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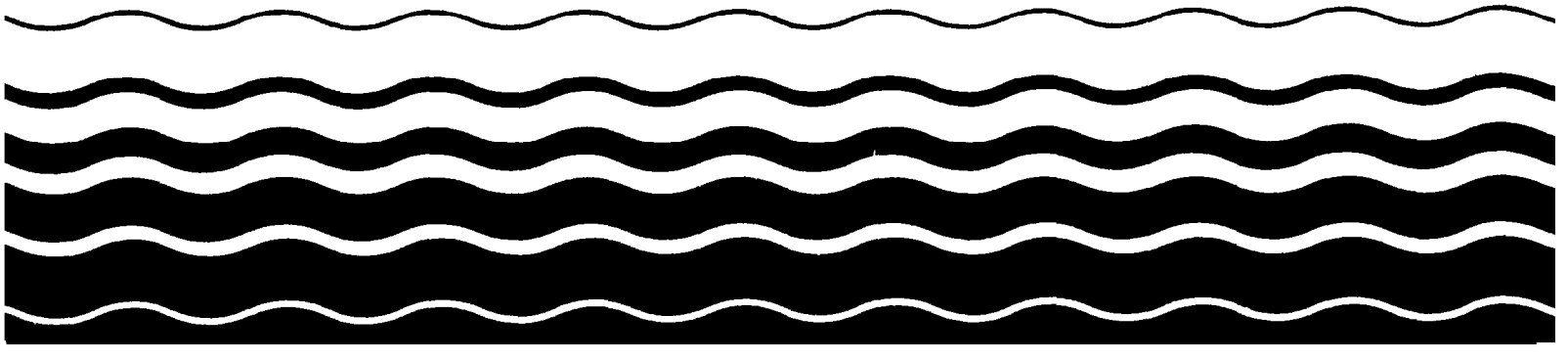

AMBIENT WATER QUALITY CRITERIA FOR

2,4-DICHLOROPHENOL

Prepared By
U.S. ENVIRONMENTAL PROTECTION AGENCY

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FOREWORD

Section 304 (a)(1) of the Clean Water Act of 1977 (P.L. 95-217), requires the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. Proposed water quality criteria for the 65 toxic pollutants listed under section 307 (a)(1) of the Clean Water Act were developed and a notice of their availability was published for public comment on March 15, 1979 (44 FR 15926), July 25, 1979 (44 FR 43660), and October 1, 1979 (44 FR 56628). This document is a revision of those proposed criteria based upon a consideration of comments received from other Federal Agencies, State agencies, special interest groups, and individual scientists. The criteria contained in this document replace any previously published EPA criteria for the 65 pollutants. This criterion document is also published in satisifaction of paragraph 11 of the Settlement Agreement in Natural Resources Defense Council, et. al. vs. Train, 8 ERC 2120 (D.D.C. 1976), modified, 12 ERC 1833 (D.D.C. 1979).

The term "water quality criteria" is used in two sections of the Clean Water Act, section 304 (a)(1) and section 303 (c)(2). The term has a different program impact in each section. In section 304, the term represents a non-regulatory, scientific assessment of ecological effects. The criteria presented in this publication are such scientific assessments. Such water quality criteria associated with specific stream uses when adopted as State water quality standards under section 303 become enforceable maximum acceptable levels of a pollutant in ambient waters. The water quality criteria adopted in the State water quality standards could have the same numerical limits as the criteria developed under section 304. However, in many situations States may want to adjust water quality criteria developed under section 304 to reflect local environmental conditions and human exposure patterns before incorporation into water quality standards. It is not until their adoption as part of the State water quality standards that the criteria become regulatory.

Guidelines to assist the States in the modification of criteria presented in this document, in the development of water quality standards, and in other water-related programs of this Agency, are being developed by EPA.

STEVEN SCHATZOW
Deputy Assistant Administrator
Office of Water Regulations and Standards

CRITERIA DOCUMENT

2,4-DICHLOROPHENOL

CRITERIA

Aquatic Life

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

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

Human Health

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

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INTRODUCTION

2,4-Dichlorophenol (2,4-DCP) is a commercially produced substituted phenol used entirely in the manufacture of industrial and agricultural products. As an intermediate in the chemical industry, 2,4-DCP is utilized principally as the feedstock for the manufacture of the herbicide, 2,4-dichlorophenoxyacetic acid (2,4-D), 2,4-D derivatives (germicides, soil sterilants, etc.) and certain methyl compounds used in mothproofing, antiseptics, and seed disinfectants. 2,4-DCP is also reacted with benzene sulfonyl chloride to produce miticides or further chlorinated to pentachlorophenol, a wood preservative (U.S. EPA, 1975).

2,4-Dichlorophenol is a colorless, crystalline solid having the empirical formula $C_6H_4Cl_2O$, a molecular weight of 163.0 (Weast, 1975), a density of 1.383 at 60°F/25°C and a vapor pressure of 1.0 mm Hg at 53.0°C (Sax, 1975). The melting point of 2,4-DCP is 45°C, and the boiling point is 210°C at 760 mm Hg (Aly and Faust, 1965; Weast, 1975).

2,4-DCP is slightly soluble in water at neutral pH and dissolves readily in ethanol and benzene (Kirk and Othmer, 1964). 2,4-DCP behaves as a weak acid and is highly soluble in alkaline solutions, since it readily forms the corresponding alkaline salt. The dissociation constant (pK_a) for 2,4-DCP has been reported to be 7.48 (Pearce and Simpkins, 1968). Unlike the monochlorophenols, 2,4-DCP is not volatile from aqueous alkaline solutions (Kirk and Othmer, 1964).

2,4-DCP is readily prepared without benefit of a catalyst using gaseous chlorine and molten phenol at 80 to 100°C. Synthesis is also attainable through the chlorination of the monochlorophenols (Kirk and Othmer, 1972).

It has been demonstrated that phenol is quite reactive to chlorine in dilute aqueous solutions over a wide pH range (Carlson and Caple, 1975; Middaugh and Davis, 1976).

Although 2,4-DCP presently has no direct commercial application, it is used as an important chemical intermediate, and it is synthesized from dilute aqueous solutions. Its identification as a metabolic intermediate and degradation product of various commercial products by plants (Kirk and Othmer, 1972), microorganisms (Kearney and Kaufman, 1972; Steenson and Walker, 1957; Bell, 1957, 1960; Evans and Smith, 1954; Fernley and Evans, 1959; Loos, et al. 1967a,b; Loos, 1969) and sunlight (Aly and Faust, 1964; Crosby and Tutass, 1966; Mitchell, 1961) has been well established.

Numerous studies on the microbial degradation of 2,4-DCP have been conducted, revealing degradation to yield succinic acid (Alexander and Aleem, 1961; Macrae, et al. 1963; Paris and Lewis, 1973; Fernley and Evans, 1959; Bell, 1957, 1960; Bollag, et al. 1968a,b; Macrae and Alexander, 1965; Chu and Kirsch, 1972; Ingols, et al. 1966; Chapman, 1972; Duxbury, et al. 1970; Loos, et al. 1967b; Tiedje, et al. 1969).

Few data exist regarding the persistence of 2,4-DCP in the environment. 2,4-DCP is slightly soluble in water, while its alkaline salts are readily soluble in aqueous solutions. Its low vapor pressure and non-volatility from alkaline solutions would cause it to be only slowly removed from surface water via volatilization. Studies have indicated low sorption of 2,4-DCP from natural surface waters by various clays (Aly and Faust, 1964).

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Aquatic Life Toxicology*

INTRODUCTION

2,4-Dichlorophenol has been widely used as a chemical intermediate in the manufacture of herbicides, germicides, temporary soil sterilants, plant growth regulators, mothproofing agents, seed disinfectants, miticides, and wood preservatives. In spite of this, there are only limited toxicity data available dealing with effects of 2,4-dichlorophenol on freshwater aquatic organisms. Flavor-impairment studies with 2,4-dichlorophenol showed that flesh tainting in fish occurred at substantially lower concentrations than those that produced other adverse effects on plant, fish, and invertebrate species. For this reason, flavor-impairment information may be an important consideration in deriving the 2,4-dichlorophenol criterion for freshwater aquatic organisms. Additional testing of 2,4-dichlorophenol is necessary to meet the minimum data base requirement, detailed in the guidelines, for the derivation of a criterion. This testing should verify if flavor-impairment of fish flesh is, indeed, the most sensitive and important parameter for protecting the presence of and the uses of freshwater aquatic organisms.

Only one test has been conducted with 2,4-dichlorophenol and saltwater organisms.

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*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and Its Uses in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are calculations for deriving various measures of toxicity as described in the Guidelines.

EFFECTS

Acute Toxicity

The data base for freshwater invertebrate species (Table 1) consists of two static tests on a single cladoceran species. The 48-hour LC_{50} values determined for Daphnia magna by Kopperman, et al. (1974) and U.S. EPA (1978) were 2,610 µg/l and 2,600 µg/l, respectively. The LC_{50} values from both tests show good reproducibility of results between investigators. Since all the invertebrate acute data available for 2,4-dichlorophenol are for only one species, it is impossible to determine the relative sensitivity of Daphnia magna with that of other invertebrate species.

Only two acute values dealing with 2,4-dichlorophenol effects on freshwater fish species were available (Table 1) and only one of these tests was conducted using flow-through conditions and measured concentrations. Phipps, et al. (Manuscript) calculated a 96-hour LC_{50} of 8,230 µg/l for fathead minnows. This is more than 4 times higher than the 96-hour LC_{50} of 2,020 µg/l for bluegills determined under static test conditions using nominal concentrations (U.S. EPA, 1978). Although differences in test methods make comparisons difficult, it appears bluegills may be slightly more sensitive to 2,4-dichlorophenol than are fathead minnows.

The LC_{50} values for Daphnia magna fall between the LC_{50} values found for bluegills and fathead minnows. From the few acute data available, it appears that there are no large differences in the sensitivity of fish and invertebrate species to 2,4-dichlorophenol.

The 96-hour LC_{50} values for chlorinated phenols and bluegills (U.S. EPA, 1978) in this and other criterion documents are directly related to the degree of chlorination. These values decrease from 6,590 µg/l for 2-chlorophenol and 3,830 µg/l for 4-chlorophenol to 60 and 70 µg/l for pentachlo-

rophenol. Data for other species do not correlate as well.

Chronic Toxicity

The freshwater chronic data base for 2,4-dichlorophenol consists of a single embryo-larval test (Holcombe, et al. Manuscript) conducted with fathead minnows. The chronic limits determined from this study (290-460 µg/l) were based on effects on larval survival, and the resulting chronic value was 365 µg/l (Table 2).

There appears to be a moderate difference between the concentration of 2,4-dichlorophenol that cause acute and chronic effects on fathead minnows. The acute-chronic ratio for this species is 23 (Table 2).

Data from an embryo exposure and an additional 4-day larval exposure with three species of fishes at two water hardnesses (Birge, et al. 1979) will be discussed in the miscellaneous section.

Species mean acute and chronic values for 2,4-dichlorophenol are listed in Table 3.

Plant Effects

The toxicity of 2,4-dichlorophenol to freshwater aquatic plants does not appear important in the derivation of a criterion since deleterious on plants (Table 4) occurred only at much higher concentrations than those which produced acute toxic effects on fish and invertebrate species. However, the knowledge that 2,4-dichlorophenol is not highly toxic to plants is important because 2,4-dichlorophenol is used in producing the commonly used herbicide, 2,4-D (2,4-dichlorophenoxyacetic acid). Some observed toxic effects of 2,4-dichlorophenol on plants were the complete destruction of chlorophyll in Chlorella pyrenoidosa at 100,000 µg/l (Huang and Gloyna, 1968) and a 50 percent reduction in chlorophyll in Lemna minor at 58,320 µg/l (Blackman, et al. 1955). Huang and Gloyna (1968) also determined that there

was a 56.4 percent reduction of photosynthetic oxygen production in Chlorella pyrenoidosa after exposure to 50,000 µg/l for 120 minutes.

Residues

No measured steady-state bioconcentration factor (BCF) is available for 2,4-dichlorophenol.

Miscellaneous

Birge, et al. (1979) determined LC_{50} values for three fish species after embryo exposures and after additional 4-day larval exposures at hardnesses of 50 and 200 mg/l as $CaCO_3$ (Table 5). The LC_{50} values after the 4-day larval exposures were 80 and 70 µg/l for rainbow trout, 390 and 260 µg/l for goldfish, and 1,350 and 1,070 µg/l for channel catfish at hardnesses does not substantially affect toxicity, although for all species tested the LC_{50} values for 2,4-dichlorophenol were slightly higher at low hardness than at high hardness. The LC_{50} value of 260 µg/l for goldfish (Birge, et al. 1979), which was based on an 8-hour total exposure of embryos and larvae, was considered to be the lowest freshwater acute value for 2,4-dichlorophenol. Rainbow trout embryos and larvae exposed in this study were fathead minnows since the chronic value for fatheads is 365 µg/l (Holcombe, et al. Manuscript). Since the rainbow trout LC_{50} of 70 µg/l (Birge, et al. 1979) was based on a relatively long-term study (24-day embryo exposure plus a 4-day larval exposure), this value was considered to be the lowest chronic value.

Flavor-impairment studies (Shumway and Palensky, 1973) showed that flesh tainting occurred when 2,4-dichlorophenol concentrations ranging from 0.4 µg/l to 14 µg/l, depending on the species of fish tested, were exceeded (Table 5). Based on the available data for 2,4-dichlorophenol, flavor-impairment in fish occurs at lower concentrations than other effects used to

evaluate toxicity and may be an important consideration in deriving a criterion. Since the purpose of the Guidelines is to set water quality criteria which protect both the presence and uses of aquatic life, a criterion which will protect against tainting of fish flesh is necessary to preserve the quality of the freshwater fishery. However, the lack of toxicity data for 2,4-dichlorophenol makes it impossible to ascertain if a criterion based on flavor-impairment of fish flesh would be low enough to protect all freshwater aquatic organisms.

The only saltwater data available on the effects of 2,4-dichlorophenol are from an acute exposure to mountain bass, a species endemic in Hawaii (Hiatt, et al. 1953). Abnormal behavioral responses, including rapid swimming in a vertical position, gulping at the surface of the water, and jerky motions, were observed in a nominal concentration of 20,000 µg/l.

Summary

Acute effects on freshwater fish and invertebrate species were observed at concentrations from 260 to 8,230 µg/l. The chronic value for the fathead minnow was 365 µg/l with an acute-chronic ratio of 23. An embryo and 4-day larval exposure of rainbow trout yielded an LC_{50} value of 70 µg/l. The lowest plant effect (50,000 µg/l) caused by exposure to 2,4-dichlorophenol was based on a reduction in photosynthetic oxygen production in an algal species. Effects on flavor-impairment of largemouth bass occurred when concentrations of 2,4-dichlorophenol exceeded 0.4 µg/l.

CRITERIA

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

life stages of one species of fish occurs at concentrations as low as 70 µg/l.

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

Table 1. Acute values for 2,4-dichlorophenol

Species	Method*	LC50/EC50 (µg/l)	Species Mean Acute Value (µg/l)	Reference
		FRESHWATER SPECIES		
Cladoceran, *Daphnia magna*	S, U	2,610	-	Kopperman, et al. 1974
Cladoceran, *Daphnia magna*	S, U	2,600	2,605	U.S. EPA, 1978
Fathead minnow (juvenile), *Pimephales promelas*	FT, M	8,230	8,230	Phipps, et al. Manuscript
Bluegill, *Lepomis macrochirus*	S, U	2,020	2,020	U.S. EPA, 1978

* S = static, FT = flow-through, U = unmeasured, M = measured

No Final Acute Values are calculable since the minimum data base requirements are not met.

Table 2. Chronic values for 2,4-dichlorophenol (Holcombe, et al. Manuscript)

Species	Method*	Limits (µg/l)	Species Mean Chronic Value (µg/l)
FRESHWATER SPECIES			
Fathead minnow, Pimephales promelas	ELS	290-460	365

* ELS = early life stage

Acute-Chronic Ratio

Species	Chronic Value (µg/l)	Acute Value (µg/l)	Ratio
Fathead minnow, Pimephales promelas	365	8,230	23

Table 3. Species mean acute and chronic values for 2,4-dichlorophenol

Number	Species	Species Mean Acute Value* (µg/l)	Species Mean Chronic Value (µg/l)	Acute-Chronic Ratio**
		FRESHWATER SPECIES		
3	Fathead minnow, *Pimephales promelas*	8,230	365	23
2	Cladoceran, *Daphnia magna*	2,605	-	-
1	Bluegill, *Lepomis macrochirus*	2,020	-	-

* Rank from high concentration to low concentration by species mean acute value.

**See the Guidelines for derivation of this ratio.

Table 4. Plant values for 2,4-dichlorophenol

Species	Effect	Result (µg/l)	Reference
	FRESHWATER SPECIES		
Alga, *Chlorella pyrenoidosa*	Complete destruction of chlorophyll	100,000	Huang & Gloyna, 1968
Alga, *Chlorella pyrenoidosa*	56.4% reduction of photosynthetic oxygen production	50,000	Huang & Gloyna, 1968
Duckweed, *Lemna minor*	50% reduction in chlorophyll	58,320	Blackman, et al. 1955

Table 5. Other data for 2,4-dichlorophenol

Species	Duration	Effect	Result (µg/l)	Reference
		FRESHWATER SPECIES		
Lymnaeid snails, Pseudosuccinea columella Fossaria cubensis	24 hrs	100% mortality	10,000	Batte & Swanson, 1952
Crayfish, Orconectes propinquus Orconectes immunis Cambarus robustus	48 hrs	100% mortality	10,000	Telford, 1974
Crayfish, Orconectes propinquus Orconectes immunis Cambarus robustus	1 wk	100% mortality	5,000	Telford, 1974
Crayfish, Orconectes propinquus Orconectes immunis Cambarus robustus	10 days	14% mortality	1,000	Telford, 1974
Crayfish, Orconectes propinquus Orconectes immunis Cambarus robustus	10 days	Increased blood glucose levels	1,000	Telford, 1974
Rainbow trout, Salmo gairdneri	48 hrs	ETC*	1	Shumway & Palensky, 1973
Rainbow trout, Salmo gairdneri	24-day embryo exposure	LC50 at hardness of 50 mg/l $CaCO_3$	80	Birge, et al. 1979
Rainbow trout, Salmo gairdneri	24-day embryo exposure	LC50 at hardness of 200 mg/l $CaCO_3$	70	Birge, et al. 1979
Rainbow trout, Salmo gairdneri	24-day embryo plus 4-day larval exposure	LC50 at hardness of 50 mg/l $CaCO_3$	80	Birge, et al. 1979
Rainbow trout, Salmo gairdneri	24-day embryo plus 4-day larval exposure	LC50 at hardness of 200 mg/l $CaCO_3$	70	Birge, et al. 1979

Table 5. (Continued)

Species	Duration	Effect	Result (µg/l)	Reference
Goldfish, *Carassius auratus*	4-day embryo exposure	LC50 at hardness of 50 mg/l $CaCO_3$	1,760	Birge, et al. 1979
Goldfish, *Carassius auratus*	4-day embryo exposure	LC50 at hardness of 200 mg/l $CaCO_3$	1,240	Birge, et al. 1979
Goldfish, *Carassius auratus*	4-day embryo plus 4-day larval exposure	LC50 at hardness of 50 mg/l $CaCO_3$	390	Birge, et al. 1979
Goldfish, *Carassius auratus*	4-day embryo plus 4-day larval exposure	LC50 at hardness of 200 mg/l $CaCO_3$	260	Birge, et al. 1979
Goldfish, *Carassius auratus*	24 hrs	LC50	7,800	Kobayashi, et al. 1979
Fathead minnow (juvenile), *Pimephales promelas*	192 hrs	LC50	6,500	Phipps, et al. Manuscript
Channel catfish, *Ictalurus punctatus*	4-day embryo exposure	LC50 at hardness of 50 mg/l $CaCO_3$	1,850	Birge, et al. 1979
Channel catfish *Ictalurus punctatus*	4-day embryo exposure	LC50 at hardness of 200 mg/l $CaCO_3$	1,700	Birge, et al. 1979
Channel catfish, *Ictalurus punctatus*	4-day embryo plus 4-day larval exposure	LC50 at hardness of 50 mg/l $CaCO_3$	1,350	Birge, et al. 1979
Channel catfish, *Ictalurus punctatus*	4-day embryo plus 4-day larval exposure	LC50 at hardness Of 200 mg/l $CaCO_3$	1,070	Birge, et al. 1979
Bluegill, *Lepomis macrochirus*	48 hrs	ETC*	14	Shumway & Palensky, 1973
Largemouth bass, *Micropterus salmoides*	48 hrs	ETC*	0.4	Shumway & Palensky, 1973

Table 5. (Continued)

Species	Duration	Effect	Result (µg/l)	Reference
		SALTWATER SPECIES		
Mountain bass** Kuhlia sandvicensis	Acute response	Moderate reaction	20,000	Hiatt, et al. 1953

* ETC = the highest estimated concentration of material that will not impair the flavor of the flesh of exposed fish.

**Endemic in Hawaii

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Mammalian Toxicology and Human Health Effects

EXPOSURE

Ingestion from Water

Absorption of 2,4-dichlorophenol (herein referred to as 2,4-DCP in this document) by biological tissues may occur upon exposure to 2,4-DCP either dissolved in water or associated with suspended matter or sediments in water. Chlorophenols such as 2,4-DCP are weak acids that become increasingly ionized under alkaline conditions; they are mainly non-ionized at physiological pH. These properties, together with the lipophilic nature of chlorophenols (including 2,4-DCP), make it likely that chlorophenols would be absorbed from the gastrointestinal tract.

Sources of 2,4-DCP in water may be diffuse (e.g., agricultural runoff) or localized (e.g., point source pollution from manufacturing waste discharges). Sidwell (1971) verified the presence of 2,4-DCP in 2,4-dichlorophenoxyacetic acid (2,4-D) manufacturing wastes. Over a 7-month period, total chlorophenol content ranged from 68 mg/l of waste to a high of 125 mg/l, with 2,4-DCP content ranging as high as 89 percent of the total. Details of these findings are presented in Table 1.

If certain assumptions are made, the data of Sidwell (1971) can be used to estimate human exposure to 2,4-DCP from drinking water obtained below a point source of this type. Assuming (1) an effluent with a 2,4-DCP concentration of 125 mg/l in water containing 100,000 mg/l of total solids (Table 1), (2) dilution in the watercourse occurring to the point where an acceptable freshwater total solids concentration of 1,000 mg/l is reached, and (3) no removal of 2,4-DCP by water treatment, drinking water will contain

TABLE 1

Industrial Plant Effluent Content of Chlorophenols[a]

Date Sampled	25 Jan	3 Mar	21 Apr	28 May	27 Aug
Total Solids (mg/l)	6,960	40,100	76,320	104,860	11,000
Temperature (^{o}C)	12	18	21	28.5	24
pH	7.5	7.6	7.4	7.4	7.0
Chlorophenols (mg/l)	68	118	125	112	74
Phenol Type (%)[b]					
Phenol-	3.4	6.2	1.7	24.8	trace
2-chloro-	2.9	6.1	trace	trace	trace
2,4-dichloro-	73.6	17.9	20.0	11.4	89.0
2,6-dichloro-	9.9	41.7	38.8	30.5	3.0
2,5-dichloro-	trace	6.2	1.7	trace	1.8
4-chloro-	2.5	12.1	18.3	20.0	2.8
2,4,6-trichloro-	2.8	9.9	19.5	13.3	3.4
2,4,5-trichloro-	4.7	trace	trace	trace	trace

[a]Modified from Sidwell, 1971

[b]Percent of total phenols present

2,4-DCP at an estimated concentration of 1.25 mg/l. If the daily water intake of a 70 kg person is assumed to be 2 liters, this would result in a daily 2,4-DCP dosage of 0.036 mg/kg body weight/day. It should be emphasized that this dosage is a worst case exposure level that would probably occur only in drinking water obtained below a 2,4-DCP-contaminated point source discharge.

Sharpee (1973) noted the presence of 2,4-DCP in soil treated with 2,4-dichlorophenoxyacetic acid (2,4-D). This presence may be accounted for by the photolytic or microbial breakdown of 2,4-D which will be discussed later in this document. Walker (1961) studied the contamination of ground water by the migration of waste products from the manufacture of chemicals at the Rocky Mountain Arsenal, Denver, Colorado. Walker reported the phytotoxic properties of water caused by either 2,4-D or an unnamed "closely related compound." The 2,4-D type compounds were not a direct product of the Arsenal operations, but rather were apparently the result of chemical reactions which occurred within basins used for the storage of effluents from a variety of Arsenal operations.

If diffuse or point source contamination of waters with 2,4-DCP is in fact occurring, the dissipation of this compound in aquatic environments becomes an important consideration. The major avenues of 2,4-DCP dissipation that have been studied are microbial degradation and photodecomposition. Aly and Faust (1964) examined the dissipation of 2,4-DCP from natural lake waters at a buffered pH of 7. In aerated lake waters, with initial 2,4-DCP concentrations of 100, 500, and 1,000 µg/l, the percentages of 2,4-DCP remaining at 9 days were 0, 0.34, and 46 respectively. By contrast, initial concentrations of 100, 500, and 1,000 µg/l in unaerated and

unbuffered waters resulted in percentages of 40, 51.6, and 56, respectively, remaining at 17 days. Aly and Faust concluded that the persistence of chlorophenol would tend to increase at lower pH and under anaerobic conditions that might result from the decomposition of excessive organic matter.

Ingols, et al. (1966) studied the degradation of various chlorophenols by activated sewage sludge and concluded that 2,4-DCP was degraded more rapidly by activated systems with previous exposure to chlorophenols than by those with no previous exposure to chlorophenols. When activated sludge was exposed to 2,4-DCP at levels of 100 mg/l of sludge, 75 percent of the chemical disappeared in two days, and essentially 100 percent was gone in five days. Hemmett (1972) showed that microorganisms acclimated to the herbicide 2,4-D could degrade 2,4-DCP without a lag period, implying similar biological pathways for the two compounds.

When 2,4-DCP was subjected to aeration basin treatment, the initial concentration of 64 mg/l dropped to an undetectable level within five days (Sidwell, 1971); this was more rapid than the rate of degradation observed for 2,4-DCP in distilled water. When an aerated lagoon alone was used, removal of all chlorophenols varied from 55 to 89 percent. Overall, removal using both lagoon and stabilization ponds ranged from 87 to 94 percent. Thus, natural degradation of 2,4-dichlorophenol may be enhanced by proper application of effluent waste management principles.

Dissipation of dichlorophenols also occurs through photodecomposition in aqueous solutions. Aly and Faust (1964) demonstrated that (1) 2,4-DCP was decomposed by ultraviolet light, and

(2) the rate of photolysis in distilled water decreased as pH decreased. Degradation of 50 percent of 2,4-DCP by ultraviolet light was accomplished in two minutes at pH 9.0, in five minutes at pH 7.0, and in 34 minutes at pH 4.0. The studies of Aly and Faust were conducted with light of wavelength 253.7 nm, which is slightly shorter than the natural ultraviolet radiation wavelength range of 292 to 400 nm.

The riboflavin-sensitized dimerization of 2,4-dichlorophenol to tetrachlorodiphenyl ethers, tetrachlorodihydroxy-biphenyls, and other products was reported by Plimmer and Klingebiel (1971). Chlorinated dibenzo-p-dioxins, which could have resulted from ring closure of these tetrachlorodiphenyl ethers, were not detected in the products of photolysis. The authors speculated that failure to detect chlorinated dibenzo-p-dioxins may have been due to the rapid photolytic breakdown of those dioxins. Rapid photolysis of chlorinated dibenzo-p-dioxins was confirmed by Crosby, et al. (1971).

That 2,4-dichlorophenol can be formed as a photolytic product of the herbicides 2,4-D and nitrofen (2,4-dichlorophenyl p-nitrophenyl ether) in aqueous suspension under sunlight or simulated sunlight, has been noted by several investigators, including Aly and Faust (1964), Zepp, et al. (1975), and Nakagawa and Crosby (1974a,b). Crosby and Tutass (1966) irradiated 2,4-D with artificial light (254 nm) and natural sunlight and observed that under both conditions 2,4-dichlorophenol was formed as a photolytic product of 2,4-D and was further degraded to 4-chlorocatechol. 2,4-DCP was photolabile, with 50 percent being lost in five minutes at pH 7.0.

Microbial decomposition of 2,4-dichlorophenol in soils and aquatic environments has been extensively studied. Alexander and A'eem (1961) found that when 80 µg 2,4-DCP/ml medium was incubated in the presence of Dunkirk silt loam and Mardin silt loam, 2,4-DCP was not detectable after nine and five days, respectively. When these soil preparations were treated with sodium azide, no disappearance of 2,4-DCP was noted, supporting the role of microbiological processes in the degradation. Loos, et al. (1967) found that extracts of Arthrobacter sp. contained enzymes capable of dehalogenating 2,4-dichlorophenol. Degradation was rapid, with 100 percent chloride release from 2,4-DCP occurring after four hours of incubation. The cells responsible for the dehalogenating process were active when cultured on the herbicides 2,4-D or 2-methyl-4-chlorophenoxyacetate (MCPA). Kearney, et al. (1972) found no tetrachlorodibenzo-p-dioxin in soils treated with up to 1,000 µg 2,4-DCP/g. Sharpee (1973) measured 2,4-DCP present in the soil as a consequence of 2,4-D application and found that 2,4-DCP did appear in soil treated with 2,4-D, but it did not persist as long as the 2,4-D.

As indicated earlier, the principal source of 2,4-dichlorophenol in soils is believed to be the herbicide 2,4-D. The various intermediates (including 2,4-DCP) in the microbial metabolism of 2,4-D have been characterized by several investigators (Spokes and Walker, 1974; Loos, et al., 1967; Bollag, et al., 1968; Evans, et al., 1971; Paris and Lewis, 1973; Ingols, et al., 1966; Alexander and Aleem, 1961). Kearney and Kaufman (1972) have shown that the organisms capable of degrading 2,4-D to 2,4-DCP continue the degra-

dation process to catechol intermediates and finally to succinic acid. Thus, the herbicide 2,4-D is eventually biodegraded to an ecologically acceptable product, succinic acid.

Recently, attention has focused on a potential chlorophenol source of a more ubiquitous nature than herbicide and pesticide applications. Both municipal and industrial wastewater are often subjected to chlorination to achieve disinfection and deodorization (Barnhart and Campbell, 1972). One result of chlorination is the reaction of chlorine with phenol to produce chlorophenols, some of which are a source of obnoxious odors and/or taste (Dietz and Traud, 1978; Barnhart and Campbell, 1972; Burttschell, et al. 1959; Hoak, 1957).

Phenol has been observed to be quite reactive with chlorine in dilute aqueous solutions. This high reactivity of phenol is attributed to the ring-activating, electron-releasing properties of the -OH functional group (Barnhart and Campbell, 1972; Morris, 1978). Halogen substitution is favored in the ortho- and para-positions (Burttschell, et al. 1959).

Chlorination of phenols results in a stepwise substitution of the 2, 4, and 6 positions of the aromatic ring. Barnhart and Campbell (1972) felt that it was probable that chlorination resulted in a complex mixture of chlorophenols. Burttschell, et al. (1959) chlorinated 1 liter of 20 mg phenol/ml solution containing 2 g/l sodium bicarbonate with 40 mg chlorine and isolated 2,4-DCP as one of three major chlorophenols. Relative chlorophenol content determined in this study was as follows:

Component	Percent of Product
Phenol	1-2
2-Chlorophenol	2-5
4-Chlorophenol	2-5
2,4-Dichlorophenol	20
2,6-Dichlorophenol	25
2,4,6-Trichlorophenol	40-50

(Absolute amounts of chlorophenols were not reported.)

Lee and Morris (1962) verified the stepwise chlorination of phenol to chlorophenolic compounds. Furthermore, they determined that the reaction rate and yield of 2,4-DCP is quite pH dependent, with a decrease in generation time and an increase in yield for 2,4-DCP as pH increases from 7 to 9. The reactions occurred at initial chlorine concentrations of 1 ug/g and phenol concentrations of 50 ng/g.

Jolley (1973) indicated that development of chlorinated organics, including chlorinated phenols, is retarded in solutions with high ammonia concentrations. In a later study, Jolley, et al. (1978) examined the chlorination of sewage waters under conditions simulating those used for disinfection of sewage effluents and/or antifoulant treatment of cooling waters of electric power generating plants. Over 50 chloro-organic constituents were separated in each analysis of concentrated sewage effluent chlorinated in the laboratory with 2.5 to 6 mg/l concentrations of chlorine. Similar studies were also done on two bodies of lake water, each receiving effluent from a coal-fired, electric-power generating plant. In the three systems examined, Jolley, et al. (1978) detected monochlorophenols at ng/g levels but found no dichlorophenols.

Glaze, et al. (1978) identified trichlorophenols, but found no dichlorophenols, in superchlorinated municipal wastewaters. Thus, in contrast to laboratory demonstrations of DCP formation, recent work under conditions simulating the natural environment has not established that 2,4-DCP is a significant product resulting from chlorination of phenol-containing waters.

Ingestion from Food

Any 2,4-dichlorophenol contamination of food products (non-aquatic) would probably result from use of the herbicide 2,4-D. As stated earlier, 2,4-DCP is a possible contaminant of 2,4-D, as well as an intermediate compound in the biological and photolytic degradations of 2,4-D.

Absorption of 2,4-DCP has been reported for some plant species (Isensee and Jones, 1971). Total DCP content in oat and soybean plants increased for approximately three weeks when the soil contained a DCP concentration of 0.07 µg/g. As these plants grew, the total tissue content of DCP remained relatively constant but decreased as the plants matured. At the time of harvest, oats contained 0.01 µg of DCP per gram of plant tissue, and soybeans contained 0.02 µg/g. The 2,4-DCP did not seem to concentrate in the plant seeds. DCP was not detected in the grain of these oat plants, and the soybean seeds contained only 1 to 2 percent of the total plant DCP. No evidence was found of DCP translocation in soybeans after foliar application.

The conversion of 2,4-D to 2,4-DCP has been demonstrated in sunflowers, corn, barley, strawberries, and kidney beans. Steen, et al. (1974) found that 2,4-DCP residues in plants treated with

2,4-D herbicide were from 20 to 100 times lower than residues of 2,4-D. Related studies by Sokolov, et al. (1974) involved application of 2,4-D to rice fields. At harvest, rice grain contained neither 2,4-D nor 2,4-DCP, even though 2,4-DCP was present in the rice plant. The 2,4-DCP content in potato tubers treated with 2,4-D amounted to less than 10 percent of the total 2,4-D content.

There is little information on the transfer of 2,4-D or its degradation compounds to food products of animal origin. Mitchell, et al. (1946) provided evidence for the gastrointestinal absorption of 2,4-D in lactating dairy cows and detected the herbicide in blood serum during a 106-day oral dosing study. However, 2,4-D was not detected in the milk. More recently, 2,4-D was not found (detection limit 0.1 µg/g) in bovine milk following oral doses of 5 µg 4-(2,4-dichlorophenoxybutyric) acid [4-(2,4-DB)] (Gutenmann, et al. 1963a) or 2,4-D (Gutenmann, et al. 1963b) or 50 µg 2,4-D (Bache, et al. 1964a) per gram of feed.

Clark, et al. (1975) studied the tissue distribution of 2,4-DCP in sheep and cattle fed 2,4-D at the relatively high concentrations of 300, 1,000, and 2,000 µg/g of feed. Given that the daily ration is approximately 3 percent of the body weight, these feed concentrations are equivalent to approximate dosages of 9, 30, and 60 mg/kg body weight, respectively. The treated diets were fed for 28 days, and resulting concentrations of 2,4-D and 2,4-DCP in several edible tissues were determined (Table 2). With a detection limit of 0.05 µg/g, analysis did not detect 2,4-DCP in the fat or muscle of cattle and sheep, even at the highest dose levels. However, the kidney and liver were found to contain large amounts of

TABLE 2

2,4-D and 2,4-DCP Residues (in mg/kg)
in Sheep and Cattle Fed 2,4-D[a]

	2,4-D (Dose mg/kg body weight/day)				
	Sheep		Cattle		
Compound	60	60[b]	9	30	60
		Muscle			
2,4-D	0.06	<0.05	<0.05	<0.05	0.07
2,4-DCP	<0.05	<0.05	<0.05	<0.05	<0.05
		Fat			
2,4-D	0.10	0.15	0.13	0.45	0.34
2,4-DCP	<0.05	<0.05	<0.05	<0.05	<0.05
		Liver			
2,4-D	0.98	0.29	<0.05	0.14	0.23
2,4-DCP	0.16	0.15	0.11	0.59	0.31
		Kidney			
2,4-D	9.17	0.37	2.53	8.67	10.9
2,4-DCP	0.26	0.07	0.56	1.17	1.06

[a]Modified from Clark, et al. 1975

[b]Dosed 28 days, then withdrawn from 2,4-D for 7 days

2,4-DCP proportional to the dose given. Furthermore, when sheep were withdrawn from 2,4-D for one week, measurable amounts of 2,4-DCP were still detected. Based on the limited evidence presented, the liver appeared to retain 2,4-DCP for longer periods than did the kidney. The data presented do not allow accurate calculation of a depletion rate, nor can the total time period of measurable residues be ascertained.

Sherman, et al. (1972) fed technical grade Nemacide® [O-(2,4-dichlorophenyl)-O,O-diethyl phosphorothioate] at 50, 100, 200, and 800 µg/g of feed to laying hens for 55 weeks. Analysis for 2,4-DCP (a metabolite of Nemacide®) by gas-liquid chromatography (detection limit 0.006 to 0.208 µg/g) resulted in detection of 2,4-DCP residues in liver and yolk, but not in muscle or fat. Details of the analytical findings are presented in Table 3. As in the studies of Clark, et al. (1975), there does appear to be some predilection of 2,4-DCP for liver, even when formed by biotransformation from two parent compounds. In the hens studied, liver 2,4-DCP concentration decreased as the dosage of Nemacide® was decreased. The highest mean liver level of 2,4-DCP found in hens was 0.56 µg/g, identical to a mean level of 0.56 µg/g found in the kidneys of cattle fed 300 µg 2,4-D/g of feed (the dosage level that most closely approximates possible field exposure) by Clark and coworkers (1975). It should be noted that the Sherman study was strictly a laboratory study, using conditions that are not likely to occur in the field. However, in a worst-case exposure situation, the consumption of 2,4-DCP-contaminated chicken liver and cattle kidney could result in approximately equivalent human exposure.

TABLE 3

2,4-Dichlorophenol Residues in Laying Hens fed Nemacide®
[O-(2,4-dichlorophenol)-O,O-diethyl phosphorothioate][a]

Nemacide® Feed Concentration in ppm	Days since Withdrawal from Nemacide®	Residues of 2,4-DCP (ppm) mean (and range)	
		Liver	Egg Yolk
800	0	0.47(0.14-0.68)	0.61(0.34-0.75)
	5	---	0.27
	7	0.50(0.25-0.75)	
	10	---	<0.12
	14	0.27(0.06-0.48)	
	21	0.19(0.11-0.27)	
200	0	0.38(0.31-0.44)	0.15(0.12-0.17)
	7	0.30(0.24-0.35)	
	14	0.14(0.10-0.18)	
	21	0.36(0.07-0.64)	
100	0	0.26(0.25-0.26)	0.15(<0.12-0.21)
	7	0.56	
	14	0.15(<0.05-0.33)	
	21	0.05	
50	0	0.31(0.16-0.46)	<0.12
	7	0.18(0.14-0.22)	
	14	0.06(<0.05-0.11)	
	21	0.05	

[a]Modified from Sherman, et al. 1972

Bjerke, et al. (1972) dosed dairy cows for three weeks with 2,4-D concentrations as high as 1,000 mg/kg of diet. 2,4-DCP was not found in the milk or cream of treated cows.

The results of Clark, et al. (1975) can be used to calculate a worst case estimate for the degree of human exposure to 2,4-DCP from consumption of contaminated meat. Assuming (1) an average forage yield of two tons (1,818 kg) per acre, (2) the retention of all herbicide on the treated plants, (3) an application rate of 1 lb (454 gms) of 2,4-D per acre, and (4) consumption by an animal of 3 percent of its body weight per day in forage, then the dosage delivered to an animal eating forage contaminated with this level of 2,4-D would be approximately 7 mg 2,4-DCP/kg body weight. This amount corresponds roughly to the lowest dosage (9 mg/kg body weight) that was administered to cattle and sheep by Clark and his colleagues. Based on the results of Clark, et al., it is reasonable to expect that cattle fed a constant diet of forage contaminated with 2,4-D applied at commercial rates would accumulate 2,4-DCP concentrations of approximately 0.11 µg/g of liver and 0.56 µg/g of kidney.

If a 70 kg person consumed 0.5 kg of kidney daily at a 2,4-DCP residue concentration of 560 µg/kg, that person would be consuming approximately 280 ug of 2,4-DCP, or 4.0 ug/kg body weight, daily. If that person also ingested 36 µg 2,4-DCP/kg body weight/day in contaminated drinking water, as calculated from 2,4-DCP levels in water downstream from a 2,4-DCP manufacturing plant (see Ingestion from Water section), the resultant daily 2,4-dichlorophenol dosages would total 40 µg/kg. It is therefore clear that the water would

contribute 90 percent of this highest calculated daily dosage of 2,4-DCP.

It should be emphasized that an exposure level of 4 µg 2,4-DCP/kg body weight/day is a worst case example for food intake. It would only occur if a person (1) ate 0.5 kg of kidney per day and (2) all the kidney consumed was contaminated with 0.11 µg 2,4-DCP/g. This contamination level would probably occur only if the cattle were fed a constant diet of 2,4-D-sprayed forage, since experimental evidence (Clark, et al. 1975; Zielinski and Fishbein, 1967) indicates that levels of 2,4-DCP in animal tissue diminish rapidly following withdrawal of 2,4-D from the diet.

A bioconcentration factor (BCF) relates the concentration of a chemical in aquatic animals to the concentration in the water in which they live. The steady-state BCFs for a lipid-soluble compound in the tissues of various aquatic animals seem to be proportional to the percent lipid in the tissue. Thus, the per capita ingestion of a lipid-soluble chemical can be estimated from the per capita consumptions of fish and shellfish, the weighted average percent lipids of consumed fish and shellfish, and a steady-state BCF for the chemical.

Data from a recent survey on fish and shellfish consumption in the United States were analyzed by SRI International (U.S. EPA, 1980). These data were used to estimate that the per capita consumption of freshwater and estuarine fish and shellfish in the United States is 6.5 g/day (Stephan, 1980). In addition, these data were used with data on the fat content of the edible portion of the same species to estimate that the weighted average percent

lipids for consumed freshwater and estuarine fish and shellfish is 3.0.

No measured steady-state bioconcentration factor is available for 2,4-dichlorophenol, but the equation "Log BCF = (0.85 Log P) - 0.70" (Veith, et al. 1979) can be used to estimate from the octanol-water partition coefficient (P) the BCF for aquatic organisms that contain about 7.6 percent lipids (Veith, 1980). Based on an average log P value of 3.19 (Hansch and Leo, 1979), the steady-state bioconcentration factor for 2,4-dichlorophenol is estimated to be 103. An adjustment factor of 3.0/7.6 = 0.395 can be used to adjust the estimated BCF from the 7.6 percent lipids on which the equation is based to the 3.0 percent lipids that is the weighted average for consumed fish and shellfish. Thus, the weighted average bioconcentration factor for 2,4-dichlorophenol and the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is calculated to be 103 x 0.395 = 40.7.

Inhalation

There is no direct evidence to indicate that humans are exposed to significant amounts of 2,4-DCP through inhalation. Although the compound is volatile, no quantitative studies of inhalation exposure or general environmental contamination have been found.

Dermal

Dermal exposure to 2,4-DCP would most likely occur during the manufacture, transport, or handling of the compound. Due to its lipophilic nature and low degree of ionization at physiologic pH, absorption of 2,4-DCP would be expected; however, no data relating to dermal absorption have been found.

PHARMACOKINETICS

Absorption

No information concerning the direct absorption of 2,4-dichlorophenol in humans or animals was found. Toxicity data to be developed later confirm the existence of systemic toxicosis, indicating that 2,4-DCP is absorbed by several routes.

Because of their high lipid solubility and low ionization at physiological pH, dichlorophenols would be expected to be readily absorbed following ingestion.

Distribution

No information was found concerning distribution of 2,4-DCP in man. The previously discussed (see Ingestion from Food section) animal studies (Clark, et al. 1975; Sherman, et al. 1972) demonstrated distribution of 2,4-DCP in liver, kidney, and egg (see Tables 2 and 3).

Metabolism

The biotransformation of 2,4-DCP in humans has not been reported. No information could be found concerning the metabolism of 2,4-DCP administered directly to experimental animals. However, a limited amount of information on the metabolism of 2,4-DCP derived from administration of gamma and beta benzene hexachloride (BHC) to mice was reported by Kurihara (1975). Mice were given C^{14}-labeled gamma- or beta-BHC by intraperitoneal injection, and the appearance of metabolites in the urine was monitored. 2,4-DCP and 2,4-DCP conjugates were found and identified primarily as glucuronides and sulfates (Table 4). Administration of gamma-BHC resulted in a majority of the 2,4-DCP being conjugated as the glucuronide, while beta-BHC administration resulted in a greater amount of sulfate

TABLE 4

Urinary 2,4-DCP Metabolites of Benzene Hexachloride in Mice[a]

	Form of 2,4-DCP		
BHC Isomer	Glucuronide	Sulfate	Total
gamma-BHC	4-5%[b]	0-1%	4-6%
beta-BHC	1-2%	3%	4-5%

[a]Source: Kurihara, 1975

[b]Percent of total metabolites

conjugate. Assuming that the mouse biotransforms 2,4-DCP resulting from endogenous metabolism in a manner similar to 2,4-DCP directly administered, then sulfate and glucuronide conjugation appear to be major metabolic pathways.

Excretion

2,4-D has been found to be rapidly excreted in the urine of mice (Zielinski and Fishbein, 1967), rats (Khanna and Fang, 1966), sheep (Clark, et al. 1964), and swine (Erne, 1966a,b) under various dosing conditions. The phenoxy herbicide MCPA was also rapidly excreted in cattle urine (Bache, et al. 1964b). However, excretion data from studies using 2,4-DCP are not extensive, and no information was found for 2,4-DCP excretion in man.

Karapally, et al. (1973) found that when rabbits were given radioactive gamma-BHC, 2.5 percent of total radioactivity in the urine was due to 2,4-DCP. Data presented did not allow for determination of body burden or half-life. Shafik, et al. (1973) administered a daily dose of Nemacide® in peanut oil orally to rats for three days. After administration of 1.6 mg Nemacide®, 67 percent of that compound appeared in urine as 2,4-DCP within three days. With a dosage of 0.16 mg Nemacide®, 70 percent of the pesticide appeared as 2,4-DCP within 24 hours. Work cited earlier (Kurihara, 1975) indicated the appearance of metabolites of 2,4-DCP in urine as a result of gamma- and beta-BHC administration (see Table 4).

EFFECTS

Acute, Subacute, and Chronic Toxicity

Farquharson, et al. (1958) indicated that the toxicity of chlorophenols tends to increase as chlorination is increased.

The mechanism of toxic action for 2,4-DCP in mammalian systems in vivo has not been well defined. Limited in vitro studies indicate two potential actions. 2,4-DCP inhibits oxidative phosphorylation in rat liver mitochondria and rat brain homogenates (Farquharson, et al. 1958; Mitsuda, et al. 1963). According to Mitsuda, et al. (1963), inhibitory activity of chlorophenols was roughly correlated with the dissociation constant of the inhibitor. In addition, chlorine atoms on the ortho position weakened the activity of mono- and dichlorophenols as oxidative inhibitors. A concentration of 4.2×10^{-5} M 2,4-DCP inhibited oxidative phosphorylation by 50 percent in rat liver mitochondria. By comparison, pentachlorophenol was approximately 40 times more active, and dinitrophenol was twice as active in inhibiting oxidative phosphorylation.

Stockdale and Selwyn (1971) reported observations suggesting that the phenol-induced mediation of the passage of protons across the inner mitochondrial membrane is sufficient to cause uncoupling of oxidative phosphorylation. They further noted that phenols have direct effects on the enzyme ATPase as well as on one or more components of the electron transport system; however, neither of these effects is actually involved in the uncoupling process.

Farquharson, et al. (1958) offered the conclusion that chlorophenols with pK values of 7.85 or less appear to be acutely associated with production of marked hypotonia and early onset of rigor mortis after death. Similar clinical effects are associated with well known oxidative uncouplers, such as 2,4-dinitrophenol and pentachlorophenol. Relatively few studies of the acute or subacute toxicity of 2,4-DCP have been reported. The acute LD_{50} values determined by several investigators are presented in Table 5.

TABLE 5

Acute Mammalian Toxicity of 2,4-DCP

Species	Route of Administration	LD_{50} (mg/kg)	Reference
Rat	Oral	580	Deichmann, 1943
Rat	Subcutaneous	1,730	Deichmann, 1943
Rat	Intraperitoneal	430	Farquharson, et al. 1958
Rat	Oral	4,000	Kobayashi, et al. 1972
Mouse	Oral	1,600	Kobayashi, et al. 1972

In the study by Farquharson, et al. (1958), acute poisoning of rats following intraperitoneal 2,4-DCP injection appeared to be characterized by the onset of hypotonia two to three minutes after dosing. This effect began in the hindlimbs and moved forward until the rats were prostrate. Eye reflexes were weakened and there was no withdrawal from toe pinch. Muscle twitches rarely occurred spontaneously and could not be evoked by auditory or tactile stimuli. Rectal temperature was only slightly decreased. Initial dose-induced polypnea was followed by slowed respiration and dyspnea as coma ensued. Rigor mortis appeared earlier in rats killed with 2,4-DCP than in control rats killed with ether.

The oral LD_{50} derived by Deichmann (1943) appears at odds with the findings of Kobayashi, et al. (1972). Deichmann used fuel oil as a solvent, which may have enhanced rapid uptake of 2,4-DCP. The vehicle for the Kobayashi studies could not be determined. Nonetheless, from the LD_{50} values, it appears that 2,4-DCP would constitute an acute hazard only following massive exposure.

In a subacute (10-day) study, Kobayashi, et al. (1972) found that all mice survived when 2,4-DCP at 667 mg/kg body weight was given orally. They derived LD_{50} figures for this study which appear similar to those listed for the acute studies (see Table 5). In the same study (Kobayashi, et al. 1972), male mice were also fed 2,4-DCP in the diet over a 6-month period. Parameters evaluated included average body weight, food consumption, organ weight, glutamic oxaloacetate transaminase, glutamic pyruvic transaminase, erythrocyte counts, leucocyte counts, and histopathological changes. The estimated dose levels were 45 mg/kg/day, 100 mg/kg/

day, and 230 mg/kg/day, corresponding to dietary 2,4-DCP concentrations of 50C, 1,000, and 2,000 µg/g, respectively. No adverse effects were noted in mice at any dose level, except for some microscopic non-specific liver changes after the maximum dose. These changes included infiltration of round cells and swelling of hepatocytes, with some differences in cell size. Two animals were reported to have "dark cells" in the liver. (To the author's knowledge, this term is not commonly used in the United States, and its meaning is not clear.) Kobayashi, et al. concluded that 100 mg/kg/day is a maximum no-effect level in mice.

No other chronic toxicity studies using 2,4-dichlorophenol have been found. One report in the literature (Bleiberg, et al. 1964) has suggested a possible role of 2,4-DCP in acquired chloracne and porphyria in workers manufacturing 2,4-DCP and 2,4,5-trichlorophenol (2,4,5-TCP). The workers involved were also exposed to acetic acid, phenol, monochloroacetic acid, and sodium hydroxide. Since various dioxins (including 2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD), which has been associated with chloracne) have been implicated as contaminants of 2,4,5-TCP, the role of 2,4-DCP in inducing chloracne and porphyria is not conclusive (Huff and Wassom, 1974).

Synergism and/or Antagonism

Reports of studies directly assessing the synergism or antagonism of 2,4-dichlorophenol by other compounds were not found. Since 2,4-DCP is an uncoupler of oxidative phosphorylation (Mitsuda, et al. 1963), it may be expected that concomitant exposure to other uncouplers (e.g., pentachlorophenol, dinitrophenol) would

enhance that effect. In addition, exposure to gamma- or beta-BHC, 2,4-D, and nitrofen could add slightly to any primary body burden of 2,4-DCP.

Any agent causing liver damage sufficient to decrease the conjugation of 2,4-DCP with glucuronide or sulfate could conceivably alter the excretion and/or toxicity of the parent compound. However, there are no specific studies to reflect such an effect.

Teratogenicity

Pertinent data could not be located in the available literature concerning the teratogenicity of 2,4-DCP.

Mutagenicity

No studies were found which addressed the mutagenicity of 2,4-dichlorophenol in mammalian systems. Amer and Ali (1968, 1969) did report some effects of 2,4-DCP on mitosis and meiosis in flower buds and root cells of vetch (Vicia faba). Changes included meiotic alterations of chromosome stickiness, lagging chromosomes, and anaphase bridges when flower buds were sprayed with 0.1 M 2,4-DCP. Mitotic changes of chromosome stickiness, lagging chromosomes, disintegration, bridging, disturbed prophase and metaphase, and occasional cytomyxis were seen in root cells exposed to 62.5 mg/l DCP. Later studies (Amer and Ali, 1974) further confirmed the effect of chromosome stickiness, lagging chromosomes, and fragmentation in 35-day-old Vicia faba. The relationship of these changes to alterations in mammalian cells has not been established.

Carcinogenicity

Repeated application of phenol and some substituted phenols has demonstrated promoting, as well as complete, tumorigenic activ-

ity (Boutwell and Bosch, 1959). In the Boutwell and Bosch study, two trials included evaluation of 2,4-DCP as a promoter. In one trial, 25 µl of a 20 percent solution of 2,4-DCP in benzene was applied twice weekly for 15 weeks to female Sutter mice two to three months of age. The other trial was identical, except that 2,4-DCP was applied for 24 weeks. Application in both trials followed an initiating dose of 0.3 percent dimethyl-benzanthracene (DMBA) in benzene.

The 2,4-DCP dose used corresponds to 5 mg of compound per mouse at each application, or 10 mg/week when applied twice weekly. Sutter mice two to three months old would be expected to weigh 35 grams, so that the dose rate would be 40.82 mg/kg body weight. Tumorigenic response was measured as follows:

1) The percentage of surviving mice bearing one or more papilloma was ascertained.

2) The number of papillomas on all surviving mice was totaled and divided by the number of survivors to give the average number of papillomas per surviving mouse.

3) The number of mice bearing malignant tumors was determined.

Results of the promoter trial with 2,4-DCP are presented in Table 6. Related promoter experiments with phenol and the two benzene controls are included for comparative purposes. Boutwell and Bosch concluded that the promoting activity of 2,4-DCP is similar to that of phenol. However, no statistical analyses nor dose-response data were included to support this comparison.

To see if there was a statistically significant difference between the 2,4-DCP-treated mice and the benzene controls, a Fisher

TABLE 6

Appearance of Skin Tumors in Mice Treated Cutaneously with Phenols following a Cutaneous Dose of 0.3% Dimethyl-benzanthracene (DMBA) in Acetone[a]

Treatments[b]	Time Animals Examined (week)	No. of Mice (survivors/total)	Survivors with Papillomas (%)	Average Papillomas per Survivor	Survivors with Epithelial Carcinomas (%)
Benzene control	15	15/20	7	0.07	0
Benzene control	24	27/32	11	0.15	0
10% phenol in benzene in DMBA	20	24/30	33	0.62	13
20% phenol in acetone	12	21/24	58	-	5
20% phenol in benzene	24	10/33	100	3.20	20
20% 2,4-DCP in benzene	15	27/33	48	1.07	11
20% 2,4-DCP in benzene	24	16/23	75	1.62	6

[a]Source: Modified from Boutwell and Bosch, 1959

[b]All received DMBA except where stated

exact test was undertaken for this document. Tumor incidence was derived with the assumption that only survivors were examined for tumors. The calculated results are presented in Table 7.

This analysis indicates that (1) the higher incidence of papillomas in both 2,4-DCP-treated groups was not attributable to chance, and (2) the carcinoma incidence was not significantly elevated over controls.

This statistical exercise should not obscure a number of considerations that could affect the meaningfulness of the results. (1) The study used dermal application of a phenolic compound at 20 percent concentration in organic solvents. This concentration is high enough to destroy hair follicles and sebaceous glands. The papillomatous response observed may have developed in response to chemical and/or physical damage from application of an irritant compound. (2) Even with the harsh treatment, no malignant neoplasia was observed, except when DMBA was used as an initiator. The only neoplasia observed was at the site of direct application. (3) Pathological identification of benign and malignant tumors was done on a gross level, with only periodic confirmation by microscopic examination. (4) The mice were housed in creosote-treated wooden cages, which themselves were capable of initiating a carcinogenic response.

The report of Boutwell and Bosch (1959) is the only one found that deals with the tumorigenicity of 2,4-DCP. However, since the study was designed primarily to detect promoting activity, the effect of 2,4-DCP as a primary carcinogen is not well defined.

TABLE 7

Results of Fisher Exact Test Applied to Data
from Boutwell and Bosch (1959)

Treatment	Duration of Treatment (wks)	Incidence of Papillomas	P-value vs. Control	Incidence of Epithelial Carcinoma	P-value vs. Control
Benzene control (I)	15	1/15	-	0/15	-
20% 2,4-DCP in benzene	15	13/27	0.61×10^{-2}	3/27	0.2548
Benzene control (II)	24	3/27	-	0/27	-
20% 2,4-DCP in benzene	24	12/16	0.36×10^{-4}	1/16	0.3721

The route of administration is not appropriate to the model for carcinogenic risk assessment and has no established relationship to oral exposure. Overall, the study does present evidence that 2,4-DCP may be a possible promotor, and the work may be applicable to evaluating the hazard of skin or respiratory exposure to 2,4-DCP, alone or concurrent with other chemicals.

Other Effects

An odor threshold for 2,4-DCP in water has been reported by at least three investigators. Hoak (1957) determined the odor threshold of 2,4-DCP to be 0.65 µg/l at 30°C and 6.5 µg/l at 60°C. Determination of the detectable odor was made by a panel of two or four people comparing flasks of test water to a flask of odor-free water. The lowest concentration detected by any panel member was taken as the odor threshold. Hoak speculated that odor should become more noticeable as temperature increases; however, in evaluating a series of chlorophenols and cresols, it was found that some compounds had higher odor thresholds at 30°C, and others were higher at 60°C.

Burttschell, et al. (1959) made dilutions of chlorophenol in carbon-filtered tap water and used a panel of four to six people to evaluate odor. Tests were carried out at room temperature, which the investigator estimated to be 25°C. If a panel member's response was doubtful, the sample was considered negative. The geometric mean of the panel responses was used as the odor threshold. For 2,4-DCP the threshold was 2 ug/l.

Dietz and Traud (1978) used a panel composed of 9 to 12 persons of both sexes and various age groups to test the organolep-

tic detection thresholds for 126 phenolic compounds. To test for odor thresholds, 200 ml samples of the different test concentrations were placed in stoppered odor-free glass bottles, shaken for approximately five minutes, and sniffed at room temperature (20-22^{o}C). For each test, water without the phenolic additive was used as a background sample. The odor tests took place in several individual rooms in which phenols and other substances with intense odors had not been used previously. Geometric mean values were used to determine threshold levels. To determine taste threshold concentrations of selected phenolic compounds, a panel of four test individuals tested water samples containing various amounts of phenolic additives. As a point of comparison, water without phenolic additives was tasted first. Samples with increasing phenolic concentrations were then tested. Between samples, the mouth was rinsed with the comparison water and the test person ate several bites of dry white bread to "neutralize" the taste. Geometric mean detection level values for both tests provided threshold levels of 0.3 µg/l for taste and 40 µg/l for odor for 2,4-DCP.

None of these three studies, however, indicated whether the determined threshold levels made the water undesirable or unfit for consumption.

CRITERION FORMULATION

Existing Guidelines and Standards

Presently, no standard for exposure to 2,4-DCP in drinking or ambient water has been set, although a standard of 0.1 mg/l for 2,4-D, a related compound, has been set [National Academy of Sciences (NAS), 1977].

Current Levels of Exposure

Human exposure to 2,4-DCP has not been monitored, but a worst case estimate of 40 µg 2,4-DCP/kg body weight/day of exposure was presented in the Ingestion from Food section.

Special Groups at Risk

The only group expected to be at risk from high exposure to 2,4-DCP is industrial workers involved in the manufacturing or handling of 2,4-DCP and 2,4-D. No data were found to relate exposure or body burden to conditions of contact with 2,4-DCP.

Basis and Derivation of Criterion

Insufficient data exist to indicate that 2,4-DCP is a carcinogenic agent. The only study performed (Boutwell and Bosch, 1959) was designed to detect promoting activity, and the effect of 2,4-DCP as a primary carcinogen could not be evaluated. Also, the route of administration (dermal) in this study is inappropriate for use in the linear model for carcinogenic risk assessment (see Carcinogenicity section).

Minimal health effects data exist on the acute and chronic effects of 2,4-DCP. Only one study of a chronic nature (Kobayashi, et al. 1972) was found. Kobayashi and colleagues determined a chronic (6-month) no-effect level for 2,4-DCP to be 1,000 ug/g of diet for mice, which was equivalent to 100 mg/kg body weight/day.

An equivalent daily dose for a 70 kg adult human would be 7,000 mg/day (100 mg/kg/day x 70 kg). Applying an uncertainty factor of 1,000 as suggested by the National Academy of Sciences Safe Drinking Water Committee (1977), the Acceptable Daily Intake (ADI) for a 70 kg adult human would be 7,000 mg ÷ 1,000 = 7 mg. Solving the equation (2L)(C) + (C)(BCF)(fish consumption/day) = ADI, the water quality criterion (C) can be computed:

$$(2L)(C) + (C)(40.7)(0.0065) = 7 \text{ mg}$$

$$(2L)(C) + (C)(0.26455) = 7 \text{ mg}$$

$$C = \frac{7 \text{ mg}}{2.26455L}$$

$$C = 3.09 \text{ mg/l}$$

where:

7 mg = the calculated daily exposure for a 70 kg person (ADI) based on the above conditions

2L = amount of drinking water consumed/day

0.0065 kg = amount of fish consumed/day

C = maximum permissible level in water based on above conditions

Thus, a criterion level based entirely upon toxicological data would be 3.09 mg/l.

Human health is a subjective measurement in many respects. The organoleptic properties of 2,4-DCP could conceivably alter human health by causing a decrease in water consumption. This might be of particular importance to individuals with certain renal diseases or in instances where dehydration occurs as a result of vigorous exercise, manual labor, or hot weather.

Since the odor and taste detection threshold concentrations for 2,4-dichlorophenol are well below any toxicity-based criterion level that may be derived, the ambient water quality criterion is based on organoleptic data. It should be emphasized that this criterion is based on aesthetic qualities rather than health effects. However, to the effect that this criterion is below the level derived from the chronic toxicity study of Kobayashi, et al. (1972), it is likely to also be protective of human health.

The data of Hoak (1957), Burttschell, et al. (1959), and Dietz and Traud (1978) all indicated that low microgram concentrations of 2,4-dichlorophenol in water are capable of producing a discernable odor. Dietz and Traud further observed a distinct flavor alteration of water at sub-microgram levels of 2,4-DCP. The Burttschell, et al. (1959) and Dietz and Traud (1978) studies did not indicate a range of responses; however, because of the variability of responses inherent in such procedures, it is certainly possible that the odor threshold for some evaluators (at least in the Burttschell group) would extend downward toward the 0.65 ug/l figure of Hoak. Thus, the data from these three studies are considered to be reasonably mutually supportive (i.e., Hoak's 0.65 µg/l for odor, Burttschell's 2.0 µg/l geometric mean value for odor, and Dietz and Traud's geometric mean values of 40 µg/l for odor and 0.3 µg/l for taste).

Therefore, based on the prevention of undesirable organoleptic qualities, the criterion level for 2,4-dichlorophenol in water is 0.3 µg/l. This level should be low enough to prevent detection of objectionable organoleptic characteristics and far below minimal

no-effect concentrations determined in laboratory animals. As more substantive and reliable data become available in the future, a criterion level based on human health effects may be more confidently postulated.

It should be emphasized that data are needed in the following areas to properly evaluate any hazard from 2,4-DCP:

1) Monitoring of worker exposure to 2,4-DCP in industries manufacturing or using the chemical.

2) Monitoring of public water supplies and industrial and municipal effluents to determine an expected range of concentrations under differing environmental conditions.

3) More definitive studies of residue kinetics of 2,4-DCP in food animals which are exposed to products capable of generating 2,4-DCP.

4) Evaluation of chronic toxicity, mutagenicity, and teratogenicity of 2,4-DCP using currently acceptable techniques.

5) A carcinogenicity study of 2,4-DCP using the oral route and evaluated according to current protocols.

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