Aedes aegyptii, larvae exposed for an unspecified period.

4.2. CHRONIC EFFECTS

Pertinent data regarding chronic toxicity of p-chlorobenzoic acid to aquatic organisms could not be located in the available literature as cited in Appendix A.

4.3. PLANT EFFECTS

Pertinent data regarding effects of p-chlorobenzoic acid on aquatic plants could not be located in the available literature as cited in Appendix A.

4.4. SUMMARY

Information regarding the toxicity of p-chlorobenzoic acid to aquatic organisms was limited. Trabalka and Burch (1978) reported that no mortality occurred in Daphnia pulex exposed to p-chlorobenzoic acid at concentrations ≤100 mg/ℓ for 96 hours under static conditions. Casida (1955) reported an LC_{50} of 8.7 mM (1362 mg/ℓ) for mosquito, Aedes aegyptii, larvae exposed for an unspecified period.

5. PHARMACOKINETICS

5.1. ABSORPTION

Pertinent data regarding the absorption of p-chlorobenzoic acid could not be located in the available literature as cited in Appendix A.

5.2. DISTRIBUTION

Lang and Lang (1956) found insignificant levels of radioactivity in the organs of rats 1-23 days after they were given intraperitoneal injections of 50 mg [carboxy-^{14}C]p-chlorobenzoic acid that had been neutralized with NaOH. The abstract from which these data were taken stated that there was no storage in the body.

5.3. METABOLISM

Kieckebusch et al. (1960) reported that p-chlorobenzoic acid is metabolized in a manner similar to benzoic acid, with p-chlorohippuric acid as the major metabolite.

5.4. EXCRETION

Nearly all of the radioactivity of a 50 mg dose of neutralized [^{14}C]p-chlorobenzoic acid administered to rats by intraperitoneal injection was recovered in the urine within 24 hours (Lang and Lang, 1956). Small amounts of radioactivity were also recovered in the feces.

5.5. SUMMARY

The only pharmacokinetic study of p-chlorobenzoic acid showed that nearly all the radioactivity of a 50 mg i.p. dose of neutralized labeled p-chlorobenzoic acid was recovered in the urine within 24 hours (Lang and Lang, 1956) Small amounts of radioactivity were recovered in the feces and insignificant levels remained in the tissues. Kieckebusch et al. (1960) state that the main metabolite of p-chlorobenzoic acid is p-chlorohippuric acid.

6. EFFECTS

6.1. SYSTEMIC TOXICITY

6.1.1. Inhalation Exposures. Pertinent data regarding the toxicity of p-chlorobenzoic acid following subchronic or chronic inhalation exposure could not be located in the available literature as cited in Appendix A.

6.1.2. Oral Exposures.

6.1.2.1. SUBCHRONIC -- In a study by Kieckebusch et al. (1960), groups of 20 male and 20 female young (40-50 g) Elberfield rats were fed diets containing p-chlorobenzoic acid at 0, 0.1 or 0.2% for 5 months. Feed intake was measured daily. The approximate daily doses determined by the authors were 13 and 26 mg for the low and high dose groups, respectively. Near the end of the experiment, a 24-hour urine sample was analyzed for protein and sugar content, and the sediment was examined microscopically. While urine samples were being taken, the rats were fasted. At the end of the experiment, the rats were sacrificed, organ weights were determined and the livers and kidneys were examined histologically. The results of the study showed no adverse effects in any of the parameters examined.

6.1.2.2. CHRONIC -- Pertinent data regarding the chronic oral toxicity of p-chlorobenzoic acid could not be located in the available literature as cited in Appendix A.

6.1.3. Other Relevant Information. D'eng et al. (1983) showed an elevation of two hepatic enzymes in the serum 0.5-5 hours after the administration of an LD_{50} dose. Daily oral administration of 1/10 the LD_{50} dose produced an inhibition of hepatic protein synthesis within 2 weeks. The significance of this study is difficult to evaluate because it was available only in abstract form.

In an abstract reported by Kaulla (1962), p-chlorobenzoic acid has been shown to have substantial in vitro fibrinolytic activity.

NIOSH (1986) lists the rat intraperitoneal LD_{50} for p-chlorobenzoic acid as 1000 mg/kg.

6.2. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of p-chlorobenzoic acid following any route of exposure could not be located in the available literature as cited in Appendix A.

6.3. MUTAGENICITY

Pertinent data regarding the mutagenicity of p-chlorobenzoic acid could not be located in the available literature as cited in Appendix A.

6.4. TERATOGENICITY

Pertinent data regarding the teratogenicity of p-chlorobenzoic acid could not be located in the available literature as cited in Appendix A.

6.5. OTHER REPRODUCTIVE EFFECTS

In the study by Kieckebusch et al. (1960), no differences in the number of litters or the development of the young were noted in rats fed p-chlorobenzoic acid in the diet at 0.1 or 0.2%. The rats were mated 8 weeks and ~4 months after the beginning of the experiment. A delay in sexual maturation of females at both exposure levels was noted; however, the authors judged the difference not to be significant.

6.6. SUMMARY

Rats fed p-chlorobenzoic acid in the diet at 0.1 or 0.2% did not develop toxic effects, and no differences in the number of litters or the development of the young were noted (Kieckebusch et al., 1960). D'eng et al. (1983) observed that a single oral LD_{50} dose produced a transient stimulation of hepatic protein synthesis whereas daily dosing with 1/10 LD_{50} and

1/50 LD_{50} resulted in inhibition of protein synthesis in the liver of rats. Kaulla (1962) found that p-chlorobenzoic acid has *in vitro* fibrinolytic activity. The rat intraperitoneal LD_{50} for p-chlorobenzoic acid is 1000 mg/kg (NIOSH, 1986).

Pertinent data regarding the carcinogenicity, mutagenicity and teratogenicity of p-chlorobenzoic acid following subchronic and chronic inhalation exposure and chronic oral exposure could not be located in the available literature as cited in Appendix A.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Pertinent guidelines and standards, including EPA ambient water and air quality criteria, drinking water standards, FAO/WHO ADIs, EPA or FDA tolerances for raw agricultural commodities or foods, and ACGIH, NIOSH or OSHA occupational exposure limits could not be located in the available literature as cited in Appendix A.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the effects of p-chlorobenzoic acid could not be located in the available literature as cited in Appendix A.

8. RISK ASSESSMENT

8.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of p-chlorobenzoic acid could not be located in the available literature as cited in Appendix A.

8.1.1. Weight of Evidence. The lack of data concerning the carcinogenicity of p-chlorobenzoic acid in either humans or animals indicates that the compound should be classified as an EPA Group D chemical (U.S. EPA, 1986b), not classifiable as to human carcinogenicity.

8.1.2. Quantitative Risk Estimates. The lack of carcinogenicity data precludes the derivation of carcinogenicity-based quantitative risk assessment values.

8.2. SYSTEMIC TOXICITY

8.2.1. Inhalation Exposure. The lack of data concerning the toxicity of p-chlorobenzoic acid following inhalation exposure precludes the derivation of inhalation risk assessment values.

8.2.2. Oral Exposure.

8.2.2.1. LESS THAN LIFETIME EXPOSURES (SUBCHRONIC) -- In the study by Kieckebusch et al. (1960), no adverse effects were noted in rats fed diets containing p-chlorobenzoic acid at 0.1 or 0.2% (13 or 26 mg/day) for 5 months. Despite the lack of an effect level, this study can be used to derive a subchronic RfD. Dividing the daily dose of 26 mg provided by the author by 0.15 kg rat body weight, estimated from rat body weights at the start and end of the study, a rat dose of 173.3 mg/kg/day is estimated. Dividing the rat NOAEL by an uncertainty factor of 100 (10 for interspecies extrapolation and 10 to protect sensitive individuals), a human subchronic oral RfD of 2 mg/kg/day, or 121 mg/day for a 70 kg human, is derived.

Confidence in this RfD is very low. The RfD is based on a freestanding NOAEL, so that it is likely that the value is unnecessarily conservative. On the other hand, the study by Kieckebusch et al. (1960) was marginally adequate because an effect level was not defined and histopathological examinations of only the liver and kidneys were completed. In addition, there are no supporting studies; no other subchronic or chronic studies are available, and p-chlorobenzoic acid has not been studied for carcinogenicity, mutagenicity or developmental toxicity.

8.2.2.2. **CHRONIC EXPOSURES** -- No chronic oral studies of p-chlorobenzoic acid are available. A chronic oral RfD of 0.2 mg/kg/day, or 12 mg/day for a 70 kg human, can be derived by dividing the subchronic oral NOAEL by an additional uncertainty factor of 10 to extrapolate from subchronic to chronic exposure.

Confidence in the chronic RfD is very low. The RfD is based on a freestanding NOAEL found in a subchronic oral study (Kieckebusch et al., 1960), and no supporting studies are available.

9. REPORTABLE QUANTITIES

9.1. BASED ON SYSTEMIC TOXICITY

The only toxicity study of p-chlorobenzoic acid (Kieckebusch et al., 1960) did not identify any adverse effects. Therefore, as indicated in Table 9-1, an RQ value cannot be derived.

9.2. BASED ON CARCINOGENICITY

No data were located regarding the carcinogenicity of p-chlorobenzoic acid in humans or animals, and the compound was placed in EPA Group D. Hazard ranking based on carcinogenicity is not possible for EPA Group D substances.

TABLE 9-1

p-Chlorobenzoic Acid

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	
Dose:	
Effect:	
Reference:	
RV_d:	
RV_e:	
Composite Score:	
RQ:	Data are not sufficient for deriving an RQ

10. REFERENCES

Alexander, M. and B.K. Lustigman. 1966. Effect of chemical structure on microbial degradation of substituted benzenes. J. Agric. Food Chem. 14: 410-413.

Anbar, M., D. Meyerstein and P. Neta. 1966. The reactivity of aromatic compounds toward hydroxyl radicals. J. Phys. Chem. 70: 2660-2661.

Boethling, R.S. and M. Alexander. 1979. Effects of concentration of organic chemicals on their biodegradation by natural microbial communities. Appl. Environ. Microbiol. 37: 1211-1216.

Casida, J.E. 1955. Toxicity of aromatic acids to the larvae of mosquito Aedes aegyptii and the counteracting influence of amino acids. Biochem. J. 59: 216-221. (CA 59:7137b)

Crosby, D.G. and E. Leitis. 1969. Photodecomposition of chlorobenzoic acids. J. Agric. Food Chem. 17: 1033-1035.

D'eng, B., A.I. Nikolaev, P.X. Khasigov, N.V. Likhacheva and N.A. Zaitsev. 1983. Evaluation of the hepatotoxic activity of several chlor-nitro derivatives of benzoic acid. Vopr. Med. Khim. 29(6): 113-117. (Rus.) (Taken from TOXBIB 84/148511)

DiGeronimo, M.J., M. Nikaido and M. Alexander. 1979. Utilization of chlorobenzoates by microbial populations in sewage. Appl. Environ. Microbiol. 37: 619-625.

Eisenrich, S.J., B.B. Looney and D.J. Thornton. 1981. Airborne organic contaminants in the Great Lakes ecosystem. Environ. Sci. Technol. 15: 30-38.

Freitag, D., L. Ballhorn, H. Geyer and F. Korte. 1985. Environmental hazard profile of organic chemicals. Chemosphere. 14: 1589-1616.

Gelfand, S. 1979. Chlorocarbons - Hydrocarbons (Toluenes). In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 5, 3rd ed., M. Grayson and D. Eckroth, Ed. John Wiley and Sons, New York. p. 825.

Gibson, D.T. 1977. Biodegradation of aromatic petroleum hydrocarbons. In: Fate and Effects of Petroleum Hydrocarbons in Marine Organism and Ecosystems, D.A. Wolfe, Ed. Pergamon Press, New York. p. 36-46.

Gibson, S.A. and J.M. Suflita. 1986. Extrapolation of biodegradation results to groundwater aquifers: Reductive dehalogenation of aromatic compounds. Appl. Environ. Microbiol. 52(4): 681-688.

Haller, H.D. 1978. Degradation of mono-substituted benzoates and phenols by wastewater. J. Water Pollut. Control Fed. 50: 2771-2777.

Hansch C. and A.J. Leo. 1985. Medchem Project. Issue No. 26. Pomona College, Claremont, CA.

Horowitz, A., D.R. Shelton, C.P. Cornell and J.M. Tiedje. 1982. Anaerobic degradation of aromatic compounds in sediment and digested sludge. Dev. Ind. Microbiol. 23: 435-444.

Horvath, R.S. 1973. Enhancement of cometabolism of chlorobenzoates by the co-substrate enrichment technique. Appl. Microbiol. 25: 961-963.

Karasevich, Y.N. and G.M. Zaitsev. 1984. Utilization of 4-chlorobenzoic and 2,4-dichlorobenzoic acids by a mixed culture of microorganisms. Mikrobiologiya. 53(3): 374-380. (Rus.) [CA 101(9):69138x]

Kaulla, K.N.V. 1962. Chemical structure and indication of fibrinolysis. In vitro studies with 126 synthetic compounds. Throb. Diath. Haemorrhag. 7: 404-420. (CA 57:15378Ff)

Kieckebusch, W., W. Griem and K. Lang. 1960. The tolerability of p-chlorobenzoic acid. Arzneimi Hel-Forsch. 10: 999-10001. (In German with English translation)

Klages, U. and F. Lingens. 1979. Degradation of 4-chlorobenzoic acid by a Nocardia species. Fem. Microbiol. Lett. 6(4): 201-203. (CA 92:2953)

Korte, F. and W. Klein. 1982. Degradation of benzene in the environment. Ecotoxicol. Environ. Saf. 6: 311-327.

Lang, H. and K. Lang. 1956. Fate of benzoic acid-C^{14} and p-chlorobenzoic acid C^{14}. Naunyn-Schmiedeberg's Arch. Exptl. Pathol. Pharmakol. 229: 505-512. (CA 51:4568d)

Lucas, S.V. 1984. GC/MS Analysis of Organics in Drinking Water Concentrates and Advanced Waste Treatment Concentrates: Vol. 2. Computer-Printed Tabulations of Compound Identification Results for Large-Volume Concentrates. Columbus Labs. Health Eff. Res. Lab., Columbus, OH. EPA 600/1-84-020B. NTIS PB85-128239.

Lyman, W.J., W.F. Reehl and D.H. Rosenblatt. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York. p. 7-4, 4-9, 5-5.

Marks, T.S., A.R. Smith, R.W. Anthony and A.V. Quirk. 1984. Degradation of 4-chlorobenzoic acid by Arthrobacter sp. Appl. Environ. Microbiol. 48(5): 1020-1025.

Mill, T., D.G. Hendry and H. Richardson. 1980. Free-radical oxidants in natural waters. Science. 207: 886-887.

Muir, W.M. 1963. Acids, carboxylic. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 1, A. Standen, Ed. John Wiley and Sons, New York. p. 224-7, 233-8.

NIOSH (National Institute for Occupational Safety and Health). 1986. RTECS (Registry of Toxic Effects of Chemical Substances). Online: December.

NIOSH (National Institute for Occupational Safety and Health). 1987. RTECS (Registry of Toxic Effects of Chemical Substances). Online. March.

Perry, R.H. and D. Green, Ed. 1984. Perry's Chemical Engineer's Handbook, 6th ed. McGraw-Hill Book Co., New York. p. 3-30.

Quimby, B.D., M.F. Delaney, P.C. Uden and R.M. Barnes. 1980. Determination of the aqueous chlorination products of humic substances by gas chromatography with microwave plasma emission detection. Anal. Chem. 52(2): 259-263.

Spokes, J.R. and N. Walker. 1974. Chlorophenol and chlorobenzoic acid co-metabolism by different genera of soil bacteria. Arch. Microbiol. 96: 125-134.

SRI (Stanford Research Institute). 1986. 1986 Directory of Chemical Producers: United States of America. SRI International, Menlo Park, CA.

Swann, R.L., D.A. Laskowski, P.J. McCall, K. vander Kuy and H.J. Dishburger. 1983. A rapid method for the estimation of the environmental parameters octanol/water partition coefficient, soil sorption constant, water to air ratio and water solubility. Res. Rev. 85: 17-28.

Thom, N.S. and A.R. Agg. 1975. The breakdown of synthetic organic compounds in biological processes. Proc. R. Soc. Long. B. 189: 347-357.

Trabalka, J.R. and M.B. Burch. 1978. Investigation of the effects of halogenated organic compounds produced in cooling systems and process effluents on aquatic organisms. In: Water Chlorination: Environmental Impact and Health Effects, R.L. Jolley, H. Gorchev and D.R. Hamilton, Jr., Ed. Conf. Proc. p. 163-173. ETIC/78/006669.

U.S. EPA. 1977. Computer Print-out of Nonconfidential Production Data from TSCA Inventory. OPTS, CID, U.S. EPA, Washington, DC.

U.S. EPA. 1980. Guidelines and Methodology Used in the Prepartion of Health Effect Assessment Chapters of the Consent Decree Water Criteria Documents. Federal Register. 45(231): 49347-49357.

U.S. EPA. 1984. Methodology and Guidelines for Reportable Quantity Determinations Based on Chronic Toxicity Data. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1986a. Methodology for Evaluating Carcinogenicity in Support of Reportable Quantity Adjustments Pursuant to CERCLA Section 102. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1986b. Guidelines for Carcinogen Risk Assessment. Federal Register. 51(185): 33992-34003.

U.S. EPA. 1987. Graphical Exposure Modeling System (GEMS). Fate of Atmospheric Pollutants (FAP). Office of Toxic Substances. U.S. EPA, Washington, DC.

USITC (U.S. International Trade Commission). 1985. Synthetic Organic Chemicals United States Production and Sales, 1984. USITC Publ. 1745, Washington, DC.

USITC (U.S. International Trade Commission). 1986. Synthetic Organic Chemicals United States Production and Sales, 1985. USITC Publ. 1892, Washington, DC.

Van, H., Ed. 1986. OPD Chemical Buyers 1987 Directory, 74th ed. Schnell Publishing Co., New York.

Vandenbergh, P.A., R.H. Olsen and J.F. Colaruotolo. 1981. Isolation and genetic characterization of bacteria that degrade chloroaromatic compounds. Appl. Environ. Microbiol. 42(4): 737-739.

Williams, A.E. 1978. Benzoic acid. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 3, M. Grayson and D. Eckroth, Ed. John Wiley and Sons, New York. p. 790.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ. p. 298.

APPENDIX A

LITERATURE SEARCHED

This HEED is based on data identified by computerized literature searches of the following:

TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in February, 1987. In addition, hand searches were made of Chemical Abstracts (Collective Indices 5-9), and the following secondary sources should be reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 1986-1987. TLVs: Threshold Limit Values for Chemical Substances in the Work Environment adopted by ACGIH with Intended Changes for 1986-1987. Cincinnati, OH. 111 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. 2878 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.

Grayson, M. and D. Eckroth, Ed. 1978-1984. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., Littleton, MA. 575 p.

IARC (International Agency for Research on Cancer). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. WHO, IARC, Lyons, France.

Jaber, H.M., W.R. Mabey, A.T. Lieu, T.W. Chou and H.L. Johnson. 1984. Data acquisition for environmental transport and fate screening for compounds of interest to the Office of Solid Waste. SRI International, Menlo Park, CA. EPA 600/6-84-010. NTIS PB84-243906.

NTP (National Toxicology Program). 1986. Toxicology Research and Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, I.N. 1984. Dangerous Properties of Industrial Materials, 6th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1986. Directory of Chemical Producers. Menlo Park, CA.

U.S. EPA. 1986. Report on Status Report in the Special Review Program, Registration Standards Program and the Data Call in Programs. Registration Standards and the Data Call in Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1985. CSB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (U.S. International Trade Commission). 1985. Synthetic Organic Chemicals. U.S. Production and Sales, 1984, USITC Publ. 1422, Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

McKee, J.E. and H.W. Wolf. 1963. Water Quality Criteria, 2nd ed. Prepared for the Resources Agency of California, State Water Quality Control Board. Publ. No. 3-A.

Pimental, D. 1971. Ecological Effects of Pesticides on Non-Target Species. Prepared for the U.S. EPA, Washington, DC. PB-269605.

Schneider, B.A. 1979. Toxicology Handbook. Mammalian and Aquatic Data. Book 1: Toxicology Data. Office of Pesticide Programs, U.S. EPA, Washington, DC. EPA 540/9-79-003. NTIS PB 80-196876.

APPENDIX B

Summary Table for p-Chlorobenzoic Acid

	Species	Exposure	Effect	RfD or q_1^*	Reference
Inhalation Exposure					
Subchronic	ID	ID	ID	ID	ID
Chronic	ID	ID	ID	ID	ID
Carcinogenicity	ID	ID	ID	ID	ID
Oral Exposure					
Subchronic	rat	0.2% in the diet for 5 months	NOAEL	121 mg/day	Keickebusch et al., 1960
Chronic	rat	0.2% in the diet for 5 months	NOAEL	12 mg/day	Keickebusch et al., 1960
Carcinogenicity	ID	ID	ID	ID	ID
REPORTABLE QUANTITIES					
Based on chronic toxicity:	ID				ID
Based on carcinogenicity:	ID				ID

ID = Insufficient data